

PETER C. GÖTZSCHE

DEADLY PSYCHIATRY AND ORGANISED DENIAL

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AMERICANS
AND
EUROPEANS
2014

People's Press

Peter C. Gøtzsche

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Abbreviations

ADHD: Attention Deficit Hyperactivity Disorder

APA: American Psychiatric Association CI: Confidence Interval

DSM: Diagnostic and Statistical Manual of Mental Disorders

EMA: European Medicines Agency

FDA: Food and Drug Agency (USA)

ICD: International Classification of Diseases

GP: General Practitioner

NICE: National Institute for Health and Care Excellence (UK)

NIMH: National Institute of Mental Health (USA)

OCD: Obsessive Compulsive Disorder

SSRI: selective serotonin reuptake inhibitor, an antidepressant

UN: United Nations

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About the author

Professor Peter C. Gøtzsche graduated as a Master of Science in biology and chemistry in 1974 and as a physician in 1984. He is a specialist in internal medicine; worked in the drug industry 1975-83, and at hospitals in Copenhagen 1984-95. With about 80 others, he helped start The Cochrane Collaboration in 1993 with the founder, Sir Iain Chalmers, and established The Nordic Cochrane Centre the same year. He became professor of Clinical Research Design and Analysis in 2010 at the University of Copenhagen.

Gøtzsche has published more than 70 papers in “the big five” (*BMJ*, *Lancet*, *JAMA*, *Annals of Internal Medicine* and *New England Journal of Medicine*) and his scientific works have been cited over 15,000 times.

Gøtzsche has an interest in statistics and research methodology. He is a member of several groups publishing guidelines for good reporting of research and has co-authored CONSORT for randomised trials (www.consort-statement.org) STROBE for observational studies (www.strobe-statement.org), PRISMA for systematic reviews and meta-analyses (www.prisma-statement.org), and SPIRIT for trial protocols (www.spirit-statement.org). He was an editor in the Cochrane Methodology Review Group 1997-2014.

Books by Peter C Gøtzsche

Gøtzsche PC. *Deadly medicines and organised crime: How big pharma has corrupted healthcare*. London: Radcliffe Publishing; 2013. Translated into several languages, see www.deadlymedicines.dk.

Gøtzsche PC. *Dødelig medicin og organiseret kriminalitet: Hvordan medicinalindustrien har korrumperet sundhedsvæsenet*. København: People's Press; 2013.

Gøtzsche PC. *Mammography screening: truth, lies and controversy*. London: Radcliffe Publishing; 2012.

Gøtzsche PC. *Rational diagnosis and treatment: evidence-based clinical decision-making*. 4th ed. Chichester: Wiley; 2007.

Wulff HR, Gøtzsche PC. Rationel klinik. Evidensbaserede diagnostiske og terapeutiske beslutninger. 5. udgave. København: Munksgaard Danmark; 2006.

Gøtzsche PC. På safari i Kenya. København: Samlerens Forlag; 1985.

1

Introduction

Psychiatry is not an easy specialty. It requires a lot of patience and understanding, and there are many frustrations. I am sure psychiatrists sometimes get frustrated at patients who continue to destroy their lives, refusing to take on board the good advice they have been offered about how they could improve on their attitude to life's many troubles.

This book is not about the psychiatrists' problems, however. It is about why psychiatry has failed to deliver what patients want, and what the consequences are of focusing on using harmful drugs of questionable benefit. Most patients don't respond to the drugs they receive and, unfortunately, the psychiatrists' frustrations at the lack of progress often lead to the prescribing of more drugs or higher doses, further harming the patients.

Psychiatric drugs are so harmful that they kill more than half a million people every year among those aged 65 and over in the United States and Europe (see Chapter 14). This makes psychiatric drugs the third leading cause of death, after heart disease and cancer.

I don't think there is anything psychiatric patients fear more than forced treatment, and this is an important reason why having close contact with the psychiatric treatment system markedly increases suicides (see Chapter 15). I shall explain why forced treatment is unethical and should be banned and also demonstrate that psychiatry is possible without it.

Many psychiatric drugs not only increase total mortality but also increase the risk of suicide and homicide, while no drug agency anywhere has approved any drug as being effective in *preventing* suicides. Lithium is an exception, as it might possibly reduce suicides (see Chapter 7).

Widespread overdiagnosis and overtreatment is another issue I take up. There is huge overdiagnosis of mental disorders, and once you receive a psychiatric diagnosis everything you do or say becomes suspect, as you are now under observation, which means that the initial, perhaps tentative diagnosis, all too easily becomes a self-fulfilling prophecy (see Chapter 2).

I believe we could reduce our current usage of psychotropic drugs by 98% and at the same time improve people's mental health and survival (see Chapter 14). The most important reason for the current drug disaster is that leading psychiatrists have allowed the drug industry to corrupt their academic discipline and themselves.

I have written this book primarily for the patients, particularly those who have desperately wanted to come off their drugs but were met with hostile and arrogant reactions from their doctors, and I shall explain how it is possible to safely taper drugs (Chapter 12).

I have also written the book for young psychiatrists in training in the hope that it could inspire them to revolutionise their specialty, which is badly needed. One sign that psychiatry is in deep crisis is that more than half the patients believe their mental disorder is caused by a chemical imbalance in the brain. They have this misperception from their doctors, which means that more than half the psychiatrists lie to their patients. I know of no other specialty whose practitioners lie to their patients. Psychiatrists also lie to themselves and to the public, and I shall give many examples of official statements that exaggerate the benefits of psychiatric interventions by five to ten times and underestimate the harmful effects by a similar factor.

Those at the top of the hierarchy I call "silverbacks," since they are almost always males and behave like primate silverbacks in the jungle, keeping others away from absolute power, which in nature carries rewards such as easy access to females – in psychiatry this translates into money and fame. These silverbacks suffer from collective, organised denial. They refuse to see the damage they cause even when the evidence is overwhelming. Further, they have united around a number of myths and misconceptions, which they defend stubbornly but which are very harmful for patients. Some of the worst, which I shall debunk in this book, are:

- psychiatric diagnoses are reliable;
- it reduces stigmatisation to give people a biological or a genetic explanation for their mental disorder;
- the usage of psychiatric drugs reflects the number of people with mental disorders;
- people with mental disorders have a chemical imbalance in their brain and psychiatrists can fix this imbalance with drugs, just like endocrinologists use insulin for diabetes;
- long-term treatment with psychiatric drugs is good, as it prevents recurrence of the disease;

- treatment with antidepressants does not lead to dependence;
- treatment of children and adolescents with antidepressants protects against suicide;
- depression, ADHD and schizophrenia lead to brain damage; and
- drugs can prevent brain damage.

I shall also explain how I have come to the conclusion that psychiatric research is predominantly pseudoscience, and why reliable research constantly tells us a very different story to the fairy tale that leading psychiatrists want us to believe in.

I am a specialist in internal medicine and took an interest in psychiatry in 2007 when Margrethe Nielsen from the Danish Consumer Council approached me with an idea for her PhD thesis: “Why is history repeating itself? A study on benzodiazepines and antidepressants (SSRIs).”

Her studies showed that, indeed, history has repeated itself. We have repeated the same mistakes with the SSRIs that we made with benzodiazepines, and before them with barbiturates. We have created a huge epidemic of drug overuse with just as many drug addicts on SSRIs as on benzodiazepines (see Chapter 12).

Margrethe’s findings were not welcomed by two of her examiners, who had turfs to defend. One, Steffen Thirstrup, worked for the Danish drug agency, the other, John Sahl Andersen, was a general practitioner. Our drug agencies have contributed substantially to the current misery, and most of the drug harms are caused by general practitioners, who prescribe about 90% of the psychiatric drugs.

They rejected her thesis for no good reason, but having appealed to the University, she defended it successfully.¹ If psychiatrist David Healy had not been the third examiner, she might not have obtained her PhD, which would have been a gross injustice, as her research is sound and her PhD thesis is considerably better than many I have seen.

Unwelcome facts are being suppressed all the time, and I shall give numerous examples of the works of the “doubt industry” where people incessantly publish seriously flawed research to provide support for their unsustainable ideas.

After having studied the science carefully, I note that some people I have met and several organisations have come to the conclusion that the way we currently use psychiatric drugs and the way we practice psychiatry cause more harm than good. The general public agrees and feels that antidepressants, antipsychotics, electroshock and admission to a psychiatric ward are more often harmful than beneficial (see Chapter 13). I have no doubt they are right, and the double-blind placebo controlled randomised trials – which are not so blind as intended – have

rather consistently shown that it is the psychiatrists that think their drugs are effective, not the patients (see Chapter 3).

Investigators who have not been blinded effectively can see the exact opposite of what is actually true when they medicate patients. They see what they want to see, which is what is convenient for them and for their specialty, not what really happens (see Chapters 3 and 6).

Cochrane reviews have shown that it is doubtful whether antidepressants are effective for depression (see Chapter 3) and whether antipsychotics are effective for schizophrenia (see Chapter 6). Some drugs can be helpful sometimes for some patients, particularly in the acute phase where a patient can be so tormented by panic or delusions that it can be helpful to dampen the emotions with a tranquilliser. However, unless doctors become much more expert in the way they use psychiatric drugs which would mean using them very little, in low doses, and always with a plan for tapering them off, our citizens would be far better off if we removed all psychotropic drugs from the market.

Some people will see this as a provocative statement, but it isn't. It is based on solid science, which I shall document. I am used to being called provocative or controversial, which I take to mean that I am telling the truth. In healthcare, the truth is rarely welcomed, as so many people have so many wrong ideas to defend. The silverbacks of psychiatry have created a fantasy world of their own, which is not evidence-based medicine and which is riddled by harmful polypharmacy (see Chapter 13).

Silverbacks in the UK exhibit psychiatry's organised denial

People critical of psychiatry are often met with *ad hominem* attacks from the psychiatric establishment or with scientific arguments of little merit. This happened to me after I gave a keynote lecture in 2014 at the opening meeting of the Council for Evidence-based Psychiatry in the House of Lords, chaired by the Earl of Sandwich, called "Why the use of psychiatric drugs may be doing more harm than good." The other speakers, psychiatrist Joanna Moncrieff and anthropologist James Davies, gave similar talks and have written critical books of mainstream psychiatry.²⁻⁵

Three months later, psychiatrist David Nutt and four male colleagues (I shall refer to them by a collective "DN") attacked me in the first issue of a new journal,

Lancet Psychiatry.⁶ Their paper is only two pages long, but it is so typical of the silverbacks' knee-jerk reactions when criticised that I shall describe it in some detail.

Anti-everything

DN started out by saying that, "Psychiatry is used to being attacked by external parties with antidiagnosis and antitreatment agendas." Silverbacks often say that those coming from another tribe ("external parties") are not allowed to criticise them. This arrogant attitude has unfortunate consequences because many psychiatrists adopt the same position towards their patients, thinking they need not listen to them or take seriously their criticism of the drugs they ingest. It is also common for silverbacks to stigmatise those who dare criticise psychiatry as being anti-something, and DN use the terms "anti-psychiatry" and "anti-capitalist" associated with "extreme or alternative political views."

"New nadir in irrational polemic"

DN were unhappy with newspaper headlines such as "Antidepressants do more harm than good, research says," which appeared in *The Times* and *The Guardian* after our council meeting, and they called this a "new nadir in irrational polemic." They found it especially worrying that I being a co-founder of the Cochrane Collaboration, an initiative set up to provide the best evidence for clinical practitioners, had apparently suspended my "training in evidence analysis for popular polemic." Silverbacks usually speak with the same voice as the drug industry because it so generously supports them financially (see Chapter 13), and DN are not an exception. We are told: "Depression is a serious and recurrent disorder that is currently the largest cause of disability in Europe and is projected to be the leading cause of morbidity in high-income countries by 2030." No British understatement here, though there is no way we can reliably count the number of people with depression. The criteria for the diagnosis are arbitrary and consensus-based, and they are now so broad that a large part of the healthy population can get the diagnosis (see Chapter 3). It is therefore misleading to say that depression is a serious disorder. Most people have mild symptoms of everyday distress that hit most of us from time to time; very few are seriously depressed. Worse still, the dramatic increase in depression-related morbidity that DN speak about has been caused by the psychiatrists themselves. The drugs they use do not cure depression but turn many self-limiting episodes into chronic ones (see Chapter 12). This is not helping patients; it is serving the interests of

psychiatry and the pharmaceutical industry.

“Impressive ability to prevent recurrence of depression”

The DN group argues that antidepressants are among the most effective drugs we have in the whole of medicine and mentions their “impressive ability to prevent recurrence of depression, with a number needed to treat of around three [to prevent one recurrence].” It certainly looks impressive but it isn’t true. The trials that have shown these effects, where half of the patients continue with their antidepressant drug after they have recovered while the other half is switched to placebo, are totally unreliable (see Chapter 11). This is because those switched to placebo have to go cold turkey, i.e. abstinence symptoms occur because their brain has adapted to the antidepressant, just like alcoholics get into trouble if they suddenly stop drinking, and these symptoms can mimic depression.

In their praise of antidepressants, DN also say they have an impressive effect on acute depression. They haven’t. It is likely that they have no effect at all (see Chapter 3).

DN note that fewer participants on an antidepressant than on placebo withdrew from the trials because of treatment inefficacy, which they interpret as evidence that antidepressants are effective. This interpretation is not appropriate. It is often the combination of the perceived benefits and harms that determines whether a patient stays in a trial. A patient who is on an active drug has often guessed this, because of the drug’s side effects, and might therefore be more inclined to continue in the trial even if the drug has no effect, particularly since psychiatrists often tell their patients that it may take a while before the effect appears. Conversely, patients on placebo have no incentive to carry on and therefore, more than in the drug group, drop out due to lack of effect.

It is therefore advised in textbooks on research methods not to focus on the number of patients who drop out because of lack of effect. It only makes sense to look at the total number of drop-outs, which is also the most *relevant* outcome for treatments that are not curative but only have an effect on the patients’ symptoms.

Patients are the best judges for deciding whether a perceived benefit of taking a drug outweighs its side effects, and they find the drugs pretty useless, as just as many patients stop treatment on antidepressants as on placebo in the trials for any reason.⁷

Does academic debate increase suicides?

The DN group mentions that many people who are not taking antidepressants commit suicide, claiming that a “blanket condemnation of antidepressants by lobby groups and colleagues risks increasing that proportion.” In my book about mammography screening,⁸ I called this the *you are killing my patients* argument. Those who raise uncomfortable questions about popular interventions are accused of being responsible for the death of many people. But let’s think. If we generalised this argument to become a common ethical standard, researchers could never question any intervention if it was believed to save lives. Thus, we would probably still be performing bloodletting in our hospitals for all kinds of diseases, even for cholera, where such treatment is deadly.

More importantly, the crux of the argument is wrong. Antidepressants don’t protect people against suicide (see Chapter 3).

DN claim that most of those who commit suicide are depressed, but the underlying data do not allow such a conclusion.⁹ A widely cited study found that most suicides were related to a diagnosis of depression, but only 26% of the people were known to have been diagnosed with depression before they killed themselves. All the others got a post-mortem diagnosis based on a so-called psychological autopsy, and it is self-evident that establishing a diagnosis of a psychiatric disorder in a dead person is a highly bias-prone process. Social acceptability bias threatens the validity of such retrospective diagnosis-making. Relatives often seek socially acceptable explanations and may be unaware of or unwilling to disclose certain problems, particularly those that generate shame or put some of the blame on themselves. It is therefore tempting to put the blame on an impersonal thing like a disease, which cannot protest although it might never have existed. It is a very popular belief among psychiatrists that most of those who commit suicide suffer from depression but it is doubtful whether this is correct – people kill themselves for many reasons other than depression.

The next argument that the DN people put forward to prove their case that antidepressants protect against suicide isn’t any better. They claim that more than 70% are not taking an antidepressant at the time of death. Obviously, when people who are not depressed kill themselves, there is no case for taking an antidepressant before they die. Furthermore, antidepressants can cause an extreme form of restlessness called akathisia, which predisposes to suicide^{10, 11} and which can make the patient stop taking the drug before the suicide. Stopping an antidepressant abruptly, e.g. because the patient ran out of pills, can also cause akathisia and suicide. Thus, there are at least three good reasons why people who kill themselves might not have taken antidepressants at the time of death.

DN’s next argument is also unconvincing. They say that in countries where

antidepressants are used properly, suicide rates have fallen substantially. Well, in countries where cars are used properly (causing few traffic accidents), birth rates have fallen substantially, but that doesn't prove anything. Scientifically sound studies have never been able to find a relationship between increased use of antidepressants and falling suicide rates, or vice versa (see Chapter 3).

“Some of the safest drugs ever made”

The hyperbole escalates towards the end of DN's article. We are told that the SSRIs are some of the safest drugs ever made and that their adverse effects are rarely severe or life threatening. The facts are that SSRIs kill one of 28 people above 65 years of age treated for one year; that half of the patients get sexual side effects; and that half of the patients have difficulty stopping antidepressants because they become dependent on them (see Chapter 3). When silverback psychiatrists call SSRIs some of the safest drugs ever made, I believe it is fair to say that it is unsafe for people who suffer from something that could be treated with an SSRI to consult a psychiatrist.

Critics “prefer anecdote to evidence”

It is surreal to me when DN say that, “Many of the extreme examples of adverse effects given by the opponents of antidepressants are both rare and sometimes sufficiently bizarre as to warrant the description of an unexplained medical symptom,” and that, “To attribute extremely unusual or severe experiences to drugs that appear largely innocuous in double-blind clinical trials is to prefer anecdote to evidence.” DN do not appreciate that the main reason that SSRIs appear innocuous in clinical trials is that the companies have manipulated the data to an extraordinary degree (see Chapter 3).¹¹⁻¹³

Furthermore, DN fail to listen to patients. That an adverse effect is “bizarre” doesn't disqualify it. Many patients have experienced the same highly bizarre adverse effects, which have returned when the patients were exposed to the same drug again. This is an accepted method for establishing cause-effect relationships in clinical pharmacology, which is called challenge, dechallenge and rechallenge. In 2010, on one of the occasions where I lectured to Danish psychiatrists, I got nowhere with this argument in a discussion with a US psychiatrist. He argued that the randomised trials had not shown an increased risk of suicide, but he didn't understand that it is not a requirement for establishment of harms that they have been confirmed in randomised trials. He might have listened too much to the industry, which downplays the harmful effects of their drugs by pointing out that

they weren't statistically significant, often after they have manipulated the data to ensure that no significant differences would see the light of day.

DN suggest that we should ignore "severe experiences to drugs," which they dismiss as anecdotes and claim might be distorted by the "incentive of litigation". This is the height of professional denial and arrogance. It is deeply insulting to those parents who have lost a healthy child and those spouses who have lost a partner whom an SSRI drove to suicide or homicide. Furthermore, members of the Council for Evidence-based Psychiatry explained in *Lancet Psychiatry* that British withdrawal-support charities report alarming numbers of people suffering disabling symptoms for multiple years following withdrawal from antidepressants.¹⁴

“Insulting to the discipline of psychiatry”

In their finishing remarks, DN say that my "extreme assertions ... are insulting to the discipline of psychiatry ... and at some level express and reinforce stigma against mental illnesses and the people who have them." I shall explain in Chapter 6 that it is the psychiatrists that stigmatise the patients, not those who criticise psychiatry.

DN also say that, "The anti-psychiatry movement has revived itself with the recent conspiracy theory that the pharmaceutical industry, in league with psychiatrists, actively plots to create diseases and manufacture drugs no better than placebo. The anti-capitalist flavour of this belief resonates with anti-psychiatry's strong association with extreme or alternative political views."

In my reply, I noted that, "This is the language of people who are short of arguments."¹⁵ It was pretty ironic that – of all their expostulations – DN lamented that critics of psychiatry believe that the pharmaceutical industry and the psychiatrists create diseases and use drugs no better than a placebo, as if this was a self-evidently absurd proposition. As I shall explain later, this is pretty much true. Whereas it is not true when DN say that those who criticise the overuse of psychiatric drugs are "extreme" or "alternative." When I wrote to the editor of *Lancet Psychiatry* and requested an opportunity to defend my academic reputation, the editor told me that the Nutt and colleagues' paper was given an independent peer review, as well as being subjected to legal review. This is difficult to understand, given its many errors, the pronounced *ad hominem* attacks, and the tough UK libel law.

I addressed the worst of DN's misconceptions in my reply.¹⁵ I also noted that Nutt and two of his co-authors, Guy M Goodwin and Stephen Lawrie, had between them declared 22 conflicts of interest in relation to drug companies, and I

wondered whether this explained their dismissal of psychotherapy, although it is effective and recommended by the UK's National Institute for Health and Care Excellence (NICE).

After having read this, you might think that – in their own words about their critics – these psychiatrists are “extreme,” as they cherish so many unsustainable opinions about their own field of work. But unfortunately they are not.

Professor David Nutt is a mainstream psychiatrist and an influential one. He was previously the United Kingdom's drug czar (the main adviser to the government) until he was sacked for claiming that ecstasy is no more dangerous than riding a horse, which he called “equasy,” short for “Equine Addiction Syndrome.”¹⁶ Nutt won the 2013 John Maddox Prize for Standing Up for Science. The judges awarded him the prize in recognition of the impact his thinking and actions have had in influencing evidence-based classification of drugs, and his continued courage and commitment to rational debate, despite opposition and public criticism. Words fail me.

Professor Guy M Goodwin is head of Oxford University's Department of Psychiatry and was President of the British Association for Psychopharmacology in 2002-2004.

Professor Dinesh Bhugra, at the Institute of Psychiatry at King's College in London, was previously President of the UK's Royal College of Psychiatry and is currently president-elect of the World Psychiatric Association.

Professor Seena Fazel is a Forensic Psychiatrist at Oxford University's Department of Psychiatry; he has an interest in violent crime and suicide.

Professor Stephen Lawrie is Head of the Division of Psychiatry at the University of Edinburgh and is on the editorial board of *Lancet Psychiatry*.

These psychiatrists are at the top of their profession and yet they hold views which are in direct contrast to the science in their field. This illustrates that psychiatry is in deep crisis and that its leaders suffer from organised denial.

My preference is to mention names because people should be held responsible for their actions and arguments. If they do something laudable, they would be disappointed if they were anonymous, but it must work both ways. If I concealed the names when people did something reproachable, or sustained an erroneous belief, I would be inconsistent, and my readers would try to guess anyway who they were. Science is not about guesswork, which is another reason why I prefer to mention names. However, it is fair to point out that when I name a person for something he or she should not be proud of, there are thousands of others that have done the same or share the same beliefs.

References

- 1 Nielsen M. Selective Serotonin Reuptake Inhibitors (SSRI) – sales, withdrawal reactions and how drug regulators reacted to this with benzodiazepines as comparator (PhD thesis). University of Copenhagen; 2013.
- 2 Moncrieff J. The bitterest pills. Basingstoke: Palgrave Macmillan; 2013.
- 3 Moncrieff J. The myth of the chemical cure: a critique of psychiatric drug treatment. Basingstoke: Palgrave Macmillan; 2007.
- 4 Moncrieff J. A straight talking introduction to psychiatric drugs. Ross-on-Wye: PCCS Books; 2009.
- 5 Davies J. Cracked: Why psychiatry is doing more harm than good. London: Icon Books; 2013.
- 6 Nutt DJ, Goodwin GM, Bhugra D, et al. Attacks on antidepressants: signs of deep-seated stigma? *Lancet Psychiatry* 2014;1:103-4.
- 7 Barbui C, Furukawa TA, Cipriani A. Effectiveness of paroxetine in the treatment of acute major depression in adults: a systematic reexamination of published and unpublished data from randomized trials. *CMAJ* 2008;178:296-305.
- 8 Gøtzsche PC. Mammography screening: truth, lies and controversy. London: Radcliffe Publishing; 2012.
- 9 Raven M. Depression and antidepressants in Australia and beyond: a critical public health analysis (PhD thesis). University of Wollongong, Australia; 2012. <http://ro.uow.edu.au/theses/3686/>.
- 10 Bass A. Side effects – a prosecutor, a whistleblower, and a bestselling antidepressant on trial. Chapel Hill: Algonquin Books; 2008.
- 11 Healy D. Let them eat Prozac. New York: New York University Press; 2004.
- 12 Gøtzsche PC. Deadly medicines and organised crime: How big pharma has corrupted health care. London: Radcliffe Publishing; 2013.
- 13 Virapen J. Side effects: death. College Station: Virtualbookworm.com Publishing; 2010.
- 14 Timimi S, Thomas P, Davies J, et al. Antipsychiatry and the antidepressants debate. *Lancet Psychiatry* 2014;1:174.
- 15 Gøtzsche PC. Why I think antidepressants cause more harm than good. *Lancet Psychiatry* 2014;1:104-6 (available at www.deadlymedicines.dk).
- 16 Hope C. Ecstasy ‘no more dangerous than horse riding.’ *The Telegraph* 2009 Feb

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What does it mean to be mentally ill?

If we first take a look at medicine in general, we may better understand the diagnostic problems in psychiatry. We put disease labels on patients with similar problems to make it easier to communicate with each other, to do research, and to treat and prevent diseases from occurring. These diagnostic labels work best when we know what causes particular diseases. It is very useful to know, for example, that a certain pneumonia is caused by pneumococci, as we may then cure it with penicillin. We therefore subdivide pneumonias according to their aetiology and may even label them this way, e.g. we talk about pneumococcal pneumonia.

There are many different kinds of diagnoses in medicine and some are preliminary and just describe a symptom, e.g. stomach pain, which may become the final diagnosis, if no cause is found, or the final diagnosis could be stomach ulcer.

Some diagnoses are syndromes, which consist of several symptoms, signs and paraclinical findings (e.g. results of blood tests or radiology).¹ Rheumatoid arthritis is a good example. We don't know yet what causes it, although we suspect it is an infection. In 1975, a cluster of cases of arthritis occurred in Connecticut that were later shown to be caused by a bacterium, *Borrelia*, which is tick-borne. Before the aetiology was known, it was a syndrome diagnosis, and the patients could have experienced a rash, headache, fever and other symptoms and signs in addition to the arthritis.

We can cure this disease with penicillin and other antibiotics, in contrast to rheumatoid arthritis, which is treated with pretty dangerous drugs. Most patients receive non-steroidal, anti-inflammatory drugs (NSAIDs) for their pain and some die because these drugs can cause stomach ulcers and heart attacks. Disease-modifying agents are also dangerous, and drug treatment is therefore an important reason why these patients don't live so long as other people.

The level of understanding of psychiatric diseases is pretty low compared to the rest of medicine, and the treatments are much more harmful and deadly than those

used for rheumatoid arthritis (see Chapter 14). We don't know much about what causes mental illnesses and the diagnostic uncertainty is far greater than in other areas of medicine.

One of the things that is part of the syndrome diagnosis of rheumatoid arthritis is the presence of rheumatoid factor in the blood, which is an antibody directed against the person's own tissues. There is no such blood test for a mental disorder, and it hasn't been possible to demonstrate that people suffering from common mental disorders have brains that are different from healthy people's brains (see Chapter 11).

It is not easy to define what we mean by being ill or having a disease and we are not consistent when we talk about these issues. People with type 2 diabetes who have no symptoms are not ill, they just have a risk factor, increased blood glucose, which predisposes them to becoming ill. And yet we call such people patients and might even say they suffer from diabetes, although they don't suffer the slightest bit. As another example, women who go to mammography screening are often called patients in information leaflets and scientific articles although they are healthy citizens, at least in relation to breast cancer.

Quite often, psychiatrists prefer to talk about a mental disorder, rather than a mental illness or disease, which is because psychiatric diagnoses are social constructs. The staff at the Mayo Clinic in Minnesota call it an illness, however:²

Mental illness refers to a wide range of mental health conditions – disorders that affect your mood, thinking and behavior ... Many people have mental health concerns from time to time. But a mental health concern becomes a mental illness when ongoing signs and symptoms cause frequent stress and affect your ability to function ... In most cases, symptoms can be managed with a combination of medications and counselling (psychotherapy).

This is how most doctors think. As we don't know what a mental disease is, we define it as a constellation of symptoms, which impair the patient's life.

Psychiatric diagnoses are made by talking to the patients, but the current checklist approach looks a bit too much like the familiar parlour game, Find Five Errors. For example, we say that a person who has at least five symptoms out of nine possible is depressed.³

If we look hard enough, we will surely find "errors" in most people. We are very quick to form an opinion about a stranger, which in an evolutionary sense has great survival value. If we come across a stranger from another tribe in the forest,

we decide instantly whether to run, fight, or start talking. In a similar vein, the doctor's intuition and experience may suggest in a matter of seconds what the problem is for a particular patient, and there is a considerable risk that the doctor from then on asks leading questions, which yields the required number of error points and leads to a misdiagnosis.

Instead of trying to understand the patients, psychiatry has developed into a checklist exercise,⁴ which one could ask a secretary or the patients themselves to carry out. Psychiatrists have told me that this is what general practitioners often do, after which they make a diagnosis. A 1993 study in the United States by the Rand Corporation showed that:⁵

Over half the physicians wrote prescriptions after discussing depression with patients for three minutes or less.

Studies have shown that doctors quite often don't use the official checklists but, rather, their hunch about what might be wrong, which increases the risk of misdiagnosis and overdiagnosis even more. Although there are 374 diagnoses in DSM-IV (*Diagnostic and Statistical Manual of Mental Disorders*), only half of people who were in treatment met diagnostic criteria for a disorder.⁶

This is a very unfortunate development. Serious mental illness is often linked to previous traumas, and childhood adversities triple the risk of developing psychosis.⁷ If the medical history isn't uncovered – which takes time – the treatments applied will usually be pretty ineffective. Even Robert Spitzer, who was the driving force behind the new checklist approach to psychiatric diagnoses in his capacity as chairman of the working group for DSM-III, now recognizes that what he introduced and believed in has had unfortunate consequences.

About 25 years ago, I discussed with a seasoned psychiatrist what the individual perspective means in comparison to what randomised trials tell us about the value of our treatments. I didn't understand what he meant about individualising the treatment, arguing that no two patients are alike. I said that we wouldn't know what we were doing unless we studied our interventions in randomised trials and treated patients with the same diagnosis in the same way even though they were different. I was influenced in my thinking by the failure of Freudian psychoanalysis, which was unscientific, as its practitioners didn't bother to test whether their theories were true. They simply felt they were confirmed again and again by their patients.⁸ Science philosopher Karl Popper has written about this way of thinking with vitriolic sarcasm:⁸

“As for Adler, I was much impressed by a personal experience. Once in 1919,

I reported to him a case which to me did not seem particularly Adlerian, but which he found no difficulty in analysing in terms of his theory of inferiority feelings, although he had not even seen the child. Slightly shocked, I asked him how he could be so sure. ‘Because of my thousand-fold experience,’ he replied; whereupon I could not help saying: ‘And with this new case, I suppose, your experience has become thousand-and-one-fold’.”

Even in contemporary psychiatry, diagnoses are sometimes made this way.

The psychiatrist and I didn’t discuss the same thing. What he meant was that all the individual circumstances that for a particular patient leads to a certain diagnosis are different from those of the next patient who gets the same diagnosis, and if we don’t take these into account, we might give the patient the wrong treatment. I think we were both right. We need the randomised trials but only as a starting point for considering all the other relevant issues for a particular patient, which requires careful listening and an open mind.

On being sane in insane places

“All the other doctors said he couldn’t control himself. He has a disorder.”

The family to a boy wrongly diagnosed with Asperger’s and wrongly treated with olanzapine⁹

My concerns about how diagnoses are made in psychiatry are not exaggerated. It is one of the major problems in psychiatry, and it can take surprisingly little to get a diagnosis. It can be risky, for example, if patients mention they hear voices. In 1973, psychologist David L Rosenhan published a famous article in *Science*, “On being sane in insane places.”¹⁰ Rosenhan and seven other healthy people showed up at psychiatric hospitals and said they heard voices. The task was to get out again by their own devices by convincing the staff that they were sane. As soon as they had been admitted, they therefore ceased to simulate symptoms and behaved completely normally. Yet they were hospitalised for 19 days on average (Rosenhan for two months before he was released), and they were prescribed drugs they avoided swallowing, a total of nearly 2,100 pills of a wide variety, although the pseudopatients presented with the same “symptom.” They were all discharged with a diagnosis of schizophrenia in remission, although their only “symptom” had been that they heard voices, which normal people can experience.

Many of the real patients suspected that the pseudopatients were sane but the staff didn’t notice the normality. This illustrates an important bias in diagnosis

making. Once a diagnosis is made, it is hard to reverse; it sticks to you. Rosenhan explained that the label was so powerful that many of the pseudopatients' normal behaviours were overlooked entirely or profoundly misinterpreted by the staff in order to make them fit with a popular theory of the dynamics of a schizophrenic reaction. A case summary prepared after a pseudopatient was discharged illustrates this fallacy:

This white 39-year-old male ... manifests a long history of considerable ambivalence in close relationships, which begins in early childhood. A warm relationship with his mother cools during his adolescence. A distant relationship to his father is described as becoming very intense. Affective stability is absent. His attempts to control emotionality with his wife and children are punctuated by angry outbursts and, in the case of the children, spankings. And while he says that he has several good friends, one senses considerable ambivalence embedded in these relationships also.

In actual fact, nothing of an ambivalent nature had been described in the pseudopatient's relations, and an entirely different meaning would have been ascribed if it were known that the man was normal.

The pseudopatients made notes and observed that patient behaviours were often misinterpreted by the staff. When a patient had gone "berserk" because he had been mistreated by an attendant, a nurse rarely asked questions but assumed his upset derived from his pathology, or from a recent family visit. The staff never assumed that it could be one of themselves or the structure of the hospital that explained the patient's behaviour.

Rosenhan explained that the diagnosis becomes a self-fulfilling prophecy. Eventually, the patient accepts the diagnosis and behaves accordingly. Rosenhan argues that we should not label all patients schizophrenic on the basis of bizarre behaviours or cognitions, but limit our discussions to behaviours, the stimuli that provoke them, and their correlates. He finds that the psychological forces that result in depersonalisation are strong and imagines what it would be like if the patients were powerful rather than powerless. If they were viewed as interesting individuals rather than diagnostic entities; if they were socially significant rather than social lepers; and if their anguish truly and wholly compelled our sympathies and concerns; would we then not seek contact with them, despite the availability of medications? Perhaps for the pleasure of it all?

Unfortunately, these wise words have been forgotten in present-day psychiatry where the patients' personal histories count for so little that the psychiatrists often fail to unravel them.

Rosenhan describes how powerlessness was evident everywhere. The patient was deprived of many of his legal rights and was shorn of credibility because of his psychiatric label. The pseudopatients observed abusive behaviour, which was terminated quite abruptly when other staff members were known to be coming; staff were credible witnesses, the patients were not.

Rosenhan concludes that we cannot distinguish the sane from the insane in psychiatric hospitals and wonders how many sane people that are not recognised as such in our psychiatric institutions? And how many patients that might be sane outside the psychiatric hospital but seem insane in it because they are responding to a bizarre setting?

A research and teaching hospital whose staff had heard of Rosenhan's findings doubted that such an error could occur at their hospital. Rosenhan therefore informed the staff that at some time during the following three months, one of more pseudopatients would attempt to be admitted into the psychiatric hospital. Each staff member was asked to judge whether a patient was a pseudopatient. Forty-one of 193 patients (21%) were alleged, with high confidence, to be pseudopatients by at least one member of the staff. However, Rosenhan had not admitted *any* pseudopatients!

Very many people are wrongly diagnosed with schizophrenia. A 1982 study found that two-thirds of 1,023 African-Americans with schizophrenia didn't have symptoms necessary for this diagnosis according to current guidelines.¹¹ In 1985, the chief psychiatrist at Manhattan State Hospital reviewed the records of 89 patients with schizophrenia and concluded that only 16 should have gotten the diagnosis.¹¹

Erroneous diagnoses can be fatal. In one such case, a child with Asperger was treated with antipsychotic drugs, which triggered schizophrenia-like symptoms, including psychosis, whereby the erroneous diagnosis became a self-fulfilling prophecy, which ultimately killed her because of the drugs that were enforced on her against her will (see Chapter 15, Dear Luise).¹²

It is not as odd as it might seem that many people are wrongly diagnosed with schizophrenia. Psychiatry is radically different from other areas of medicine, as normal people have similar symptoms and feelings as patients have; it is mostly a matter of degree. Even for schizophrenia, this is the case. Psychosis is not a biological illness like arthritis, and many normal people have psychotic experiences – including delusions and hallucinations – from time to time.

The demons attack you

When we have made a diagnosis, whether right or wrong, we blow life into our social construct, e.g. the Mayo Clinic staff said that the disorder affects you, as if it had some independent existence.

The patient's symptoms are real, but the diagnostic label is not real in the sense that it defines something that exists independent of us. An elephant truly exists and may attack us if we come too close. We also say that diseases attack us, e.g. "she had an asthma attack," like if asthma had some real existence in nature, like an elephant.

You may feel I am getting too philosophical, so I shall therefore explain in Chapter 5, about ADHD, why these distinctions can be very important. Here is another example. When a friend of mine was admitted to hospital in her twenties with acute psychosis, the psychiatrist said: "You are schizophrenic!" At that point, she felt she stopped existing as a person, with autonomy and dignity. She was no longer someone that her carers needed to respect, she was a bag of symptoms they took control over, and the following years were devastating for her.

Subtle differences can be important. If her psychiatrist had said: "You are a person who currently has symptoms, which we usually call schizophrenia," it would have indicated that the person was still there and was so much more than her symptoms, and that the disease would not necessarily last for the rest of her life, which, unfortunately, is often how psychiatrists have perceived this disease. They haven't realised that it is them, with their antipsychotic drugs, who have made the troubles lifelong (see Chapter 11).

Let there be disorder

"And DSM said: Let there be disorder"

KLRK, GOMERY AND COHEN IN *MAD SCIENCE*¹³

In its fourth edition, the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) from the American Psychiatric Association tried to define what a mental disorder is.¹⁴ I have highlighted in italics some of the more problematic bits:

A clinically significant behavioral or psychological syndrome or pattern that occurs in an individual and that is associated with present distress (e.g., a painful symptom) or disability (i.e., impairment in one or more important areas of functioning) or with a significantly increased

risk of suffering death, pain, disability, or an important loss of freedom. In addition, this syndrome or pattern must not be merely an *expectable and culturally sanctioned response* to a particular event, for example, the death of a loved one. Whatever its original cause, *it must currently be considered* a manifestation of a behavioral, psychological, or biological *dysfunction* in the individual. Neither deviant behavior ... nor conflicts that are *primarily* between the individual and society are mental disorders unless the deviance or conflict is a *symptom of a dysfunction* in the individual.

This definition is extremely elastic and includes many judgments, also with regard to the degree of the phenomena being described. This ambiguity results in large observer variation when independent psychiatrists assess whether a given person has a mental disorder or not and which one it is.^{14, 15}

It is quite impossible to make all this ambiguity and subjectivity operational, and it would be easy to suggest a more meaningful and robust definition. The DSM is a consensus document, however, and its diagnoses are unscientific and arbitrary. Real sciences do not decide on the existence and nature of the phenomena they are dealing with via a show of hands with a vested interest and pharmaceutical industry sponsorship.¹⁶

The claim that the extensive new diagnostic checklist system introduced in DSM-III in 1980 is reliable has been convincingly refuted in a book.¹⁵ The disappointing results when two psychiatrists evaluated the same people have been buried in a smoke of positive rhetoric in surprisingly short articles, given the importance of the subject. The documentation is hard to find, but the book says it all. Its two authors did a formidable job in casting light on this issue that no one has wanted to debate in the American Psychiatric Association. Even the largest study, of 592 people, was disappointing despite the fact that the investigators took great care in training the assessors.¹⁷ For bulimia nervosa, which is extremely easy to diagnose, the kappa values when two physicians interviewed the same people were above 0.80, but for major depression and schizophrenia, two of the most important diagnoses, the kappas were only 0.64 and 0.65, respectively.

Since we cannot say decisively what a mental disorder is, we could try the accepted diagnostic procedures on healthy people to see whether they also get psychiatric diagnoses. Indeed they do. I looked up Psych Central, a large website that has been highly praised by neutral observers and has won awards.¹⁸ We were eight normal and successful people who tried the tests for depression, ADHD and mania, and none of us survived all three tests. Two had depression and four had

definite, likely or possibly ADHD. Seven suffered from mania; one needed immediate treatment, three had moderate to severe mania, and three had milder degrees.

My results have been confirmed by others, which suggests that there is one or more psychiatric diagnoses awaiting each of us. Rosenhan showed that American psychiatry had no clothes, which was confirmed in another study from the 1970s.¹¹

When researchers interviewed 463 people, they found that all of them experienced thoughts, beliefs, moods, and fantasies that, if isolated in a psychiatric interview, would support a diagnosis of mental illness.

Denmark recently introduced a new law that specifies that patients admitted to hospital are guaranteed a diagnosis within four weeks. This can be helpful in reducing the stressful waiting time for people who don't know if they have cancer or not, but the law was much criticised, for good reasons. For example, many ailments are self-limiting, and as all treatments can lead to harm, it is often in the patients' best interest *not* to get a diagnosis, as doctors have difficulty in *not* treating when they have a diagnosis. They have learned a lot about using drugs for everything one can possibly imagine, and also for what one cannot imagine, during their medical studies, but very little about when it would be best to just wait and see. My own take on the new law is that if you approach a doctor with a mental health problem, you are guaranteed at least one diagnosis!

It's not surprising that when therapists were asked to use DSM criteria on healthy people, a quarter of them also got a psychiatric diagnosis.¹⁶ Imagine if you tested healthy people for cancer with a test that gave a quarter of them an erroneous diagnosis, which led to treatment with chemotherapy for a cancer that wasn't there. We wouldn't allow such a poor test to be used in any other area of healthcare except psychiatry.

DSM-III from 1980 was replaced by DSM-IV in 1994, which was even worse than its predecessor and lists 26% more ways to be mentally ill.¹⁶ Allen Frances, chairman for the DSM-IV task force, now believes the responsibility for defining psychiatric conditions needs to be taken away from the American Psychiatric Association (APA) and argues that new diagnoses are as dangerous as new drugs: "We have remarkably casual procedures for defining the nature of conditions, yet they can lead to tens of millions being treated with drugs they may not need, and that may harm them."¹⁹ Frances noted that DSM-IV created three false epidemics because the diagnostic criteria were too wide: ADHD, autism and childhood bipolar disorder.

Psychologist Paula Caplan was involved with the DSM-IV and fought hard to get the silliest ideas out.¹⁴ In 1985, when the APA decided to introduce Masochistic Personality Disorder to be used for women who were beaten up by their husbands, Caplan and her colleagues mockingly inventing Macho Personality Disorder that evolved into Delusional Dominating Personality Disorder for the violent males, which they suggested would apply if a man fulfilled 6 of 14 criteria, of which the first was “Inability to establish and maintain meaningful interpersonal relationships.”

A crucial question in the clinical encounter is: Do I have a good reason to believe that it would help to give this person a diagnosis? Some of us still remember Minimal Brain Damage Dysfunction, which was thrown in the faces of millions of parents although it could only be harmful, as there was nothing they could do.

Professionals other than psychiatrists are also keen to overdiagnose and overtreat people. When my wife was pregnant for the first time, my main role was to keep the professionals away from her, and I demonstrated time and again for them that the interventions they suggested were either useless or harmful, with reference to an evidence-based book based on systematic reviews of the randomised trials.²⁰ This was how the Cochrane Collaboration, to which I belong, was born; it started literally with pregnancy and childbirth. Shortly after our first daughter was born, my wife and I were visited by a nurse who declared that our daughter would have difficulty talking, as the ligament under her tongue was too tight. We had a big laugh after the nurse was gone. She didn't know what she was talking about, and even if it had been true, there was no treatment, so why invent a false diagnosis?

Very few leading psychiatrists are willing to admit that their specialty has spiralled out of control and when issues of overdiagnosis and overtreatment are brought up, their standard reply is that many patients are underdiagnosed. Of course there will always be some overlooked patients, but the main problem is not underdiagnosis but overdiagnosis, which those psychiatrists that are not silverbacks know perfectly well. In a 2007 survey, 51% of 108 Danish psychiatrists said they used too much medicine and only 4% said they used too little.²¹

I consider it organised denial, whose purpose is to protect guild interests, that silverbacks all over the world ignore the clear results of the loose diagnoses and the loose hand at the prescription pad. Sales of drugs for the nervous system in Denmark are so high that one-quarter of the whole population could be in treatment. In the United States, the most sold drugs in 2009 were antipsychotics,

and antidepressants came fourth, which cannot possibly reflect genuine needs, but it gets worse all the time.¹⁸

Our children have not avoided the disease mongering. In the United States, 1% of children up to only four years of age are on psychotropic drugs, although the first three years of life are a period of rapid neurodevelopment,²² and about a quarter of the children in American summer camps are medicated for ADHD, mood disorder or other mental health problems.¹⁸

It is psychiatry that has become insane, not our children. Some child psychiatrists brag that they can make an initial assessment of a child and write a prescription in less than 20 minutes, and for some paediatricians it takes only five minutes.²³

Why is it that leading psychiatrists cannot get enough? Isn't this behaviour so bizarre, abnormal, socially dysfunctional, and harmful towards others, that, in accordance with the psychiatrists' own way of thinking, it would be legitimate to invent a diagnosis for it? An appropriate name could be *Obsessive Compulsive Disease Mongering Disorder, OCDMD*, which could also be short for *Obvious Common Desire of Money-making Diagnoses*. The diagnostic criteria could be a disturbance of at least six months during which at least five of the following are present:

1. Has been on industry payroll within the last three years.
2. Is willing to put his or her name on ghost-written manuscripts.
3. Believes that getting a diagnosis cannot hurt.
4. Believes that screening cannot hurt, as the drugs have no side effects.
5. Believes that people with psychiatric disorders have a chemical imbalance in the brain.
6. Tells patients that psychiatric drugs are like insulin for diabetes.
7. Believes that depression and schizophrenia destroy the brain and that drugs prevent this.
8. Believes that antidepressants protect children against suicide.
9. Believes information from drug companies is useful.

I have come across psychiatrists who have a full house, i.e. for whom all nine criteria apply. I am against forced treatment (see Chapter 15), but I am in favour of forced retirement for doctors who suffer from OCDMD in order to protect other people from harm.

You may think I am being unfair to psychiatry, but my criteria are actually more

reasonable than the criteria in DSM-III for Oppositional Defiant Disorder in children.¹⁵

“A disturbance of at least six months during which at least five of the following are present:

1. Often loses temper.
2. Often argues with adults.
3. Often actively defies or refuses adult requests or rules, e.g., refuses to do chores at home.
4. Often deliberately does things that annoy other people, e.g., grabs other children's hats.
5. Often blames others for his or her own mistakes.
6. Is often touchy or easily annoyed by others.
7. Is often angry and resentful.
8. Is often spiteful and vindictive.
9. Often swears or uses obscene language.”

These criteria are totally subjective and arbitrary, and “often” is part of them all. How often is “often” supposed to be? Many children fulfil all nine criteria, and yet only five are needed for a “diagnosis.” For what purpose? As far as I can see, this is pretty normal behaviour.

I am sure that naivety, ignorance and the urge to do good play a role for the silly diagnoses, but there is a darker side to it. Many of those who develop DSM have heavy conflicts of interest in relation to the drug industry and creating many diagnoses means money, fame and power for those at the top.¹⁴ It is also about getting control over others, which is inherent in our biology. Putting diagnoses on people is a powerful instrument that makes them dependent on what their psychiatrists feel and think, and it leads to abuse (see Chapter 15). A patient told me that when she felt her psychiatrist behaved in a God-like manner and asked him whether he thought he was God, he punished her by adding an additional diagnosis, borderline personality disorder.

The most prominent American child psychiatrist, Joseph Biederman, who has likely done more than anybody else to overdose our children with antipsychotics through his invention of juvenile bipolar disorder,^{13, 24} has also behaved in a God-like manner. At a court trial, an attorney asked him about his rank at Harvard Medical School. “Full professor,” he replied. “What’s above that?” the attorney asked. “God,” Biederman replied.²⁴

Some psychiatrists cannot even resist the temptation of putting diagnostic labels on their opponents in public debates. Henrik Day Poulsen is probably the doctor in Denmark who collaborates the most with drug companies. In 2013, he was an Advisory Board member or a consultant for six companies, and “educated” other doctors for nine companies. Like his benefactors, he didn’t like my book about deadly medicines and organised crime in the drug industry,¹⁸ and wrote in a newspaper article that I, “in my usual paranoid manner,” had showed off with examples how the ugly drug industry cheats and defrauds people.²⁵ Usual paranoia means having a chronic psychosis characterised by delusions, i.e. being insane.

On another occasion, when a politician with a background as a psychiatric nurse said that, given his income from the drug industry, she was in doubt about whether he worried about the patients or provided a sales pitch for using more pills, he called her “desperate.”²⁶ Poulsen has more diagnoses up this sleeve; he has published the book “Everyday’s psychopaths.”

Psychiatric drugs lead to many wrong diagnoses

There are several reasons – but few good ones – why many mental health patients have more than one diagnosis. First, the diagnostic criteria are very broad and highly unspecific for the problems patients have. Second, there is a lot of overlap between the different diagnostic categories and a propensity of one condition to change into another over time. This is often called high comorbidity, although the problem is not that the patient has several “diseases” but that the diseases are so vaguely defined that it is like a biologist looking at a shadow at a distance who says: “It is an elephant and a wildebeest and possibly also a rhinoceros.” Third, the drugs’ side effects are often misinterpreted as new disorders. Prescribing one drug therefore often leads to prescribing of other types of drugs in cascade fashion. For example, an antipsychotic may cause the patient to feel lethargic and depressed, which leads to an antidepressant; and if started on an antidepressant, the patient may develop symptoms of mania, which leads to an antipsychotic.^{9, 24}

Doctors need to realise that it’s impossible to judge whether a patient truly also suffers from these additional “illnesses,” as long as the patient is under influence of mind-altering chemicals.⁹ The adverse effects can come and go, which is an important reason why people think it cannot be the drug.²⁷ In this way, not only routine treatment but also attempts at withdrawing a drug – which often elicit these side effects – can lead to more diagnoses, more drugs and more harm.

Addiction experts know perfectly well that it is futile to diagnose underlying psychiatric disorders when a patient is abusing drugs. Drug abuse and dependence with their cycles of intoxication and withdrawal mimic every possible psychiatric problem. Then why don't psychiatrists abstain from making diagnoses when people are under influence of those brain-active chemicals we call psychiatric drugs?²⁷

It should be forbidden to make new diagnoses while the patient is in treatment with psychotropic drugs, and if psychiatrists cannot resist the temptation, they should by default call it a likely drug-induced disorder. This will put the blame on themselves and not on the patient, and will increase the likelihood that psychiatrists would taper the drugs, as they would be afraid of litigation, if they did nothing after having diagnosed a drug-induced disorder.

A fourth important reason for the far too many diagnoses is that the diagnoses are often made at the first visit, when the patients may turn up with sadness, stress at work, marital problems, a recent trauma or so much else that many of us will experience from time to time. Doctors tend to forget that the diagnosis is a snapshot, and that the patients might be fine both before and after their visit to the doctor. Obviously, the more a person visits a doctor, the higher the risk of getting a false diagnosis.

Doctors should be patient and should try to avoid putting diagnostic labels on people at their first visit, also because diagnoses are sticky. Even when proved wrong later on, diagnoses are almost impossible to get rid of again, and they stigmatise people (see Chapter 6) and may have implications for employment, insurance, and many other important issues.

Doctors should also avoid prescribing drugs at the first encounter unless the situation is very acute. If a patient insists on getting a drug, e.g. an antidepressant, an honest discussion of its many harms and its doubtful benefits (see Chapter 3) should convince most patients that it is not a good idea to rush into action.

The diagnostic labels psychiatrists use fit very poorly with the type of patients general practitioners meet, but any challenge to specialist perspectives on mental disorders in primary care usually generates incredulity among psychiatrists and a reinforcement of their belief that retraining of primary care workers is the solution.²⁸ Retraining in what? Not in the DSM, I hope!

The Goodness Industry

Our “doing good” culture poses a major health risk in the psychiatric field. Institutions such as kindergartens and schools may put pressure on parents to accept dubious diagnoses like ADHD to obtain additional funding, and other institutions may put pressure on psychiatrists to obtain a diagnosis of post-traumatic stress disorder.

It can also be rewarding for people themselves to play sick to get a diagnosis, which can open the floodgates for all sorts of benefits in terms of increased social services, educational support, flex jobs, light jobs, early retirement, disability allowance, insurance claims, and whatever else. As an example, education benefits in Denmark can be 2.4 times higher for people who have been diagnosed with schizophrenia, schizotypal personality disorder, persistent psychotic condition, short-term psychotic condition, schizoaffective disorder, unspecified psychosis of nonorganic origin and emotionally unstable personality structure of borderline type. The borderline diagnosis in particular is a pretty elastic one.

In the Goodness Industry, too many therapists are too tempted to do too much for too many people, and patient representatives – often supported by the pharmaceutical industry – are often wrong when they claim that their members are underdiagnosed, undertreated, and underprioritised.

I have heard several senior psychiatrists say it cannot hurt anyone to get a diagnosis. Such people shouldn't work as psychiatrists. All professional interventions in citizens' lives, including giving people diagnoses, can cause harm. It is a paradox that public debates and reports in the news media are dominated by the beneficial effects of diagnoses and interventions when the first thing we know about any intervention is that it can be harmful. If this were not the case, it could not have any potentially beneficial effect.

Patients are not consumers

In the Anglo-Saxon tradition, patients are often called consumers, but it is a strange term. Patients don't consume anything; in fact, the psychiatric drugs consume them, as they take their personality away. Consumption was the old term for tuberculosis, which “eats” the tissues. Similarly, psychiatric drugs “eat” the brain if taken for a long time, as they cause chronic brain damage (see Chapter 11).

When patients with breast cancer, prostate disease, fractures and HIV were asked, they preferred to be called patients, not consumers, clients, customers, or anything else.²⁹ Many alternatives to “patient” incorporate assumptions (e.g. a market relationship), which care recipients may find objectionable. We should

respect this and drop the term consumer. It was introduced with good intentions about empowering patients but this can be done without calling them something they don't want to be called and which is pretty misleading, too.

More funny and fake diagnoses

When life gets too absurd, a good laugh can help. Two funny videos illustrate how easy it is to convince healthy people to take drugs they don't need for a disease they don't have. The Australian artist Justine Cooper invented a TV commercial that advertises Havidol (have it all), with the chemical name avafynetyne HCl (have a fine time plus hydrochloric acid).^{30, 31} Havidol is for those who suffer from dysphoric social attention consumption deficit anxiety disorder (DSACDAD).

Feel empty after a full day of shopping? Enjoy new things more than old ones? Does life seem better when you have more than others? Then you may have the disorder, which more than 50% of adults have. Havidol should be taken indefinitely, and side effects include extraordinary thinking, dermal gloss, markedly delayed sexual climax, inter-species communication and terminal smile. "Talk to your doctor about Havidol." Some people believed it was for real and folded it into real websites for panic and anxiety disorder or for depression.

Another video featured journalist Ray Moynihan.³² A new epidemic – motivational deficiency disorder – was first announced in the BMJ's 1 April issue in 2006,³³ and like for Havidol, some people believed the disease existed. In its mild form, people cannot get off the beach or out of bed in the morning, and in its most severe form it can be lethal as the sufferer may lose the motivation to breathe. Moynihan says: "All my life people have called me lazy. But now I know I was sick." The drug is Indolebant, and its champion, neuroscientist Leth Argos, reports how a patient's wife telephoned him and was in tears. After having used Indolebant, her husband had mowed the lawn, repaired the gutter and paid an electricity bill – all in one week.

I showed these two videos as an introduction to my talk about overdiagnosis and overtreatment when I lectured for over 100 psychiatrists in 2012. They laughed out loud but not when I added that what they had just seen wasn't far from their everyday practice.

A patient once told me she suffered from chronic fatigue syndrome but she described many weird symptoms that couldn't possibly be disease symptoms. A little later, I told the company I was in about the video with Moynihan's motivational deficiency disorder; everyone laughed but her. Perhaps she got my

hint.

There is a cartoon where the doctor says to the patient: “We can’t find anything wrong with you, so we’re going to treat you for Symptom Deficit Disorder.” I also came across *Disorder Fabrication Syndrome*, invented by Barry Turner, Lecturer in Medical Ethics and Law:³⁴

A new psychiatric condition has been observed by psychiatrists working at the Brandt-Sievers Institute for Eugenics. The condition, Disorder Fabrication Syndrome, is a kind of paranoid delusional disorder where the sufferer believes in their own infallibility and superiority and is often associated with comorbid narcissistic personality disorder. The sufferer will incessantly classify all manner of normal human behaviour as a disorder or syndrome.

The disorder is thought to be caused by a chemical imbalance brought on by studying psychology and psychiatry at an institute funded by big pharma. The constant handling of money doled out by the drug companies seems to affect the way the psychologists and psychiatrists process neurotransmitters. Another theory is that this might be a kind of hysteria induced by chronic avarice.

The most effective treatment for this group of patients is to strike them off any professional registers which makes their craving for pharmaceutical company money remit. In extreme cases, prosecuting them for research fraud is another alternative. This sometimes controversial method has just been applied with great success at the University of Vermont.

It is believed that the condition is underdiagnosed in psychiatrists and clinical psychologists and that a screening programme ought to be introduced in this high risk population.

There are many silly diagnoses in psychiatry that could be used to label many people, e.g. *Premenstrual Dysphoric Disorder*, which is also harmful, as the diagnosis might prevent women from getting a job or have custody of their children in case of a divorce.¹⁴ The criteria for this diagnosis are so unspecific that they cannot distinguish between women with severe premenstrual symptoms and other women, and even men give answers similar to women with severe symptoms,¹⁴ so I take it that men should be treated indefinitely, as they have no periods. The FDA didn’t care. It approved fluoxetine for this non-disease, which the US psychiatrists had the gall to call depression!¹⁸ Eli Lilly gave the drug another name, Sarafem, which was a repainted Prozac with attractive lavender and pink colours. Pretty ironic colours on a pill that ruins people’s sex lives (see next chapter). In Europe, Lilly was forbidden to promote fluoxetine for something

that wasn't a disease, and the European Medicines Agency fiercely criticised Lilly's trials, which had major deficiencies. The Cochrane review of this non-disease included 31 trials and it found antidepressants to be effective.³⁵ Of course. Everything that has side effects (and there were plenty)³⁵ seems to work when the outcomes are subjective (see next chapter), both for diseases and non-diseases.

References

- 1 Gøtzsche PC. Rational diagnosis and treatment: evidence-based clinical decision-making, 4th edition. Chichester: Wiley; 2007.
- 2 Mayo Clinic Staff. Mental illness. <http://www.mayoclinic.org/diseases-conditions/mental-illness/basics/definition/con-20033813> (accessed 11 Nov 2014).
- 3 Østergaard SD, Jensen SOW, Bech P. The heterogeneity of the depressive syndrome: when numbers get serious. *Acta Psychiatr Scand* 2011;124:495-6.
- 4 McHugh PR, Slavney PR. Mental illness - comprehensive evaluation or checklist? *N Engl J Med* 2012;366:1853-5.
- 5 Medawar C. The antidepressant web - marketing depression and making medicines work. *Int J Risk & Saf Med* 1997;10:75-126.
- 6 Kessler RC, Demler O, Frank RG, et al. Prevalence and treatment of mental disorders, 1990 to 2003. *N Engl J Med* 2005;352:2515-23.
- 7 Varese F, Smeets F, Drukker M, et al. Childhood adversities increase the risk of psychosis: a meta-analysis of patient-control, prospective and cross-sectional cohort studies. *Schizophr Bull* 2012;38:661-71.
- 8 Wulff HR, Pedersen SA, Rosenberg R. *Philosophy of medicine*. London: Blackwell; 1986.
- 9 Breggin P. *Psychiatric drug withdrawal: a guide for prescribers, therapists, patients, and their families*. New York: Springer Publishing Company; 2013.
- 10 Rosenhan DL. On being sane in insane places. *Science* 1973;179:250-8.
- 11 Whitaker R. *Mad in America*. Cambridge: Perseus Books Group; 2002.
- 12 Christensen DC. *Dear Luise: a story of power and powerlessness in Denmark's psychiatric care system*. Portland: Jorvik Press; 2012.
- 13 Kirk SA, Gomory T, Cohen D. *Mad science: psychiatric coercion, diagnosis and drugs*. New Brunswick: Transaction Publishers; 2013.
- 14 Caplan PJ. *They say you're crazy: how the world's most powerful psychiatrists decide who's normal*. Jackson: Da Capo Press; 1995.
- 15 Kirk SA, Kutchins H. *The selling of DSM: the rhetoric of science in psychiatry*. New York: Aldine de Gruyter; 1992.
- 16 Watts G. More psychiatrists attack plans for DSM-5. *BMJ* 2012;344:e3357.
- 17 Williams JB, Gibbon M, First MB, et al. The Structured Clinical Interview for DSM-III-R (SCID). II. Multisite test-retest reliability. *Arch Gen Psychiatry* 1992;49:630-6.
- 18 Gøtzsche PC. *Deadly medicines and organised crime: How big pharma has corrupted health care*. London: Radcliffe Publishing; 2013.

- 19 Moynihan R. Medicalization. A new deal on disease definition. *BMJ* 2011; 342:d2548.
- 20 Enkin M, Keirse MJNC, Renfrew M, et al. A guide to effective care in pregnancy & childbirth. Second edition. Oxford: Oxford University Press; 1995.
- 21 [Work environment and treatment modalities in Danish psychiatry]. *Nordjyske Medier*; 2007.
- 22 Garfield LD, Brown DS, Allaire BT, et al. Psychotropic drug use among preschool children in the Medicaid program from 36 states. *Am J Public Health* 2015;105:525-9.
- 23 Roberts EJ. A rush to medicate young minds. *Washington Post* 2006 Oct 8.
- 24 Whitaker R. Anatomy of an Epidemic. New York: Broadway Paperbacks; 2010.
- 25 Poulsen HD. [2014: The year where the patient will be the loser]. *Berlingske* 2013 Dec 25.
- 26 Schmidt AL. [Two hats? Özlem Cekiç doesn't trust Henrik Day Poulsen]. *Politiken* 2014 Sept 10.
- 27 Breggin P. Medication madness. New York: St. Martin's Griffin; 2008.
- 28 Jacob KS, Patel V. Classification of mental disorders: a global mental health perspective. *Lancet* 2014;383:1433-5.
- 29 Deber RB, Kraetschmer N, Urowitz S, et al. Patient, consumer, client, or customer: what do people want to be called? *Health Expectations* 2005;8:345-51.
- 30 Coombes R. Having the last laugh at big pharma. *BMJ* 2007;334:396-7.
- 31 HAVIDOL: female testimonial. www.youtube.com/watch?v=sQw_cdhXGco (accessed 27 Dec 2014).
- 32 A new epidemic (motivational deficiency disorder). Available online at: www.youtube.com/watch?v=RoppJOtRLe4 (accessed 27 Dec 2014).
- 33 Moynihan R. Scientists find new disease: motivational deficiency disorder. *BMJ* 2006;332:745.
- 34 Turner B. Disorder Fabrication Syndrome. <http://www.adhdtesting.org/history%20of%20add-adhd.htm> (accessed 27 Dec 2014).
- 35 Marjoribanks J, Brown J, O'Brien PMS, et al. Selective serotonin reuptake inhibitors for premenstrual syndrome. *Cochrane Database Syst Rev* 2013;6: CD001396.

3

Depression

Screening for depression

The diagnostic criteria in psychiatry are very broad, and they should therefore not be applied on healthy people. Such screening is a sure way to make us all crazy. A notorious programme in the United States was TeenScreen, which came up with the result that one in five children suffer from a mental disorder, leading to a flurry of discussions about a “crisis” in children’s mental health.¹

It wasn’t a crisis in children’s mental health but a crisis in the standard of psychiatric research² plus a chronic impairment of the intellectual capacity or honesty of some leading psychiatrists. We usually say that a screening tool shouldn’t lead to too many false negative findings, but for depression, it doesn’t really matter if we overlook some cases. It is so easy to spot the severe cases of depression. Therefore, what is being overlooked are the mild cases, which are self-limiting and for which there is consensus that antidepressant drugs don’t work.

What is important is that there should not be too many false positives, i.e. healthy people who are diagnosed with depression, but this is exactly what we get. The screening test recommended by the World Health Organization is so poor that for every 100,000 healthy people screened, 36,000 will get a false diagnosis of depression.^{3,4} When I criticise my colleagues for using such poor tests, I am told that they are only a guideline in the diagnostic work-up and that additional testing will be performed. In an ideal world perhaps, but this is not what most doctors do. Many patients report that there was no further testing and that they got a diagnosis and a prescription in about ten minutes.⁵ This is expected, as 80-90% of prescriptions are written by general practitioners,^{5,6} and they don’t have much time.

That the standard of psychiatric research is very poor is illustrated by the fact that in only 5% of the studies assessing the false positive and false negative results of screening for depression had the researchers excluded patients who were already diagnosed with depression.² This flaw is inexcusable. If we want to

know how good ultrasound is to pick up cancers in the stomach of people who look healthy, we don't study people who have already been diagnosed with large cancers with ultrasound, the very technique we want to test.

The Cochrane review on screening for depression recommends firmly against it, after having examined 12 trials with 6,000 participants.⁷ Nonetheless, the Danish National Board of Health recommends screening for depression.⁸ Our health authorities are masterminds in the sport of eating a cake and still having it. After dutifully quoting the Cochrane review, the authorities recommend screening for various poorly defined “risk groups,” which are:

- Previous depression
- Depression in the family
- Heart disease
- Stroke
- Chronic pain
- Diabetes
- Smoker's lungs
- Cancer
- Parkinson's disease
- Epilepsy
- Other mental disorders (because of comorbidity with depression)
- Pregnant women
- Women who just had a baby
- Refugees
- Immigrants.

This impressive list of people in “risk groups” cover a considerable part of the population. Unsurprisingly, there were many psychiatrists in the working group that came up with these recommendations.

When I – as invited speaker at large scientific meetings for psychiatrists – have pointed out how harmful screening for depression is, they didn't pay the slightest attention. This organised denial has shocked me, particularly when the professor at my own hospital – in reply to my remark that screening leads to treatment of many healthy people with antidepressants – said that it didn't matter because antidepressants have no side effects! Really? To a substantial extent, the “risk groups” comprise those who have lived more than five decades, and antidepressants kill many of these (see Chapter 14).⁹

It is difficult to know how much overdiagnosis of depression there is, as we don't even know what a true diagnosis is. If we count elephants, we don't suddenly decide to include also wildebeests in our count just because they are also greyish and have four legs. For depression, however, the criteria for the diagnosis have been broadened enormously over the years. Based on the nine criteria in DSMIV, psychiatrists have calculated that one can be depressed in at least 1,497 different ways.¹⁰ Some of these variations are not really what most people would call depression.

In 2010, the US Centers for Disease Control and Prevention stated that 9% of the interviewed adults met the DSM-IV criteria for current depression.³ However, very little is required. You are depressed if you have had little interest or pleasure in doing things for eight days over the past two weeks plus one additional symptom, which can be many things, for example:

- trouble falling asleep
- poor appetite or overeating
- being so fidgety or restless that you have been moving around a lot more than usual.

This is unreasonable. A boy whose sweetheart abandoned him will feel miserable for all fourteen days and cannot sleep or eat much. Little pleasure in doing things will happen to most of us, no matter how positive, active and outgoing we are, and most Americans overeat.

With such an approach to diagnosis, it is not surprising that the prevalence of depression has increased dramatically since the days when we didn't have antidepressant pills.¹¹ And there is a substantial risk of circular evidence in all this. If a new class of drugs affect mood, appetite and sleep patterns, depression may be defined by industry supported psychiatrists as a disease that consists of just that; problems with mood, appetite and sleep patterns.¹²

I have listened to many pseudo-academic discussions where people have tried to explain why there are more depressed people now than previously. The usual explanation is that our society has become more hectic and puts greater demands on people. As far as I can see, we are more privileged than ever before, our lives are less stressful, social security is far better, and there are far fewer poor people. It is more reliable to estimate whether the prevalence of severe depression has increased, and psychiatrists constantly tell me that this is not the case.¹³

One of the signs that US silverbacks have run amok is the medicalisation of grief. In DSM-III, bereavement was a depressive disorder only if it had lasted for

more than a year, in DSM-IV it was two months, and in DSM-5 from 2013 it is only two weeks. Few marriages are so bad that the person left behind will rejoice after only two weeks that the partner is gone. We should allow people to be unhappy at times – which is completely normal – without diagnosing them. In clinical practice, these “limits” are immaterial of course. A clinician will not tell a sad person to wait another week before he fulfils the diagnostic criteria for depression and can get a prescription.

The major change in DSM-III from 1980 was the introduction of a symptom-based approach for diagnosis. It has been criticised for creating diseases and for classifying normal life distress and sadness as mental disease in need of drugs. Expected reactions to a situational context, for example the loss of a beloved person, divorce, serious disease or loss of job, are no longer mentioned as exclusion criteria when making the diagnosis. These changes, which are so generous towards the drug industry, could be related to the fact that all the DSM-IV panel members on mood disorders had financial ties to the pharmaceutical industry.³

Public debates on overdiagnosis are frustrating, and I wonder whether leading psychiatrists don't understand the issues, or whether they display organised denial in order to cover up for their failures. We use so many antidepressants in Denmark that every one of us could be in treatment for six years of our lives, and the increased use of these drugs doesn't reflect an increasing need; it is closely related to the marketing pressure.¹⁴ This should make everybody's alarm bells ring, but when the TV host asked us during a panel discussion how we could reduce the high consumption and expressly pointed out that we should not discuss whether the consumption was too high, Professor Lars Kessing didn't reply to the question but said the consumption wasn't too high because the prevalence of depression had increased greatly during the last 50 years.

The denial seems epidemic. Another chief physician at Kessing's department recently told a journalist that the consumption of antidepressants corresponds to the number of sick patients.¹⁵ Such statements that all is fine are misleading. Although there is international agreement that antidepressants don't work for mild depression and shouldn't be used, most of those treated have rather mild depression.⁶

Studies have shown that more people are overdiagnosed than underdiagnosed for depression,¹³ which shouldn't surprise anyone who is not a psychiatrist. In fact, the term “major depressive disorder” has become contradictory in terms, as it now includes cases of mild major depressive disorder. This is also meaningless since such cases are neither major, nor depression, nor even a disorder.¹³

Antidepressant drugs don't work for depression

We have used antidepressant drugs for more than 50 years, but it is unlikely that they have a real and useful effect on depression, whereas their many harmful effects are not in doubt. Antidepressants cause more harm than good,¹⁶ which the remainder of this chapter is about.

The US Food and Drug Administration (FDA) found in a metaanalysis of randomised trials with 100,000 patients, half of whom were depressed, that about 50% got better on an antidepressant and 40% on a placebo.¹⁷ A Cochrane review of depressed patients in primary care reported slightly higher benefits,¹⁸ but didn't include the unpublished trials, which have much smaller effects than the published ones.¹⁹

Most doctors call the 40% in the placebo group a placebo effect, which it isn't. Most patients would have gotten better without a placebo pill, as this is the natural course of an untreated depression. Therefore, when doctors and patients say they have experienced that the treatment worked, we must say that such experiences aren't reliable, as the patients might have fared equally well without treatment.

It is important to understand these issues. When I point out in public debates that antidepressants are pretty ineffective, psychiatrists often say that they are still useful, as the patients benefit from the placebo effect. But how large is the placebo effect? One of my senior researchers, Asbjørn Hróbjartsson, wanted to find this out, so he collected all trials where the patients were randomised to placebo or nothing (often called a waiting list control group). In the most recent update of our review, we included 234 trials investigating 60 clinical conditions in all areas of healthcare.²⁰ We found that placebo interventions do not have important clinical effects, apart from a few areas such as pain and nausea. However, we cannot know whether these effects are real or the patients just tried to be kind to the experimenting doctor. The problem with such studies is that we cannot blind a trial where half the patients get something that looks like an active treatment, and the other half don't get anything. Psychological research has taught us that in such settings, and when the outcome is subjective, positive effects will be considerably exaggerated.

Hróbjartsson recently published another important study. He wanted to see to what extent observers who had not been blinded to the treatment patients received exaggerate the effect, and he collected all trials that had both a blinded and a non-blinded observer.²¹ He included 21 trials for a variety of diseases and found that

the treatment effect was overestimated by 36% on average (measured as odds ratio) when the non-blinded observer assessed the effect compared to the blinded observer. Most of the studies had used subjective outcomes, and as the effect of antidepressants is also assessed on subjective scales (e.g. the Hamilton scale), Hróbjartsson's results are directly relevant for antidepressant trials, as these trials have not been effectively blinded. Antidepressant medications have conspicuous side effects, and many patients and doctors will therefore know if the blinded drug contains an active substance or placebo. If we assume that the blinding is broken for all patients in the antidepressant trials, and adjust for the bias the loss of blinding causes, we will find that antidepressants have no effect (odds ratio 1.02).^{3, 16}

The blinding needs not be broken for all patients, however. All that is required for the effect to disappear is that 5% of the patients are misclassified in terms of whether they became better or not, as the 50% effect on active drug then becomes 45%, and the 40% effect on placebo also becomes 45%. The blinding is generally broken for many patients in psychotropic drug trials, in some cases for all patients, as in a trial of alprazolam versus placebo.²² The authors of a review of blinding problems ended their paper by saying that, "The time has come to give up the illusion that most previous research dealing with the efficacy of psychotropic drugs has been adequately shielded against bias."²² This was in 1993, but the psychiatrists have chosen to totally ignore this fundamental problem in their research on drugs. Organised denial again.

Many years ago, trials were performed with tricyclic antidepressants that were adequately blinded, as the placebo contained atropine.²³ This substance causes dryness in the mouth and other side effects similar to those seen with antidepressants, and the trials are therefore much more reliable than those using conventional placebos. The mouth can become so dry on an antidepressant that one can hear the tongue scraping and clicking, which is an important reason that some patients lose their teeth because of caries. A Cochrane review of nine trials (751 patients) with atropine in the placebo, failed to demonstrate an effect of tricyclic antidepressants.²³ The measured effect, a standardised mean difference of 0.17, was not only statistically uncertain, but also so small that even if it were true, it would have no clinical relevance. It corresponds to 1.3 on the Hamilton scale, which ranges from 0 to 52, and the smallest effect that can be perceived on this scale is 5-6.²⁴ (In the clinical study reports we obtained from the European Medicines Agency, the median standard deviation on the Hamilton scale after treatment was 7.5; thus, a standardised mean difference of 0.17 corresponds to $0.17 \times 7.5 = 1.3$.) The minimal *clinically relevant* effect is of course larger than

the bare minimum that can be perceived. That you can see light at the end of the tunnel doesn't necessarily mean that there is enough light to read a newspaper.

Given the poor blinding and the subjective scales used to assess the outcome, it is not surprising that everything that numbs people or makes them euphoric seems to “work” for depression, including antipsychotics, anti-epileptic drugs and stimulants. Three of the 17 items on the Hamilton scale, for example, are about insomnia and this problem alone can yield six points on the scale.²⁵ And if a person goes from maximum anxiety to no anxiety, eight points can be earned. Thus, alcohol would clearly “work” for depression, but we don't prescribe alcohol for people.

Other meta-analyses, which, like the Cochrane review, were not financially supported by the drug industry, have also shown disappointing results. Virtually all psychiatrists say that antidepressants work for severe and moderate depression, but that isn't correct. According to the American Psychiatric Association, moderate depression has a Hamilton score between 14 and 18, severe depression is between 19 and 22, and very severe depression is above 22.²⁶ A meta-analysis with individual patient-level data from six trials (718 patients) found that selective serotonin reuptake inhibitors (SSRIs) were ineffective for both mild, moderate and severe depression, and even for patients with very severe depression, the effect corresponded to only 3.5 on the Hamilton scale,²⁶ which is well below what is a minimal clinically relevant effect. Furthermore, these trials were not adequately blinded, as they had used conventional placebos. If we adjust for this bias, the small effect for very severe depression disappears.

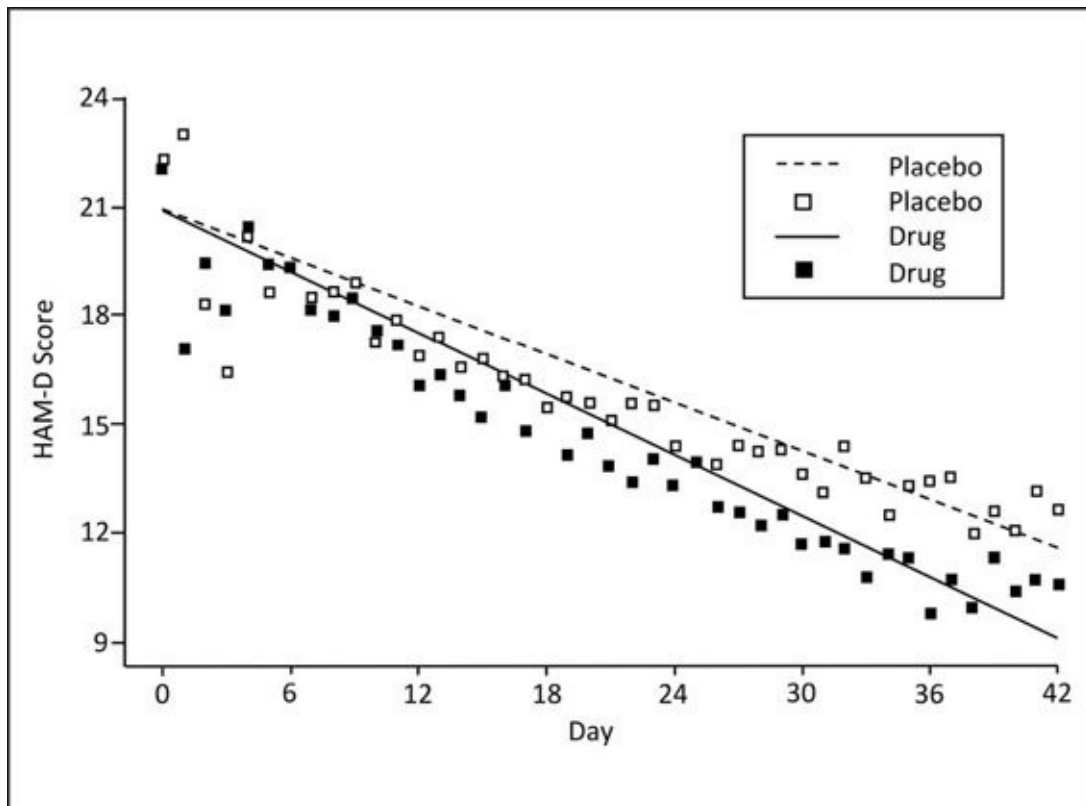
Even if we assume that antidepressants *might* have a trivial effect on those who are very severely depressed,^{26, 27} this is a small fraction of the patients that are treated. A study found that only 8% of depressed patients treated in routine clinical practice could be included in a standard efficacy trial.²⁸ As half the patients in depression trials have very severe depression,^{26, 29} and as many of the trials have been performed in hospitals, it cannot be correct when some psychiatrists claim that antidepressants are highly effective for the melancholic depression they treat in their hospitals.

A highly revealing way of judging the efficacy issue is to see how much faster people improve on an active drug than on a placebo, and a meta-analysis of 37 industry-sponsored trials of fluoxetine and venlafaxine showed just that.³⁰ The patients were severely depressed to begin with and after four weeks on the drug, they were only mildly depressed (Figure 3.1). It took eight more days before patients on the placebo had improved to the same extent. However, as these trials

were not adequately blinded, the true difference is likely to be a few days,²³ since a Hamilton score difference of 1.3 amounts to less than a week on the figure.

Thus, there is no good reason to use antidepressant drugs or to claim that they work like insulin for diabetes. The effect of insulin is instantaneous; in contrast, what we see in the figure is the slow, spontaneous remission in both groups, plus some bias in the active group.

Figure 3.1. Depression severity over time in 37 trials of fluoxetine or venlafaxine versus placebo. HAM-D is the Hamilton Depression Scale. Redrawn.



These sobering facts are in stark contrast to a 2013 statement from the president of the American Psychiatric Association, Jeffrey Lieberman, who claimed that antidepressants are highly effective and alleviate the symptoms substantially, if not completely, in 50-80% of the patients who suffer from major depression.³¹ In 2014, the medical director at the Norwegian drug agency, Steinar Madsen, said at a meeting that antidepressants work for 50-60% of the patients. I replied that his statement illustrated why we cannot trust our drug regulators and reminded him that the FDA had found in their analysis of 100,000 patients that antidepressants worked for only 10% of the patients.

These monstrous exaggerations are everywhere. A Cochrane review of electroconvulsive therapy claimed precisely the same, that antidepressants work for 50-60% of the patients.³² I wonder who invented these numbers and why people are so eager to ruin their credibility by citing them?

Reboxetine is approved in Europe but not in the United States, where Pfizer's application was rejected.³³ A German health technology assessment institute wanted to evaluate the drug³³ but, despite several requests, Pfizer steadfastly refused to provide the institute with a list of all published and unpublished trials.³⁴ The institute therefore concluded that it couldn't assess whether the drug was of any benefit,³⁵ but as this could lead to lack of reimbursement, Pfizer responded immediately, now with the ludicrous claim that they had provided the institute with sufficient data, which from Pfizer's point of view were "suited for a benefit assessment." Pfizer ultimately had to hand over all the trials, however, and the analysis showed that reboxetine produced no significant effect. The European Medicines Agency (EMA) did nothing,³⁶ apart from criticising the institute for having left out a statistical outlier from its analysis, but that criticism was inappropriate. The odds ratio for response in the seven included trials was 1.24 (95% confidence interval (CI) 0.98 to 1.56) whereas it was 11.4 (95% CI 3.1 to 42.1) for the excluded trial. When two confidence intervals are so far apart, we suspect fraud, and even one of the included trials was suspiciously positive.

Patients are the best judges for deciding whether a subjective effect of a drug outweighs its side effects, but the trials showed no such benefit. Just as many patients stopped treatment prematurely when they were on antidepressants as when they were on placebo.³⁷ This shows that the patients find the drugs useless, and it is likely even worse than this. Most patients on active drug will experience side effects, and although they might have preferred to quit, they could have decided to go on till the end of the trial hoping that a beneficial effect would eventually show up. Conversely, patients who have guessed they are on placebo might be more keen to stop, as it isn't meaningful to take placebo and waste time on control visits. Therefore, the fact that the patients in these poorly blinded trials stop their trial drug just as often when it's active as when it's placebo could mean that in reality they feel the drug is worse than the disease. I find this likely.

US court cases that have temporarily opened the companies' archives, and also our own archive of trials submitted to the EMA, have revealed that the industry has measured the patients' quality of life in many trials but has failed to publish the results.¹¹ It doesn't take a genius to work out that if the results had been positive the world would have heard about them.

An analysis of a prescription database showed that after only two months half the patients had stopped taking the drug.³⁸ Nonetheless, the psychiatrists love the pills and often say they work in 70-80% of patients,³⁹ which is mathematically impossible when only 50% continue taking the drug after two months.

There are many reasons why the effect of antidepressants has been overrated. It provides prestige and cool cash to claim large effects. The psychiatrists know that if they report major benefits and few harms in the trials, the drug companies will ask them again, which will enhance their academic standing and income. This distortion is sometimes done deliberately. It is very telling that psychiatrists found a positive effect of SSRIs in eight depression trials, including 1,756 children and adolescents (effect size 0.25), whereas no effect was reported by patients (effect size 0.05).⁴⁰ The Cochrane review of newer antidepressants in children and adolescents found exactly the same (effect sizes of 0.29 and 0.06, respectively, calculated by me based on standard deviations from other reviews).⁴¹

Trials in adults show the same. The clinicians' assessments in trials of old drugs like amitriptyline showed an effect size 0.25 whereas the patients' effect size was 0.06.⁴² This could be because clinicians know the side effects better than patients and therefore are better at unblinding the trials, but it could also simply be a matter of academic corruption.

Other important flaws in placebo controlled trials

Virtually all trials of psychotropic agents are flawed, not only because of the unblinding problem, but also because of their design. This was recognised decades ago, but drug trials and reviews have continued to pretend the problem doesn't exist,⁴³ likely because the psychiatrists choose to ignore everything that threatens the myths they have built up around their profession.

The design problem is that people are being randomised who have already been treated with the same type of drugs, which means that harms are inflicted on the placebo group because of withdrawal effects. Three-quarters of the depression trials have an initial placebo lead-in period of 1-2 weeks where patients can be excluded before randomisation,²⁹ but this time is too short because some withdrawal symptoms come much later or last much longer.^{44, 45} In nine trials of depression, patients were abruptly switched to an inactive placebo for 1-2 weeks after they had been treated for 8-9 weeks with duloxetine, which has a half-life of only 12 hours.⁴⁵ About half the patients experienced withdrawal effects, and about

half had not yet resolved after the 1-2 weeks of observation. Eli Lilly didn't report what happened to these patients after the formal observation period, but in the first quarter of 2012 more reports were submitted to the FDA on serious drug withdrawal effects for duloxetine than for any other regularly monitored drug, including two opioids.⁴⁴

There are many tricks. Some studies exclude patients who improve in the placebo lead-in period; some studies use active drugs in this period and exclude people who experience troublesome side effects; and some studies have both types of "cleansing," which was the case for one of the very few trials of fluoxetine in children that purported to have shown an effect.⁴⁶ But even with this extremely biased design, the children fared no better on the drug than on placebo on self-rating scales or on ratings by their parents. The "effect" was only seen on a secondary scale filled out by the psychiatrists who were paid by Lilly to run the trial!⁴⁶

What is particularly interesting is that SSRIs don't work for children and adolescents.⁴⁷ This might be because many of these patients were not exposed to an antidepressant before they were recruited to the trials, which are therefore less biased than trials in adults.⁴⁸

Why do doctors treat depression with drugs? Not to improve the score on some highly bias-prone subjective scale. What we want to achieve more than anything else is to reduce the patients' risk of committing suicide, and I shall discuss this issue below. It is also important to help people cope with their lives, e.g. getting them back to work and saving their marriages and other social relations, but although there are thousands of trials of antidepressants we don't know whether drugs are helpful for this. This means that either it hasn't been studied, or the results have been so disappointing that the industry has buried them. Given the drugs' lack of efficacy on depression and their pronounced side effects, especially their damaging effect on people's sex lives (see below), it seems unlikely that they have such effects.

Fluoxetine, a terrible drug, and bribery in Sweden

The SSRIs are pretty terrible drugs and patients aren't too happy taking them. But doctors choose to ignore how bad these pills are. When the first best-selling SSRI, fluoxetine, appeared in the late 1980s, there was no doubt about it. Senior management in Eli Lilly wanted to shelve it after having considered to market it

for eating disorders,¹¹ but Lilly was in serious financial trouble and had to make it a success.³

Initially, the FDA was sceptical and noted serious flaws in Lilly's trials. An FDA officer wrote in 1984 that patients who didn't do well after two weeks had their code broken, and if they were on placebo, they were switched to fluoxetine.³⁹ In this way, six weeks of fluoxetine was compared to two weeks on placebo, which is a hopelessly flawed comparison and, with the blinding broken, more bias was introduced. It also turned out that 25% of the patients had taken an additional drug, and when the FDA in 1985 removed patients on other drugs from Lilly's trials, there was no significant effect of fluoxetine. By adding benzodiazepines, Lilly broke the rules for its trials but didn't inform the FDA, and when the FDA later learned about it, the agency permitted it and thereby broke its own rules.⁴⁹ The public and the doctors were never informed about this ruse.

The FDA went to extremes to make it look like fluoxetine worked.³⁹ Perhaps the fact that Lilly is an American company played a role. Fluoxetine was approved when Bush senior was president and he had been a member of the board of directors of Lilly. Vice President Dan Quayle was from Indiana where Lilly's headquarters are, and he had former Lilly personnel on his own staff and sat on an FDA oversight committee.⁴⁹

The German drug regulator found fluoxetine "totally unsuitable for the treatment of depression," and furthermore noted that according to the patients' self-ratings there was little or no response, in contrast to doctors' ratings.⁴⁶

Despite the formidable odds, Lilly turned this awful drug into a blockbuster, which contributed to making the company one of the world's ten biggest. It's pretty clear to me that without the help of corrupt psychiatrists, big pharma wouldn't have sold many of its psychotropic drugs.³ Corruption is widespread in American healthcare, but even in Sweden there was corruption. When the director of Lilly in Sweden, John Virapen, showed some of the data on the drug to Swedish psychiatrists, they laughed and didn't think he was serious about seeking approval for it.⁵⁰ But that didn't last long.

Virapen, who had good reasons to feel his future career at Lilly depended on approval of fluoxetine, solved his problem with bribery. He found out who the independent expert was who would examine the clinical documentation for the Swedish drug agency. This expert was Anders Forsman, forensic psychiatrist and member of the legal council in the Swedish National Board of Health.⁵¹ Forsman didn't like fluoxetine at all and had laughed about it just two weeks earlier, but when Virapen asked him what was required to get the drug approved quickly, Forsman suggested \$20,000, which shouldn't become known to the taxman, plus a

good deal of research money for his department. Half the money to be paid at once and the other half when the mission was accomplished, just like when the mob orders a murder.

After the deal, deaths disappeared in footnotes and, according to Virapen, it went something like this:⁵⁰ “Five had hallucinations and tried to commit suicide, which four of the test subjects succeeded in doing” was changed to: “Five of the other test subjects had miscellaneous effects.” On top of this, Forsman wrote his own personal letter of recommendation.

I have met with Virapen and his account of the events has been confirmed by official documents. For example, the chairman for the Institute against Bribery wrote to the Department of Justice that the agreement was that Forsman would get the money in return for a positive report about the drug, and that he cashed the money. Forsman didn't cover his tracks. While the work was ongoing, he wrote to Virapen that he hoped he would be able to function as a sort of lubricant for the processes, “You understand what I mean.”

When confronted with his misdeeds many years later, Forsman lied about them.⁵² He claimed he had informed the director of the Swedish drug agency verbally about his collaboration with Lilly before he got the official assignment and that he also mentioned his conflict of interest in his report and explained about his contacts with Lilly in detail, but that someone at the Board of Health had erased it. This seems pretty far-fetched and the director and another professor at the drug agency both declared that they would never have accepted an expert with such conflicts of interest.

The approval in Germany also followed what Virapen calls “unorthodox lobbying methods exercised on independent members of the regulatory authorities.”

After having been so helpful to Lilly, Virapen was fired. This is like a script from the mob. When a hitman has murdered a well-known citizen, it is safest to leave no witnesses and kill the assassin. The official explanation was that Lilly had certain ethical principles! Two other people who knew about the bribery were also fired.

Virapen tried to prosecute Forsman, but it wasn't possible because he wasn't an employee of the health authority. The Swedish anticorruption law was later amended, as a direct consequence of this affair.

Forsman's career didn't suffer. He came to work for the court, as a psychiatric assessor for Sweden, and probably felt at home there, as there is nowhere where people lie as much as in court.

There weren't many truly depressed people in the mid-1980s when the criteria

for the diagnosis were much more stringent and relevant than today. Fluoxetine was therefore marketed as a mood lifter. Not much difference to street pushers here, as fluoxetine can have cocaine- and amphetamine-like stimulant effects on some people.⁴⁹ The warnings on the labels for antidepressant drugs, such as anxiety, agitation, panic attacks, insomnia, irritability, hostility, aggressiveness, impulsivity, akathisia, hypomania, and mania leave no doubt that these drugs can induce medication madness just as street drugs can.⁴⁹

Harms of antidepressant drugs are denied or downplayed

It took some years after I started my research on antidepressant drugs before I fully realised how dangerous they are. This is because their most serious harms have been denied or downplayed by all the major players in the field who have a joint interest in obscuring them. The drug industry earns billions by selling lies about their pills; the drug regulators won't admit that they erred when they approved the drugs, so they are very slow in issuing warnings and don't withdraw the drugs from the market; and the leading psychiatrists earn millions by dutifully defending all the illusions the drug industry has created.

Fluoxetine quickly became America's most complained-about drug.⁴⁶ In the first nine years, the FDA received 39,000 adverse event reports, far more than for any other drug. There were hundreds of suicides, horrendous crimes, hostility, psychoses, confusion, abnormal thinking, convulsions, amnesia and sexual dysfunction. Already in 1991, only three years after its launch, fluoxetine led the harms list, and yet, at an FDA hearing the same year, after many witnesses had told stories about out-of-character suicides and homicides, the advisory committee members, many of whom had financial ties to the drug makers, unanimously rejected this proposal: "There is credible evidence to support a conclusion that antidepressant drugs cause the emergence and/or the intensification of suicidality and/or other violent behaviors."

There is also widespread professional denial among psychiatrists of the drug harms patients tell them about. This was displayed when I mentioned on TV news that antidepressants can change the personality. In a commentary to this, the president of the Danish Psychiatric Association wrote it was misleading to focus on a side effect that is so scary for patients and which was extremely rare.⁵³ It isn't. Six years earlier, Danish psychiatrists had conducted a study in which approximately 500 patients told them what they thought about their antidepressant treatment, and about half the patients agreed that the treatment could alter their

personality and that they had less control over their thoughts and feelings.⁵⁴ Four-fifths agreed that as long as they took antidepressants, they didn't really know if they were actually necessary.

The patients' replies correspond closely with what other researchers have found,⁵⁵ but the Danish psychiatrists refused flatly to believe what the patients had told them. They considered it wrong and called the patients ignorant. They also felt that the patients needed "psychoeducation." However, the relatives had the same opinion as the patients about antidepressants. Perhaps they should also be taught they were wrong?

A British study was similarly discouraging: no less than 78% of 2,003 lay people considered antidepressants addictive, but according to the psychiatrists who had conducted the study, the patients only needed to be told that addiction wasn't a problem!⁶

In a survey of 1,829 patients on antidepressants in New Zealand, 62% reported sexual difficulties, 60% felt emotionally numb, 52% felt not like themselves, 39% cared less about others, 47% had experienced agitation and 39% had experienced suicidality.⁵⁵ *None were told anything by their doctors about feeling less like themselves or about possible effects on relationships with other people (other than sexual dysfunction).*

It is very strange that these terrible drugs have become so popular but it's likely because both patients and doctors consider the natural remission of the depression a drug effect. For example, 83% of the patients believed that the drugs had reduced their depression.⁵⁵

An international survey of 3,516 patients from 14 patient advocacy groups showed that nearly half had stopped taking a psychotropic drug at some point in their life because of side effects, and the main problem was agitation in a quarter of the patients.⁵⁶

In the mythology of antidepressants, the combined effects of crime, corruption, fraud, bias, substandard research, irrational arguments and organised denial among psychiatrists have had particularly devastating consequences for patients in relation to suicide. It is a true horror story and the most tragic area of healthcare I have ever encountered. Our most vulnerable patients – the children – are driven into suicide with "happy pills" that should never have been prescribed to them, as they don't work for depression. And drug companies and doctors push children into suicide while claiming that their drugs *protect* them against suicide. Can anything be worse than this? And, as usual, the drug agencies have protected the drug companies.

The FDA protects Eli Lilly

In 1990, only two years after fluoxetine came on the market, Martin Teicher and colleagues described six patients who had become suicidal and reacted in bizarre ways on fluoxetine, which was something completely new to them. Teicher's observations were very convincing, but Lilly lied to Teicher telling him that no data existed confirming his observations of suicidality.⁴⁹ Internal Lilly documents revealed that the FDA worked with the company on the suicide issue, and at the subsequent 1991 FDA hearing, Lilly's scientist left out information that demonstrated that fluoxetine increases the risk of suicide.⁵⁷ Earlier, Lilly had submitted data to the German drug agency showing that suicide attempts almost doubled on fluoxetine compared to placebo. But at the hearings, the chair of the FDA committee, psychiatrist Daniel Casey, brutally interrupted Teicher so that he couldn't present his findings. He was allowed to present a few slides while Lilly staff presented many. A few years later, Teicher's wife was offered a top job at Lilly without having applied, which was hardly a coincidence. The standard procedure is to blacklist and haunt critical people and if that doesn't work, to buy them or their close relatives. Teicher's wife divorced him and went to work for Lilly.

In 1989, a man shot eight people dead, wounded another 12 and killed himself one month after he started fluoxetine.³ Lilly won a jury verdict and claimed it was "proven in a court of law ... that Prozac is safe and effective." However, the trial judge suspected a secret deal had been struck and pursued Lilly and the plaintiffs, eventually forcing Lilly to admit that it had made a secret settlement with the plaintiffs during the trial. Infuriated by Lilly's actions, the judge ordered the finding changed from a verdict in Lilly's favour to one of "dismissed as settled with prejudice," saying, "Lilly sought to buy not just the verdict but the court's judgment as well." Peter Breggin was an expert in support of the family, but his own attorney betrayed him and presented a weak case, as he knew about the secret deal.⁴⁹ After this deal, Breggin's attorney sent back the revealing documents to Lilly where they disappeared against the law impeding other lawsuits. Incriminating documents also disappeared at the FDA.

Lilly bought FDA panel members, too. An FDA advisory panel concluded in 1991 that fluoxetine was safe despite concerns raised by FDA's safety officer David Graham and others, which led critics to point out that several of the panellists had financial ties to Lilly.

Throughout the 1990s, while swearing publicly that fluoxetine didn't increase the risk of suicide or violence, Lilly quietly settled lawsuits out of court and kept the incriminating evidence hidden by obtaining court orders to seal the documents.

Lilly's internal papers indicate that the company had an explicit strategy to blame the disease and not the drug for violence and suicide, and Lilly excluded 76 of 97 cases of suicidality on fluoxetine in a post-marketing surveillance study it submitted to the FDA. In relation to lawsuits, David Healy found early drafts of fluoxetine's package insert that stated that psychosis might be precipitated in susceptible patients; the warning wasn't included in the package insert for the United States but the German drug agency required it.

By 1999, the FDA had received reports of over 2,000 suicides associated with fluoxetine, and a quarter of them specifically referred to agitation and akathisia. A severe form of agitation, akathisia, is extreme restlessness that some patients describe as wanting to jump out of their skin. These people behave in an agitated manner which they cannot control and can experience unbearable rage, delusions, and disassociation.⁴⁹ Akathisia was likely much underreported in the trials and misdiagnosed as nervousness, agitation or "agitated depression."³⁹

The FDA said it would not have allowed a company to put a warning about akathisia or suicide on the label; it would have considered it mislabelling! This was a red herring, however, as drug companies can change the labelling without prior approval by the FDA if they feel it is needed for safety reasons.⁵⁸ In a court case of sertraline, a judge called Pfizer's arguments "perverse" and another judge said that it would be "inconceivable to argue that an additional warning regarding suicidality would be false or misleading" and that the law "allows, even encourages, manufacturers to be proactive when learning of new safety information related to their drug."⁵⁹ The court case was about a 15-year old girl, Shyra Kallas, who was prescribed sertraline by her primary care doctor for warts – yes for warts – and shot and killed herself. Daniel Troy of the FDA filed an FDA brief that claimed that the agency's authority pre-empts state drug safety requirements, which Pfizer used to its advantage to fend off lawsuits involving suicides. Interestingly, Troy counted Pfizer as one of his clients in the year before he took public office.

FDA's expert on safety matters, David Graham, noted that fewer than 10-20% of fatal effects were reported for fluoxetine, but the data nevertheless showed relatively more suicidality among patients on fluoxetine than among those on tricyclic antidepressants or placebo.⁵⁹ However, the safety officers' admonitions were largely ignored by FDA top brass, and although the FDA had been aware of an apparent seven times greater rate of suicidal behaviour in children taking sertraline since 1996, the agency did nothing about it. At long last, in July 2005, the FDA issued an advisory to healthcare professionals stating that antidepressant drugs increase suicidal thinking or behaviour in about one out of 50 children.

Lilly concealed the increased risk of suicide and violence and kept suicides from public view.³ In 2004, a healthy 19-year-old student who had taken duloxetine in order to help pay her college tuition hanged herself in a laboratory run by Lilly.⁶⁰ It turned out that there had been 41 deaths and 13 suicides among people taking duloxetine but missing in FDA's files was any record of the college student and at least four other volunteers known to have committed suicide. Lilly admitted that it had never made public at least two of those deaths, and anonymous sources told a journalist that duloxetine caused suicidal tendencies in patients who took the drug for urinary incontinence and weren't depressed.

The FDA said that federal regulation prohibited it from releasing study data for a drug that fails to win FDA approval, and the FDA didn't approve duloxetine for incontinence. This is absurd, as duloxetine was approved for depression. The FDA's argument for secrecy is that, "failed efforts at drug development need protection lest entrepreneurs suffer a competitive disadvantage when other companies aren't forced to expend the same time and money exploring dead ends."⁶⁰ FDA's disregard for research ethics means that patients will die in drug trials because other companies won't know that a particular type of drug is lethal. The FDA says it's compelled to maintain secrecy by law, but there is no such law.⁶¹

It is also unethical to allow the same molecule to be sold under two different trade names (Yentreve and Cymbalta), as it leads to overdosing. As many doctors and patients wouldn't know it's the same drug, it might be prescribed twice, for two different illnesses.

Later, the FDA announced that 11 of 9,400 women on duloxetine in the stress urinary incontinence trials had a suicide attempt.⁶² These studies show that SSRIs increase the risk of suicide also in middle-aged people, as the suicide attempt rate was 2.6 times higher than for other women of similar age.⁶²

Drug regulators in Europe also protected Eli Lilly. When we contacted the UK drug regulator in 2011 to get data on fluoxetine, which the EMA didn't have, as the UK was the EU Reference Member State for fluoxetine, we were told the agency had destroyed the files! The UK agency destroys the files after 15 years, "unless there is a legal, regulatory, or business need to keep them, or unless they are considered to be of lasting historic interest."³ So does that mean there is no legal or historical interest for unpublished clinical study reports on a drug that is still on the market and kills many people?

Massive underreporting of suicides in the

randomised trials

There are many reasons why the randomised trials have seriously underestimated the risk of suicide and suicidal events from antidepressant agents. Above all, almost all placebo controlled trials have been conducted by drug companies, which have a profound interest in hiding that their drugs push people into suicide. This became particularly clear when, in 2004, the *BMJ* received a series of internal Lilly documents and studies on fluoxetine from an anonymous source, which had been available ten years earlier in a litigation case.⁶³

These documents revealed that Lilly had known since 1978 – ten years before fluoxetine came on the market – that fluoxetine can produce in some people a strange, agitated state of mind that can trigger in them an unstoppable urge to commit suicide or murder. In 1985, two years before fluoxetine was approved, the FDA's safety reviewer noted under the headline "Catastrophic and Serious Events" that some psychotic episodes had not been reported by Lilly but were detected by the FDA by examining case reports on microfiche. The reviewer noted that fluoxetine's profile of adverse effects resembled that of a stimulant drug.

The documents also showed that Lilly was keen to root out the word "suicide" altogether from its database and its headquarters suggested that, when doctors reported a suicide attempt on fluoxetine, Lilly should code it as an "overdose." This is highly misleading, as it is hardly possible to kill oneself by overdosing fluoxetine and as the suicides occur on normal doses. Lilly wasn't alone with this misconduct. Independent investigators that looked at UK regulator data found that several companies had coded suicide attempts as overdoses.⁶⁴

Lilly's instructions furthermore were that "suicidal ideation" should be coded as "depression." But two internal Lilly researchers in Germany were unhappy with these directions, and one of them wrote in 1990: "I do not think I could explain to the BGA [the German regulator], to a judge, to a reporter or even to my family why we would do this, especially on the sensitive issue of suicide and suicide ideation."⁶⁵ Already in 1985, an in-house analysis of placebo controlled trials found 12 suicide attempts on fluoxetine versus one each on placebo and a tricyclic antidepressant, but after the code was broken, Lilly's hired consultants threw out six of the attempts on fluoxetine!⁴⁹

One of the leaked documents noted that 38% of the fluoxetine-treated patients reported new activation in the clinical trials, compared to only 19% of placebo-treated patients. This means that fluoxetine causes activation in one of five patients treated with the drug (as the difference to placebo is about 20%). Akathisia is a severe form of agitation, so this was bad news for Lilly, as akathisia increases the risk of suicide. Early on, Lilly therefore recommended that

such patients should also take benzodiazepines, which reduce the symptoms. The FDA approved fluoxetine based on four pivotal studies, but three of them permitted the use of concurrent psychotropic medication, and one-quarter of the enrolled patients took benzodiazepines or chloral hydrate.⁶⁶ Other companies adopted the same strategy, and minor tranquillisers were permitted in 84% of placebo controlled trials of antidepressants.²⁹

This means not only that suicides and suicide attempts on antidepressants have been seriously underestimated in the trials; it also means that we don't know what the true effect on depression is, as benzodiazepines have an effect on depression.

Further obfuscating the suicide risk, at least three companies, Glaxo-SmithKline (GSK), Lilly and Pfizer, added cases of suicide or suicide attempts to the placebo arm of their trials, although they didn't occur while the patients were randomised to placebo. Some of these events occurred in the run-in period, before the patients had been randomised, other events happened in the active drug group after the randomised phase was over.⁶⁷ These fraudulent manoeuvres can be important for the companies in court cases. For example, 60-year old Donald Schell murdered his wife, daughter and granddaughter and committed suicide after two days on paroxetine, but in its defence, GSK said that its trials didn't show an increased risk of suicide on paroxetine.⁶⁸

The FDA continued to protect the companies.³ In 2003, FDA officials noticed a curious thing while examining the results of a trial with youngsters on paroxetine.⁶⁹ GSK had reported that substantially more kids had shown "emotional lability" on the drug than on a placebo, but what was that? When the FDA officials asked GSK, it turned out that almost all of these events were related to suicidality. So what did the FDA do about it? The FDA bosses suppressed this information!

When FDA's safety officer Andrew Mosholder, a child psychiatrist, concluded that SSRIs increase suicidality among teenagers, the FDA prevented him from presenting his findings at an advisory meeting and suppressed his report. When the report was leaked, the FDA's reaction was to do a criminal investigation into the leak. Drug companies react in the same way when an employee leaks information about drug harms that the company has buried in its archives.⁷⁰

David Healy noted in 2002 that, based on data he had obtained from the FDA, two suicides and three suicide attempts that were ascribed to the placebo group in a paroxetine trial had occurred in the run-in period, before the patients were

randomised.⁷¹ This wasn't denied by GSK, but the company stated that Healy's analysis – which was the correct one – was scientifically invalid and misleading. GSK also masqueraded as wounded patients when it pompously stated that, “Major depressive disorder is a potentially very serious illness associated with substantial morbidity, mortality, suicidal ideation, suicide attempts and completed suicide. Unwarranted conclusions about the use and risk of antidepressants, including paroxetine, do a disservice to patients and physicians.”⁷²

Psychiatrist Joseph Glenmullen studied these documents for the lawyers and said that it's virtually impossible that GSK simply misunderstood the data, and Martin Brecher, the FDA scientist who reviewed paroxetine's safety, said that this use of the run-in data was scientifically illegitimate. But GSK was right about one thing: “Unwarranted conclusions about the use and risk of antidepressants, including paroxetine, do a disservice to patients and physicians.” GSK has done exactly what they warn against. Even in 2011, GSK denied that paroxetine can cause people to commit suicide.⁶⁸

FDA's meta-analysis of suicides in trials with 100,000 patients is deeply flawed

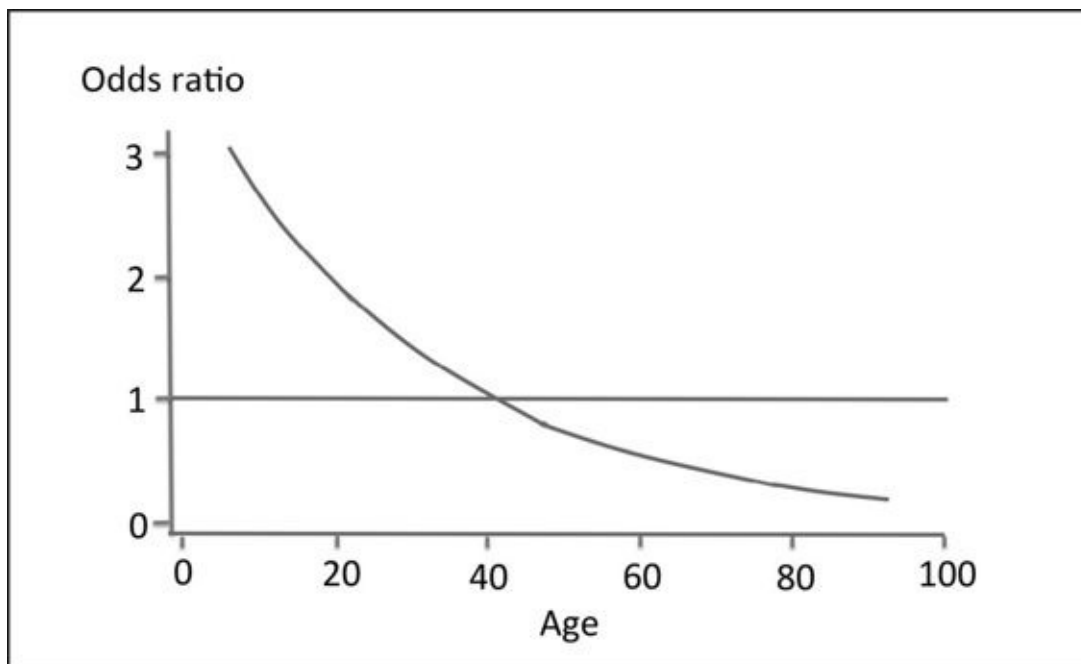
Though we cannot trust the randomised trials of antidepressants, they can still be of value, e.g. it would be highly convincing if they demonstrated an increased risk of suicide events despite all the bias and fraud.

The pressure on drug agencies to find out if SSRIs cause suicide began in 1990, but although it mounted during the following years, very little happened. It took 16 years before the FDA published a meta-analysis addressing this issue. The FDA's analysis included 372 placebo controlled trials of SSRIs and similar drugs involving 100,000 patients.¹⁷ It showed that up to 40 years of age, the drugs increased suicidal behaviour, and in older patients they decreased it (see Figure 3.2.).

However, it is far worse. Although the FDA knew that the companies had cheated on them earlier in relation to suicidal events, the agency asked them to adjudicate possibly suicide-related adverse events in their trials and send them to the FDA. This is weird. Why would the companies not continue cheating when they knew the FDA wouldn't check their work? If they didn't cheat this time, it would be too obvious how much they had cheated earlier. And why didn't the FDA behave as we do in other walks of life? The police wouldn't ask a criminal to go back to the scene of the crime and look for evidence while being assured that the judge would believe him. The FDA's official excuse was that the companies

knew best and that the large number of participants was also a reason to let the sponsors adjudicate the events.⁷³ It was very convenient for the FDA to accept that the evidence it would get would very likely be flawed, as it would lessen the risk of accusations that the agency had failed earlier.

Figure 3.2. FDA's meta-analysis of 372 placebo controlled trials of SSRIs and similar drugs involving 100,000 patients. Odds ratios for suicidal behaviour for active drug versus placebo by age. Redrawn.



Another major problem with the FDA's approach was that, although it was widely known at the time that any dose change increases the risk of suicide,⁷⁴ suicidal events that occurred later than one day after stopping randomised treatment were not registered. As stopping an SSRI may increase the risk of suicide for weeks, this approach seriously underestimates the suicidal effects of SSRIs.

The actual data demonstrate that the FDA's analysis grossly underestimates suicide risk. In trials on certain drugs included in the FDA's analysis, there were sometimes *more* suicides than in the *whole* FDA analysis of *all* the drugs. There were only five suicides in FDA's analysis of 52,960 patients on SSRIs (one per 10,000 patients), but an internal Lilly memo from 1990 described nine suicides in 6,993 patients on fluoxetine in the trials (13 per 10,000).⁷⁵ In a 1995 meta-analysis, there were five suicides on paroxetine in only 2,963 patients (17 per

10,000; this meta-analysis wrongly reported two suicides on placebo, which had occurred in the washout period).⁷⁶ Adding the suicides on both drugs, we get 14 suicides in only 9,956 patients. Other data are equally disturbing. In 1984, the German drug agency described two suicides among only 1,427 patients on fluoxetine in the trials (14 per 10,000).⁷⁷

An analysis of prescriptions written by general practitioners in the UK immediately after the drugs came on market is similarly revealing.⁷⁸ The patients had an average age of 50 and 79% had depression, but although the follow-up was only about six months, there were 90 suicides among patients receiving fluoxetine, sertraline or paroxetine, which was 24, 17 and 27 suicides per 10,000, respectively.

The data I found are remarkably consistent and show, based on the randomised trials alone, that:

There are likely to have been 15 times more suicides on antidepressant drugs than reported by the FDA in its meta-analysis of 100,000 patients in 2006, which is an error of 1,400%!

What I get out of this colossal underreporting of suicides is that SSRIs likely increase suicides in all ages. One would have thought that suicide is a hard endpoint but in drug trials, it is highly subjective how many suicides there are. Thomas Laughren was responsible for the FDA's meta-analysis of 100,000 patients from 2006, and he published a paper using FDA data in 2001 where he reported 22 suicides in 22,062 patients randomised to antidepressants, which is 10 per 10,000,⁷⁹ or 10 times as many as he reported five years later.¹⁷ There were only two suicides in 8,692 patients on placebo, which Laughren interprets thus: "There is obviously no suggestion of an excess suicide risk in placebo-treated patients." No, there surely isn't, but why didn't Laughren comment on the fact that hits us in the face, namely that there were four times as many suicides on antidepressants as on placebo, which was statistically significant ($P = 0.03$, my calculation)?⁷⁹

In its large analysis, the FDA found that paroxetine increased suicide attempts significantly in adults with psychiatric disorders, odds ratio 2.76 (95% CI 1.16 to 6.60).¹⁷ GSK limited its analysis to adults with depression, but it also found that paroxetine increases suicide attempts, odds ratio 6.7 (95% CI 1.1 to 149.4).⁸⁰ GSK USA sent a "Dear Doctor" letter that pointed out that:⁸¹

The risk of suicidal behaviour was increased also above age 24.

In contrast, the FDA has claimed that it is only in those below 24 years of age that these drugs are risky.⁷³

In GSK’s analysis, there were 11 suicide attempts on paroxetine (3,455 patients) and only one on placebo (1,978 patients). Five of the 11 suicide attempts were in patients aged 25 to 30 years, which illustrates how dangerous it is to operate with age limits when we are dealing with suicides on antidepressants. I really wonder why GSK reported no suicides, as there were five suicides in the 1995 meta-analysis of paroxetine in fewer patients, as just mentioned. But there is so much that doesn’t add up in this “creative” area where 2 + 2 is not necessarily 4.

Table 3.1. Sertraline trials in adults; n: number of suicides and suicide attempts; N: number of patients; follow-up: time after the randomised phase ended; RR: relative risk; CI: confidence interval.

	Follow-up	sertraline		placebo		RR	(95% CI)
		n	N	n	N		
FDA 2006 ¹⁷	24 hours	7	6,950	7	6,047	0.87	(0.31 to 2.48)
Pfizer 2009 ⁸³	24 hours	5	6,561	8	5,480	0.52	(0.17 to 1.59)
Pfizer 2009 ⁸³	30 days	25	10,917	14	9,006	1.47	(0.77 to 2.83)
Gunnell 2005 ⁸⁴	> 24 hours	24	7,169	8	5,108	2.14	(0.96 to 4.75)

A 2005 meta-analysis conducted by independent researchers of the published trials included 87,650 patients and all ages and they found double the suicide attempts on drug than on placebo (odds ratio 2.28, 95% CI 1.14 to 4.55).⁸² They also found out that many suicide attempts must have been missing; some of the investigators responded that there were suicide attempts they had not reported in their trials, while others replied that they didn’t even look for them. Further, events occurring shortly after active treatment was stopped were not counted.

The bias this omission causes has been demonstrated in meta-analyses of sertraline used in adults (Table 3.1). The FDA’s meta-analysis didn’t find an increase in suicide, suicide attempt or self-harm combined, relative risk = 0.87 (FDA’s Table 30), whereas Pfizer’s own meta-analysis suggested a halving of this, relative risk = 0.52, when all events that occurred after 24 hours were omitted.⁸³ When Pfizer included events occurring up to 30 days after the randomised phase was over, there was an increase in these suicidality events of about 50% (relative risk = 1.47). A 2005 meta-analysis conducted by independent

researchers using UK drug regulator data found a doubling in suicide or self-harm when events after 24 hours were included (relative risk = 2.14, 95% CI 0.96 to 4.75, my calculation).⁸⁴ These researchers noted that the companies had underreported the suicide risk in their trials, and they also found that non-fatal selfharm and suicidality were seriously underreported compared to the reported suicides.

In a meta-analysis conducted by GSK,⁸⁵ suicide-related events occurred more often on paroxetine than on placebo in children and adolescents (odds ratio 3.86, 95% CI 1.45 to 10.26), whereas suicide items on rating scales like Hamilton's didn't show this difference. A meta-analysis carried out by the FDA in children and adolescents found the same; suicide items on depression scales misleadingly showed a slightly *less* risk of suicidality with SSRIs (relative risk 0.92, 95% CI 0.76 to 1.11) whereas company data showed a doubling of this risk (relative risk 1.95, 95% CI 1.28 to 2.98).⁸⁶

In absolute terms, two out of 100 children experience suicidality because of the drugs they take.

Robert Gibbons and colleagues used individual patient data obtained from Eli Lilly and they claimed that fluoxetine didn't increase suicide risk in children and adolescents with major depressive disorder,⁸⁷ but this only illustrates that it is misleading to use too small samples. The FDA researchers found that fluoxetine increased the risk of suicide, suicide attempt or preparation for suicide in people under 25, relative risk 2.32 (95% CI 0.78 to 6.87) and when all drugs were included, this increased risk was the same but it was now statistically significant, relative risk 2.35 (95% CI 1.35 to 4.09).¹⁷ Gibbons declared in his paper that he had served as an expert witness for Wyeth and Pfizer in cases related to antidepressants and suicide. I am sure Gibbons improved his chances of also becoming an expert witness for Lilly and I shall say more about this remarkable man later.

It is clear that the risk of suicide caused by SSRIs has been grossly underestimated in the trials, and there are many reasons for this:

- 1) Fraud.
- 2) The investigators didn't report suicide attempts or didn't even look for them.
- 3) Many suicidal events have been coded as something else.
- 4) By only recruiting people at very low risk of committing suicide for their trials, the drug industry has taken great care not to get in trouble.

- 5) The companies have urged investigators to use benzodiazepines in addition to the trial drugs, which have prevented some of the violent reactions that would otherwise have occurred.
- 6) Some trials have run-in periods on active drug, and patients who don't tolerate it aren't randomised.
- 7) Most patients were in antidepressant treatment before they were randomised, which leads to withdrawal symptoms in the placebo group that predispose to suicide.
- 8) Events occurring shortly after active treatment is stopped were rarely registered.
- 9) Many trials are buried in company archives and these are not the most positive ones.
- 10) Patients are carefully monitored in trials and drug intake is likely to be stopped before a serious problem develops. In clinical practice, patients may not be monitored at all, and may miss doses, which increases the risk of suicide because of withdrawal effects. A study of prescription refill data indicated that 30% of patients on SSRIs may miss 4-15 days of therapy between prescription refills.⁸⁸
- 11) Patients in trials have contact with other people and get hope, therefore the risk of suicide is less than in real life.

The FDA failed us again. Despite the vast number of missing suicide events, its meta-analysis showed an increase in suicidal behaviour up to age 40, but when the FDA published its results in 2009, it had used 10-year age groups and now claimed that the risk was only increased in people under age 25.⁷³ In contrast, the 2005 meta-analysis conducted by independent researchers mentioned above⁸² found that the risk of a suicide attempt in those below 60 years of age was 2.4 times larger in the SSRI group than in the placebo group, and when they took length of treatment into account, they found that for every 1,000 patients treated for one year, there were 5.6 additional suicide attempts on active drug compared to placebo.⁸² Thus, by treating only 180 patients for a year with an SSRI, one additional patient will attempt suicide.

My deep mistrust of the FDA is shared by FDA's own scientists and is based on solid evidence of FDA's inappropriate actions in other matters of great importance for public health.³ Thomas Laughren, who was in charge of FDA's misleading meta-analysis of 100,00 people, established the Laughren Psychopharm Consulting in 2013 with himself as director.⁸⁹ He says:

“I have 29 years of experience at the FDA protecting the public health by assuring the safety and efficacy of psychiatric drugs. I accomplished this goal in part by working with pharmaceutical companies to help shape their development programs in order to meet FDA’s goals for assuring the development of safe and effective psychiatric drugs. I also worked to advance public health by helping to speed innovations to make psychiatric drugs safer and more effective.”

Laughren furthermore says his goal is to help pharmaceutical companies so that they can “meet the high standards of FDA and other regulatory agencies.” He certainly knows how to speak like a drug company.

The pervasive scientific misconduct has led to a research literature where one has to dig deeply to find the few gems among all the garbage. A 2004 UK systematic review of trials in childhood depression showed that, when unpublished trials were included, a favourable harm-benefit profile changed to an unfavourable one for several SSRIs.⁹⁰ None of the companies (Eli Lilly, GSK, Pfizer, Lundbeck and Wyeth) was forthcoming when asked for unpublished data, so the authors got them from a drug agency. Unfortunately, this review was highly misleading for fluoxetine, which was praised as being the only drug that had a “favourable risk-benefit profile.” It hasn’t. We are supposed to believe that *fewer* serious adverse events occurred on fluoxetine than on placebo (less than 1% versus 3.6%)! And also that the rate of discontinuation because of adverse events was similar (5.7% versus 6.3%). Given what we know about SSRIs, this is impossible and only tells us how deeply flawed the fluoxetine trials are. Another major blunder in the review is that the authors only included suicidal behaviour if it was a serious adverse event (causing death, permanent damage, or hospitalisation).

The Dutch drug agency had a more sober view on these trials.⁹¹ They defined suicidality as suicide, suicide attempt or suicide thoughts and found that there was a signal for all the drugs, with no difference between them. They also pointed out that the UK review had excluded a large trial of fluoxetine that found that this drug also increases suicidality. The Dutch were strangely secretive; they didn’t name the drugs but it is clear from their table that fluoxetine was one of them, and which one it was.

Even the EMA finally woke up. It announced in 2006 that parents and doctors should carefully monitor children and youth being treated with fluoxetine and watch out for suicidal tendencies.³ A fake fix. It’s impossible for parents to monitor their children closely. Children leave home virtually every day, so what would a monitoring schedule look like? And children have hanged themselves next door while their parents watched TV, shortly after they appeared totally

normal and said everything was okay. I once discussed this with a child psychiatrist on TV and told her it didn't make sense to me that psychiatrists treated depressed children with a drug that doubled their risk of suicide. Her reply was that the children should be monitored closely, particularly in the beginning. What then about children who forget to take a couple of pills or who stop taking their drug because of its side effects? It just cannot be done. Suicides on SSRIs can occur totally unexpectedly.

Numerous studies have shown that SSRI can be deadly at any age. Many people, also in the later decades of life, have changed their personality completely upon taking an SSRI and have killed themselves, others, or both. This raises an interesting hypothesis that the authors of the FDA meta-analysis also mentioned.⁷³ Antidepressant drugs could have two separate effects: an undesirable effect in some patients that promotes suicidal behaviour (and violence towards others), and a therapeutic effect in some that decreases suicidal behaviour. Clinical and animal studies provide support for this hypothesis. A review of 84 animal studies showed that reduced aggression upon treatment with SSRI was most common, but sometimes the animals became more aggressive.⁹²

In 1994, a trial showed that 18 of 54 patients made a suicide attempt on fluoxetine and 18 of 53 made one on placebo.⁹³ The included patients did not have major depression but had nonetheless made at least two previous suicide attempts. We are not told whether some patients already were on an antidepressant drug and therefore went cold turkey when randomised to placebo, or what age the patients had, but they were likely adults, as researchers almost always say if their patients were children. Thus, the trial showed that fluoxetine doesn't protect adults against suicide attempts.

Fluoxetine should never have been approved for children, or indeed for any creature. It is FDA-approved for "separation anxiety" in dogs, which is when dogs howl too much when their owner leaves home. Perhaps this is not such a fantastic idea. Eli Lilly showed in 1978 that cats who had been friendly for years began to growl and hiss on fluoxetine and became distinctly unfriendly.⁹⁴ After cessation of fluoxetine, the cats returned to their usual friendly behaviour in a week or two.

These drugs quite seriously impair normal development. The package insert for fluoxetine mentions that after only 19 weeks of treatment, children had lost 1.1 cm in height and 1.1 kg in weight compared to children treated with placebo.

I assume the FDA had an off-day in 2007 – or all the bosses were away at the same conference – when it admitted that:

SSRIs can cause suicide at all ages.

The FDA humbly “proposed” to the drug makers that they update their black box warning.⁹⁵

“All patients being treated with antidepressants for any indication should be monitored appropriately and observed closely for clinical worsening, suicidality, and unusual changes in behavior, especially during the initial few months of a course of drug therapy, or at times of dose changes, either increases or decreases. The following symptoms, anxiety, agitation, panic attacks, insomnia, irritability, hostility, aggressiveness, impulsivity, akathisia (psychomotor restlessness), hypomania, and mania, have been reported in adult and pediatric patients being treated with antidepressants.”

The FDA also noted that, “Families and caregivers of patients should be advised to look for the emergence of such symptoms on a day-to-day basis, since changes may be abrupt.”

The FDA finally admitted that SSRIs can cause madness at all ages and that the drugs are very dangerous; otherwise daily monitoring wouldn't be needed. But since this is a fake fix, the FDA, instead of “proposing” label changes, should have taken the drugs off the market. Particularly since the problems are much worse in clinical practice than what is reported in industry sponsored trials. Peter Breggin reports that of 184 patients in hospital starting fluoxetine, sertraline or paroxetine, 11 developed mania and eight became psychotic, and in Yale, 8% of 533 consecutive admissions were for mania or psychosis caused by antidepressants, and two patients heard voices commanding them to kill themselves.⁴⁹ Breggin also notes that two of the three most common adverse reactions to fluoxetine were depression and abnormal thoughts, but the FDA scratched out depression from its prominent place in the label and it went from being frequent to being non-existent, and abnormal thoughts became abnormal dreams, which is not quite as bad.

Some of our drug regulators appear to be smarter than others, though. As far as I know, Australia is the only country that has refused to approve any SSRI for use in children.⁶ Some countries have approved fluoxetine based on deeply flawed trials, but it's very unlikely that any SSRI works for children, and in the large TAD trial there were six suicide attempts in the fluoxetine groups and one in the other groups.⁹⁶ Hardly a drug you would want to expose your child to.

It is totally reprehensible, outright dangerous and disrespectful towards all those who have lost a child or a spouse to an antidepressant drug

that leading psychiatric professors and the drug companies continue to deny⁹⁷⁻⁹⁹ – in direct contrast to the most reliable data we have – that antidepressants increase the risk of suicide among children and adolescents.

Another dirty trick: using patient-years instead of patients

In their submissions to drug agencies, several companies obscured the suicide risk by using patient-years as the denominator instead of the number of randomised patients. This introduced considerable bias because several of these trials had a follow-up phase where all patients could receive the active drug. As those who continue with the drug for a long time are those who tolerate it, patient-years are added “for free” to the drug group in terms of suicidality.

Based on FDA documents David Healy has obtained, it is possible to see how large this bias is. In Pfizer’s submission on sertraline, there was a 26% decrease in suicide attempts on active drug compared with placebo.¹⁰⁰ However, three of five suicide attempts ascribed to placebo happened in the wash-out phase before randomisation, and when the FDA excluded these, sertraline caused a 29% increase in suicide attempts. Using patients as the denominator, the increase in suicide attempts on sertraline was 73%. For paroxetine, suicide attempts decreased by 52% using patient-years, but increased by 25% using patients.¹⁰¹ David Graham reported in an internal FDA document that the suicidality rate on fluoxetine was 0.52% versus 0.18% on placebo, i.e. almost three times higher on drug.¹⁰²

These dirty tricks were not harmless, as it appears they contributed to convincing the FDA that it should not worry about a suicide risk with SSRIs.¹⁰³

Psychiatrists on company payrolls also use these dirty tricks. In 1995, Stuart Montgomery and colleagues examined paroxetine in a series of meta-analyses and came up with the astounding result that there were 5.6 times *fewer* suicides on paroxetine than on placebo!¹⁰⁴

Case stories of suicide on SSRIs

Thousands of people have committed suicide because of the antidepressants they took and many of them have been reported in scientific articles, in newspaper reports and on websites.

The tragic loss of a loved one can influence people's memories and could make them forget about any significant psychological problems preceding the suicide, which would move some of the self-blame from the family to the doctor. However, although such recall bias can occur, we also know that antidepressants can cause healthy volunteers to become suicidal. Furthermore, people close to the victim have in many cases noticed a fundamental change in personality, which is so bizarre that they don't have the slightest doubt that the pills caused it.

We owe it to those who died because of a greedy, criminal and fraudulent drug industry, assisted by overly industry-friendly drug regulators and corrupt psychiatrists, to tell some of their stories. A thousand deaths are a faceless statistic. But there are real people behind the numbers and when we describe what happened to real people, other people tend to take notice.

Here are eight of the tragic deaths I know about at a personal level because I have met with the parents or spouses left behind. They all wished me to tell their story to alert other people to the dangers of SSRIs and also wanted me to show photos of those they lost (see photos). I met four of them in Los Angeles at a scientific meeting on the theme that biological (drug-based) psychiatry does more harm than good (see photos) (see Chapter 14). Their determination in publicizing their own tragedies has spared others from experiencing the same, as they realised before it was too late that their loved one was heading for disaster.

Unfortunately, many doctors and other people believe that when our authorities have issued a warning against using a drug up to a certain age, it is safe to use the drug above that age. In Denmark, the package inserts warned against the increased risk of suicide up to age 18, although there never were any data to suggest that the risk wasn't increased in people slightly older than this. Such misleading recommendations have had tragic consequences.

Danilo Terrida

Danilo Terrida was only 20 when he hanged himself in a crane at a shipyard in 2011, two hours after having assured his family on the phone that he was fine and had small-talked with them. A month earlier, he had celebrated his birthday and everything apparently went well. Two weeks later, he made contact with the emergency medical service because *he did not feel well psychologically* and was sent home with tranquillising pills and was advised to see a doctor the next day. The local doctor he contacted refused to see him and asked him to call his family doctor several hundred kilometres away. This he did, and after eight minutes on the phone, he was prescribed sertraline, although he wasn't depressed, and with no follow-up. A few moments before he killed himself, Danilo talked to a friend

and said he didn't know what was happening to him, but he didn't mention anything about suicide. This is rather typical of an SSRI-induced suicide, and the timing is also typical. Many people kill themselves early on. The involved doctors have been officially criticised for amending Danilo's files, up to a year after his death, so that it looked more plausible that he killed himself because of a depression, which wasn't true (see www.daniloforlivet.dk).

At least 11 other people, 10 of them adults, who have committed suicide in Denmark on antidepressants have acquired economic compensation from the Patient Insurance Association.¹⁰⁵

Toran Bradshaw

Maria Bradshaw's son Toran who was her only child was prescribed fluoxetine in January 2007 despite having been assessed as being a healthy adolescent who had an expected reaction to a stressful life event, which was a *breakup with his girlfriend*. A month later, Toran experienced a severe cluster of adverse reactions including suicidal behaviour, self-harm, aggression, hostility, hallucinations, lack of concentration and impaired functioning. The symptoms were so severe that he dropped out of school. His psychiatrist's response was to increase his dose, which worsened the adverse reactions. Toran withdrew from fluoxetine and from mental health treatment of his own volition.

The following year, a psychiatric registrar prescribed fluoxetine to him again, against his mother's wishes. The registrar recorded no diagnosis after having conducted a mental state exam and finding no evidence of depression, anxiety or any other mental disorder. The next day, a multi-disciplinary team reviewed Toran's file and recorded "diagnosis deferred" noting that there was no evidence of a mental disorder. Toran initially refused the prescription on the basis that fluoxetine should not be taken with alcohol, but the registrar recorded that he had reached an agreement with Toran that he would stop taking the fluoxetine on a Friday, could drink up to six bottles of beer a night over the weekend and restart his fluoxetine on Mondays. Given fluoxetine's long half-life in the body, 2 to 4 days for the drug and 7 to 15 days for its active metabolite norfluoxetine, this was a foolish recommendation. Toran followed this regime but suffered a repeat of the former adverse reactions and suddenly hanged himself after 15 days on fluoxetine. He was only 17 years old.

After Toran's death, Maria had genetic testing conducted, which confirmed that Toran metabolised drugs slowly and had therefore been overdosed. Maria also established CASPER, an organisation where those bereaved by suicide help

others bereaved by suicide (<http://www.casper.org.nz/>). Maria has told me that the national media have credited CASPER with a 20% drop in youth suicide and that it means more to her than anything else that her son continues to do good in the world despite not being here physically.

Maria wrote to the District Health Boards in New Zealand and found out that 8% of those who died and who had gotten a recent prescription for an antidepressant, had no diagnosis of any mental disorder. *In the district responsible for Toran's care, 75% of those under 18 who died had no diagnosis.*

In New Zealand, psychiatrists and suicidologists have managed to convince the government that publishing information on suicides causes copycat suicide. She has reviewed the evidence for this, which is extremely weak, but it's nonetheless a criminal offense for Maria to tell Toran's story, punishable by a fine of up to \$5,000 each time and a fine for the media of up to \$20,000. These threats have not stopped Maria and she has had support from the media.

Maria sold her home to pay for Toran's inquest when the doctors dragged it out over 18 days of hearings, thinking she would walk away because of the cost. She now lives in Dublin, in the home of another mother who lost her child to SSRI-induced suicide, Leonie Fennell, and has everything she owns in two suitcases.

There have been ten government enquiries into Toran's death but, as in all such cases, the issue is not whether the child received a good standard of care but whether the psychiatrist departed from generally accepted practice. And of course, since the usual practice in psychiatry is terribly poor, most of the investigations found that Toran's care was not a departure from usual practice. However, both the government and Mylan Pharmaceuticals have resolved that it is probable that fluoxetine caused Toran's suicide.

Maria is still fighting to achieve justice and has persuaded the police to review Toran's file to see whether manslaughter charges can be laid. She hopes that her efforts will be a deterrent, which could make other doctors consider that their patients may have a stropmy mother like her and be more careful. Maria has spent seven years on meticulously documenting Toran's case learning everything she could about psychopharmacology, psychiatry, neurochemistry, genetics, randomised trials and anything else that could help not only her but also all the other parents that have stories similar to hers.

When I gave a lecture at the Department of Psychology at the Manooth University outside Dublin, to which Maria had invited me, another bereaved mother was in the audience, Stephanie McGill Lynch, who also lost her child to fluoxetine. Maria wrote to me:

"I know you agree with me that this has got to stop." Yes, and this is

why I have written this book. I won't accept that doctors and drug companies push our children into suicide with drugs that have no benefits for them. I cannot see it's any different to killing our children by pushing narcotic drugs in the street.

Jake McGill Lynch

Jake's mother, Stephanie, describes her son as a beautiful, bright 14-year old boy. In late 2011, he was diagnosed with Asperger's syndrome in an extremely mild form (the diagnosis of Asperger's was eliminated in 2013 in DSM-5 and replaced by a diagnosis of autism spectrum disorder).

Jake started counselling with a psychologist in 2011 due to some dark thoughts that had appeared in an essay he wrote in school. In January 2013, *he had counselling again, for anxiety*, and his psychologist decided to refer him to her colleague, a psychiatrist, as she felt his anxiety would be heightened when he was to sit his state exams. Jake's parents didn't even know what a psychiatrist was but just thought it was the psychologist's colleague.

Jake's dad took him to his appointment because no big deal was made of it, and they met with the psychiatrist for ten minutes, after which they left with a prescription for fluoxetine. Jake had never been on medication before, but the family was not given any literature or any description from the psychiatrist or the pharmacist, and they didn't even know what sort of drug Prozac was but simply trusted the psychiatrist.

Six days later, Jake had his first reaction. He walked out of an exam half-way through it and cried for about 2-3 hours that night, saying, "You don't know what it's like in my head." His parents thought this was from the stress of the exams. They never imagined that a drug could do this to a person.

About a week later, they got Jake back to the psychiatrist and told her all about what happened, but she said that it would wear off after three or four weeks and that Jake would be fine. But Jake was not fine, and on day 46 he was a bit restless after school and looked a bit flush in the face, although he never had a colour in his cheeks. His parents thought he had a row with his little online girlfriend.

The family had a legally held rifle in the house, as Jake and Stephanie were members of a shooting club. They would often take the gun down, and Jake asked if he could take it down that night, which was nothing out of the ordinary, so his request was granted. Stephanie forgot to take the box from his room with bolt and ammunition.

Jake placed the gun in his mouth and pulled the trigger. He had no history of suicide ideation or self-harm, and no diagnosis of anything but Asperger's.

However, the National Health Service in Ireland is now trying to say he had severe anxiety – although this isn't true – and it fights the parents from every corner with this. It is the same story all the time: put all the blame on the disease, never on the drug. The parents asked David Healy to do a second opinion based on Jake's medical files, which he did.

Stephanie and her husband have attended their son's inquest three times so far and are still in the middle of a legal argument about which medical expert the Coroner's court will allow them to consult. The court has refused their request to use David Healy, as he is considered to be not impartial due to his papers and books about the relation between SSRIs and suicide!

Stephanie finds the whole thing absolutely disgusting. This was a 14-year old child who had plans for the future. He had no illness, he had a condition that no medication would fix, and he was living quite happily with just counselling for his anxiety. Stephanie and her husband were never told about the dangers of drugs like fluoxetine. Had they known about them, they would never have kept a firearm in the house.

Shane Clancy

Shane was 22 years old when *he broke up with his girlfriend*.¹⁰⁶ He found it difficult without her and was prescribed citalopram. He became agitated and rang the doctor four days later, as his tongue felt very swollen (a recognized side effect of citalopram). He left a message but got no response, and five days later he took the remainder of the tablets in an attempted suicide.

His mother brought him back to the clinic where he saw a different doctor who continued the prescription for citalopram.

Ten days later, Shane called his ex-girlfriend, who was in her new boyfriend's house, and asked her to send her boyfriend outside, as Shane was hurt and needed help. Shane stabbed the boyfriend fatally and also stabbed his ex-girlfriend and her boyfriend's brother who emerged from the house to find out what was going on. Shane was later found dead in the garden having stabbed himself about 20 times.

The case was all over the media for several days and every scrap of evidence that pointed to pre-meditation was a nail in Shane's coffin. At first there was no mention of antidepressants, but Leonie Fennell, Shane's mother, feeling guilty for having engineered her son onto citalopram, raised the issue. The Irish College of Psychiatrists responded that there was absolutely no evidence that antidepressants could cause problems and to say so was dangerous and irresponsible.

Before the inquest, an extraordinary letter to the national newspapers was sent from all professors of psychiatry in Ireland (or so they said), stating that there was no evidence that antidepressants could cause harm.

The jurors were asked to recommend whether the verdict should be suicide or an open verdict. An open verdict means that there is no clear evidence that the act was planned, but could, for example, have happened under the influence of a substance like LSD. In European countries, an open verdict is not an initial step to suing a pharmaceutical company, as it is almost impossible to sue a pharmaceutical company in Europe.

Returning an open verdict in England or Ireland does not blame the drug. It simply means in this case that the jurors have decided that the killings and the events did not permit a straightforward answer. But even so, the pharmaceutical companies sent academics such as Professor Guy Goodwin from Oxford (see Chapter 1), along with several high-powered lawyers to ensure the coroner or the jury would return a suicide verdict. It didn't. David Healy provided expert testimony and the jury returned an open verdict.

Just like the American Psychiatric Association (APA) before and since, The Irish College of Psychiatrists went into overdrive issuing statements left, right, and centre that there was no evidence that citalopram could cause suicide or violence. This led a retired professor of psychiatry, Tom Fahy, send this letter to the Irish College:

“I am afraid the College is plain wrong. There is no such thing as a college statement which is circulated to the membership simultaneous with its publication, without opportunity for comment or vote and ‘in unison’ with a body 100% financed by drug companies, and with personal hostile references to expert testimony at an inquest with families still in grief. And this on the heels of a dreadful multiprofessorial letter even before the inquest began. Extraordinary and outside my experience. If I were not retired I’d dissociate and publicly resign.”

Stewart Dolin

Stewart Dolin had the perfect life.¹⁰⁷ He was married to his high school sweetheart for 36 years. He was the father of two grown children with whom he had a very close and meaningful relationship. He was a senior partner of a large international law firm, managing hundreds of corporate lawyers. He enjoyed his work and derived satisfaction from cultivating relationships with his clients, as well as helping them achieve the results they desired. He enjoyed travel, skiing, dining, joking around with his family and friends and an occasional cigar. He was 57 years old and high on life.

In the summer of 2010, Stewart *developed some anxiety regarding work*. He was prescribed paroxetine. Within days, Stewart’s anxiety became worse. He felt restless, had trouble sleeping and kept saying, “I still feel so anxious.”

Six days after beginning the medication, following a regular lunch with a business associate, Stewart left his office and walked to a nearby train platform. A registered nurse later reported seeing Stewart pacing back and forth and looking very agitated. As a train approached, Stewart took his own life. This happy, funny, loving, wealthy, dedicated husband and father who loved life left no note and no logical reason why he would suddenly want to end it all. The package insert for paroxetine did not list suicidal behaviour as a potential side effect for men of Stewart’s age.

Stewart’s wife did not know it then, but Stewart was suffering from akathisia. She started MISSD (The Medication-Induced Suicide Education Foundation in

Memory of Stewart Dolin), which is a non-profit organisation dedicated to honouring the memory of Stewart and other victims of akathisia by raising awareness and educating the public about the dangers of akathisia so that needless deaths are prevented.

Candace Downing

Candace was a happy 12-year-old girl who had never been depressed or had suicidal ideation.¹⁰⁸ She was prescribed sertraline because *she suffered from school anxiety*. Her mother Mathy found her beautiful little girl hanging, her knees drawn up. Her father knew the minute he saw her that it was too late but tried to administer CPR, which continued for another 45 minutes at the hospital but in vain.

“Do you know what that’s like, to see your happy little girl hanging? There was no note, no warning, not for her, not for us.”

When Candace entered middle school, she began having problems on tests and frustration over certain homework assignments. She would block on answers she knew on tests, or write so illegibly that some answers were marked incorrect, even if she had them correct. Because of her parents’ concern, she saw her paediatrician, who recommended that she see a child psychiatrist. He immediately wanted to give her sertraline. Mathy was opposed, but he reassured her that it was safe and that he would recheck her in three weeks. After three weeks, he wanted to double the dose, from 12.5 mg to 25 mg, which Mathy opposed. Because of her vehemence, the medication was not increased at that time.

Right before school started following summer vacation, Mathy and Candace returned to the child psychiatrist, who once again wanted to increase the dose. When Mathy voiced concern, he stated, “What are you worried about? Kids take 100-200 mg of Zoloft a day without any problems.”

“Why was so much hidden from us? Why were we not ever informed about the contraindications or adverse reactions of Zoloft, or for that matter, antidepressants in children? Didn’t we have the right to be informed? ... Shouldn’t it have been our choice to place Candace on medications that involved risk rather than the pharmaceutical companies or the FDA?”

Candace had many friends. Everybody loved her and when she died, more than a thousand people attended her service. Candace was everybody’s little girl, and if it could happen to her, it could happen to anyone’s child.

After Candace’s death, the Downings became aware that an abrupt withdrawal from an antidepressant can prove fatal, as it can create psychotic states, with

decreased powers of reasoning. No one ever told them that their daughter was going in and out of psychotic states and needed to be watched closely every second.

“If we had been able to make our own choices, if we had been aware of the risks, this would never have happened, as we would never have allowed Candace to be placed on such a risky and controversial medication,” said Andrew Downing.

“What happened to our daughter and so many others like her is a travesty. We have since met other families who have lost their child after Zoloft was prescribed for test anxiety. Those in a position to create positive change can go home to their children at night. We will never have that opportunity with Candace again. Our therapist referred to what happened to Candace as abduction. She was taken away from us with no warning and died in the process. What gave them that right?” Mathy stated tearfully.

The Downings, and other families, charge that drug makers knew from premarketing studies that these drugs made some children and teens suicidal but hid the study results.

“This is about the right of the American people to make their own decisions. I can’t sit back as an American citizen and watch children continue to die. And that is why we hope the documentary ‘Prescription: Suicide?’¹⁰⁸ will help to get that message out where it counts: among the American families whose biggest concern is to protect and nurture their children,” said Mathy.

The Downings have testified at FDA hearings and are lobbying Congress to make all research public. Mathy has also addressed the US Drug Safety Systems Committee, which is reviewing the numerous allegations against the FDA’s inadequate handling of policy regarding antidepressants.

The Downings’ psychiatrist had not told them that he was on Pfizer payroll making speeches touting Zoloft. Pfizer said in a statement for CBS News that “it’s paid consulting work with doctors helps the company learn how to reduce adverse reactions.”¹⁰⁹ No condolences were apparently offered.

People have called the Downings and said that because of Candace their child is alive, as they knew what to look for.

Cecily Bostock

Cecily Bostock was a musician, an artist, an over-achiever in almost everything she did.^{110, 111} *She was having a lot of trouble sleeping*, had racing thoughts, was over-analysing, and was overly sensitive. This prompted a prescription of

paroxetine.

Her mother Sara said that within three weeks of taking Seroxat, Cecily became a totally different person.

“The last two days she was just a complete zombie I have to say. She was just agitated, jumping at every noise and not making sense. I was very concerned. We were very close to Cecily. I just loved her deeply.”

Sara found her daughter lying on the kitchen floor. There was a large chef’s knife on the floor by her and just a trickle of blood from her chest. Cecily had stabbed herself twice through the heart. Her autopsy revealed she had a very high blood level of paroxetine, which reflects poor metabolism and is a feature common to many of these suicides.

Cecily killed herself about 20 days after she had started taking Seroxat. She was just 25 years old.

Since her daughter’s death, Sara has been campaigning and creating awareness about the dangers of antidepressants. She spoke at a charity conference in 2008 about her experience and knowledge from years of research, which is on video (<http://vimeo.com/16727219>). Sara has also given testimony at an FDA hearing and has co-founded www.ssristories.com, which is a collection of over 5,000 stories that have appeared in the media.

Woody Witczak

In 2003, Tim, known to most as Woody, died of a sertraline-induced suicide at age 37.¹¹² He was not depressed, nor did he have any history of mental illness. He died after taking the drug for five weeks with the dosage being doubled shortly before his death. *He was given the antidepressant by his general physician for “insomnia.”*

Woody loved life and all that this world has to offer. He had endless energy, a constant smile and truly cared for others. He had a successful career in sales and was active in the community, socially and politically, always willing to fight against injustice. Woody truly inspired others to be the best they could be.

Woody went to his regular internist because he was having trouble sleeping, in part because he had just started a new position as vice president of sales with a start-up company about two months earlier. He was excited about this dream opportunity to make his mark on the business world. Along with this excitement came some stress and difficulty sleeping.

This was the first time he’d ever gone to a doctor for this sort of issue. Woody’s doctor gave him three weeks’ worth of sertraline samples and told him to

come back for a follow-up appointment after the samples were finished. There was no discussion about the risks or the need to be closely monitored because of this mind-altering drug. The first three weeks Woody was taking sertraline his wife was out of the country on business and no one was monitoring him. Within a couple of days, he experienced many of the known side effect of sertraline, e.g. night sweats, diarrhoea, trembling hands, and worsened anxiety.

One of the most significant side effects Woody had was akathisia. He was very restless, which caused him not to sleep, and irritable and felt he always needed to keep moving.

Shortly before his death, Woody came home crying after driving around all day. He sat in a foetal position on the kitchen floor profusely sweating with his hands pressing around his head saying, “Help me. Help me. I don’t know what’s happening to me. I am losing my mind. It’s like my head is outside my body looking in.” The next day, Woody called his doctor to tell him what happened and was told to be patient because it could take four to six weeks before the drug worked.

Over the course of the next week, in typical Woody fashion, he was looking for ways to “beat this feeling in my head all while still running three to four miles a day. Two weeks later, when sertraline should have worked according to his doctor, Woody was found hanging from the rafters in the garage. Woody’s family and friends only wish they knew then what they know now. It wasn’t Woody’s head. It was the drug.

Never once did Kim, Woody’s wife, or Woody question the drug. Why would they? It was FDA approved, heavily promoted as safe and effective, and it was given by his doctor. People trust their doctors, who assume the FDA and the drug companies did their job to ensure that the drugs they prescribe are safe and effective.

The day Woody died, the front page of the local newspaper had an article that people in the UK had found a link between antidepressants and suicide in teens. Kim’s quest for the truth has led her to testify about the dangers of SSRIs at hearings in the US Senate, at the FDA, the Health Department, Congress and the courts. Together with other campaigners, she was active in getting black box warnings added to antidepressants.

Through my work, I have met with relatives of many more people killed by SSRIs than those described here. It makes me particularly angry that most of those who died or killed others shouldn’t have had a prescription for an SSRI. This is true for all of the eight victims I have mentioned, as the “indications” were:

anxiety regarding work or school work (3)
break up with girlfriend (2)
trouble sleeping (2)
didn't feel well psychologically (1).

Here are brief accounts of additional tragedies, which I have described before.³ Again, in none of the cases was there a reasonable indication for an antidepressant.

Matt Miller had moved to a new neighbourhood and a new school and was prescribed sertraline and told to call his doctor in a week. After taking his seventh pill, Matt went to his bedroom closet and hanged himself, having to lift his legs off the floor and hold himself there until he passed out. He was only 13 years old.

Jeremy Lown was a teenager with Tourette's syndrome. To treat his uncontrollable tics and verbal outbursts, his neurologist prescribed fluoxetine. Three weeks later, Jeremy hanged himself in the woods behind his house.

Vicky Hartman was given a sample pack of sertraline by her child's doctor. She didn't suffer from any mental disorder but mentioned she needed a "pick-me-up" to help with stress. Soon after starting the medication, she shot her husband and herself.

A man hanged himself after taking fluoxetine, which his cardiologist had prescribed for chest pain, and a woman shot herself after taking the fluoxetine her family doctor had prescribed for migraine.

Twenty-year-old student Justin Cheslek had trouble sleeping and was prescribed sleeping pills. When he complained to the doctor that the pills made him groggy and "depressed," the doctor gave him paroxetine, on which he developed akathisia. Two weeks later, the doctor gave him venlafaxine, and after three weeks on antidepressants, he hanged himself.

Brennan McCartney went to his family doctor with a chest cold and mentioned he felt sad over breaking up with a girl. He left with a script for an antibiotic and a sample pack of citalopram. On the fourth day, Brennan seemed agitated when he left the house and failed to come home. He hanged himself in a local park, only 18 years old.

Caitlin Hurcombe, a teenager, had visited relatives in the United States where she saw an ad for fluoxetine and wanted to try it. She went to her local doctor and it took her eight minutes to get the prescription. She descended into unprecedented chaos, including neural twitches, violent nightmares and self-harm, and hanged herself 63 days later. As this story shows, advertising prescription drugs to the public, which is legal only in the United States and New Zealand, can kill healthy

people who don't need them. Caitlin's story is particularly tragic, as the name Prozac was developed for Eli Lilly by a branding agency.¹¹³ Prozac suggested positive and professional and zappy, and conveyed the idea that taking antidepressants did not have to mean you were mentally ill. Instead, you could be young and troubled by the world, in need of an aspirin for an existential hangover. Prozac was eagerly grasped as the embodiment of a dream, the idea that an antidote to the pain of modern living could exist in a simple pill. This marketing killed Caitlin.

Akathisia is the main culprit

It is a longstanding belief that the “activating effect” antidepressants cause in some patients gives them the energy to follow through on suicidal impulses before the mood improvement takes effect. This myth should have been scrapped ages ago, but the drug industry has perpetuated it, as it has been useful for their storytelling. According to Lundbeck,¹¹⁴

“When the leaflet says that suicidal thoughts may worsen when you first start taking antidepressants, it is because ... it can take some time before the drug works (sometimes up to two weeks). Therefore, the disease is not necessarily attenuated at once, and it may therefore trigger enhanced suicidal thoughts, but this is because of the disease, not the drug.”

This information is totally misleading. The disease is *never* attenuated at once; it *doesn't* take two weeks before the drug works – the patients slowly become better whether they are treated or not (see Figure 3.1); and it is *not* the disease that triggers enhanced suicidal thoughts, it is the *drug*.

The many proofs that this is a myth come from several types of research, e.g. data show that both the incidence of akathisia and deliberate self-harm increase with dose, unlike the effect on the depression.¹¹⁵ Further, people kill themselves or others without being depressed; even healthy people can do it when they are exposed to these drugs. We also know what the mechanism of action is. In addition to akathisia, drug-induced psychosis and emotional blunting play a role; people act in an out-of-character fashion.

In 2000, David Healy published a study he had carried out with 20 healthy volunteers – all with no history of depression or other mental illness – and to his big surprise two of them became suicidal when they received sertraline.¹¹ One of them was on her way out the door to kill herself in front of a train or a car when a phone call saved her. Both volunteers remained disturbed several months later and seriously questioned the stability of their personalities. Pfizer's own studies of

sertraline in healthy volunteers had shown similarly deleterious effects, but most of these data are hidden in company files.¹¹ We have collected around 150 healthy volunteer studies from the literature and from the EMA and they show that SSRIs double the incidence of effects that can predispose to suicide.¹¹⁶

There are numerous accounts of suicides in healthy people and in people who had minor problems with their lives that should never have caused their doctor to prescribe an antidepressant for them. When I discuss this with leading psychiatrists, very few admit that the drug can cause suicide. Most display their specialty's organised denial and have typically used these arguments:

- 1) The suicides are just anecdotes (in my opinion, they are tragic losses for the families and they don't see them as "anecdotes").
- 2) The randomised trials didn't show a statistically significant increase in suicides (the psychiatrists reward the drug industry for all its frauds).
- 3) The trials only showed an increase in suicidal behaviour, not in suicides (I shall debunk this argument below).
- 4) The randomised trials were not designed to detect suicide and suicide attempts (which is correct, but the effect of this is that the true risk has been much underestimated).
- 5) Observational studies have shown that antidepressants protect against suicide, e.g. suicides increased when the usage of the drugs went down (all reliable studies have rejected this relation, see next section).
- 6) Those who killed themselves suffered from an unrecognised depression (this is a tautology, a bit like Freud's postulate that we are all homosexuals and those who deny it are latent homosexuals).

Prior to its meta-analysis of 100,000 patients, the FDA held a meeting with public representation.¹¹⁷ Understandably, the families were outraged that they had not been informed of the increased risk of suicide and said that such a notification would have led them to refuse such dangerous treatment, preventing their tragic, unnecessary losses. Here are two of the testimonies.

Lisa van Syckel mentioned that: "The FDA and the pharmaceutical industries have repeatedly stated that it is the disease, not the drug, that causes our children to become violent and suicidal. It wasn't the disease that caused my daughter to viciously mutilate herself; it was the drug. It wasn't the disease that caused my daughter to become violent and suicidal and out of control. It wasn't the disease that caused her to scream the words 'I want to die.' And, it sure as hell was not the disease that caused Christopher Pittman to kill the two people he loved the

most, his grandparents. He had been on Zoloft just three weeks and he was 12 years old. Christopher is now facing life in prison as an adult. Pfizer refers to me and others as a detractor of SSRIs and that I am misinforming legislators with oversight responsibilities. As an adult, I am considered fair game for verbal attacks but, ladies and gentlemen, Pfizer crossed the line the day they attacked a dead child. They viciously attacked a dead child and you all know it. And you, ladies and gentlemen, as adults, need to tell Pfizer that they need to stop.”

Mathy Downing (see above) noted that little Candace’s death was “entirely avoidable, had we been given appropriate warnings and implications of the possible effects of Zoloft. It should have been our choice to make and not yours. We are not comforted by the insensitive comments of a corrupt and uncaring FDA or pharmaceutical benefactors such as Pfizer who sit in their ivory towers, passing judgments on the lives and deaths of so many innocent children. The blood of these children is on your hands. To continue to blame the victim rather than the drug is wrong. To make such blatant statements that depressed children run the risk of becoming suicidal does not fit the profile of our little girl.”

Lundbeck: Our drugs protect children against suicide

Lundbeck sells five antidepressants (amitriptyline, nortriptyline, citalopram, escitalopram and vortioxetine) and the company should therefore know a lot about such drugs. However, in 2011 its CEO, Ulf Winberg, claimed in a radio programme that SSRIs reduce suicides in children and adolescents. When the stunned reporter asked him why the package inserts warned against suicide attempts, also for Lundbeck’s drugs, he replied that he expected the leaflets would be changed by the authorities!

The radio interview took place while Lundbeck’s US partner, Forest Laboratories, was negotiating compensation with 54 families whose children had committed or attempted suicide under the influence of Lundbeck’s antidepressant drugs.

The head of the Institute for Rational Pharmacotherapy in the Danish drug agency, Steffen Thirstrup, mentioned in the programme that he was surprised that Lundbeck contested the facts about the products in the package inserts that authorities both in the EU and the United States had decided the industry must disclose. Winberg replied that Thirstrup talked about suicide thoughts and behaviours whereas he himself talked about suicides, to which the interviewer pointed out that Thirstrup didn’t talk about suicide thoughts and behaviour but

about the increased risk of suicide and suicide attempts.

Wiinberg argued, as companies and silverback psychiatrists always do, that it is the depression and not the drugs that increase the risk of suicide.¹¹

I had already seen and heard much nonsense about psychiatric drugs but felt this was so much over the top that I published an open letter to Lundbeck.¹¹⁸ The next day, Lundbeck's research director, physician Anders Gersel Pedersen, responded in a way that looked like an acute attack of industry pomposititis.¹¹⁴ His reply begins thus:

We have – with regret – read Peter Gøtzsche's open letter, which unfortunately seems characterized by a limited professional insight into the complicated and extremely important issue of suicide and suicidal behaviour associated with depression in children and adolescents, and a possibly increased suicide risk in relation to treatment of depression with antidepressants ... In our view, any dialogue on this important topic should be evidence-based and not just take the form of superficial polemic on an insufficient basis.

Pedersen argued that it had never been shown that there is a clear relationship between suicidal behaviour, suicide attempts and suicide. Such arguments are utterly stupid. A suicide starts with a thought about suicide, which leads to preparations for suicide, a suicide attempt and suicide. It should surprise no one – apart from the pill pushers, including corrupt psychiatrists who say the same as Lundbeck – that the risk factors for serious suicide attempts are very similar to those for suicide.^{119, 120}

Totally misleading observational studies of suicide

Pedersen wrote that untreated depression leads to more suicides and mentioned that epidemiological studies in the United States and the Netherlands had shown that suicides among children and adolescents had increased after the usage of antidepressant drugs went down.¹¹⁴ He stated that suicide rates went up by 14% and 49%, respectively, and that the rates doubled among boys in the Netherlands.

Pedersen's references do not provide any support to his opinion. He quoted a 2007 paper by Robert Gibbons for an increase in suicide rates after the FDA and the EMA warned against using antidepressants in young people in 2003 and 2004.¹²¹ However, critics quickly pointed out the dishonest science Gibbons had

employed to make his case.¹²² He didn't use the same calendar years for SSRI prescriptions as for suicides! In fact, the number of suicides for persons ages five to 24 *declined* when there was a significant decrease in the prescribing of SSRIs to youth. In other words, the data indicated that SSRIs cause suicide.¹²²

This is not the sort of error a scientist accidentally makes. It looks like a deliberate attempt to tell a story that fits a preconceived end.¹²² In the Netherlands, the academics were incensed with Gibbons and his statistical antics (Gibbons is actually a statistician, which is hard to believe) and they noted that the increase in suicides in the Netherlands was so small that it wasn't statistically significant. They described Gibbons' conclusions as "astonishing" and "misleading" and stated that he and his co-authors had been "reckless" to publish such claims.¹²²

Gibbons has published at least ten papers telling stories that just aren't true, e.g. that the FDA's warnings have increased suicides, that antidepressants are highly effective in youths, and that the randomised placebo controlled trials of fluoxetine and venlafaxine didn't find evidence of an increased suicide risk in youths.¹²² Critics submitted letters to *Archives of General Psychiatry* pointing out that Gibbons' claim about fluoxetine and venlafaxine were based on inappropriate data selection, opaque methodology, obvious arithmetic errors, methodological errors, misinterpretation of the data, deceitful presentation, and misleading conclusions, and that, if properly analysed, Gibbons would have found the same harmful effects as the FDA did. However, the editor-in-chief Joseph Coyle refused to publish the letters but relegated them to the journal's website where few people will ever find them.

Gibbons' dishonest research has received a lot of media attention and the silverbacks love to cite it. This is how societal delusion and organised denial is created. Gibbons isn't honest about his conflicts of interest either. In 2012, he published a paper about the development of a computerised test for depression, but he and his four co-authors failed to inform the readers that Gibbons is the president and founder of a commercial company that owns the rights to the test and several related tests and that they all own shares in this company.¹²³

Pedersen's other references are similarly disastrous. A study from 2006 only included data between 1992 and the first half of 2003, and did therefore not cover the period after FDA's warning.¹²⁴ This study found that the risk of suicide attempt was highest in the month before starting antidepressant treatment and declined progressively after this. This shouldn't surprise anyone, as a suicide attempt may precisely be the reason for initiation of treatment, which the authors also acknowledged. The authors also noted that there were few adolescents in their

study and that they accounted for only three suicides and 17 serious suicide attempts. They therefore resolved that their “data contribute nothing to the debate regarding the efficacy or clinical appropriateness of antidepressant treatment for adolescents.” Perhaps Pedersen hadn’t read the paper he cited, as my discussion with Lundbeck was about suicides in children and adolescents.

Pedersen’s third reference was to an absurd study by Göran Isacson from 2005.¹²⁵ It was about forensic toxicological screening of 14,857 suicides in Sweden from 1992 to 2000. Already the premise for the study was wrong: “If treatment with SSRIs increased the suicide risk in depressed individuals, they should be found in suicides more often than otherwise expected.” Patients committing suicide might very well have stopped their treatment because of development of akathisia, which predisposes to suicide, and they might therefore not have traceable drug in the blood post mortem. Isacson actually admits this in the Discussion section of his paper (as this admission contrasts sharply with his premise, I assume the editors forced him to write it).

Just like Gibbons, Isacson has also published a long series of papers that I won’t characterise as honest attempts at finding out whether SSRIs can cause suicide; in fact, other researchers have noted that both Gibbons and Isacson conclude the opposite of what their data show.¹²⁶

Five years later, Isacson published a similar paper with the declarative title, “Antidepressant medication prevents suicide in depression.”¹²⁷ Most of the data were the same but he now included three more years, up to 2003. This article was retracted two years later in a non-transparent manner. It wasn’t clear who requested the retraction but I doubt the initiative was Isacson’s. Furthermore, readers weren’t informed what the errors were, which is bizarre. A Swedish reporter submitted a Freedom of Information request to the Karolinska Institutet in Stockholm where Isacson worked but was told it was confidential information and that no data could be released. It took five months of legal process to get access to the correct data. In its final statement to the court, Karolinska Institutet claimed that the correct figures didn’t exist at the time of the reporter’s request, but that they had now been produced.

Isacson had made much of his finding that only 15% of patients admitted for psychiatric care for depression had measurable amounts of antidepressants in their blood at the time of suicide, but it turned out that the correct number was 56%, which leads to the opposite conclusion about the effect of antidepressants than the one Isacson reached.

The correct number appeared in a short statement to the court in Stockholm, which researchers won’t find again. The reporter therefore wrote to the

Karolinska Institutet again requesting the document where the 56% was listed, but was now told that, “The data were produced owing to the request from the Administrative Court of Appeal and are not saved in any document. As the requested documents do not exist at Karolinska Institutet they cannot be released.”

Not only was the cover-up bizarre, Isacsson’s paper is, too. It is close to impossible to understand, and I found several contradictions in it. It was published in the same journal as his 2005 paper but this time the editors appeared to have woken up, as there were many more caveats, some of which are rather amusing and tell us that studies like Isacsson’s are worth absolutely nothing and only increases the scientific pollution. For example: “Definitive conclusions cannot be drawn,” “Individuals prevented from suicide cannot be identified, only their absence among suicides can be estimated,” “A limitation of the study was that we lacked information on diagnoses and other possibly confounding variables,” and the best one, “Since individuals characterized by a non-event cannot be identified, a definite proof is impossible.”

Although Isacsson’s papers were both published in *Acta Psychiatrica Scandinavica*, and 79% of the suicides were likely the same, there was no reference in the 2010 paper to the 2005 paper. Many would regard this as scientific misconduct. Isacsson quoted, however, two other of his papers, also from *Acta Psychiatrica Scandinavica*,^{128, 129} which were very similar to his 2005 and 2010 papers.

Isacsson’s pollution of the literature is substantial. A PubMed search on his name and “suicide” in the title yielded 41 references in November 2014. The retraction of his 2010 paper from *Acta Psychiatrica Scandinavica* wasn’t a hindrance for publishing more of the same in this journal. In 2013, for example, he published another forensic analysis and again claimed that the warning may have led to an increase in suicides.¹³⁰ In this paper, Isacsson has a section called “Comparison with other studies” where he cherry-picks references to Gibbons’ work and to other unreliable studies and claims that suicidality is not necessarily a risk factor for suicide. Of course it is.^{119, 120}

Isacsson has not restrained himself in the media either. In 2013, he wrote in a Swedish newspaper that those people who could have reduced their suicide risk with SSRIs were excluded from the randomised trials, which included people who were more suicide prone.¹³¹ Total nonsense. The truth is that only people at *low* risk of suicide were recruited for the trials.

Pedersen had four more references in his article. One was to a study of suicides in Danish children by Professor Lars Kessing, a psychiatrist on the

Lundbeck payroll, and others.¹³² It found that none of 42 children aged 10-17 was treated with SSRIs within two weeks prior to suicide, but since SSRIs were not approved for use in children in the study period, this isn't surprising. There was also a five-year follow-up, and now some of those who committed suicide were on SSRIs. Those treated had a "highly statistically significant and strongly increased rate of suicide compared to those not treated with SSRIs" (rate ratio 19.21; 95% CI 6.77 to 54.52). This result didn't look good for Lundbeck's drugs, and the authors presented another analysis where they had corrected for psychiatric hospital contact. The rate ratio was still increased, 4.47, but bingo, it "was no longer quite significant (95% CI 0.95 to 20.96)."

It is terribly misleading to correct for psychiatric hospital contact, as such contact *in itself* increases the risk of suicide for psychiatric patients 44 times.¹³³ A correction will therefore spuriously attenuate a true relationship. The authors found that SSRIs dramatically increase the risk of suicide in children, but they concluded the opposite:

"Not treating severely depressed children and adolescents with SSRIs may be inappropriate or even fatal." What may be fatal for children are psychiatrists who conclude like this and use SSRIs in children. The study was funded by The Lundbeck Foundation.

These researchers made other inappropriate statements. They acknowledged that the large FDA analysis found that treatment of *non-depressive* children and adolescents with SSRIs led to a higher risk of attempted suicide, but argued that, "in such analyses, it cannot be excluded that the suicidal ideations among children and adolescents with depression might reflect confounding by indication, i.e., those treated tend to have more serious conditions." This is nonsense, as the FDA analysis was based on randomised trials. There cannot be confounding by indication in randomised trials.

Another paper cited by Pedersen argued that the FDA had not found an increase in suicides and that suicidal behaviour (which *was* increased) should not count, as it is a poor surrogate marker.¹¹⁷ But the author of the paper contradicted himself, as he also wrote that, "A history of a prior suicide attempt is one of the strongest predictors of completed suicide," and that the rate of suicide is 30 times greater in previous attempters than in non-attempters.

Pedersen also quoted an unsystematic review by Robert Goldney who had cherry-picked those studies that supported his idea that antidepressants protect against suicide.¹³⁴ The paper is a classic example of how one should *not* do a review. Both Gibbons and Isacson featured there, including Isacson's meaningless 2005 study,¹²⁵ without the slightest critical comment. Goldney cited

studies in the Nordic countries that linked antidepressant prescribing with a reduction of suicide, but these studies are unreliable. Other researchers, e.g. Per-Henrik Zahl and colleagues, have showed that there is no statistical association ($P = 1.0$) between the increase in sales of SSRIs and the decline in suicide rates in the Nordic countries.¹³⁵ These authors reported that the decline in suicides in Denmark and Sweden pre-dated the introduction of SSRIs by ten years or more. I have great confidence in Zahl's research. He is both a doctor and a statistician and I have published several pivotal papers with him on mammography screening. Zahl and colleagues declared that they had no conflicts of interest while Goldney had "received honoraria and research grants from a number of pharmaceutical companies." Of course he had. With such reviews, he lives up to his name and must be worth his weight in gold for them. Other authors have confirmed Zahl et al's findings in the Nordic countries and found that a large fall in autopsy rates could entirely account for the observed changes in suicide rates.¹³⁶

Pedersen's last reference was to a paper published in a journal called *Expert Opinion on Drug Safety*.¹³⁷ I had never heard of this journal before and would never read anything in a journal with such a name. We already know we cannot trust scientific papers about drug safety, and on top of that we have experts who opine something about drug safety. If the journal had been called *Systematic Reviews of Drug Harms*, I would have been more keen to read it. The article leads absolutely nowhere. It mentions FDA's warning and the subsequent decline in the usage of antidepressants in children, which the author finds alarming without any justification whatsoever for the alarm. Truly an "expert opinion." Then there was also the usual criticism of using suicidality as a surrogate for suicides, which I suppose is listed in some *User's Guide to Eternal Happiness on the Antidepressant Drug Market*, and an alarming touch of wishful thinking:

"Most studies examined excluded patients at high risk for attempting suicide. Had such high-risk patients been included in the analyses, it is possible that antidepressants would have been found to confer a protective effect."

Remember that we are talking about children here. And yet this author has the nerve to suggest that antidepressants might have protected children from suicide if only we had done the randomised trials better! The advice of such "experts" is lethal.

Pedersen isn't just anybody. He is the research director in one of the major drug companies selling antidepressants. I have therefore dissected his seven references, as they tell us a lot about how people in drug companies and their paid prostitutes among academics actually think. Industry propaganda talks about a

research-intensive industry but those responsible in the companies aren't interested at all in research. They are only interested in pushing drugs and they do this by selling lies about their drugs, with no respect for science, human suffering and deaths, as evidenced by the fact that they prefer junk science that supports sales for true science. Science to them is just window dressing.

Silverbacks all over the world claim that antidepressants protect against suicide,⁹⁷⁻⁹⁹ and some of them struggle hard to convince the FDA to remove its black box warning against suicide in young people. The junk science they refer to seems endless. The most recent study was published in the *BMJ* in 2014,¹³⁸ but as all the previous ones, it was so flawed that nothing could be inferred from it.¹³⁹ The US researchers didn't even study their primary endpoint, suicide attempts on SSRIs, but used a poor surrogate, poisoning with all psychotropic substances. People on SSRIs who attempt suicide don't usually poison themselves (and cannot really do so with SSRIs), they tend to use violent methods like hanging.^{49, 140} The researchers also ignored the fact that any dose change with SSRIs increases the risk of suicide. Thus, the risk of suicide increases if people suddenly stop taking SSRIs because of the warning, but this would be due to withdrawal symptoms and not a sign that SSRIs protect against suicide. The researchers' assertion that FDA's warning had been harmful was completely refuted by other researchers with real data on suicide attempts from five different databases, also from the United States.¹⁴¹

Some studies are involuntarily comical. For example, a study of trends in use of antidepressants and suicides claimed that there was a clear protective effect from the drugs when it was obvious by looking at the graphs that there wasn't.¹⁴²

As just noted, properly performed observational studies have dispelled the myth that antidepressants protect against suicide.^{135, 141, 143} Such a study from Sweden, where people were their own controls,¹⁴³ found a three-to-four times increased risk of suicide after starting an SSRI, with the greatest risk in the second week of treatment (odds ratio 9.7, 95% CI 3.0 to 31.7). This is exactly what we would expect, based on other studies.

It is worth remembering that, with all the randomised trials we have, it wasn't really necessary to look at the next best evidence, the observational studies, because the trials, despite all their flaws that tend to obscure the relationship, nonetheless showed that the use of antidepressants doubles the number of suicide attempts.⁸²

One of the reasons drug companies give for using their drugs is that untreated patients may commit suicide. Therefore, according to drug company logic,

abruptly stopping what they believe is a lifesaving drug, which drug companies do when they perform placebo controlled trials, increases the patients' risk of suicide. This must mean that the companies' trials are unethical. Drug companies obviously don't take seriously their own arguments; they merely use the argument that fits the situation and don't care that their arguments are contradictory.

Antidepressant-induced homicides

That antidepressants can cause homicide is beyond doubt.^{3, 11, 49, 144, 145} We should ignore people who tell us otherwise. Some say that the randomised trials didn't show this, but the drug companies have a keen interest in hiding if anyone committed homicide while on their drug. Furthermore, homicide caused by drugs is rare, which could also be a reason why we don't see them in the trials.

As stated earlier, we know what the main mechanism of action is for suicide and homicide, the extreme form of restlessness we call akathisia. There are three strong indications that it is the drug and not some unrecognised fault with the person that leads to these violent actions: They occur for people who by all objective and subjective measures were completely normal before the act, with no precipitating factors; they were preceded by clear symptoms of akathisia; and people returned to their normal personality when they came off the antidepressant.¹⁴⁶

Antidepressants are not safe in any age group. There are numerous reports in the literature and on websites that middle-aged and even old people have killed themselves or others after having experienced akathisia. Many of these people were healthy and took the drug for non-disease-related reasons, e.g. for fun, stress, insomnia, being bullied or marital problems.^{3, 11, 145, 147}

I describe here briefly a detailed Australian report on ten forensic cases.¹⁴⁵ All patients had mutations in their CYP450 genes that changed their drug metabolism; none had been violent before; all developed akathisia; and all were able to stop taking antidepressants – frequently against medical advice – and to return to their normal personalities after the violent action. None of them had any history of mental illness, and the indications for treatment with antidepressants seemed to be non-existent:

Female, 35 years, nortriptyline, distress due to husband's drinking, killed teenage daughter in toxic delirium after three days.

Male, 18 years, fluoxetine, sister was comatose after a car crash, violent akathisia for 14 days, killed his father four days after he ran out of pills.

Male, 35 years, paroxetine, distressed by “on and off” relationship with

mother of his child, stabbed former partner 30+ times to death after 11 weeks of akathisia.

Male, 46 years, paroxetine, anxiety about not making enough money to support the family, killed his son in a manic-shift akathisia and delirium after 42 days.

Male, 16 years, sertraline and fluoxetine, depressed, struggled at school, and the girlfriend left him, attempted suicide on both drugs, killed therapist in hospital after 11 weeks.

Male, 50 years, venlafaxine, distress over divorce, shot a stranger four days after stopping drug.

Male, 24 years, escitalopram, anxiety and illicit substance use, several suicide attempts and assaults, nearly killed partner, 12 years in jail for attempted murder.

Female, 26 years, several SSRIs, difficulties with in-laws, two attempts to kill her two children.

Female, 52 years, paroxetine and citalopram, harassment at work, suicide attempt and tried to kill her two children.

Female, 25 years, citalopram and venlafaxine, marital distress, several suicide attempts on both drugs, jumped in front of a train with her child while on citalopram.

Some of these patients received other drugs or substances, e.g. cannabis, but it is highly likely that the violence was caused by the SSRIs. In several cases, the treatment provided by the psychiatrists was grossly inadequate and contributed directly to the violent actions.

An example of this is the 26-year old woman who tried to kill her two children on two occasions. She was prescribed paroxetine for stress but experienced an episode of rage and attempted suicide by inhalation of carbon monoxide, and then stopped taking the drug. Despite this, *she was prescribed paroxetine again and reassured about its safety* two years later. This time she experienced intense restlessness, surges of rage and anger, panic attacks, impulsive spending sprees, and constant suicidal ideation. She reasoned that her low self-esteem, insomnia, and suicidal behaviour were due to difficulties with her in-laws. She overdosed and was *admitted to hospital where paroxetine was increased*. She tried to kill herself again and was diagnosed with an “adjustment disorder.”

She was switched to venlafaxine, which was increased over three months until the dose was eight times higher than the initial dose. Each dose increase occasioned a week spent in bed with exhaustion, as she was unable to get up (akinesia). Her mental state deteriorated and violent outbursts and suicidal ideation became frequent and severe. Unable to stay in one place, she drove several hundred miles with her children and tried to kill them and herself by car exhaust. A few days later she tried to kill her children and herself again.

There were no interacting drugs in her regimen and many of the harms described in the FDA-approved product information for venlafaxine fit well with her experiences,¹⁴⁵ e.g. intentional injury, malaise, suicide attempt, depersonalization, abnormal thinking, akathisia, apathy, ataxia, CNS stimulation, emotional lability, hostility, manic reaction, psychosis, suicidal ideation, abnormal behaviour, adjustment disorder (which became a psychiatric diagnosis for her, although it was a side effect), akinesia, increased energy, homicidal ideation, impulse control difficulties.

In contrast to the FDA-approved product information, citizens in Australia were kept in the dark about these serious harmful effects of venlafaxine, which could drive people into committing suicide and homicide. But that doesn't pardon the psychiatrists who treated her so badly; they should have known better.

Akathisia homicides have been defended as instances of involuntary intoxication both with and without genetic evidence, and some people have succeeded in receiving damages from the manufacturers for failure to warn.¹⁴⁵

Other forensic cases are also convincing,¹⁴⁷ and the documentary evidence in a legal case on paroxetine against SmithKline Beecham included an unpublished company study of incidents of serious aggression in 80 patients, of which 25 resulted in homicide. These cases confirm that there is no upper age limit where antidepressants are safe. A man aged 74 strangled his wife, and another was 66 when he became delusional on fluoxetine and killed his wife who was found with 200 stab wounds.

According to internal company documents, 0.65% of the patients in clinical trials became hostile on paroxetine compared with 0.31% on placebo, and in healthy volunteer studies, three of 271 people (1.1%) became hostile on paroxetine and none of 138 on placebo.¹⁴⁷ As noted above, we found in our healthy volunteer studies that SSRIs double the incidence of effects that can predispose to homicide.¹¹⁶ And an analysis of the 1,374 e-mails the BBC received after its Panorama programme about paroxetine (see below) showed that the self-reports of violence from patients with no apparent background of violent behaviour were clearly linked with dosage.¹⁴⁷

In 2001, for the first time, a jury found a pharmaceutical firm liable for deaths caused by an antidepressant. Donald Schell, aged 60, had been taking paroxetine for just 48 hours when he shot and killed his wife, his daughter, his granddaughter and himself.¹⁴⁸ Central to the case were SmithKline Beecham internal documents showing the company was aware that a small number of people could become agitated or violent from paroxetine. Despite this knowledge, paroxetine packaging

did not include a warning about suicide, violence or aggression, which made the company liable. The internal documents, stamped “confidential,” list the results of tests involving more than 2,000 healthy volunteers taking either paroxetine or placebo. Some volunteers experienced anxiety, nightmares, hallucinations and other side effects – definitely caused by the drug – within two days of taking it. Two volunteers attempted suicide after 11 and 18 days, respectively.

The blatant lies of GSK, which took over SmithKline Beecham, just continued. Even in 2011, ten years after the verdict, GSK denied that paroxetine can cause people to commit homicide and suicide and that there are withdrawal problems.⁶⁸ GSK’s director of US regulatory affairs insisted that David Healy – who testified as an expert witness – had not seen all the data and said there was “no credence” to the 25% agitation rate that he gave in court. However, Healy had examined every one of the healthy volunteer studies carried out before the drug was licensed except for some material that was unaccountably not there. Furthermore, GSK contradicted itself. During Healy’s deposition for the court case, the company conceded he had seen a representative sample.

In sertraline paediatric trials, eight of 189 patients (4%) discontinued the drug because of aggression, agitation, or hyperkinesia (a coding term for akathisia), compared with none in 184 patients on placebo.¹⁴⁷

On the Internet, there is a collection of over 5,000 media stories of massacres, homicides, suicides, and school and college shootings dating back to 1966 that involve antidepressants and ADHD drugs, in some cases detailing the drugs and legal defences.¹⁴⁹ The violent actions have often been linked to akathisia, emotional blunting, and manic or psychotic reactions,¹⁴⁷ also in court cases. In 2011, for example, a Canadian judge ruled that fluoxetine induced a 16-year old boy to commit murder; he knifed a friend to death.¹⁴⁶

The organised denial in psychiatry also clouds this issue, however. It is very tragic that leading psychiatrists opine that homicidal ideation and behaviour is something entirely different from homicide.

It is particularly dangerous to take antidepressants in the United States. As mentioned above, Christopher Pittman became manic and shot his two grandparents to death two days after his dose of sertraline had been doubled.¹⁵⁰ Despite being only 12 years old when he did this, he was sentenced to 30 years of prison.

David Crespi was on fluoxetine and three other drugs, which he had taken for a couple of weeks, when he killed his two twin daughters with a knife.¹⁵¹ He pleaded guilty to avoid the death penalty and got a life sentence with no chance of parole, although he became his old self after coming off the drugs.

Canadians seem to be less inhumane than Americans. David Carmichael, who killed his 11-year old son while on antidepressants, was ruled “not criminally responsible on account of a mental disorder,” and today, Carmichael is a free man who writes and speaks on the dangers of antidepressants.

Kurt Danysh didn't have the luck to live in Canada. He was 18 years old when he was prescribed fluoxetine by a general practitioner who failed to perform any psychological testing.¹⁵² He became restless and violent and shot his father, the person he loved the most, 17 days later in a totally out-of-character mood. Kurt had no history of violence prior to fluoxetine, but in 1996, he was convicted of murdering his father and sentenced to 22.5 to 60 years in prison. Eli Lilly lied in court, claiming that fluoxetine would not cause aggressive behaviour. However, it was revealed in 2004 that Lilly had concealed data from 1988, which linked fluoxetine to violence, and the FDA recognised that SSRIs can cause violent behaviour, particularly in children and adolescents. Even though more than 70 cases of homicide linked to fluoxetine have now been reported to the FDA, the judge has dismissed all appeals, referring to a rule that requires evidence of innocence to be presented within 60 days. In official documents and letters, the prosecution's own expert stated that Kurt's criminal actions were based on insanity caused by a mind-altering drug, which should have provided Kurt a concrete defense.⁶¹ The state itself, however, forced Kurt to take more of these drugs before and during his confession. Kurt has gained a paralegal degree whilst incarcerated and has launched the SAVE campaign (Stop Antidepressant Violence from Escalating) in the hope of saving other children from his fate. It was later found out that Kurt is a poor metaboliser of SSRIs.

This is as absurd, tragic and unfair as it can possibly get. People have been released from prison decades after their confinement when a DNA test showed that they couldn't possibly have been the killer. Kurt's family cannot afford a new trial and therefore try to raise funds to make it happen.¹⁵² Why isn't there a lawyer who offers to take on this trial for free? Eli Lilly executives should have been put behind bars for decades, not Kurt, but the real villains always go free in healthcare.

I often wonder why Americans are so cruel to their own people. Where is the societal benefit in locking people up for life who wouldn't have killed if they hadn't been on drugs? It is important to realise, as few psychiatrists do, that although a misdeed may look entirely rational and planned in cold blood, this can be a totally wrong interpretation because the drug may remove the usual inhibitions people have.¹⁴⁶ We cannot say in an individual case beyond reasonable doubt that the drug *didn't* play a role. The American way of handling these killings

is inhumane to the extreme. The Crespi family, for example, has three other children. Why not let the father come home to them, which the mother wants and fights for? And why not release Kurt Danysh and the other victims of drug harms?

The pills that ruin your sex life

Antidepressants are often called happy pills, but there isn't much happiness in pills that ruin your sex life. As this might be their most common effect, they should have been called unhappy pills and marketed as a formidable disrupter of your sex life, but that wouldn't have sold many pills.

The drug companies have kept pretty quiet about this sales-threatening effect. An FDA scientist found out that they had hidden sexual problems by blaming the patients rather than the drug, e.g. female anorgasmia was coded as "Female Genital Disorder."³

The companies have claimed that very few patients become sexually disturbed, e.g. only 1.9% in the registration application for fluoxetine,⁶⁶ but the true occurrence is 30 times higher. A Spanish study designed to look at this problem found that sexual disturbances developed in 59% of 1,022 patients who all had a normal sex life before they started on drug.¹⁵³ For the five most commonly used drugs (fluoxetine, paroxetine, sertraline, citalopram and venlafaxine), I calculated the weighted average occurrence of sexual problems:

57% experienced decreased libido

57% experienced delayed orgasm or ejaculation

46% experienced no orgasm or ejaculation

31% experienced erectile dysfunction or decreased vaginal lubrication.

About 40% of the patients considered their sexual dysfunction unacceptable. Some patients yawn during orgasm, which isn't the most fantastic way of starting an intimate relationship. In another survey, of 3,516 members of patient advocate groups, sexual dysfunction was cited among the most common (51%) side effects leading to treatment drop out.¹⁵³

Imagine what it is like for a boy to encounter problems with erections the first time he is going to have sex. And that he wasn't told it was due to the drug. He will think there is something wrong with *him*, and so will his sweetheart, particularly when the problem returns next time they try. This is cruel, and a child psychiatrist in Brisbane told me about three boys who had attempted suicide for

this reason.

The delayed orgasm is being used therapeutically by men who have premature ejaculation. However, the randomised trials that have shown an effect on this don't appear to be fully reliable, as they didn't report on other sexual harms.¹⁵⁴

¹⁵⁵ For example, a large randomised trial from Iran with only one author reported no cases of impotence in 138 patients on escitalopram, but in three of 138 patients on placebo.¹⁵⁵

Sexual problems are easily overlooked if the clinical interview is not directed towards revealing them. Patients aren't likely to bring them up spontaneously and might not even think they could be drug-related; e.g. in the Spanish study, only 20% of the patients reported their sexual dysfunction spontaneously.¹⁵³

Some men become impotent on SSRIs but Eli Lilly has the answer.¹⁵⁶ The company sells tadalafil (Cialis) against erectile dysfunction and recommends to take it not on demand but every day "so that the sexually active can obtain constant spontaneity." As the scouts say: "Be prepared!" Your eternal erection, which may give your wife eternal headache, is just a pill away. "Ask your doctor whether an erection is right for you."

Damage to the foetus

As already noted, the Danish National Board of Health recommends routine screening of pregnant women for depression and treatment with antidepressant drugs, although the available data do not support these recommendations.⁸ It acknowledges that SSRIs increase the occurrence of spontaneous abortions, decrease the birth weight, likely increase the occurrence of birth defects, increase neonatal complications such as irritability, tremor, hypertonia and difficulty sleeping or breast feeding, and increase the risk by a factor of five for developing pulmonary hypertension, which is a lethal harm estimated to occur in 6-12 newborns per 1,000.⁸

Given these facts, the Board's recommendation is so absurdly harmful that I wrote a little sketch about it. In 2013, I listened to a brilliant lecture by a former patient with schizophrenia, Olga Runciman, who told us how she had found her way out of psychiatry, abandoned the drugs and now lived a normal life. My lecture came right after hers, and I had never met her before but was so impressed that I asked if she would be interested in playing the part of the young pregnant woman in my sketch. She accepted and we read it aloud from my computer as an introduction to my lecture, which is on you tube with English subtitles (https://www.youtube.com/watch?v=i1LQiw_ZIQ/). I must have hit something

essential, as it was seen by over 10,000 people in two weeks:¹⁵⁷

“How’s the pregnancy going?”

“Fine, I don’t have any problems.”

“And you’re not worried about whether you can manage to look after the baby?”

“No, not at all. I am a housewife and have time to take care of it.”

“Are you aware that it’s possible to have depression without knowing it?”

“No, I didn’t know that, but I’m fine.”

“Yes, but ... uhmmm ... well ... you see ... if you are suffering from a depression, it would be good to find out.”

“But I’m perfectly fine.”

“I still think you should undergo this test for depression.”

“So what if it’s positive?”

“Then you may get a drug that will help you.”

“Well ... I ... There is nothing wrong with me, so why should I undergo this test?”

“The National Board of Health recommends it.”

“Can’t you just unsubscribe from the Board of Health? Sorry, that was a joke!”

“No, we are obliged to follow the Board’s recommendations.”

“But a test like that is a screening test, is there a Cochrane review about it?”

“Yes, and it recommends that one shouldn’t screen healthy people for depression.”

“Then why on earth does the Board of Health recommend screening pregnant women like me who are healthy?”

“I cannot understand this either, but the Board of Health has consulted experts in psychiatry who think it’s a good idea.”

“And how many of those who are healthy will get a wrong depression diagnosis with this screening test?”

“About one third.”

“Holy smoke! How does the drug work?”

“It works like amphetamine. It’s also difficult for the patients to quit, just like for amphetamine and other narcotic drugs; half the patients have difficulty stopping.”

“And what do these experts say about the side effects of the drug? What is the most common side effect?”

“Sexual problems. They occur in half the patients. It can be lack of sex drive, impotence and lack of orgasm, even for the man if he receives the drug.”

“And how about suicide? Depressed people are at high risk of committing suicide, and I assume that this medication will prevent that from happening?”

“No, on the contrary, for someone as young as you, the drug increases the risk of suicide. It can also cause birth defects. The risk is small but it is clearly increased.”

“My goodness! Many thanks for all this information, doc. Count me out. You don’t need to give me that test. I don’t want to risk getting a wrong diagnosis of depression and get treated. My husband and I love sex. And I have no wish to get a narcotic on prescription, or to commit suicide, or to give birth to a deformed child.”

In my opinion, this contrived dialogue, with its fortunate outcome, requires three things: The doctor needs to be exceptionally well informed about the facts; the woman needs to ask relevant questions; and the doctor needs to reply adequately to them. The clinical reality is not like this very often. On the other hand, some doctors will not settle for a questionnaire but will ask clarifying questions and possibly use an additional instrument. Obviously, the use of additional instruments will reduce the proportion of false positives, but they are also very uncertain. Screening healthy people will therefore inevitably lead to many false positive diagnoses, and many healthy people will be treated with antidepressants that harm them.

Birth defects have been studied in a large Danish cohort study of 500,000 children, which showed that the risk of heart septum defect is doubled.¹⁵⁸ This is not a trivial harmful effect, as 1% of the foetuses treated will get a septum defect. Cardiac birth defects are exactly what we would expect to see because serotonin plays a major role for the functioning of the heart. We have seen deadly valvular defects and pulmonary hypertension in adults who took diet pills that increase serotonin levels, and these drugs have been withdrawn from the market.³

It is of great value for people with vested interests to spread doubt about whether SSRIs cause birth defects, and many substandard studies claim they don’t. It is often wise to read the critical letters that are subsequently published, e.g. those related to a 2014 study from the *New England Journal of Medicine* claiming there was no problem.¹⁵⁹

There is no doubt that SSRIs cause birth defects. A lawyer in Houston sent me a 56-page expert report prepared for a court case by epidemiologist Nicholas Jewell who meticulously went through the scientific literature. It is superb work that demonstrates that cardiac birth defects are a class effect of SSRIs.¹⁶⁰ Confounding by indication was ruled out, which means that depression in itself

doesn't cause birth defects (which would also have been very strange, but there seems to be no limits as to what psychiatrists are prepared to suggest to avoid incriminating their drugs). Animal studies support the human studies and show a dose-response relationship. SSRIs also double the risk of preterm birth.¹⁶¹ The conclusion is inescapable:

Under no circumstances should pregnant women be screened for depression or treated with antidepressant drugs.

The fraud and lies of GlaxoSmithKline

GlaxoSmithKline (GSK) is one of the most criminal drug companies in the world.³ It has committed numerous offences that fulfil the criteria for organised crime under US law, and in 2011, GSK pleaded guilty to having marketed a number of drugs illegally for off-label use and was to pay \$3 billion, the largest healthcare fraud settlement in US history. Many of the crimes that have been most harmful for the patients and have caused many deaths have involved psychotropic drugs, but GSK has also killed many patients with rosiglitazone, a diabetes drug, which the company touted had cardiovascular benefits although it increases cardiovascular mortality and was taken off the market in Europe for this reason.³

The criminal activities seem to have involved making false statements to state officials, obstructing a federal investigation into illegal marketing, lying to the FDA about illegal promotion, withholding incriminating documents, paying kickbacks to doctors, and misreporting prices to Medicaid.

SmithKline Beecham, later merged into GSK, started marketing paroxetine in 1992. The company falsely claimed for the next ten years that paroxetine isn't habit-forming despite the fact that it led to withdrawal reactions in 30% of the patients in the original licence application.³ In 2001, the World Health Organization had found paroxetine to have the hardest withdrawal problems of any antidepressant drug and in 2002, the FDA published a warning. A year later GSK quietly and in small print revised its previous estimate of the risk of withdrawal reactions in the prescribing instructions from 0.2% to 25%,¹⁶² a 100 times increase.

Our drug regulators did absolutely nothing to protect patients for ten years, although the rate of withdrawal reactions was 100 times larger in the registration material than in the material GSK presented to patients and doctors.

From 2002 onwards, the BBC presented four documentaries about SSRIs in its Panorama series, the first one called *Secrets of Seroxat*. They are very good. The

journalist, Shelley Joffe, showed that the GSK spokesperson, Dr Alastair Benbow, lied in front of a running camera. He denied, for example, that paroxetine could cause suicidality or self-harm while he sent data to the drug regulator one month later that showed exactly this, and which immediately led to a ban on using paroxetine in children. The UK drug regulator also lied to the public and covered up for GSK, which is based in the United Kingdom, when it said that this information was completely new to the company (which had known about it for around ten years). Worst of all:

The head of the UK drug agency echoed the drug companies' untruthful assertion that it was the disease, not the drug, that increased the suicide risk.

Later, when US senator Charles Grassley asked GSK for how long the company had known that paroxetine increases the suicide risk, the company lied again. GSK replied that they detected no signal of any possible association between paroxetine and suicidality in adult patients until late February 2006. However, government investigators found that the company had the data back in 1998 and David Healy found evidence in internal company documents that 25% of healthy volunteers experienced agitation and other symptoms of akathisia while taking paroxetine.

Other studies have shown similar results.¹⁶³ Peter Breggin was an expert witness in a case where a man drowned his two children and himself under influence of paroxetine, but in 2001, GSK prevented Breggin from publishing his findings about suicidality and violence, which showed that GSK had been negligent.⁴⁹ The judge contributed to the absurdity by calling this proprietary information. GSK listed suicide attempts as emotional lability and disguised cases of akathisia by using many subcategories for overstimulation such as nervousness and anxiety.

The BBC asked the public to submit emails about their experiences with paroxetine, and 1,374 emails were read by clinical pharmacologist Andrew Herxheimer and researcher Charles Medawar, cofounder of Social Audit. Though GSK had fiercely denied that SSRIs cause dependence and can lead to suicide, it was clear that both claims were wrong, and that, furthermore, the drugs can lead to hostility and murder, e.g. "After 3 days on paroxetine, he sat up all night forcing himself to keep still because he wanted to kill everyone in the house."¹⁶²

The richness of the patients' own reports was impressive. Many described electric shock sensations in the head and visual problems when they tried to stop,

but such reactions had been coded by the authorities as dizziness or paraesthesia. Because of these revelations, drug agencies in many other countries now accept adverse events reports submitted directly by patients, without needing to pass the doctors' obstacles first.

In 2004, a researcher used the comprehensive internal reports of GSK's trials, made available on the internet as a result of litigation, and found that paroxetine increased significantly suicidal tendencies, odds ratio 2.77 (95% confidence interval 1.03 to 7.41).¹⁶⁴ He included the unpublished trial 377, which didn't find an effect of paroxetine and which GSK had stated in an internal document that there were no plans to publish.¹⁶⁵ He also included the infamous trial 329.

Trial 329 of paroxetine in children and adolescents

GSK published trial 329 in 2001.¹⁶⁶ The paper falsely stated that paroxetine was effective in children and adolescents and had minimal side effects, and GSK also lied to its sales force, telling them that the trial had shown remarkable efficacy and safety.¹⁶⁷

The trial was widely believed and cited,³ but it was fraudulent. What it showed was remarkable inefficacy and harms, but after extensive manipulations, the ghost-written paper, which had 20 doctors as "authors," ended up being positive.^{167, 168} The statistical alchemy created four statistically significant effects after splitting the data in various ways, and many variations were tried before the data confessed to the torture.

The paper falsely stated that the new outcomes were declared *a priori*, and for harmful effects, the manipulations were even worse. The internal, unpublished study report showed that at least eight children became suicidal on paroxetine versus one on placebo ($P = 0.035$). In the published paper only one headache was considered to be related to paroxetine treatment. Cases of suicidal thoughts and behaviour on paroxetine were called emotional lability, hospitalisation, exacerbated depression or aggression,^{3, 164} and at least three adolescents who threatened or attempted suicide weren't described in the paper whose first author, Martin Keller, wrote that they were terminated from the study because of non-compliance.⁵⁷ When the FDA demanded the company to review the data again, there were four additional cases of intentional self-injury, suicidal ideation or suicide attempt, all on paroxetine.

Keller seems to be the typical sort of guy we find at the top of the much touted

public-private “partnerships.” He double-billed his travel expenses; the psychiatry department he chaired received hundreds of thousands of dollars to fund research that wasn’t being conducted; Keller himself received hundreds of thousands of dollars from drug companies every year he didn’t disclose; and a social worker found a list of adolescents who indicated they had been enrolled in a study, which wasn’t true.³ It seemed they were made up, which would have been tempting, given that \$25,000 was offered by the drug company for each vulnerable teenager Keller delivered.

Keller’s misdeeds didn’t harm his career, likely because his department had received \$50 million in research funding. This isn’t a printing error: \$50 million! A spokesperson from Brown, where Keller worked, said that Keller’s research on paroxetine complied with their research standards. Fraudulent research that has contributed importantly to pushing thousands of children and adolescents into suicide all over the world is said to live up to a US hospital’s research standards. Welcome to America!

The *Journal of the American Academy of Child and Adolescent Psychiatry*, which published the fraud, refused to convey to their readers that the article misrepresented the science and refused to retract it.¹⁶⁸ An explanation for this passivity can likely be found by following the money that goes to the journal’s owner.

GSK pushed its drug for use in children, although it didn’t work, was immensely harmful, and wasn’t approved for use in children. The illegal marketing involved withholding trials showing paroxetine was ineffective,¹⁶⁹ and the fraud was deliberate: “It would be commercially unacceptable to include a statement that efficacy had not been demonstrated, as this would undermine the profile of paroxetine.”¹⁶⁸

The fraud and lies paid off, which is why there is so much of it. From 1998 to 2001, five million prescriptions a year were being written for paroxetine and sertraline for children and adolescents.¹⁷⁰ Even in 2013, hundreds of suicides later, GSK had the audacity to state: “GSK does not agree that the article is false, fraudulent or misleading.”¹⁷¹

Why does anyone pay attention to what drug companies or their hired allies among doctors say about anything? Unfortunately, publicly funded studies can be just as deceptive as those funded by the drug industry. The huge STAR*D study is one such example.

The STAR*D study, a case of consumer fraud?

This STAR*D study was highly relevant. The set-up and entry criteria reflected normal clinical practice and real patients seeking care were included.¹⁷² It was the largest antidepressant effectiveness study ever conducted. Funded by the NIMH at a cost of \$35 million, it was designed to test whether a multistep, flexible use of medications, including add-on drugs to augment the effect of the antidepressant, would help people recover and stay well, also in the year after recovery.

The investigators announced that the study would produce results with “substantial public health and scientific significance.” It surely did, but not in the way the investigators had imagined. It didn’t show the expected positive results, and all the initial brouhaha ended as a story of a scientific scandal and dishonest science.¹⁷³

There was no placebo group, and all patients started on citalopram. This was motivated by citalopram’s “absence of discontinuation symptoms” and “safety” in elderly patients.¹⁷⁴ It defies belief that some of the prominent psychiatrists in America chose to display this level of ignorance about the drugs they use, considering that the trial protocol was from 2002.

When the study was over, NIMH announced that, “about 70% of those who did not withdraw from the study became symptom-free.” NIMH repeated this false claim 36 times in various press releases,¹⁷⁵ and the investigators also made numerous false claims, e.g. that the patients who scored as remitted had “complete absence of depressive symptoms” and had “become symptom-free.”¹⁷⁶ The truth was that a “remitted” patient could have a Hamilton score of 7. The only Hamilton suicide question, “feels like life is not worth living,” is scored as 1, and other symptoms that are scored as 1 include “feels he/she has let people down” and “feels incapable, listless, less efficient.” No professional would describe such patients as having become symptom-free; in fact, each of these symptoms are used in diagnosing major depression.¹⁷⁶

The researchers noted in their abstract that, “The overall cumulative remission rate was 67%.” In the main text, however, they acknowledged that this was a “theoretical” remission rate assuming that those who exited the study would have had the same remission rates as those who stayed in the protocol. That assumption is extremely unlikely to be true. There are usually many more treatment failures among those who drop out than among those who continue. Furthermore, the investigators cherry-picked the data they reported. Instead of using the Hamilton scale as planned, they used a scale that added another 152 patients to the remitted group.¹⁷² They also included 607 patients with mild depression and 324 patients with no baseline Hamilton score who, according to their own protocol and a flow

chart they published,^{172, 174} should have been excluded from their analyses, and which further inflated the number of remitted patients. If the study protocol had been followed and the results honestly reported, the researchers would have announced that 38% of the patients remitted during the four steps of treatment, and that the remaining 62% either dropped out or failed to remit.¹⁷²

The investigators stated that most of the remitted patients stayed well also throughout the final year of “continuing care” where the physicians could change the patients’ medications, alter dosages, and add new medications.¹⁷³ Science journalist Robert Whitaker did his best to figure out the precise number of patients who remitted and stayed well throughout the study, but the data were presented in such a confusing manner that he gave up. Ed Pigott and colleagues succeeded in cracking the nut and reported that only 108 of the 4,041 patients¹⁷⁴ had a “sustained remission,”¹⁷⁴ which means that only 3% of the patients who entered the trial remitted, stayed well and stayed in the trial during the year of follow-up.

Even this low percentage is likely exaggerated.¹⁷² Many of the 108 stay-well patients must have come from the group of 607 patients with mild depression that shouldn’t have been included in the analysis. Furthermore, the investigators took great care to deliver an optimal treatment, so the success rate in routine clinical care must be lower than 3%.

Pigott and colleagues advised that, in light of the meagre results of STAR*D, we should not use the term “treatment-resistant depression.”¹⁷² We should focus on what is wrong with our treatment rather than using language that wrongly implies that there is an exceptional subgroup of patients with an exotic form of depression who are refractory to an otherwise effective treatment.

This publicly funded study bombarded the doctors and the public with the monstrously misleading message that antidepressants enable about 70% of depressed outpatients to recover, and the medications were said to be “far more effective” than placebo,¹⁷³ which is equally untruthful, and apart from this there was no placebo group to compare with! A journalist asked one of the investigators, Maurizio Fava, a prominent psychiatrist from Massachusetts General Hospital in Boston, whether the analysis by Pigott and colleagues¹⁷² was correct. “I think their analysis is reasonable and not incompatible with what we had reported,” Fava said.¹⁷³ This is a remarkable admission. Fava acknowledged that the 3% success rate is accurate and also, at least indirectly, that this is the real result. He also acknowledged that the investigators knew this all along, and that this information was in their published reports. However, it is not honest science to play hide and seek to such an extent that it is close to impossible for others to unravel the truth.

The STAR*D study results show that the practice guideline of the American Psychiatric Association that advise that “following remission, patients who have been treated with antidepressant medications in the acute phase should be maintained on these agents to prevent relapse” is harmful. Antidepressants don’t cure depressions and don’t prevent them; they cause them. They are depressogenic agents when used long term (see Chapter 11). The STAR*D study provides convincing evidence of the drug-induced tardive dysphoria other investigators have described.^{177, 178}

Psychiatrists often respond to Whitaker’s talks by quoting the fake STAR*D results, which they take as evidence that antidepressants – if you just keep trying them – work for most people. This collective delusion is terribly harmful.

The many STAR*D papers display highly selective reporting of outcomes, numerous false claims, contradictory statements, and even pure fiction.¹⁷⁶ As of mid-2011, despite over 100 papers having been published, 11 prespecified outcomes had still not been reported!¹⁷⁹ One paper stated in the abstract that suicidal ideation was seen in only 0.7% of the patients, and the authors said that their study “provides new evidence to suggest little to no relation between use of a selective serotonin reuptake inhibitor and self-reported suicidal ideation.” This misleading statement was *contradicted by some of the same authors* who, in other papers, mentioned suicidal ideation in 6.3% and 8.6% of those on citalopram in STAR*D, i.e. 10 times more.

Is the STAR*D study so fraudulent that all its 100+ papers should be retracted? Ed Pigott says about this:¹⁷⁶

“In my five plus years investigating STAR*D, I have identified one scientific error after another. Each error I found reinforced my search for more ... These errors are of many types, some quite significant and others more minor. But all of these errors – without exception – had the effect of making the effectiveness of the antidepressant drugs look better than they actually were, and together these errors led to published reports that totally misled readers about the actual results. As such, this is a story of scientific fraud, with this fraud funded by the National Institute of Mental Health at a cost of \$35 million.”

As already noted, all patients tried citalopram first, and its remission rate was inflated by 45% in the summary article. In their disclosure statements, ten of STAR*D’s authors report receiving money from Forest, Lundbeck’s partner in the United States. In 2011, Pigott filed a whistleblower complaint that alleges that Forest bribed a principal investigator to fix the results in favour of citalopram.¹⁸⁰ The complaint alleges that because of this bribe, citalopram was the only antidepressant employed in the first part of the study, and it led to falsification and

overstatement of the drug's effectiveness.

I searched PubMed for STAR*D in January 2015 and got 290 hits, so I really appreciate that Pigott and others did the detective work for me. It is scandalous that a hugely expensive study funded by taxpayers can be so shamelessly misleading, but it illustrates once again that top psychiatrists are prepared to defend their organised illusions, whatever the costs in terms of money, deception, dishonesty, lack of public trust in their specialty, and harm to the patients. In my opinion, the STAR*D papers should be retracted and an independent group of scientists who are not psychiatrists should be funded to write a paper that explains what this study really showed: That antidepressants are ineffective and harmful.

References

- 1 Petersen M. *Our daily meds*. New York: Sarah Crichton Books; 2008.
- 2 Thoms BD, Arthurs E, El-Baalbaki G, et al. Risk of bias from inclusion of patients who already have diagnosis of or are undergoing treatment for depression in diagnostic accuracy studies of screening tools for depression: systematic review. *BMJ* 2011;343:d4825.
- 3 Gøtzsche PC. *Deadly medicines and organised crime: How big pharma has corrupted health care*. London: Radcliffe Publishing; 2013.
- 4 Henkel V, Mergl R, Kohnen R, et al. Identifying depression in primary care: a comparison of different methods in a prospective cohort study. *BMJ* 2003; 326:200-1.
- 5 Frances A. *Saving normal*. New York: Harper Collins; 2013.
- 6 Raven M. *Depression and antidepressants in Australia and beyond: a critical public health analysis (PhD thesis)*. University of Wollongong, Australia; 2012. <http://ro.uow.edu.au/theses/3686/>.
- 7 Gilbody S, House A, Sheldon T. Screening and case finding instruments for depression. *Cochrane Database Syst Rev* 2005;4:CD002792.
- 8 [Guidelines for unipolar depression in adults]. København: Sundhedsstyrelsen; 2007.
- 9 Coupland C, Dhiman P, Morriss R, et al. Antidepressant use and risk of adverse outcomes in older people: population based cohort study. *BMJ* 2011;343:d4551.
- 10 Østergaard SD, Jensen SOW, Bech P. The heterogeneity of the depressive syndrome: when numbers get serious. *Acta Psychiatr Scand* 2011;124:495-6.
- 11 Healy D. *Let them eat Prozac*. New York: New York University Press; 2004.
- 12 The creation of the Prozac myth. *The Guardian* 2008;Feb 27.
- 13 Dowrick C, Frances A. Medicalising unhappiness: new classification of depression risks more patients being put on drug treatment from which they will not benefit. *BMJ* 2013;347:f7140.
- 14 Nielsen M, Gøtzsche P. An analysis of psychotropic drug sales. Increasing sales of selective serotonin reuptake inhibitors are closely related to number of products. *Int J Risk Saf Med* 2011;23:125-32.
- 15 Bruun KS. [Chief physician: Those who get antidepressants need them]. *Netavisen* 2014 Oct 21.
- 16 Gøtzsche PC. Why I think antidepressants cause more harm than good. *Lancet Psychiatry* 2014;1:104-6.
- 17 Laughren TP. Overview for December 13 Meeting of Psychopharmacologic Drugs Advisory Committee (PDAC). 2006 Nov 16. Available online at:

- www.fda.gov/ohrms/dockets/ac/06/briefing/2006-4272b1-01-FDA.pdf (accessed 22 October 2012).
- 18 Arroll B, Elley CR, Fishman T, et al. Antidepressants versus placebo for depression in primary care. *Cochrane Database Syst Rev* 2009;3:CD007954.
 - 19 Turner EH, Matthews AM, Linardatos E, et al. Selective publication of antidepressant trials and its influence on apparent efficacy. *N Engl J Med* 2008; 358:252-60.
 - 20 Hróbjartsson A, Gøtzsche PC. Placebo interventions for all clinical conditions. *Cochrane Database Syst Rev* 2010;1:CD003974.
 - 21 Hróbjartsson A, Thomsen AS, Emanuelsson F, et al. Observer bias in randomised clinical trials with binary outcomes: systematic review of trials with both blinded and non-blinded outcome assessors. *BMJ* 2012;344:e1119.
 - 22 Fisher S, Greenberg RG. How sound is the double-blind design for evaluating psychotropic drugs? *J Nerv Ment Dis* 1993;181:345-50.
 - 23 Moncrieff J, Wessely S, Hardy R. Active placebos versus antidepressants for depression. *Cochrane Database Syst Rev* 2004;1:CD003012.
 - 24 Leucht S, Fennema H, Engel R, et al. What does the HAMD mean? *J Affect Disord* 2013;148:243-8.
 - 25 Hamilton M. A rating scale for depression. *J Neurol Neurosurg Psychiatr* 1960; 23:56-62.
 - 26 Fournier JC, DeRubeis RJ, Hollon SD, et al. Antidepressant drug effects and depression severity: a patient-level meta-analysis. *JAMA* 2010;303:47-53.
 - 27 DeRubeis, Fournier JC. Depression severity and effect of antidepressant medications. *JAMA* 2010;303:1599.
 - 28 Zimmerman M, Mattia JI, Posternak MA. Are subjects in pharmacological treatment trials of depression representative of patients in routine clinical practice? *Am J Psychiatry* 2002;159:469-73.
 - 29 Walsh BT, Seidman SN, Sysko R, et al. Placebo response in studies of major depression: variable, substantial, and growing. *JAMA* 2002;287:1840-7.
 - 30 Gibbons RD, Hur K, Brown H, et al. Benefits from antidepressants: synthesis of 6-week patient-level outcomes from double-blind placebo-controlled randomized trials of fluoxetine and venlafaxine. *Arch Gen Psychiatry*. Published online March 5, 2012.
 - 31 Johnson LA. Pfizer disputes claim against antidepressant. *USA Today* 2013 Jan 31.
 - 32 StekML, Wurff van der FFB, Hoogendijk WJG, et al. Electroconvulsive therapy for the depressed elderly. *Cochrane Database Syst Rev* 2003;2:CD003593.
 - 33 Eyding D, Lelgemann M, Grouven U, et al. Reboxetine for acute treatment of major depression: systematic review and meta-analysis of published and unpublished placebo and selective serotonin reuptake inhibitor controlled trials. *BMJ* 2010;341:c4737.
 - 34 Press release. Antidepressants: benefit of reboxetine not proven. 2009 Nov 4. <http://www.iqwig.de/index.981.en.html> (accessed 4 Feb 2010).
 - 35 Wieseler B, McGauran N, Kaiser T. Finding studies on reboxetine: a tale of hide and seek. *BMJ* 2010;341:c4942.
 - 36 European Medicines Agency. Pharmacovigilance Working Party (PhVWP), July 2011 plenary meeting. 2011 July 28.
 - 37 Barbui C, Furukawa TA, Cipriani A. Effectiveness of paroxetine in the treatment of acute major depression in adults: a systematic re-examination of published and unpublished data from randomized trials. *CMAJ* 2008;178:296-305.
 - 38 Serna MC, Cruz I, Real J, et al. Duration and adherence of antidepressant treatment (2003 to 2007) based on prescription database. *Eur Psychiatry* 2010; 25:206-13.

- 39 Breggin P. Talking back to Prozac. New York: E-reads, 1994.
- 40 Spielmans GI, Gerwig K. The efficacy of antidepressants on overall well-being and self-reported depression symptom severity in youth: a meta-analysis. *Psychother Psychosom* 2014;83:158-64.
- 41 Hetrick SE, McKenzie JE, Cox GR, et al. Newer generation antidepressants for depressive disorders in children and adolescents. *Cochrane Database Syst Rev* 2012;11:CD004851.
- 42 Greenberg RP, Bornstein RF, Greenberg MD, et al. A meta-analysis of antidepressant outcome under “blinded” conditions. *J Consult Clin Psychol* 1992;60:664-9.
- 43 Moncrieff J. The bitterest pills. Basingstoke: Palgrave Macmillan; 2013.
- 44 Moore TJ, Cohen MR, Furberg CD. Quarter Watch Monitoring FDA MedWatch Reports. Why reports of serious adverse drug events continue to grow. 2012 Oct 3.
- 45 Perahia DG, Kajdasz DK, Desai D, et al. Symptoms following abrupt discontinuation of duloxetine treatment in patients with major depressive disorder. *J Affect Disord* 2005;89:207-212.
- 46 Whitaker R. Anatomy of an Epidemic. New York: Broadway Paperbacks; 2010.
- 47 Timimi S. Child psychiatry and its relationship with the pharmaceutical industry: theoretical and practical issues. *Adv Psychiat Treat* 2008;14:3-9.
- 48 Mellinger K. Our powerful mind, and hope. 2014 Oct 25.
<http://www.madmamerica.com/2014/10/powerful-mind-hope/> (accessed 19 Dec 2014).
- 49 Breggin P. Medication madness. New York: St. Martin’s Griffin; 2008.
- 50 Virapen J. Side effects: death. College Station: Virtualbookworm.com Publishing; 2010.
- 51 Hedlund F. [National expert was bought by drug giant]. *Dagens Nyheter* 2007 Nov 30.
- 52 Hedlund F. [Drug company used pretty dirty methods]. *Dagens Nyheter* 2007 Nov 30.
- 53 Bauer J, Karle J. [Wrong information about depression]. *Politiken* 2011 Apr 1.
- 54 Kessing L, Hansen HV, Demyttenaere K, et al. Depressive and bipolar disorders: patients’ attitudes and beliefs towards depression and antidepressants. *Psychological Medicine* 2005;35:1205-13.
- 55 Read J, Cartwright C, Gibson K. Adverse emotional and interpersonal effects reported by 1829 New Zealanders while taking antidepressants. *Psychiatry Res* 2014;216:67-73.
- 56 Wang PS, Gilman SE, Guardino M, et al. Initiation of and adherence to treatment for mental disorders. *Med Care* 2000;38:926-36.
- 57 Bass A. Side Effects – a prosecutor, a whistleblower, and a bestselling antidepressant on trial. Chapel Hill: Algonquin Books; 2008.
- 58 Gale Zikis, Individually and as administrator of the estate of Donald R. Zikis plaintiff, v. Pfizer Inc., a Delaware corporation, defendant. In the United States district court for the northern district of Illinois eastern division. No. 04 C 8104. 2005 Nov 8.
- 59 Alliance for Human Research Protection. 2005 Aug 29. FDA response to Utah court must be truthful. <http://www.ahrp.org/infomail/05/08/29.php> (accessed 10 Jan 2015).
- 60 Lenzer J. Drug secrets: what the FDA isn’t telling. *Slate* 2005 Sept 27.
- 61 Corriher SC. The pharmaceutical tragedy of Kurt Danysh.
<http://healthwyze.org/index.php/component/content/article/163-kurt-danysh.html> (accessed 2 Jan 2015).
- 62 FDA. Historical Information on duloxetine hydrochloride (marketed as Cymbalta).
<http://www.fda.gov/Drugs/DrugSafety/PostmarketDrugSafetyInformationforPatientsandProviders/ucm114970.htm> (accessed 15 Sept 2013).
- 63 Lenzer J. FDA to review ‘missing’ drug company documents. *BMJ* 2005;330:7.
- 64 Medawar C, Herxheimer A. A comparison of adverse drug reaction reports from professionals and

- users, relating to risk of dependence and suicidal behaviour with paroxetine. *Int J Risk Saf Med* 2003/2004;16:5-19.
- 65 Bouchy C. Internal Eli Lilly memo. 1990 Nov 13.
- 66 Medawar C. The antidepressant web – marketing depression and making medicines work. *Int J Risk Saf Med* 1997;10:75-126.
- 67 Healy D. Did regulators fail over selective serotonin reuptake inhibitors? *BMJ* 2006;333:92-5.
- 68 Boseley S. Murder, suicide. A bitter aftertaste for the ‘wonder’ depression drug. *Guardian* 2011 June 11.
- 69 Stipp D. Trouble in Prozac. *CNN Money* 2005 Nov 28.
- 70 Rost P. *The Whistleblower: confessions of a healthcare hitman*. New York: Soft Skull Press; 2006.
- 71 Healy D. SSRIs and deliberate self-harm. *Br J Psychiatry* 2002;180:547.
- 72 Rockhold F, Metz A, Traber P. Response from GlaxoSmithKline. *Br J Psychiatry* 2002;180:548.
- 73 Stone M, Laughren T, Jones ML, et al. Risk of suicidality in clinical trials of antidepressants in adults: analysis of proprietary data submitted to US Food and Drug Administration. *BMJ* 2009;339:b2880.
- 74 Valuck RJ, Orton HD, Libby AM. Antidepressant discontinuation and risk of suicide attempt: a retrospective, nested case-control study. *J Clin Psychiatry* 2009;70:1069-77.
- 75 Eli Lilly memo. Suicide Report for BGA. Bad Homburg. 1990 Aug 3.
- 76 Montgomery SA, Dunner DL, Dunbar GC. Reduction of suicidal thoughts with paroxetine in comparison with reference antidepressants and placebo. *Eur Neuropsychopharmacol* 1995;5:5-13.
- 77 Internal Eli Lilly memo. Bad Homburg. 1984 May 25.
- 78 Mackay FJ, Dunn NR, Wilton LV, et al. A comparison of fluvoxamine, fluoxetine, sertraline and paroxetine examined by observational cohort studies. *Pharmacoepidemiol Drug Safety* 1997;6:235-46.
- 79 Laughren TP. The scientific and ethical basis for placebo-controlled trials in depression and schizophrenia: an FDA perspective. *Eur Psychiatry* 2001;16:418-23.
- 80 GlaxoSmithKline. Briefing Document. Paroxetine adult suicidality analysis: major depressive disorder and non-major depressive disorder. 2006 April 5.
- 81 GlaxoSmithKline. Important prescribing information. 2006 May.
- 82 Fergusson D, Doucette S, Glass KC, et al. Association between suicide attempts and selective serotonin reuptake inhibitors: systematic review of randomised controlled trials. *BMJ* 2005;330:396.
- 83 Vanderburg DG, Bazar E, Fogel I, et al. A pooled analysis of suicidality in double-blind, placebo-controlled studies of sertraline in adults. *J Clin Psychiatry* 2009;70:674-83.
- 84 Gunnell D, Saperia J, Ashby D. Selective serotonin reuptake inhibitors (SSRIs) and suicide in adults: meta-analysis of drug company data from placebo controlled, randomised controlled trials submitted to the MHRA’s safety review. *BMJ* 2005;330:385.
- 85 Apter A, Lipschitz A, Fong R, et al. Evaluation of suicidal thoughts and behaviors in children and adolescents taking paroxetine. *J Child Adolesc Psychopharmacol* 2006;16:77-90.
- 86 Hammad TA, Laughren T, Racoosin J. Suicidality in pediatric patients treated with antidepressant drugs. *Arch Gen Psychiatry* 2006;63:332-9.
- 87 Gibbons RD, Brown CH, Hur K, et al. Suicidal thoughts and behavior with antidepressant treatment: reanalysis of the randomized placebo-controlled studies of fluoxetine and venlafaxine. *Arch Gen Psychiatry* 2012;69:580-7.
- 88 Rosenbaum JF, Fava M, Hoog SL, et al. Selective serotonin reuptake inhibitor discontinuation syndrome: a randomised clinical trial. *Biol Psychiatry* 1998;44:77-87.

- 89 Thomas Laughren. <https://www.linkedin.com/pub/thomas-laughren/64/683/1b5> (accessed 2 Dec 2014).
- 90 Whittington CJ, Kendall T, Fonagy P, et al. Selective serotonin reuptake inhibitors in childhood depression: systematic review of published versus unpublished data. *Lancet* 2004;363:1341-5.
- 91 Wohlfarth TD, van Zwieten BJ, Lekkerkerker FJ, et al. Antidepressants use in children and adolescents and the risk of suicide. *Eur Neuropsychopharmacol* 2006;16:79-83.
- 92 Carrillo M, Ricci LA, Coppersmith GA, et al. The effect of increased serotonergic neurotransmission on aggression: a critical meta-analytical review of preclinical studies. *Psychopharmacology (Berl)* 2009;205:349-68.
- 93 Montgomery DB, Roberts A, Green M, et al. Lack of efficacy of fluoxetine in recurrent brief depression and suicidal attempts. *Eur Arch Psychiatry Clin Neurosci* 1994;244:211-5.
- 94 Slater IH, Jones GT, Moore RA. Inhibition of REM sleep by fluoxetine, a specific inhibitor of serotonin uptake. *Neuropharmacology* 1978;17:383-9.
- 95 FDA. Antidepressant use in children, adolescents, and adults. <http://www.fda.gov/drugs/drugsafety/informationbydrugclass/ucm096273.htm> (accessed 22 Apr 2014).
- 96 March J, Silva S, Petrycki S, et al. Fluoxetine, cognitive-behavioral therapy, and their combination for adolescents with depression: Treatment for Adolescents With Depression Study (TADS) randomized controlled trial. *JAMA* 2004;292:807-20.
- 97 Nutt DJ, Goodwin GM, Bhugra D, et al. Attacks on antidepressants: signs of deep-seated stigma? *Lancet Psychiatry* 2014;1:103-4.
- 98 Videbech P. [The debate about antidepressants – do they work and does one become dependent]? *BestPractice Psykiatri/Neurologi* 2014 May;nr 25.
- 99 Kessing L, Nordentoft M, Middelboe T. [Right and wrong about psychotropic drugs]. *Politiken* 2014 Jan 17.
- 100 FDA. Statistical review and evaluation of sertraline (addendum 2). 1991 Jan 31.
- 101 FDA. Review and evaluation of clinical data. Original NDA 20-031. Paroxetine (Aropax) safety review. 1992 Oct 5.
- 102 Graham D. Memorandum about fluoxetine surveillance. Internal FDA document. 1990 Sept 11.
- 103 Healy D. Antidepressants and suicide: briefing paper. 2003 June 20.
- 104 Montgomery SA, Dunner DL, Dunbar GC. Reduction of suicidal thoughts with paroxetine in comparison with reference antidepressants and placebo. *Eur Neuropsychopharmacol* 1995;5:5-13.
- 105 Thomsen S. [Danilo wasn't the first: at least 11 others have obtained compensation after suicide]. *Politiken* 2014 Jan 8.
- 106 Healy D. Professional suicide – the Clancy case. <http://davidhealy.org/professional-suicide-the-clancy-case/> (accessed 22 Dec 2014).
- 107 Missd. <http://missd.co/> (accessed 22 Dec 2014).
- 108 Prescription: suicide? <http://www.prescriptionsuicide.com/downingmedia.html> (accessed 22 Dec 2014).
- 109 CBS evening news. Drug companies paying doctors? 2008 June 27. http://www.cbsnews.com/sections/i_video/main500251.shtml?id=4214033n (accessed 22 Dec 2014).
- 110 Fiddaman B. Mother speaks out [Seroxat Suicide]. 2010 Nov 14. <http://ehealthforum.com/blogs/fiddy/mother-speaks-out-seroxat-suicide-b13161.html> (accessed 22 Dec 2014).
- 111 Fighting childhood depression. 2004 May 28. http://www.pbs.org/newshour/bb/health-jan-june04-depression_5-28/ (accessed 22 Dec 2014).
- 112 WoodyMatters. 1965-2003. Using Woody's life and deaths to save others.

- <http://www.woodymatters.com/> (accessed 22 Dec 2014).
- 113 The creation of the Prozac myth. *The Guardian* 2008 Feb 27.
- 114 Pedersen AG. [Lundbeck's reply to Peter Gøtzsches's open letter about antidepressants and suicide]. *Videnskab dk* 2001 July 8. <http://videnskab.dk/krop-sundhed/lundbecks-svar-pa-peter-gotzsches-abne-brev-om-antidepressiva-og-selvmoerd> (accessed 8 July 2011).
- 115 Miller M, Swanson SA, Azrael D, et al. Antidepressant dose, age, and the risk of deliberate self-harm. *JAMA Intern Med* 2014;174:899-909.
- 116 Bielefeldt AØ, Danborg P, Gøtzsche PC. Systematic review of adverse effects of antidepressants in healthy volunteer studies. *Proceedings of the 23rd Cochrane colloquium*. Vienna, Austria; 2015.
- 117 Klein DF. The flawed basis for FDA post-marketing safety decisions: the example of anti-depressants and children. *Neuropsychopharmacology* 2006; 31:689-99.
- 118 Gøtzsche PC. [Open letter to Lundbeck about antidepressants and suicide]. *Videnskab dk* 2011 July 7. [hap//videnskab//krop-sundhed/abent-brev-til-lundbeck-om-antidepressiva-og-selvmoerd](http://videnskab.dk/krop-sundhed/abent-brev-til-lundbeck-om-antidepressiva-og-selvmoerd) (accessed 8 July 2011).
- 119 Beautrais AL. Suicide and serious suicide attempts in youth: a multiple-group comparison study. *Am J Psychiat* 2003;160:1093-9.
- 120 Michel K. Suicide risk factors: a comparison of suicide attempters with suicide completers. *Br J Psychiatry* 1987;150:78-82.
- 121 Gibbons RD, Brown CH, Hur K, et al. Early evidence on the effects of regulators' suicidality warnings on SSRI prescriptions and suicide in children and adolescents. *Am J Psychiatry* 2007;164:1356-63.
- 122 Whitaker R. The triumph of bad science. 11 July 2012. <http://www.madmamerica.com/2012/07/the-triumph-of-bad-science/> (accessed 12 July 2012).
- 123 Gibbons RD, Weiss DJ, Pilkonis PA, et al. Failure to report financial disclosure information. *JAMA Psychiatry* 2013 Nov 20.
- 124 Simon GE, Savarino J, Operskalski B, et al. Suicide risk during antidepressant treatment. *Am J Psychiatry* 2006;163:41-7.
- 125 Isacson G, Holmgren P, Ahlner J. Selective serotonin reuptake inhibitor antidepressants and the risk of suicide: a controlled forensic database study of 14,857 suicides. *Acta Psychiatr Scand* 2005;111:286-90.
- 126 Isacson G, Rich CL, Jureidini J, et al. In debate: The increased use of antidepressants has contributed to the worldwide reduction in suicide rates. *Br J Psychiatry* 2010;196:429-33.
- 127 Isacson G, Reutfors J, Papadopoulos FC, et al. Antidepressant medication prevents suicide in depression. *Acta Psychiatr Scand* 2010;122:454-60.
- 128 Isacson G, Holmgren P, Druid H, et al. The utilization of antidepressants – a key issue in the prevention of suicide: an analysis of 5281 suicides in Sweden during the period 1992-1994. *Acta Psychiatr Scand* 1997;96:94-100.
- 129 Isacson G, Holmgren A, Osby U, et al. Decrease in suicide among the individuals treated with antidepressants: a controlled study of antidepressants in suicide, Sweden 1995-2005. *Acta Psychiatr Scand* 2009;120:37-44.
- 130 Isacson G, Ahlner J. Antidepressants and the risk of suicide in young persons – prescription trends and toxicological analyses. *Acta Psychiatr Scand* 2014;129:296-302.
- 131 Isacson G. [Depressed youngsters should not be denied medication]. *Svenska Dagbladet* 2013 Nov 3.
- 132 Søndergård L, Kvist K, Andersen PK, et al. Do antidepressants precipitate youth suicide? A nationwide pharmacoepidemiological study. *Eur Child Adolesc Psychiatry* 2006;15:232-40.
- 133 Hjorthøj CR, Madsen T, Agerbo E, et al. Risk of suicide according to level of psychiatric treatment: a

- nationwide nested case-control study. *Soc Psychiatry Psychiatr Epidemiol* 2014;49:1357-65.
- 134 Goldney RD. Suicide and antidepressants: what is the evidence? *Aust N Z J Psychiatry* 2006;40:381-5.
- 135 Zahl PH, De Leo D, Ekeberg Ø, et al. The relationship between sales of SSRI, TCA and suicide rates in the Nordic countries. *BMC Psychiatry* 2010;10:62.
- 136 Reseland S, Le Noury J, Aldred G, et al. National suicide rates 1961-2003: further analysis of Nordic data for suicide, autopsies and ill-defined death rates. *Psychother Psychosom* 2008;77:78-82.
- 137 Gören JL. Antidepressants use in pediatric populations. *Expert Opin Drug Saf* 2008;7:223-5.
- 138 Lu CY, Zhang F, Lakoma MD, et al. Changes in antidepressant use by young people and suicidal behavior after FDA warnings and media coverage: quasiexperimental study. *BMJ* 2014;348:g3596.
- 139 Gøtzsche PC. Study of changes in antidepressant use after FDA warnings is not reliable. *BMJ* 2014;349:g5623.
- 140 Frankenfield DL, Baker SP, Lange WR, et al. Fluoxetine and violent death in Maryland. *Forensic Sci Int* 1994;64:107-17.
- 141 Barber CW, Miller M, Azrael D. Re: Changes in antidepressant use by young people and suicidal behavior after FDA warnings and media coverage: quasiexperimental study. *BMJ* 2014;348:g3596.
- 142 Gusmão R, Quintao S, McDaid D, et al. Antidepressant utilization and suicide in Europe: an ecological multi-national study. *PLoS One* 2013;8:e66455.
- 143 Björkenstam C, Möller J, Ringbäck G, et al. An association between initiation of selective serotonin reuptake inhibitors and suicide – a nationwide register-based case-crossover study. *PLoS One* 2013;8:e73973.
- 144 Moore TJ, Glenmullen J, Furberg CD. Prescription drugs associated with reports of violence towards others. *PLoS One* 2010;5:e15337.
- 145 Lucire Y, Crotty C. Antidepressant-induced akathisia-related homicides associated with diminishing mutations in metabolizing genes of the CYP450 family. *Pharmgenomics Pers Med* 2011;4:65-81.
- 146 Breggin P. *Psychiatric drug withdrawal: a guide for prescribers, therapists, patients, and their families.* New York: Springer Publishing Company; 2013.
- 147 Healy D, Herxheimer A, Menkes DB. Antidepressants and violence: problems at the interface of medicine and law. *PLoS Med* 2006;3:e372.
- 148 Thompson A. Paxil maker held liable in murder/suicide. http://www.baumhedlundlaw.com/media/ssri/Paxil_murder.htm (accessed 24 Jan 2015).
- 149 SSRI Stories: antidepressant nightmares. <http://ssristories.org/> (accessed 21 Nov 2014).
- 150 Christopher Pittman. http://en.wikipedia.org/wiki/Christopher_Pittman (accessed 2 Jan 2015).
- 151 Kim Crespi wants husband David Crespi, who murdered their twin daughters, out of prison. *Huffington Post* 2012 Oct 3.
- 152 The Kurt Danysh Legal Aid Fund. <http://www.kurtdanysh.com/about.htm> (accessed 2 Jan 2015).
- 153 Montejo A, Llorca G, Izquierdo J, et al. Incidence of sexual dysfunction associated with antidepressant agents: a prospective multicenter study of 1022 outpatients. Spanish Working Group for the study of psychotropic-related sexual dysfunction. *J Clin Psychiatry* 2001;62 (suppl 3):10-21.
- 154 Bahrick AS. Persistence of sexual dysfunction side effects after discontinuation of antidepressant medications: emerging evidence. *Open Psychol J* 2008;1:42-50.
- 155 Safarinejad MR. Safety and efficacy of escitalopram in the treatment of premature ejaculation: a double-blind, placebo-controlled, fixed-dose, randomized study. *J Clin Psychopharmacol* 2007;27:444-50.
- 156 Eli Lilly advertisement for Cialis. *Dagens Medicin* 2012 Oct 26:25.

- 157 Gøtzsche PC. Screening pregnant women for depression. 2014 May 5. <http://www.madinamerica.com/2014/05/screening-pregnant-women-depression/> (accessed 9 Dec 2014).
- 158 Pedersen LH, Henriksen TB, Vestergaard M, et al. Selective serotonin reuptake inhibitors in pregnancy and congenital malformations: population based cohort study. *BMJ* 2009;339:b3569.
- 159 Huybrechts KF, Palmsten K, Avorn J, et al. Antidepressant use in pregnancy and the risk of cardiac defects. *N Engl J Med* 2014;370:2397-407.
- 160 Expert Report of Nicholas Jewell, PhD. Exhibit A: Regarding a causal association between maternal exposure to Zoloft during pregnancy and cardiovascular birth defects. 2014 Oct 1. Courtesy of Shawn P. Fox, Tracey Law Firm, Houston, Texas.
- 161 Huybrechts KF, Sanghani RS, Avorn J, et al. Preterm birth and antidepressant medication use during pregnancy: a systematic review and meta-analysis. *PLoS ONE* 2014;9:e92778.
- 162 Medawar C, Hardon A. Medicines out of control? Antidepressants and the conspiracy of goodwill. Netherlands: Aksant Academic Publishers; 2004.
- 163 Riddle MA, King RA, Hardin MT, et al. Behavioral side effects of fluoxetine in children and adolescents. *J Child Adolesc Psychopharmacol* 1990/1991;1:193-8.
- 164 Furukawa TA. All clinical trials must be reported in detail and made publicly available. *Lancet* 2004;329:626.
- 165 Seroxat/Paxil Adolescent Depression. Position piece on the phase III clinical studies. GlaxoSmithKline document 1998 Oct.
- 166 Keller MB, Ryan ND, Strober M, et al. Efficacy of paroxetine in the treatment of adolescent major depression: a randomized, controlled trial. *J Am Acad Child Adolesc Psychiatry* 2001;40:762-72.
- 167 Jureidini JN, McHenry LB, Mansfield PR. Clinical trials and drug promotion: selective reporting of study 329. *Int J Risk Safety Med* 2008;20:73-81.
- 168 Jureidini JN, McHenry LB. Conflicted medical journals and the failure of trust. *Accountability in Research* 2001;18:45-54.
- 169 More fraud from drug giant GlaxoSmithKline companies – court documents show. *Child Health Safety* 2010 Dec 1.
- 170 Moynihan R, Cassels A. Selling sickness: how the world's biggest pharmaceutical companies are turning us all into patients. New York: Nation Books; 2005.
- 171 Doshi P. Putting GlaxoSmithKline to the test over paroxetine. *BMJ* 2013;347:f6754.
- 172 Pigott HE, Leventhal AM, Alter GS, et al. Efficacy and effectiveness of antidepressants: current status of research. *Psychother Psychosom* 2010;79:267-79.
- 173 Whitaker R. The STAR*D scandal: a new paper sums it all up. 2010 Aug 27. <http://www.madinamerica.com/2010/08/%EF%BB%BFthe-stard-scandal-a-new-paper-sums-it-all-up/> (accessed 13 Jan 2015).
- 174 Trivedi MH, Rush AJ, Wisniewski SR, et al. Evaluation of outcomes with citalopram for depression using measurement-based care in STAR*D: implications for clinical practice. *Am J Psychiatry* 2006;163:28-40.
- 175 Pigott E. An introduction: the story of bias in the STAR*D trial and more. <http://www.madinamerica.com/2011/03/%EF%BB%BFan-introduction-the-story-of-bias-in-the-stard-trial-more/> (accessed 18 Jan 2015).
- 176 Pigott E. STAR*D: Adding fiction to fiction. 2011 April 10. <http://www.madinamerica.com/2011/04/%EF%BB%BFstard-adding-fiction-to-fiction/> (accessed 18 Jan 2015).

- 177 Fava GA. Do antidepressant and anti-anxiety drugs increase chronicity in affective disorders? *Psychother Psychosom* 1994;61:125-31.
- 178 El-Mallakh RS, Gao Y, Roberts JR. Tardive dysphoria: the role of long term antidepressant use in inducing chronic depression. *Med Hypotheses* 2011;76:769-73.
- 179 Kirk SA, Gomory T, Cohen D. *Mad science: psychiatric coercion, diagnosis and drugs*. New Brunswick: Transaction Publishers; 2013.
- 180 Bribery alleged in largest antidepressant study ever conducted. 2012 Feb 2. <http://www.madinamerica.com/2012/02/bribery-alleged-in-largest-antidepressant-study-ever-conducted/> (accessed 18 Jan 2015).

4

Anxiety

We all get anxious from time to time, which means a huge market potential for drugs. Because of ineffective blinding in trials (see Chapter 3), we would expect almost anything to “work” for anxiety. We would also expect psychological interventions to work, but for better reasons. It is obviously a good idea to teach people how to cope with their anxiety.

In terms of the balance between benefits and harms, psychotherapy is far better than drugs (see Chapter 10), but drugs are much more commonly used. So much that our citizens are drugged to about the same extent today as 50 years ago, as a large decline in the use of benzodiazepines has been compensated by a similar increase in the use of SSRIs,¹ which are used for many of the same conditions. Psychiatrists now say, pretty conveniently, that much of what they previously called anxiety – when it was still okay to use benzodiazepines – in reality was depression, so that they can now use SSRIs for the same patients.

It is true that there is much overlap in symptoms between anxiety and depression, but the change in treatment of anxiety disorders has happened despite the fact that a benzodiazepine is likely the better option. A systematic review from 2013 found that the trials were generally of poor quality, but benzodiazepines were more effective and better tolerated than tricyclic antidepressants, and they had similar or better effect than SSRIs, with fewer harmful effects.² The main reason for abandoning benzodiazepines is that there is very little money to be earned from drugs that ran out of patent ages ago.

Another systematic review, of 48 placebo controlled trials in patients with generalised anxiety disorder, found similar effects of benzodiazepines (effect size 0.32), azapirones (0.30) and venlafaxine (0.33).³ For depression, the effect sizes were 0.28 for benzodiazepines and 0.22 for azapirones (there were no data for venlafaxine, although this drug is an antidepressant). The effect decreased as the trials got bigger, but the results were reasonably robust.

The authors wondered why azapirones worked for depression, as these drugs are not usually considered effective for depression, but that’s not surprising. A drug that reduces anxiety will also be expected to reduce anxiety-related

depression, as there is less to be depressed about when your anxiety is gone. In addition, some of the items on Hamilton's depression scale are about anxiety, and the relatively small effect of the drugs could be explained by unblinding bias (see Chapter 3). According to the published trials, which exaggerate the effect in comparison with the unpublished trials,⁴ the effect size is 0.37 for the newer antidepressant drugs for depression,⁴ which is about the same as their effect for generalised anxiety disorder,³ and this effect can be explained by unblinding bias. Whether the patients are helped by the drugs to get on with their lives is totally obscure, not only because of this bias, but also because we are only told about the effect on scales with uncertain clinical relevance.

A 2008 systematic review found only one trial comparing benzodiazepines with an SSRI whereas there were 22 comparisons with older antidepressants. Again, the antidepressants were not better than benzodiazepines.⁵

An additional problem with these trials is that some of them have included patients on benzodiazepines, and when they are switched to placebo, some of them may go through the horrors of benzodiazepine withdrawal, which can be extremely anxiety-provoking.^{6,7} Such trials are fatally wounded from the start, but the FDA approved alprazolam (Xanax) – one of the worst of all benzodiazepines⁷ – for panic disorder even though a very large trial of 526 patients was carried out this way, with only a one-week run-in period without drugs.^{8,9} After the eight-week double-blind phase was over, the medication was tapered over four weeks, but many patients on alprazolam tolerated this so poorly that they were likely on their way to a lifetime addiction, and the patients had more panic attacks five weeks after having stopped alprazolam than when they entered the trial, whereas those on placebo continued to fare well.

This horrible drug became the fifth most prescribed drug in the United States, which is remarkable, as it *causes* panic in long-term use and has led to many suicides and homicides.⁷

I shall mention a few other meta-analyses. A Cochrane review of anxiety disorders in children and adolescents had included 21 trials of antidepressants and two of benzodiazepines, but, as I believe none of these drugs should be used in children, I find the reported short-term results uninteresting.¹⁰

I am also sceptical of a Cochrane review that assessed the effects of adding an extra drug (or placebo) when anxious patients had not responded adequately to first-line drug therapies.⁶ Twenty of the 28 trials were in patients with obsessive compulsive disorder (OCD), but the trials were very heterogeneous, and much of the data were based on antipsychotics. The authors didn't make firm conclusions, and I agree: antipsychotics are far too dangerous to be used for OCD (see Chapter

6). A similar Cochrane review wasn't limited to patients who hadn't responded to the antidepressant;¹² no positive recommendations were made.

It's too much for me that psychiatrists are willing to give people with OCD both antidepressants and antipsychotics. These patients should get psychotherapy. OCD isn't deadly, but the drug cocktail is.

A Cochrane review of social phobia included 37 trials,¹³ but I consider it invalid. Some of the effects reported were surprisingly large, around 0.6, given the moderate effect sizes of around 0.3 usually reported for SSRIs, and one of the problems was that all the scales appeared to have been rated by clinicians and not the patients, which creates a large bias (see Chapter 3). Further, the trials were of very poor quality, and the effect decreased so dramatically with the number of patients in the trial that any meta-analysis of these data would be grossly unreliable. The authors reported a relative risk of nonresponse of 0.64 (95% CI 0.57 to 0.73) on the Global Impression Scale, but they also showed in a figure that the largest trials found an effect close to zero! The review also reported an effect in relapse prevention studies, but such trials are highly unreliable because abstinence symptoms are introduced in the placebo group when the patients come off their drug cold turkey (see Chapter 11).

Although psychotherapy is highly effective for social phobia (see Chapter 10), patients haven't been spared all sorts of trials of dangerous drugs, including anticonvulsants and antipsychotics.¹³ Luckily for the patients, a Cochrane review of antipsychotics wasn't positive, although the review was very large (11 trials and 4,144 participants).¹⁴ Seven trials of quetiapine found the drug to be better than placebo for generalised anxiety disorder, but more patients dropped out due to adverse events, and more patients gained weight and suffered from sedation and extrapyramidal (muscular) side effects. There were two small studies of olanzapine and two studies of add-on treatment with risperidone, and they didn't find an effect.

Sleeping pills

One of the classic uses of benzodiazepines is as sleeping pills, but this usage also does more harm than good. The trials are biased, and a meta-analysis found that after adjustment for this, patients at least 60 years of age with insomnia slept 15 minutes longer on benzodiazepines or similar drugs than on placebo.¹⁵ Adverse cognitive events were five times more common, adverse psychomotor events three times more common, and daytime fatigue four times more common than if the

patients received placebo. After a few weeks, the pills don't work any longer and all that is left are the harmful effects.

Patients who took such drugs had a higher risk of falls – which cause many deaths because of hip fractures – and motor vehicle crashes. A study showed that benzodiazepines doubled the risk of injurious falls in people at least 80 years of age; these falls cause almost 1,800 deaths every year in France.¹⁶ In another large cohort, benzodiazepines or Z drugs doubled the death rate.¹⁷

The measured short-term effects in these trials are likely exaggerated. It is highly subjective to record sleeping times and sleeping quality, and the side effects of the drugs must have compromised the blinding, e.g. in a trial of alprazolam versus placebo for panic attacks, the blinding was broken for *all* patients.¹⁸

Why would anyone take such dangerous drugs instead of reading a book until falling asleep naturally? Psychotherapy is also a better option than pills (see Chapter 10).

References

- 1 Nielsen M, Gøtzsche P. An analysis of psychotropic drug sales. Increasing sales of selective serotonin reuptake inhibitors are closely related to number of products. *Int J Risk Saf Med* 2011;23:125-32.
- 2 Offidani E, Guidi J, Tomba E, et al. Efficacy and tolerability of benzodiazepines versus antidepressants in anxiety disorders: a systematic review and meta-analysis. *Psychother Psychosom* 2013;82:355-62.
- 3 Mitte K, Noack P, Steil R, et al. A meta-analytic review of the efficacy of drug treatment in generalized anxiety disorder. *J Clin Psychopharmacol* 2005;25:141-50.
- 4 Turner EH, Matthews AM, Linardatos E, et al. Selective publication of antidepressant trials and its influence on apparent efficacy. *N Engl J Med* 2008;358:252-60.
- 5 Berney P, Halperin D, Tango R, et al. A major change of prescribing pattern in absence of adequate evidence: benzodiazepines versus newer antidepressants in anxiety disorders. *Psychopharmacol Bull* 2008;41:39-47.
- 6 Whitaker R. *Anatomy of an Epidemic*. New York: Broadway Paperbacks; 2010.
- 7 Breggin P. *Psychiatric drug withdrawal: a guide for prescribers, therapists, patients, and their families*. New York: Springer Publishing Company; 2013.
- 8 Ballenger JC, Burrows GD, DuPont RL Jr, et al. Alprazolam in panic disorder and agoraphobia: results from a multicenter trial. I. Efficacy in short-term treatment. *Arch Gen Psychiatry* 1988;45:413-22.
- 9 Pecknold JC, Swinson RP, Kuch K, et al. Alprazolam in panic disorder and agoraphobia: results from a multicenter trial. III. Discontinuation effects. *Arch Gen Psychiatry* 1988;45:429-36.
- 10 Ipser JC, Stein DJ, Hawkrige S, et al. Pharmacotherapy for anxiety disorders in children and adolescents. *Cochrane Database Syst Rev* 2009;3:CD005170.
- 11 Ipser JC, Carey P, Dhansay Y, et al. Pharmacotherapy augmentation strategies in treatment-resistant anxiety disorders. *Cochrane Database Syst Rev* 2006;4: CD005473.
- 12 Komossa K, Depping AM, Meyer M, et al. Second-generation antipsychotics for obsessive compulsive disorder. *Cochrane Database Syst Rev* 2010;12:CD008141.

- 13 Stein DJ, Ipser JC, van Balkom AJ. Pharmacotherapy for social anxiety disorder. *Cochrane Database Syst Rev* 2000;4:CD001206.
- 14 Depping AM, Komossa K, Kissling W, et al. Second-generation antipsychotics for anxiety disorders. *Cochrane Database Syst Rev* 2010;12:CD008120.
- 15 Glass J, Lanctôt KL, Herrmann N, et al. Sedative hypnotics in older people with insomnia: meta-analysis of risks and benefits. *BMJ* 2005;331:1169-73.
- 16 Pariente A, Dartigues J-F, Benichou J, et al. Benzodiazepines and injurious falls in community dwelling elders. *Drugs Aging* 2008;25:61-70.
- 17 Weich S, Pearce HL, Croft P, et al. Effect of anxiolytic and hypnotic drug prescriptions on mortality hazards: retrospective cohort study. *BMJ* 2014;348:g1996.
- 18 Fisher S, Greenberg RG. How sound is the double-blind design for evaluating psychotropic drugs? *J Nerv Ment Dis* 1993;181:345-50.

ADHD

Childhood ADHD

This diagnosis was invented for DSM-III in 1980 but what is it? At a 1998 consensus conference, the chairman asked a leading ADHD expert in America, Mark Vonnegut, but although he talked for two to three minutes, Vonnegut couldn't explain what ADHD is:¹

“They cannot sit still ... they are difficult and they aggravate their parents ... the diagnosis is a mess but there is, there is, uhm, we all have a belief that we are dealing with a very serious core problem and that we have a diagnosis that allows us to communicate and gives us research, uhm, generates, uhm, sort of ideas for research, and I think, you know, we, uhm, I, I do, I think, part of the problem is that the profession keeps changing the diagnoses.” Vonnegut's *we-are-all-in-the-dark-together* utterings taught us something, namely that the psychiatrists don't know what they are doing.

Vonnegut also explained that a teacher might say that a kid was two standard deviations different from the other kids in the classroom. But hold on; 5% of us are by definition beyond two standard deviations from the average of everything that follows the normal distribution, but this doesn't mean we are sick. If we take a group of normal people and measure their height, 5% are beyond two standard deviations from the average height, but we don't invent some “disorder” for those 5% who are small or tall.

NIMH also gets into trouble when it uses 15 pages to tell us what ADHD is.² On the first page, we are told that ADHD is one of the most common childhood brain disorders and that imaging studies have shown abnormalities in the brain. But this is totally wrong. ADHD is not a “brain disorder” and the brains of these children are not different from the brains of other children (see Chapter 11). The first page also says:

“Inattention, hyperactivity, and impulsivity are the key behaviors of ADHD. It is normal for all children to be inattentive, hyperactive, or impulsive sometimes, but for children with ADHD, these behaviors are

more severe and occur more often. To be diagnosed with the disorder, a child must have symptoms for 6 or more months and to a degree that is greater than other children of the same age.”

This is about as weak as it can get and it certainly doesn't justify calling ADHD a brain disorder. The children are normal variants of normal behaviour, and we cannot all have average behaviour or average height. Many children qualify for the diagnosis simply because they are talented and therefore bored and cannot sit still in poorly disciplined classrooms, or because they have emotional problems generated at home.

Since we are talking about degrees of development and not a brain disease, we would expect more of those children born in December to have an ADHD diagnosis and be in drug treatment than those born in January in the same class, as they have had 11 fewer months to develop their brains. This is exactly the case. A Canadian study of one million school children showed that the prevalence of children in treatment increased pretty much linearly over the months from January to December,³ and 50% more of those born in December were in drug treatment. This study shows that if we approach the children with a little patience that allows them to grow up and mature, fewer would receive drugs.

However, the diagnosis arises primarily from teacher complaints and parents are often told that their kid cannot come back to school unless he or she is on an ADHD drug. A general practitioner told me that a schoolmistress had sent most of her pupils for examination on suspicion of ADHD; clearly, she was the problem, not the kids. As soon as the kids are branded with ADHD, it relieves everyone of any responsibility or incentive to redress the mess they have created. We have decided as a society that it is too laborious or expensive to modify the kid's environment, so we modify the kid's brain instead. This is cruel.

NIMH says that, “With treatment, most people with ADHD can be successful in school and lead productive lives,” but NIMH has proved itself in a very large trial that the statement is wrong (see below).

Doctors can expect to get complaints if they *don't* diagnose ADHD, particularly if they decide not to use any of the silly checklists that abound for the diagnosis. My stepfather was a school psychologist and his attitude was that we shouldn't put diagnostic labels on kids. I wish it were so, but unfortunately ADHD has become yet another false disease epidemic.

Names create what they describe. Parents or school teachers who have experienced that a boy is troublesome and disturbing may feel the ADHD diagnosis gives them an explanation. It can also lead to greater acceptance in the

boy's surroundings of his behaviour, as he cannot help it because he has this particular "disorder." However, it is circular evidence to argue this way. We have merely given the boy's behaviour a name, ADHD. With the same violation of the rules of logic we might say that Brian behaves badly, and because his name is Brian he behaves badly. ADHD is *not* an explanation, it is only a name given to a clustering of symptoms.

It is ironic that "attention deficit" is part of the name, as it can be an attention deficit in the children's social surroundings that is the real problem. If these children got more attention, there would be fewer diagnoses, which is why Peter Breggin has called it DADD: Dad Attention Deficit Disorder. As early as in the 1990s, a quarter of the children in an elementary school in Iowa were on ADHD drugs and in California the diagnosis rates increased sharply as school funding declined.⁴

Some parents contribute to the epidemic by seeking out a diagnosis for their children to obtain social benefits. Institutions such as kindergartens also contribute by putting pressure on parents to accept dubious diagnoses to obtain additional funding.

It's a tragedy and a fraud to take entirely normal children and make patients out of them while telling their parents that the children suffer from a chemical imbalance. But unfortunately many clinicians find it easier to tell parents their child has a brain disorder than to suggest parenting changes.

In 2011, an enterprise – evidently working on behalf of an anonymous drug company – sent a most bizarre invitation to Danish specialists treating children and adolescents for ADHD.⁴ The doctors would be divided into two groups for an exercise called Wargames where they should defend their product (two different ADHD medicines) with arguments and a visual presentation. Their efforts would be filmed and the company's anonymous client might be watching from another room. This Orwellian "Big brother is watching you" exercise was illegal. Danish doctors are not allowed to help companies market their products.

Also in 2011, my wife and I got very angry when our youngest daughter told us that a big ADHD bus had visited her school and distributed brochures to "raise awareness of the ADHD disorder in children." It wasn't about raising awareness but about pushing pills. The ADHD bus is owned by the Danish ADHD Association, which receives financial support from companies selling ADHD pills and other psychotropic drugs, and four of the pill brochures were produced by a company that sells methylphenidate (Ritalin), the most commonly used ADHD drug.

One of the brochures called "Girls and ADHD" had a section about treatment,

but only drugs were mentioned. A journalist asked the president of the ADHD Association why the association distributes materials from a pharmaceutical company to schoolchildren, but despite a promise to call back, this didn't happen. A press release about the appointment of the association's new director earlier the same year stated that the association "needed a director who is commercially oriented" and who should "focus on creating partnerships with private companies." The principles for such collaboration were listed on the association's homepage and the partnerships were about establishing an "advantageous and binding, often lengthy business relationship between the ADHD association and a business, built on shared expectations about input and output." Sponsorships involved a "commercial agreement between the ADHD Association and a company, with an expected return, often in relation to marketing or social responsibility perspectives." Total corruption to the detriment of our children.

The media also distort the issues. Two ADHD studies showed obvious discrepancies between the results and the conclusions, which were that ADHD patients lack dopamine and that stimulants improve long-term school outcomes. But only one of 61 media articles adequately described the results and thus questioned the conclusion.⁵ The erroneous conclusion about lack of dopamine as the cause of ADHD was uncritically propagated also in subsequent scientific papers.⁵

Similarly, a survey of the 10 most cited ADHD papers in the media showed that the media paid virtually no attention when the findings were later shown to be false.⁶ Only one newspaper article of 57 describing subsequent studies mentioned that the previous finding had been refuted.

Figure 5.1. Test for Adult ADHD.

Adult ADHD Self-Report Scale (ASRS-v1.1) Symptom Checklist

Patient Name	Today's Date				
Please answer the questions below, rating yourself on each of the criteria shown using the scale on the right side of the page. As you answer each question, place an X in the box that best describes how you have felt and conducted yourself over the past 6 months. Please give this completed checklist to your healthcare professional to discuss during today's appointment.					
	Never	Rarely	Sometimes	Often	Very Often
1. How often do you have trouble wrapping up the final details of a project, once the challenging parts have been done?					
2. How often do you have difficulty getting things in order when you have to do a task that requires organization?					
3. How often do you have problems remembering appointments or obligations?					
4. When you have a task that requires a lot of thought, how often do you avoid or delay getting started?					
5. How often do you fidget or squirm with your hands or feet when you have to sit down for a long time?					
6. How often do you feel overly active and compelled to do things, like you were driven by a motor?					
Part A					
7. How often do you make careless mistakes when you have to work on a boring or difficult project?					
8. How often do you have difficulty keeping your attention when you are doing boring or repetitive work?					
9. How often do you have difficulty concentrating on what people say to you, even when they are speaking to you directly?					
10. How often do you misplace or have difficulty finding things at home or at work?					
11. How often are you distracted by activity or noise around you?					
12. How often do you leave your seat in meetings or other situations in which you are expected to remain seated?					
13. How often do you feel restless or fidgety?					
14. How often do you have difficulty unwinding and relaxing when you have time to yourself?					
15. How often do you find yourself talking too much when you are in social situations?					
16. When you're in a conversation, how often do you find yourself finishing the sentences of the people you are talking to, before they can finish them themselves?					
17. How often do you have difficulty waiting your turn in situations when turn taking is required?					
18. How often do you interrupt others when they are busy?					
Part B					

Adult ADHD

A journalist from the Danish Broadcasting Corporation who had been diagnosed with ADHD in childhood tried the screening test recommended by the World Health Organization on eight of his colleagues and found that seven of them had the “disease.”

The test is hopeless. Two members of my close family tried it and got the diagnosis, one with a full house, as she consistently chose the gray boxes in both the A and B test (see Figure 5.1). Try it yourself.⁷ Successful, pioneering and energetic people, who don't like wasting their time on unproductive meetings listening to unfocused people who talk endlessly, might end up getting the diagnosis. All that is required in order to have “symptoms highly consistent with ADHD in adults” is to have four marks in the gray boxes for the first six questions in part A of the test.

If a dynamic person *sometimes* has trouble wrapping up the final details of a project, once the challenging parts have been done; and *sometimes* has difficulty getting things in order when he or she has to do a task that requires organization; and *sometimes* has problems remembering appointments or obligations, then this is already three of the four points needed for the diagnosis. Don't many overworked people fit these descriptions? Some of the most talented and wonderful people I know are like that and they can get the last point if they *often* avoid or delay getting started when they have a task that requires a lot of thought; or if they *often* fidget or squirm with their hands or feet when they have to sit down for a long time; or if they *often* feel overly active and compelled to do things, as if driven by a motor.

In 2004, the New York University School of Medicine Adult ADHD programme offered a free screening day for adults at a hotel, announcing that, “Despite wide recognition as a children's disorder, ADHD ... affects millions of adults who are undiagnosed and untreated.”⁸ Yes, what a catastrophe that a few people are still wandering around freely who are *not* medicated for a conduct disorder! Two years later, an article reported that *85% of those who attended screened positive for ADHD.*⁹ The researchers surveyed 51 of these people who had all been given a list of doctors to contact to help treat their so-called illness, but 27 of them admitted they never followed through. The director of the programme, Dr. Lenard Adler (yes, the surname *was* Adler), interpreted the results this way: “This data shows that people with ADHD need help to get help.” The Goodness Industry has no limits. I assume that people who won't accept the

help to get help will then be exposed to do-gooders who want to help them understand that they need help to get help so that they can help themselves. Help!

Of course there are caveats in the adult ADHD checklist that advises that further investigation is warranted and that we should consider work, school, social and family settings. However, this is not how clinical practice usually is. These additional investigations take time and may never be carried out, and even if they were, there is a high risk that leading questions will yield results that “confirm” the diagnosis. We are told, for example, to “look for evidence of early-appearing and long-standing problems with attention or self-control,” also in childhood, which could be many years back. Such explorations will suffer from recall bias and confirmatory bias tending to “validate” the provisional diagnosis, somewhat like the scandals related to alleged sexual abuse in childhood that turned out to be nothing other than false memories caused by the interviewer. Here is an account of what this can lead to, which I received from a psychiatrist:

I sit with the files from a woman whom I and my staff nurse assess as immature and disturbed. The patient was referred from a general practitioner for a possible ADHD diagnosis. A psychologist has described the patient as girlish, with emotional and identity-related problems, regressive social behaviour, and an unbalanced relationship with her mother who seems cold and irritable. The infamous ADHD tests were carried out, but amazingly, she scored very low on both inattention and hyperactivity as a child (only 1 of 9 symptoms was positive). When the psychologist told her that an ADHD diagnosis cannot be made, she and her boyfriend reacted very negatively, and the patient cried, very discouraged and demoralized, in a somewhat dramatizing and theatrical way, and proclaimed she had nothing left to live for. Then the psychologist wrote in her files that “it has only been concluded that it is doubtful whether the diagnosis ADHD can be made ... and I will examine whether in this case one can make an exception and pay more attention to symptoms in childhood (although this involves a risk of a distortion, as there may now, if possible, be even stronger forces at play than before to get the diagnosis).” This is discussed with a psychiatrist and it is decided to give the patient the benefit of the doubt and do a new test. Now 6 of 9 symptoms are considered positive for both hyperactivity and inattention in childhood, and suddenly the overall result suggests that the patient meets the criteria for the diagnosis of ADHD in childhood. And then comes the finale: “This concludes the diagnostic process and the patient will be transferred for drug treatment of the attention deficit by the ADHD team’s psychiatrist”.

This surrealistic tragedy could appropriately be called: How to weep to get a false diagnosis. In the psychiatric supermarket, there are diagnoses on all shelves with no expiry dates. It’s better not to visit and better to resist letting tears become

decisive.

The checklist for adult ADHD is only five pages long, but its authors, two professors from Harvard Medical School and one from New York University Medical School, tell us that adult ADHD can have “a significant impact on the relationships, careers, and even the personal safety of your patients who may suffer from it.” They provide four references related to this statement, which are all irrelevant. One is to the DSM-IV, another is to a textbook, the third is to a review that contradicts what the professors say, as it notes that few studies have examined the status of core symptoms beyond 14 months of treatment and recommends longer-term studies.¹⁰ The fourth paper is an interview study of 84 people with adult ADHD.¹¹ This is my usual experience with psychiatric research. I get disappointed almost every time I look up references to statements I find interesting. The quoted papers don’t support what is being claimed. It is as if the veracity and relevance of the references don’t matter the slightest bit; it’s only window dressing.

What isn’t said directly – but implicitly understood – is that the idea of diagnosing people with adult ADHD is to treat them with drugs: “Because this disorder is often misunderstood, many people who have it do not receive appropriate treatment and, as a result, may never reach their full potential.” The professors’ statement smells like a drug ad, and it has never been shown either that drug treatment has any impact on what they call “the relationships, careers, and even the personal safety of your patients who may suffer from it.”

The questions in the checklist are consistent with DSM-IV criteria and are said to address the manifestations of ADHD in adults. Yet again, the psychiatrists have blown life into a social construct that is nothing but a variation of normal behaviour and have given this construct a name, as if it existed in nature and could attack people.

ADHD drugs

Drugs used for ADHD are amphetamine derivatives, or have effects like amphetamine and cocaine. They are stimulants and can cause mania, other psychoses, brain damage and death.^{12, 13} Use of amphetamines may lead to drug dependence and people treated with methylphenidate are much more likely to abuse cocaine in young adulthood compared to those diagnosed with ADHD without drug exposure.¹⁴ This is not surprising, as stimulants are known to cause alterations in the reward centres of the brain.

The most used drug is methylphenidate (Ritalin); another is atomoxetine (Strattera), which is a noradrenaline reuptake inhibitor. Eli Lilly failed to get it approved for depression but sells it as “non-stimulant” treatment for ADHD, which is a lie. It causes dangerously stimulating symptoms in many children and the package insert has a black box warning.¹⁴ It warns that suicidal ideation was seen in 5 of 1,357 patients receiving atomoxetine versus none of 851 patients receiving placebo. Many children have developed suicidal and homicidal impulses on atomoxetine, which can also cause liver failure.¹⁵

As always, it is far worse in clinical practice than companies have reported in their trials. In a consecutive cohort of 153 children treated with atomoxetine, 51 children (33%) developed extreme irritability, aggression, mania, or hypomania.¹⁶

Which parents would want their child to take atomoxetine, methylphenidate, or any other ADHD drug, if they were honestly informed about the lack of any long-term benefits and all the serious harms?

ADHD drugs for children

Psychiatry has ... become pharma’s goldmine, with a simple business plan. Seek a small group of specialists from a prestigious institution. Pharma becomes the professional kingmaker, funding research for these specialists. Research always reports underdiagnosis and undertreatment, never the opposite. Control all data and make the study duration short. Use the media, plant news stories, and bankroll patient support groups. Pay your specialists large advisory fees. Lobby government. Get your pharma sponsored specialists to advise the government. So now the world view is dominated by a tiny group of specialists with vested interests. Use celebrity endorsements to sprinkle on the marketing magic of emotion. Expand the market by promoting online questionnaires that loosen the diagnostic criteria further. Make the illegitimate legitimate.

DES SPENCE, GENERAL PRACTITIONER, GLASGOW¹⁷

Spence mentions that a small Harvard group of world specialists admitted undisclosed personal payments from drug companies totalling \$4.2 million. A review of 43 drug trials in ADHD, of which 39 were sponsored by the companies, confirms Spence’s kingmaker tale. Very few drug reactions were called serious, although many children dropped out of the studies because of serious adverse drug reactions.¹⁸ Moreover, adverse drug reactions were only reported if the incidence was above 2% or 5%. Many studies were conducted by the same core group of

authors and we worked out how much inbreeding there was: 21 papers (49%) came from Harvard Medical School or Massachusetts General Hospital, both in Boston, and Joseph Biederman was the great fertilizer, co-authoring no less than 13 of the papers (30%).

Many of the studies are also rigged, either by dropping all children who improve on placebo before the trial starts, or the opposite, studying only children who have tolerated the drug before they are randomised to drug or placebo,¹⁹ or both.²⁰

ADHD drugs are popular with school teachers, as they make their work easier. But it is bad medicine to drug children in order to make them less disturbing and the children pervasively dislike stimulants,²⁰ which is easy to understand if one reads the long list of harmful effects in the package inserts. To outweigh the harms, benefits would therefore need to be substantial, but this is not the case.

A 2013 systematic review included 43 trials, of which 37 assessed the effect of methylphenidate compared with placebo.²¹ The review's results are obscure, however. They were only reported as percentages on an undefined scale without standard deviations. Furthermore, the quality of the trials was poor. Two-thirds couldn't be included in the meta-analysis, and most had problems with missing data and didn't report an adequate randomisation method. There was a huge scope for reporting bias, and there must have been unblinding bias, as the drugs have conspicuous side effects. Finally, an unreported number of trials were biased by design, as they had only included participants known to have responded to stimulants. I wouldn't dare conclude anything based on these drug company trials.

A systematic review from 2002 done by people from the McMaster University in Canada with a senior author whom I trust, was also pretty negative.²² It included 14 trials of at least 12 weeks' duration but only five were of adequate methodological quality and no less than 25 different outcomes and 26 different rating scales had been used. Stimulants reduced ADHD symptoms but didn't improve academic performance. The trials didn't address outcomes that are important. These authors also assessed other systematic reviews and found that they had extensive flaws due to poor description of the methods used to find, select, assess, and synthesise the information.

The FDA approved these drugs with absurdly little documentation.²³ Only 32 clinical trials were conducted for the approval of 20 ADHD drugs; the median number of participants studied per drug was 75; and the median trial length was four weeks.

The adverse effects of stimulants include tics and twitches and other behaviours consistent with obsessive compulsive symptoms, which can be quite

common.^{24, 25} Stimulants also reduce overall spontaneous mental and behavioural activity, including social interest, which causes apathy or indifference, and many children – in some studies more than half – develop depression and compulsive, meaningless behaviours.^{12, 14} Numerous animal studies have confirmed this.¹⁴ The compulsive behaviour is often misinterpreted as an improvement at school, although the child may obsessively just copy everything shown on the board without learning anything. Some children develop mania or other psychoses.^{12, 13}

When these adverse drug effects are mistaken for a worsening of the “disease,” the children are often given additional diagnoses, e.g. depression, OCD or bipolar, and additional drugs, leading to chronicity.¹⁴ But as explained in Chapter 2,

*It is bad medicine to come up with additional diagnoses when a person is under influence of a brain-active chemical, as the symptoms are most likely drug-induced.*¹⁴

There seems to be no long-term benefits from ADHD drugs, only harms. I have heard psychiatrists argue that the drugs improve occupational outcomes and reduce the risk of committing crime, but there are no reliable data in support of this wishful thinking. According to Whitaker, the American Psychiatric Association’s *Textbook of Psychiatry* stated already in 1994 that, “Stimulants do not produce lasting improvements in aggressivity, conduct disorder, criminality, education achievement, job functioning, marital relationships, or long-term adjustment.”²⁰

In 1999, NIMH published 14-month results of the first longterm trial, the Multimodal Treatment study of ADHD (MAT), in which 579 children were randomized to methylphenidate, behavioural therapy, both, or routine community care.²⁶ Many scales and outcomes were used, with no less than 19 primary outcomes, but the only differences between drug and behavioural therapy were that the children were less hyperactive or impulsive and paid more attention when on drug. Combined treatment was no better than drug alone for core ADHD symptoms.

What I find most interesting is that the improvement in symptoms over time in all four groups was sometimes considerably larger than the differences between the treatments, e.g. for inattention and social skills.

The patient sample was probably biased in favour of drugs, as patients who had previously been on an ADHD drug were excluded if they had not tolerated the drug. The authors considered ADHD a chronic disorder and advocated ongoing

treatment, which agreed poorly with the improvement in all four groups, and with the Canadian study of schoolchildren where the risk of being on the drug depended on the month the child was born (see above).

It is difficult to know what to make of all these scores in the MAT trial. Did the reported differences translate into anything important for the children? They actually didn't, as judged by the long-term results, which the psychiatrists weren't eager to publish. It took another eight years before the three-year results were published!²⁷ The investigators now revealed their financial conflicts of interest, which were astonishing. Sixteen authors listed a total of 214 drug companies, or 13 per author. These relationships were mostly described as research funding, "unrestricted grants" (a euphemism for corruption),⁴ consulting and being on speakers' bureaus and advisory boards. Not exactly a bunch of people we would entrust to give us an unbiased view of the value of methylphenidate.

The four treatment groups didn't differ significantly for any of the numerous ADHD outcomes. The investigators partly ascribed this to the fact that many children in the two non-drug groups took drugs, so the treatment contrast was low. But they also mentioned that the results were possibly due to an age-related decline in ADHD symptoms.

A companion paper is close to impossible to interpret, as the findings were drowned in advanced statistics, but the limited relevant data showed a lower rate of substance abuse in the behaviour therapy group than in the drug group after three years.²⁸ Methylphenidate didn't protect against delinquency and substance abuse; if anything, it caused them.

The results after six and eight years were also discouraging.²⁹ The treatment groups didn't differ significantly for grades earned in school, arrests, psychiatric hospitalizations, or other clinically relevant outcomes. Medication use decreased by 62% after the 14-month controlled trial, but adjusting for this didn't change the results.

These follow-up papers are also difficult to grasp, as they confuse readers with unnecessarily complicated statistics. I shall take it all down to earth by quoting one of the investigators:³⁰ "I think that we exaggerated the beneficial impact of medication in the first study ... The children had a substantial decrease in their rate of growth ... there were no beneficial effects – none ... that information should be made very clear to parents."

It wasn't. The public was duped, seduced and lied to.³¹ A news release issued by NIMH presented the negative results in a favourable light; the title was: "Improvement following ADHD treatment sustained in most children." One of the authors on the payroll of many drug companies, Peter Jensen, said, "We were

struck by the remarkable improvement in symptoms and functioning across all treatment groups.” And rather than saying that the growth of children on medication was stunted, the press release said that children who were not on medication “grew somewhat larger.”

The drug industry uses the same dirty tricks. When Merck found out that its arthritis drug Vioxx was deadly and caused more thromboses than another arthritis drug, naproxen, the company invented the hoax that naproxen was protective rather than Vioxx being harmful.⁴

ADHD drugs for adults

The benefits are also doubtful when the drugs are used for adults. As for children, many trials have been carried out by the same small group of Harvard psychiatrists who have numerous financial ties to the drug makers. And most trials are flawed by design in the same way, e.g. by including only patients that have tolerated the drug, and often also only patients who improved while on the drug. The industry calls this an “enriched design.” I call it a design that makes them rich.

We are currently doing a Cochrane review on extended release methylphenidate in adults and have found that every trial has a flawed design. A medical student we involved with this research was shocked when he saw this; he had never imagined that clinical trials could be of such poor quality, also with many missing patient-relevant outcomes. For example, he wondered why blood pressure was missing when we know that stimulants increase blood pressure and that many people die from high blood pressure.

A 2014 Cochrane review of immediate-release methylphenidate showed the expected positive effects for hyperactivity, impulsivity and inattention, but the trials were of short duration and there was a risk of bias in many cases.³² The results varied so hugely that I would not have performed meta-analyses on these data. Most worryingly, the authors could not determine whether adverse effects were not discussed because none occurred, or because no data on adverse effects were collected!

Harms from ADHD drugs

Numerous papers have described short-term harmful effects of the drugs. The increased classroom manageability comes at a high cost in terms of reductions in curiosity and social interactions. The children become more isolated, which is

hardly a good thing for a developing brain that builds many new synapses through stimulation.

As noted above, the large NIMH trial showed that methylphenidate stunts growth³³ and stimulants have many other harmful effects, including sudden cardiac death. Their side effects are similar to the criteria for bipolar disorder, and in the United States many ADHD children are diagnosed also with bipolar disorder, which I consider a medical error, as one cannot diagnose an additional disorder reliably in a drugged person (see above). Joseph Biederman and his co-workers nonetheless made a diagnosis of bipolar in 23% of 128 children with ADHD and reported this in a paper with a telling title: “Attention-deficit hyperactivity disorder and juvenile mania: an overlooked comorbidity?”³⁴ Bipolar is a serious condition often treated with antipsychotics.

We know far too little about long-term harms from ADHD drugs, but we know they can cause chronic brain damage (see Chapter 11) and can damage the heart, in the same way as seen in long-term cocaine addicts, and lead to death, also in children.¹⁹ Neurologist Fred Baughman, who has a website called ADHDfraud.org,³⁵ has explained that Adderall – a mixture of amphetamine salts – was a weight reduction drug called Obetrol, which was so addictive that it fell into disrepute and was withdrawn from the market. This addictive drug is now being sold to little children who are said to have ADHD. It was withdrawn from the Canadian market in 2005 after 14 children suddenly died and two had strokes.³⁶ The FDA did nothing, apart from trying to convince their Canadian colleagues not to withdraw the drug.

FDA trial data show that ADHD drugs cause psychosis or mania in 2-5% of people treated for one year, whereas no such cases were reported for patients on placebo.³⁷ These drugs – including atomoxetine – also cause hallucinations and violence, including homicide, and many children have killed themselves or suddenly dropped dead while playing with friends.^{37, 38} One of my friends who is a child psychiatrist told me that she had been exposed to serious threats from a criminal stuffed with methylphenidate from a reputable psychiatrist. She also said that methylphenidate is easily available on the black market, which isn't surprising, as it is a narcotic on prescription.

Millions of people are now in treatment with ADHD drugs based on mainly small, short-term, poor-quality industry trials, full of bias in the design, analysis and reporting of the data. There are very few trials of psychosocial interventions. A 2011 Cochrane review of social-skills training of children found 11 trials, but in eight of them, drugs were given to both the training group and to the control group,

and most trials had a high risk of bias.³⁹

As far as I can see, ADHD drugs do more harm than good, which is not surprising, as most of them cause similar harms as amphetamine and cocaine. People dependent on amphetamine can experience severe withdrawal symptoms that can last for weeks and which include dysphoria, irritability, melancholia, anxiety, hypersomnia, marked fatigue, intense craving for the drug and paranoia.⁴⁰

It is a paradox that some schools have posted signs saying “drugfree zones” while its teachers act as more effective drug pushers than those in the streets. The drugs may solve problems for schools but not for the children who merely act in ways that bother adults, which are very much the “symptoms” that define childhood ADHD.

All “education” of teachers, social workers, kindergarten attendants and others in how to spot symptoms of ADHD and test for it must stop, as such initiatives are harmful and prevent many of our children from having a normal childhood.

People with symptoms they think qualify for the ADHD diagnosis should avoid consulting doctors, as they very likely will diagnose them and prescribe drugs. It may even be risky to consult psychologists, as some of them collaborate closely with psychiatrists and think that drug treatment is what it should all be about. Psychologists in several countries now fight for the right to prescribe psychotropic drugs, although we prescribe them far too much already.

As ADHD is not a disease, we should ignore the criteria for the diagnosis in the DSM and ICD (International Classification of Diseases) manuals and should stop using this diagnosis. Further, it is not enough that Adderall has been removed from the market; all ADHD drugs should be removed, which would ensure that doctors can no longer use them. The idea of treating behavioural problems with drugs is an utterly sick one, which a very detailed review from the Oregon Evidence-based Practice Center leaves no doubt about.⁴¹

ADHD is a disaster area, both in terms of diagnosis, clinical research, and the harms inflicted on millions of healthy people.

References

- 1 Looking Back at the 1998 NIH Consensus Conference: Pediatrician Dr. Mark Vonnegut at a loss for words, defining ADHD as “children who can’t sit still.” <https://www.youtube.com/watch?v=MKZXH7MOwjl> (accessed 7 Dec 2014).
- 2 National Institute of Mental Health. Attention deficit hyperactivity disorder. www.nimh.nih.gov/health/publications/attention-deficit-hyperactivity-disorder/index.shtml (accessed 25 Oct 2014)
- 3 Morrow RL, Garland EJ, Wright JM, et al. Influence of relative age on diagnosis and treatment of attention-deficit/hyperactivity disorder in children. *CMAJ* 2012;184: 755-62.

- 4 Gøtzsche PC. *Deadly medicines and organised crime: How big pharma has corrupted health care*. London: Radcliffe Publishing; 2013.
- 5 Gonon F, Bezard E, Boraud T. Misrepresentation of neuroscience data might give rise to misleading conclusions in the media: the case of Attention Deficit Hyperactivity Disorder. *PLoS One* 2011;6:e14618.
- 6 Gonon F, Konsman J-P, Cohen D, et al. Why most biomedical findings echoed by newspapers turn out to be false: the case of Attention Deficit Hyperactivity Disorder. *PLoS One* 2012;7:e44275.
- 7 Adult ADHD Self-Report Scale-V1.1 (ASRS-V1.1) Symptoms Checklist from WHO Composite International Diagnostic Interview; 2003.
- 8 Adult ADHD screening day. <http://www.addforums.com/forums/showthread.php?t=6445> (accessed 29 Dec 2014).
- 9 Campanile C. Attention-deficit adults fail to get help. *New York Post* 2006 June 12.
- 10 Schweitzer JB, Cummins TK, Kant CA. Attention-deficit / hyperactivity disorder. *Med Clin North Am* 2001;85:757-77.
- 11 Biederman J, Faraone SV, Spencer T, et al. Patterns of psychiatric comorbidity, cognition, and psychosocial functioning in adults with attention deficit hyperactivity disorder. *Am J Psychiatry* 1993;150:1792-8.
- 12 Breggin P. *Psychiatric drug withdrawal: a guide for prescribers, therapists, patients, and their families*. New York: Springer Publishing Company; 2013.
- 13 Cherland E, Fitzpatrick R. Psychotic side effects of psychostimulants: a 5-year review. *Can J Psychiatry* 1999;44:811-3.
- 14 Breggin PR. The rights of children and parents in regard to children receiving psychiatric diagnoses and drugs. *Children & Society* 2014;28:231-41.
- 15 Whitely M. Strattera's sad story – (warning it may make you want to kill yourself). <http://speedupsitstill.com/strattera> (accessed 20 Jan 2015).
- 16 Henderson TA, Hartman K. Aggression, mania, and hypomania induction associated with atomoxetine. *Pediatrics* 2004;114:895-6.
- 17 Spence D. Bad medicine: adult attention-deficit/hyperactivity disorder. *BMJ* 2011;343:d7244.
- 18 Aagaard L, Hansen EH. The occurrence of adverse drug reactions reported for attention deficit hyperactivity disorder (ADHD) medications in the pediatric population: a qualitative review of empirical studies. *Neuropsychiatr Dis Treat* 2011;7:729-44.
- 19 Petersen M. *Our daily meds*. New York: Sarah Crichton Books; 2008.
- 20 Whitaker R. *Anatomy of an epidemic*. New York: Broadway Paperbacks; 2010.
- 21 Prasad V, Brogan E, Mulvaney C, et al. How effective are drug treatments for children with ADHD at improving on-task behaviour and academic achievement in the school classroom? A systematic review and meta-analysis. *Eur Child Adolesc Psychiatry* 2013;22:203-16.
- 22 Schachar R, Jadad AR, Gauld M, et al. Attention-deficit hyperactivity disorder: critical appraisal of extended treatment studies. *Can J Psychiatry* 2002;47:337-48.
- 23 Bourgeois FT, Kim JM, Mandl KD. Pre-market safety and efficacy studies for ADHD medications in children. *PLoS One* 2014;9:e102249.
- 24 Moncrieff J. *The bitterest pills*. Basingstoke: Palgrave Macmillan; 2013.
- 25 Borchering BG, Keysor CS, Rapoport JL, et al. Motor/vocal tics and compulsive behaviors on stimulant drugs: is there a common vulnerability? *Psychiatry Res* 1990;33:83-94.
- 26 The MTA Cooperative Group. A 14-month randomized clinical trial of treatment strategies for

- attention-deficit/hyperactivity disorder. *Arch Gen Psychiatry* 1999;56:1073-86.
- 27 Jensen PS, Arnold LE, Swanson JM, et al. 3-year follow-up of the NIMH MTA study. *J Am Acad Child Adolesc Psychiatry* 2007;46:989-1002.
- 28 Molina BS, Flory K, Hinshaw SP, et al. Delinquent behavior and emerging substance use in the MTA at 36 months: prevalence, course, and treatment effects. *J Am Acad Child Adolesc Psychiatry* 2007;46:1028-40.
- 29 Molina BS, Hinshaw SP, Swanson JM, et al. The MTA at 8 years: prospective follow-up of children treated for combined-type ADHD in a multisite study. *J Am Acad Child Adolesc Psychiatry* 2009;48:484-500.
- 30 Miranda C. ADHD drugs could stunt growth. *Daily Telegraph* 2007 Nov 12.
- 31 Vedantam S. Debate over drugs for ADHD reignites. *Washington Post* 2009 March 27.
- 32 Epstein T, Patsopoulos NA, Weiser M. Immediate-release methylphenidate for attention deficit hyperactivity disorder (ADHD) in adults. *Cochrane Database Syst Rev* 2014;9:CD005041.
- 33 Swanson JM, Elliott GR, Greenhill LL, et al. Effects of stimulant medication on growth rates across 3 years in the MTA follow-up. *J Am Acad Child Adolesc Psychiatry* 2007;46:1015-27.
- 34 Biederman J, Faraone S, Mick E, et al. Attention-deficit hyperactivity disorder and juvenile mania: an overlooked comorbidity? *J Am Acad Child Adolesc Psychiatry* 1996;35:997-1008.
- 35 Adams M. Neurologist Dr. Fred Baughman talks about the fraud of ADHD and the poisoning of U.S. children. 2006 Aug 30. http://www.naturalnews.com/020227_adhd_psychiatry.html (accessed 7 Dec 2014).
- 36 Canada regulators order ADD drug withdrawn. *Associated Press* 2015 Feb 10.
- 37 FDA. Minutes of the Pediatric Advisory Committee. 2006 March 22.
- 38 Moore TJ, Glenmullen J, Furberg CD. Prescription drugs associated with reports of violence towards others. *PLoS One* 2010;5:e15337.
- 39 Storebø OJ, Skoog M, Damm D, et al. Social skills training for Attention Deficit Hyperactivity Disorder (ADHD) in children aged 5 to 18 years. *Cochrane Database Syst Rev* 2011;12:CD008223.
- 40 Shoptaw SJ, Kao U, Heinzerling K, et al. Treatment for amphetamine with-drawal. *Cochrane Database Syst Rev* 2009;2:CD003021.
- 41 McDonagh MS, Peterson K, Dana T, et al. Drug class review on pharmacologic treatments for ADHD. Final Report. Oregon Health & Science University 2007.

Schizophrenia

When I first heard Robert Whitaker explain why antipsychotics do more harm than good at a meeting in Copenhagen in 2012, I was reasonably sceptical because it went counter to my training in clinical pharmacology and psychiatry. However, after having read his two books,^{1,2} some of the most important papers quoted in them, and a lot else, I know that he is right.

Schizophrenia has always been the darkest chapter in psychiatry's history book. For centuries, patients were exposed to the cruelest treatments, often against their will.¹ These included inflicting excruciating pain to distract the lunatic from his raving thoughts; dropping blindfolded patients in cold water or temporarily drowning them to create a shock effect; and putting them in a swinging chair, which produced vomiting and violent convulsions.

Many of these treatments were feared by the lunatics and the fear was used actively in order to make them behave. The rantings and ravings that appeared to define the mad – the tearing of clothes, the smearing of faeces, the screaming – were primarily antics of protest over inhumane treatment. To understand this is no less important today where many of the symptoms that seemingly define the disease are caused by the drugs or are protests over forced treatment. The threat of forced treatment is still being used to discipline patients in a power game where the patient is always the weakest (see Chapter 15).

What is particularly tragic about all this is that the psychiatrists have consistently hailed all their harmful treatments as being effective. The treatments knocked the disturbing patients down and made them docile, confused and inactive, which was convenient for the staff. The patients have rarely agreed with the staff's success criteria, but their views are often ignored even today, though the treatments imposed on them are harmful and cause many deaths.

In the 1800s, patients were sedated with opium and morphine, which were the chemical “restraints” of the time. But there was also a very different kind of treatment.¹ The Quakers treated the insane with kindness and respect and organised activities for them. Historians have concluded that this type of treatment

was surprisingly effective. Only about one third of the patients became chronically ill, and more than half remained well throughout their lives.

The first half of the 1900s was a particularly dark era in America. Eugenics was fashionable, and it began with flawed research that “proved” that insanity is an inherited disease. It progressed into sterilising and sometimes also castrating the insane, which were seen as worthless members of society. Leading scientists raised the possibility of killing the insane, and in 1935 Alexis Carrel, a Nobel Prize winning physician at the Rockefeller Institute, which supported research in eugenics, wrote in his book that the insane, or at least those who committed any sort of crime, “should be humanely and economically disposed of in small euthanistic institutions supplied with proper gases.”¹ Hitler was inspired by the American view on the insane and Nazi Germany began gassing its mentally ill patients five years later.

Many treatments introduced in this period killed large numbers of patients. The idea that insanity could be caused by an infection led to removal of the patients’ teeth and many other body parts including the large intestine. Mental patients were deliberately infected with malaria, as some effect of high fever had been seen in insanity caused by syphilis.

The barbiturates became a success after German scientists in 1903 had shown that they were good at putting dogs to sleep, and they were sometimes used to keep patients asleep for days to “restore their nervous system,” but this usage ended after it was shown that 6% of the patients died.

Around the middle of the last century, drastic treatments were introduced – insulin coma therapy, metrazole, and electroshock, which caused convulsions and often broken bones – and prefrontal lobotomy, all of which “worked” by causing brain damage, shutting down the higher functions first.¹ After emerging from insulin coma, patients acted in infantile ways, e.g. sucking their thumbs and calling out for their mummies, which behaviour was interpreted by the treating psychiatrists as a return to lucidity. Yet again, what was valued by the staff was that the patients became friendlier, less noisy and more “sociable” and spent more time sleeping, and that the nurses could behave in a more motherly fashion towards these people turned into children. However, about 5% of the patients died and the long-term outcomes were devastating.

Metrazole was no less barbaric. It is a derivative of camphor, which had been used for treatment of the insane already in the 1700s, as it also caused convulsions. The patients were terrified by the treatment, which caused spinal fractures in almost half the cases and other fractures, muscle ruptures, broken teeth and haemorrhages in internal organs. The injections were typically given two to

three times a week, but already after the first one, patients were forcibly treated, as they begged their torturers not to kill them.

Electroshock was invented by an Italian psychiatrist.¹ He first put the electrodes in the mouth and anus of dogs, but half of them died. He then visited a slaughterhouse and saw how the pigs were stunned with electroshock applied to their heads, which made it easy for the butcher to stab and bleed the animals.

It was understood right from the beginning that electroshock works by damaging the brain. Psychiatrists observed that patients lost their memories, took weeks to recover and often remained fatigued, intellectually impaired and disoriented, and acted in submissive, helpless ways. The reason that it “worked” for psychosis was simply that the patients were stripped of the higher cognitive processes and emotions that give rise to fantasies, delusions and paranoia.

This “effect” rather quickly dissipated and the illness returned. The shocks should therefore have been abandoned, but instead, a perverse idea was introduced: repeating the shocks numerous times, sometimes on a daily basis, even though the psychiatrists very well knew that the worse the brain damage, the better the “effect.”

Children weren't spared. Starting in 1942, 98 children aged four to eleven received shocks twice daily for 20 days. A ten-year old boy wanted to kill the physicians who had treated him, another attempted to hang himself. But the psychiatrists considered their treatment a success and one wrote that she had successfully given a two-year old child the usual forty shocks.

Some psychiatrists didn't delude themselves, however. They found that electroshock produced similar changes in the brain as physical trauma, with haemorrhages both in animals and people, particularly in the cortex, which led to permanent impairment of learning capacity, perception of reality, inventiveness, intuition and imagination. However, this was not what the psychiatrists told the public, which got the message that electroshocks are safe and effective. Some patients were scared to death even after curare was introduced to prevent fractures and tried to escape going through windows; some were dragged screaming into the shock rooms; and many patients regarded the shocks as punishment administered by cruel and heartless doctors. At the same time, the Red Cross determined that prisoners in Nazi concentration camps who had been electroshocked should be compensated for having suffered “pseudomedical” experiments against their will!

The frontal lobes are what make us human, but prefrontal lobotomy was introduced at about the same time, and it earned its inventor the Nobel Prize. This operation also made the patients childish and apathetic, and they lost their capacity to make sound judgments. Some died, others behaved in bizarre ways,

and about a quarter never progressed beyond the surgically induced childhood.

Money clearly played a role. By distorting completely the results of lobotomy, Rockefeller-funded scientists ensured that the money kept rolling in, although some patients had descended to the level of animals, refusing to wear clothes and urinating and defaecating in the corner. As might be expected, lobotomy was chosen as the “Endlösung” (ultimate solution) for people who couldn’t be quieted enough with electroshock, and although it wasn’t the full-blown Nazi euthanasia, a critic aptly called it “partial euthanasia.”

Also in this case, “troubled” children weren’t spared, although in one series, which included a four-year old child, two of 11 children died.

Just after the Second World War, the scandal broke in American psychiatry. Mental hospitals had developed into a sort of prison where some patients didn’t see a doctor for years and were cuffed, strapped to chairs, and beaten by the attendants, sometimes with a lethal outcome.¹

Human guinea pigs in America

In a most disturbing chapter in *Mad in America*, Robert Whitaker documents that the Nürnbeg code didn’t apply in America where the psychiatrists abused their patients for research purposes without their informed consent even as recently as in the 1990s.¹

Shortly after the war crime trials in Nürnbeg were over, Paul Hoch and his colleagues in New York experimented with LSD and mescaline, which they gave to mentally ill patients in order to develop a model for schizophrenia in humans. Both drugs worked, and Hoch also studied if electroshock and lobotomy would block the drug-induced psychosis. No one questioned the ethics of his experiments, and when he presented his results, he was congratulated for his imaginative work, which was copied by other psychiatrists.

In 1973, another researcher studied whether amphetamine and methylphenidate – the most commonly used drug for ADHD – could stir hallucinations and delusions in the mentally ill, and indeed they could, methylphenidate being the worst drug. In a New York hospital, 70 people who came into the emergency room with a first episode of psychosis were put on methylphenidate, which caused most of them to become so much worse that it took long to stabilise them on antipsychotics afterwards. Other researchers followed suit, but generally didn’t describe in medical journals how the patients had fared. However, in 1987, NIMH scientists admitted that methylphenidate injections had caused episodes of frightening intensity in patients.

These cruel experiments – which were almost exclusively an American affair – accelerated and were conducted in relative obscurity, unnoticed by the general public. In the mid-1990s, however, a citizens group led by a Holocaust survivor, Vera Sharav, and biology professor Adil Shamoo alerted the public to the unethical experiments, and Sharav remarked that such research could only be done on the powerless.

The researchers' defence was astonishing. They claimed that patients with schizophrenia volunteered for the experiments to make a contribution to science, but this was belied when independent researchers got access to the consent forms, which were unbelievably misleading. When the human guinea pigs learned about what they had been exposed to in 1998, they found it appalling and likened the experiments to those done by Nazi doctors. The researchers' explanations were pathetic, as illustrated by this statement from a patient, which is about having the cake and eating it, too:

“Do you think people really say, ‘Gee, I’ll sign up for more suffering?’ Many of us suffer enough on our own. And these [researchers] are the same people who say we don’t have enough insight and so there have to be involuntary commitment laws because we can’t see that we are ill.”

Fifty years of such research had led nowhere. Just like the military torture in Guantanamo led nowhere. There is a good reason why we have laws, and no good reason why some people are above the law if they have a high enough position that gives them total power over others.

A different type of crime that I have heard a good deal about is tampering with the patient's files, either by adding something after the event or deleting facts that would look bad in a court case. In a Danish court case I am involved with, the general practitioner added false information to the chart after the patient had hanged himself on sertraline.

The chemical lobotomy

When chlorpromazine came on the market in 1954, it was first considered a chemical lobotomy, as it produced many of the same effects as lobotomy. It was also called a chemical straitjacket, as it kept the patients under control, and later called a neuroleptic.

Psychiatrists noted that chlorpromazine didn't have any specific antipsychotic properties; the patients continued to have delusions and hallucinations but were less disturbed by them.¹ However, this truth was quickly buried, and in 1955, the president of the US Society of Biological Psychiatry, Harold Himwich, came up

with the totally weird idea that antipsychotics work like insulin for diabetes.³ The hype was extreme. A series of laudatory articles in *New York Times* hailed chlorpromazine as being curative, capable of healing the mind and bringing people back to normal life. A study in the *American Journal of Psychiatry* claimed that only two of 1,090 patients, followed for up to three years, showed faint signs of Parkinsonism, whereas a more honest psychiatrist had reported two years earlier that he had seen Parkinsonism in *all* of his patients.

According to psychiatric folklore, which we still hear today, it was the advent of antipsychotics that emptied the asylums, but this emptying started earlier, both in the United States and in the UK, and was driven by economic considerations.^{1, 3, 4}

NIMH contributed in a remarkable way to the organised delusion.^{1, 5} In 1964, NIMH funded investigators reported on a six-week study of newly admitted patients with schizophrenia in treatment with phenothiazines like chlorpromazine. None of the 270 patients became worse and the drugs reduced apathy, improved motor movement and made patients less indifferent – exactly the opposite of what these drugs do to patients, and which the psychiatrists had admitted a decade earlier – and side effects were said to be “mild and infrequent ... more a matter of patient comfort than of medical safety.” The investigators felt that the phenothiazines were curative and should no longer be called tranquillisers but anti-schizophrenic drugs. The study was double-blind and there was a placebo group. What the study tells us is that,

Investigators who have not been blinded effectively can see the exact opposite of what is actually true when they medicate patients. They see what they want to see, which is what is convenient for them and for their specialty, not what happens.

Isn't this what psychiatrists have always done? Convinced themselves that their primitive treatments were effective although they weren't? Is it any different to the current misconception that psychotropic drugs are highly effective? This study contributed to shaping the erroneous beliefs that schizophrenia can be cured with drugs and that antipsychotics should be taken indefinitely. I shall say more about the trial below, as the one-year follow-up data are very telling.

The psychiatrists didn't ask the patients how they felt about the drugs but they have suffered the consequences of the fairy tale ever since, although the scientific facts about what the drugs do to the brain are sobering.¹ The drugs hinder brain function by partially shutting down vital dopaminergic pathways. This leads to

altered behaviour and thinking, emotional isolation where the patients feel like “zombies,” development of Parkinson’s disease, and chemical lobotomy because the dopaminergic pathways to the frontal lobes get partially blocked.

What these drugs produce in the patient is a disaster, and almost all of the traits people think are caused by schizophrenia are in fact caused by the drugs: the awkward gait, the jerking arm movements, the vacant facial expression, the sleepiness, the lack of initiative.¹ These symptoms were seen more than a hundred years ago in encephalitis lethargica, which is caused by a virus, and which predicted the worst outcome in patients rendered psychotic by an infection. The term schizophrenia was coined for these patients in 1908.¹ This historical background explains why drug-induced harms are sometimes interpreted as disease symptoms even today.

The bizarre involuntary and irreversible muscular symptoms are known as tardive dyskinesia. It is seen in 5% of patients within the first year of treatment and increases by an additional 5% with each additional year of exposure,^{1, 6} which explains why about half the patients in long-term facilities have it.⁷ Tardive akathisia is a particular virulent form of tardive dyskinesia where the patient is driven by a torture-like inner agitation that compels them into moving their hands and feet nervously or pace frantically about in an effort to relieve the distress.⁷

Big pharma and psychiatry kept the public ignorant about this harm for about 20 years after chlorpromazine came on the market,⁷ as such knowledge might have pricked the whole drug balloon, which was such a cash cow for both parties.¹ The American Psychiatric Association’s astounding silence only stopped when lawsuits were filed with claims of negligence for failing to warn patients. The risk of developing malignant neuroleptic syndrome was also largely ignored, but it has been estimated that 100,000 Americans died from it in a 20-year period and that 80,000 might have lived if the physicians had been warned against it.¹

Some patients appreciate being calmed down with antipsychotics but the more common view is that the drugs are awful. They are seen as a means of torture and control that may prevent patients from thinking clearly and reading an entire book for years. The muscle contractions can lead to physical pain, people are alienated from themselves and emotionally flattened, and their will may be paralysed. A 1999 survey showed that 90% of patients on neuroleptics were depressed, 88% felt sedated, and 78% complained of poor concentration.¹

Two physicians have described how a single dose of haloperidol knocked them down.⁸ They experienced a marked slowing of thinking and movement, profound inner restlessness, a paralysis of volition and a lack of physical and psychic energy, being unable to read or work. David Healy found the same in 20

staff from his hospital who received droperidol.³ Everyone felt anxious, restless, disengaged and demotivated to do anything; a psychologist volunteer found it too complicated just to obtain a sandwich from a sandwich machine. Some felt irritable and belligerent and many were unable to recognise the altered mental state they were in and to judge their own behaviour, something which Peter Breggin calls medication spellbinding.⁷

It is easy to understand why patients often report in surveys that the drugs are worse than the disease.³ Yet, there is very little reference to the terrible drug effects in the psychiatric literature, which conveys the erroneous idea that these drugs are specific for psychosis.³ Drug regulators are complicit in this. In its report on risperidone, the UK drug agency said nothing about how the drug affects mood, attention, clarity of thought, memory, mental speed, emotional responsiveness, motivation, creativity or any other intellectual quality.³ It's unbelievable.

Right from the beginning, patients were hiding pills in their cheeks and spitting them out into toilets and when they were discharged, they were unwilling to purchase the drug. About half the patients avoided taking drugs.¹ Such disobedient patients are called non-compliant in the drug literature, although they do everything they can to comply with their own view of things. To circumvent this “problem,” Smith, Kline & French developed liquid chlorpromazine that could be secretly mixed into the patients' food. Later, long-acting injectables were developed to control the rationally acting patients and, although they killed many of them, they were also hailed as a “major tactical breakthrough.”

We know from telephone help lines that what medicated persons miss the most are themselves, and even the most deluded persons are often capable of reporting the harmful effects of the drugs objectively. They know that the drugs have robbed from them a full sense of being and that their lives have been rendered meaningless.¹ At a Senate hearing in 1975, a witness said that medicated people suffer a deadlier confinement than prisoners had ever known, and a senator called neuroleptics chemical handcuffs that assured solitary confinement of the mind.¹

Drug trials in schizophrenia

There is a rather remarkable attitude prevalent that if a paper is published then its contents become authoritative, even though before publication the same contents may have been considered nonsense.

PFLZER PHYSICIAN HASKELL WELNSTEIN¹

Drug trials in schizophrenia is a disaster area, and this is not only because the patients can be more difficult to handle than other patients in trials, e.g. patients with high blood pressure. When the Cochrane Schizophrenia Group reviewed 2,000 trials in 1998 it found half a century of trials of limited quality, duration, and clinical utility.⁹ Over half the trials had 50 or fewer patients, over 600 different interventions were studied, and 640 different rating scales were used to measure the outcome, 369 of them only once. The clinical meaning of these outcomes was obscure, and the quality of reporting was exceptionally poor and didn't improve over time. Only 4% of the trials clearly described the methods of allocation, only 22% described blinding adequately, while some description of treatment withdrawals was given in 42%.

The authors mentioned that it is difficult to blind the trials, but this is possible if one puts something in the placebo that has side effects (see Chapter 3).

So how are we supposed to find out what the drugs do to patients and whether some are better than others? The Cochrane Schizophrenia Group has published over one hundred reviews of drugs and some run over hundreds of pages, but we can take some shortcuts to get to the bottom of all this. Most importantly, virtually all of the trials are unreliable and there are five major reasons for this.

1) The placebo controlled trials are highly flawed in favour of the active drug, as they have been close to 100% unblinded and as the assessment of the outcome is highly subjective.

2) Almost all trials have included patients already in drug treatment, and the active drug has often been stopped abruptly, which can cause terrible harms, including psychosis, akathisia and death, in those randomised to placebo.

3) The outcomes are not relevant. The trials don't tell us whether drug treatment helps the patients get back into society and lead a normal life.

4) Almost all trials are short-term, although we know that a beneficial acute effect is replaced by harmful long-term effects when the drugs are used for more than a few weeks (see below).

5) The head-to-head comparisons of different drugs are also flawed. The typical trick is to compare newer antipsychotics with a too-high dose of haloperidol, which makes it possible for the companies to claim that their drug is better tolerated.^{1, 10}

Using such manoeuvres, it has been possible to hail the newer, so-called atypical, antipsychotics as breakthrough drugs, although they are equally bad as, or sometimes worse than, the old drugs.¹¹ The reason that Janssen could claim that its bestseller risperidone didn't have more extrapyramidal (muscular) side effects

than placebo was the abrupt withdrawal of the previous antipsychotic drug, which inflicted such effects on the placebo group to such an extent that one in six “placebo” patients got them!¹ The companies needed to show that their drugs reduced psychotic symptoms and they made the placebo patients psychotic by withdrawing their drug cold turkey.

This withdrawal design is lethal. One in every 145 patients who entered the trials for risperidone (Janssen), olanzapine (Eli Lilly), quetiapine (AstraZeneca) and sertindole (Lundbeck) died, but none of these deaths were mentioned in the scientific literature, and the FDA didn't require them to be mentioned.¹ Many of these patients killed themselves; the suicide rate in the trials was two to five times the usual rate for patients with schizophrenia, and a major reason was the withdrawal-induced akathisia.¹ An analysis of patient reports on the Internet showed that suicidal thoughts when taking antipsychotics are strongly associated with akathisia; 13.8% of respondents reporting akathisia also reported suicidal thoughts, compared with 1.5% of those who didn't mention akathisia ($P < 0.001$).¹² This harm would be expected to be related to the dose of the previous drug, and it clearly is.¹³ It could be argued that those who were most ill got the highest doses and were therefore more prone to experience these effects, but the dose effect was so strong that this couldn't have been the major factor.

The FDA reviewers repeatedly pointed out that the companies used a biased design that didn't contain a true placebo group to show that their drugs worked, but the drugs were approved.¹ This cruel design has not only been lethal for some patients, it has also produced misleading results for those who survived, as the patients in the placebo group had been harmed.¹

The FDA forbade Janssen to claim in advertisements that risperidone was superior to haloperidol with regard to safety or effectiveness, but this was exactly the message Janssen conveyed in scientific papers, which the FDA had no control over, although it was wrong. A meta-analysis of 52 trials (12,649 patients) concluded that there was no basis for claiming that atypical antipsychotics were any better than old antipsychotics.⁴

These industry-sponsored drug trials are terribly deceptive. Whitaker conducts the thought experiment that if olanzapine had been the old drug and haloperidol the new one, it would have been easy for a company to create the impression in its trials that haloperidol was the superior drug, which it very likely is.¹

The most reliable placebo controlled trials are those of first episode schizophrenia where none of the patients have ever received drugs before. There is a Cochrane review that approaches this ideal, but even this review is biased, as

the trials are not limited to first episode patients; it includes studies “with a majority of first and second episode schizophrenia spectrum disorders.”¹⁴

Nonetheless, the review is highly interesting and the most relevant one. In three trials of chlorpromazine, the participants were half as likely to leave the study early when they were on drug than when they were on placebo. Only one of the trials (463 patients), which was sponsored by NIMH, reported on harms, and there were of course more harms on chlorpromazine than on placebo.

Another of the trials (127 patients) reported on rehospitalisations, and double as many patients on chlorpromazine than on placebo were rehospitalised within three years, relative risk 2.3 (95% CI 1.3 to 4.0). When the patients who dropped out were included in the analysis, the result was even more striking, relative risk 3.1 (95% CI 1.6 to 5.8). There were also fewer rehospitalisations in the placebo group at the one-year follow-up in the NIMH trial, but the difference wasn't quantified and the original data appear to have been lost.¹⁴

In a fourth randomised trial, Loren Mosher compared 55 patients in hospital, all of whom received antipsychotics, with 45 patients who were treated in a non-hospital milieu where 67% received no antipsychotics. The Cochrane review says nothing about the rationale for Mosher's trial, although it was unique for its time.¹ Mosher was the chief of the Center for Studies of Schizophrenia at NIMH and he wasn't against using antipsychotics. He opened a 12-room Soteria house in 1971, as he wanted to study whether treating acutely psychotic people in a humanistic way that emphasised empathy and caring and avoided antipsychotics could be as effective as drug treatment. There were no locks on the doors, and the idea was to treat people with respect. His staff were not mental health professionals but people who had social skills and empathy and who listened to the patients' crazy stories (which often revealed traumas with abuse and extreme social failure).¹⁵ Thus, Mosher paved the way for what later became known as Open Dialogue (see Chapter 10).

The results in Mosher's trial after six weeks were virtually the same in the two groups for psychopathology. Thus, as outlined further below, antipsychotics can be avoided in most patients, even in acute psychosis, where the rationale for their use is otherwise most obvious. The authors of the Cochrane review were pretty direct about this, as they pointed out that the available evidence doesn't support a conclusion that antipsychotic treatment in an acute early episode of schizophrenia is effective.¹⁴ They felt this was worrying given the widespread use of antipsychotics in the acute treatment of early episode schizophrenia-type psychoses, and also because the use of antipsychotics for millions of people with an early episode appears based on the trials for those with multiple previous

episodes (which we know are so flawed that the evidence is useless). What the Cochrane authors didn't write about was: What does this mean for use of antipsychotics more generally, also for multiple episodes of psychoses? Doesn't it mean that we don't have the evidence to support using antipsychotics at all?

The harmful effect of antipsychotics in terms of an increased risk of relapse (hospital admission) was expected. The drugs block dopamine transmission, and the brain's response to this is to produce more dopamine receptors. This means that the brain becomes supersensitive to dopamine and at greater risk for a new and more severe episode of psychosis, whether or not the patients still take drugs.^{2, 16}

In 2014, a huge Cochrane review (55 trials and 5,506 patients) was published of trials comparing chlorpromazine with placebo for schizophrenia, which was not limited to early episodes.¹⁷ I find it astonishing that the authors mention in the abstract without any reservation that akathisia didn't occur more often in the chlorpromazine group than in the placebo group. Worse still, the largest trial that contributed data to this outcome found *significantly less* akathisia in the active group than in the placebo group, relative risk 0.57, 95% CI 0.37 to 0.88. Since we know that antipsychotics *cause* akathisia¹ and that placebo *cannot* cause akathisia, this result speaks volumes about how ridiculously flawed trials in schizophrenia are. What was seen in the placebo group were cold turkey symptoms *caused* by withdrawal of antipsychotics!

There are other problems with Cochrane reviews in schizophrenia. They routinely include trials in a meta-analysis where half of the data are missing. I believe this is highly likely to produce misleading results. Furthermore, it is recommended to use completers-only data, if there are no data on those who dropped out. As there are often many drop-outs, it can bias the review in favour of the active drug to analyse the data in this way, as shown by an analysis of trials of antidepressants performed by the Swedish drug agency.^{18, 19}

We cannot turn garbage into gold with statistical alchemy. I therefore believe that the Cochrane schizophrenia reviews that have allowed so much data to be missing should be redone, with stricter criteria about which trials to include. But the question is whether it would be worth the effort when virtually all these trials are biased by design.

Even helped by all these formidable biases, the outcome of schizophrenia trials has been poor. During the first week of treatment, clinicians cannot detect any effect of antipsychotics on the symptoms,²⁰ and even later, the effect may lack clinical relevance. Based on data from 5,970 patients in trials, Stefan Leucht and colleagues have shown that minimal improvement on the Clinical Global

Impressions Ratings correspond to about 10 points on the Brief Psychiatric Rating Scale (BPRS) and 15 points on the Positive and Negative Syndrome Scale (PANSS).²⁰ What is obtained in recent placebo controlled trials in submissions to the FDA is far below these minimum improvements, e.g. only 6 points on the PANSS score,^{3, 21} even though it is easy for scores to improve quite a bit if someone is knocked down by a tranquilliser and express their abnormal ideas less frequently.³

A huge NIMH-funded trial, the CATIE trial, of 1,493 patients with schizophrenia randomised to four different antipsychotics, also showed pretty disappointing results.²² Although it was planned that patients should take their drug for 18 months, and although patients are constantly coerced to take their drugs, 74% of patients discontinued it earlier, and olanzapine, risperidone and quetiapine weren't any better than an old drug, perphenazine. This trial, and other large, publicly funded trials,²³ undercut completely the legitimacy of psychiatry's treatment guidelines for schizophrenia, which recommend the newer, expensive drugs.

Since the randomised trials are so flawed, naturalistic studies become interesting. The World Health Organization (WHO) launched a study in 1969 that lasted eight years.¹ It turned out that patients fared much better in poor countries – India, Nigeria and Colombia than in the United States and four other developed countries. At five years, about 64% of the patients in the poor countries were asymptomatic and functioning well compared to only 18% in the rich countries.

Western psychiatrists dismissed the results with the argument that patients in poor countries might have milder disease. The WHO therefore did another study, focusing on first episode schizophrenia diagnosed with the same criteria in ten countries.¹ The results were pretty similar, about two-thirds were okay after two years in the poor countries versus only one third in the rich countries.

The WHO investigators tried to explain this huge difference by various psychosocial and cultural factors but didn't succeed. The most obvious explanation, drug use, was so threatening to western medicine that it went unexplored. Antipsychotics are very expensive and people in poor countries couldn't afford them, so only 16% of patients with schizophrenia were regularly maintained on antipsychotics as compared with 61% in rich countries.¹ A 20-year study from Chicago by Martin Harrow showed the same; patients who were untreated for many years had substantially better outcomes than those on antipsychotics.^{16, 24} Confounding by indication was probably an issue, but Harrow found the same when he compared patients with similar prognoses.²⁵

These results are very convincing and fit all too well with what we know from the Cochrane review of first episode schizophrenia¹⁴ and about how the drugs destabilise the dopaminergic systems so that patients become more vulnerable to relapse.¹ The WHO studies showed that recovery is more likely without drugs. A more recent study performed by Eli Lilly failed to find differences between poor and rich countries, but in this study all patients were treated with drugs, half of them with olanzapine, the other half with other antipsychotics.²⁶

Apart from avoiding the damaging effects of antipsychotics, there are other good reasons why people with schizophrenia fared so well in poor countries.²⁷ The medical model of schizophrenia – that it is a brain disease – lowers self-esteem and increases despair, hopelessness, negative public attitudes and stigmatisation (see further below). In poor countries, however, the illness is often seen as the result of external forces, e.g. evil spirits, and people are much more likely to keep the sufferer in the family and to show kindness, which helps patients recover and participate in social life again.

Few psychiatrists know about this. They have asked me whether it would be better than drugs to deprive people of their liberty by tying them to a tree. This may happen in Africa, but overall, the communities did a far better job in Africa than we do in the western world where we have institutionalised deprivation of liberty through legal means and forced treatment. Over the years, we have killed hundreds of thousands of patients with antipsychotic drugs (see Chapter 14). Is this really supposed to be a more “humane” system?

Freedom from neuroleptics should be the desired therapeutic goal, but psychiatry has again protected its self-made delusions rather than the patients. In 1998, 92% of patients with schizophrenia in America were being routinely maintained on antipsychotics.¹

What about the benzodiazepines? Since the main justification for using antipsychotics is to calm the patients down in the acute phase, one would expect benzodiazepines to be a better alternative.

But big pharma has shied away from comparing their horrendously expensive and dangerous antipsychotics with offpatent benzodiazepines that can be acquired almost for free, and psychiatrists have failed to live up to their professional responsibility by neglecting to perform such trials themselves. In 1989, 35 years after chlorpromazine came on the market, only two trials had compared the two types of drugs, and they produced similar improvements.² There are now more trials, summarised in a 2011 Cochrane review, and its conclusion is revealing:²⁸

“There is currently no convincing evidence to confirm or refute the practice of

administering benzodiazepines as monotherapy.”

In actual fact, we should use benzodiazepines instead of antipsychotics. In 14 trials that had compared them, the desired sedation occurred significantly more often on benzodiazepines. Benzodiazepines were compared with placebo in eight trials, and the authors reported that the proportion of treatment failures wasn't significantly lower with benzodiazepines than with placebo (six trials, 382 patients, relative risk 0.67, 95% CI 0.44 to 1.02). My interpretation of these data is entirely different. *Of course* benzodiazepines calm patients down, which a relative risk of 0.67 also implies. Whether or not it is statistically significant doesn't matter; it would have become significant with a few more patients.

So why don't psychiatrists use benzodiazepines instead of antipsychotics? And if they find the trials of poor quality,²⁸ then why haven't they done better trials themselves? The problem is that the psychiatrists have allowed big pharma to control their specialty to the great impediment of rational treatment for their patients. We don't even know whether antipsychotics or benzodiazepines are any better than morphine in knocking patients down. Probably not, and they are likely worse, as it is far easier to withdraw opioids from people than to withdraw antipsychotics or benzodiazepines (see Chapter 12).

Antipsychotics kill many people

People with schizophrenia have about 20-year shorter lives than others.²⁹ Suicides play a minor role, and most deaths are likely caused by antipsychotic drugs. Curiously, about two-thirds of the excess deaths are called “natural deaths,”^{29, 30} which they aren't, as they are mainly drug deaths.

A meta-analysis of placebo controlled trials showed that it's indisputable that antipsychotics kill people (see Chapter 14).³¹ There are many reasons for this. They can cause QT interval prolongation on the ECG and life-threatening ventricular arrhythmias, and large US studies have shown that antipsychotics double the risk of sudden cardiac deaths in a dose-dependent manner.^{32, 33} From 2004 to 2012, antipsychotics topped the list for reports of QT interval prolongation in the FDA's Adverse Event Database, and antidepressants came second.³⁴ Antipsychotics also cause falls and hip fracture due to orthostatic hypotension, sedation and loss of consciousness, and they increase cerebrovascular adverse events.³¹ The FDA added warnings of increased cerebrovascular adverse events to the US prescribing information for risperidone in 2003, for olanzapine in 2004 and for aripiprazole in 2005.

Other reasons why antipsychotics kill people include the huge weight gains and

diabetes many experience, which shorten life expectancy substantially. A systematic review of mortality in schizophrenia showed that mortality had increased in recent decades compared with the general population; the median standardised mortality ratio for the 1970s, 1980s and 1990s were 1.84, 2.98 and 3.20, respectively.³⁵ As the authors pointed out, an obvious explanation for this development is the increased use of newer antipsychotics, which are more likely to cause weight gain and metabolic syndrome than the old drugs.

Antipsychotics can also lead to smoking and alcohol abuse because the patients may seek relief from these dopamine-releasing substances to counteract the decrease in dopamine caused by the drugs.

In agreement with the meta-analysis of the randomised trials,³¹ a Finnish study found that the mortality risk for people with schizophrenia was more than double that for other people; after adjustment for other risk factors, including smoking, the relative risk was 2.50 (95% CI 1.46 to 4.30) per increment of one neuroleptic.³⁶ This study found a strong inverse relationship between serum HDL cholesterol and the number of neuroleptics prescribed, i.e. the more drugs, the greater the risk of dying from heart disease. A large Dutch study confirmed these results.³⁷ The mortality risk for having a diagnosis of schizophrenia and using an antipsychotic compared to a control group of patients who did not have schizophrenia was more than doubled (hazard ratio 2.6, 95% CI 2.0 to 3.2), and it was 8.4 (95% CI 3.1 to 24.1) if a “mood stabiliser” (anti-epileptic drug) was used. In a cohort of 88 in-patients followed over 10 years, 39 died, with no instances of suicide. Reduced survival was predicted by the number of antipsychotics given concurrently (relative risk 2.46, 95% CI 1.10 to 5.47).³⁸ Patients with tardive dyskinesia also have higher mortality rates, and this harm is directly related to dose.¹

But the doubt industry never sleeps.³⁹ One of the worst studies was published in *Lancet* in 2009 by Finnish researchers.²⁹ It found that use of antipsychotics for 7-11 years was associated with lower mortality than no drug use (adjusted hazard ratio 0.81, 95% CI 0.77 to 0.84). This is too good to be true, and the other main result, that the longer people had used drugs the lower the mortality, cannot be true either. Other researchers have addressed the fatal flaws in this study, e.g. 64% of the deaths were not accounted for and the mortality in patients who were not on drugs was very high and didn't concur with other Finnish data.⁴⁰

In a debate I had with Norwegian psychiatrists who claimed that antipsychotics reduce mortality in patients with schizophrenia,⁴¹ they referred to the misleading Finnish study²⁹ and also to a Swedish study. The Swedish study found higher mortality with higher doses than with lower doses,⁴² entirely as expected, but the psychiatrists didn't mention this finding. They also failed to mention that an

obvious reason why untreated patients had a high mortality was that they were different from those on drugs, which the study authors wrote themselves. They had a higher mortality of cancer and heart disease than those who received antipsychotics and were thus at high risk of dying, regardless of whether or not they were treated.

Another “doubt industry study” used FDA data and was similarly misleading.⁴³ Arif Khan and colleagues reported that antipsychotics lowered mortality in schizophrenia by more than 50% and that the drugs also lowered suicides. The authors used person-years instead of persons and included not only the double-blind phases of the placebo controlled randomised trials but also the safety extension phases in which the patients only received active drug (the average duration of placebo exposure was only 33 days, as compared to 132 days on drug). Such analyses are totally unreliable, as those who continue on active drug are those who tolerate it (I described this dirty trick in Chapter 3). What Khan’s study really showed – when counting persons – was that antipsychotics killed people (relative risk 1.65, i.e. a 65% increase in total mortality), and also caused three times as many suicides (relative risk 2.83). Khan has been principal investigator of more than 340 clinical trials sponsored by more than 65 pharmaceutical companies and 30 contract research organizations.⁴³ Is this why he published a fatally flawed meta-analysis in favour of drugs?

A patient history

Bertel Rüdinger is a 40-year old pharmacist who was admitted to a psychiatric hospital ward for the first time in 2001. The diagnosis was very severe depression. He was admitted several times, and the chief of staff resolved that he was in a deep life crisis and needed to get away from his family and everyday life to recover. While he stayed at a rehabilitation centre, one of his assigned contact persons became aware that he heard voices and the diagnosis was changed to schizoaffective disorder.

The following year, he was hospitalised most of the time and in 2005, he went on life-long disability pension because he was considered hopelessly ill. From 2003 to 2011, he was exposed to about 40% of the drugs used in psychiatry and was subjected to extensive polypharmacy, both antipsychotics, antidepressants and “mood stabilisers.”

He is very grateful that his mother never lost hope and continued to believe he could have another life. If she hadn’t softened psychiatry’s violent messages about the hopelessness of his situation, he would have committed suicide. His other

stroke of luck came when his mother needed a website. The woman who did the programming was pretty hopeless with a computer, so Bertel thought that if she could do it, he could, too. He learned how to programme websites, which became his way out of psychiatry.

In 2009, he moved from his protected housing facility and was fortunate that another such facility sought a webmaster with user experience. He got the job, and now met patients who had recovered and also people who had an entirely different approach to mental disorders than he had been accustomed to. He learned about the Hearing Voices Network and it dawned on him just how much of his personality the pills had eaten. He furthermore realised that, being a pharmacist with personal knowledge of mental disorders and psychiatric drugs, he could help promote other people's recovery process. Bertel believes he was the first pharmacist in continental Europe to start and develop clinical pharmacy in a social psychiatry setting. He stopped the last psychotropic drug in 2011, but, as for so many others treated with antipsychotics, it is impossible for him to get rid of the marked obesity his drugs caused. Therefore, his risk of dying early will forever be considerably increased because of the drugs he was prescribed.

During Bertel's "career" as a psychiatric patient, he received eight different diagnoses: depression, depression with psychotic symptoms, borderline, schizoaffective disorder with only depressive affective symptoms, paranoid schizophrenia, simple schizophrenia, schizoaffective disorder with bipolarity, and bipolar I with schizoid personality.

One of the things he learned during his stay in psychiatry was that in order for the staff to understand people with mental disorders and their psychological context, trust needs to be established. Many professionals don't see a connection between current suffering and life history. There is rarely time to establish the necessary confidence and all the forced treatments in psychiatry creates insecurity. Patients don't open up to therapists who one day exercise coercion and the next day try to build a therapeutic alliance.

Bertel says that the psychotropic drugs prevented him from committing suicide, but only because he was so heavily drugged that he was totally apathetic and couldn't do anything. He wouldn't recommend such treatment, which had formidable physical, psychological and social consequences, as prophylaxis against suicide.

Today, Bertel helps people with mental disorders to use their medication as part of their recovery process and with stopping drugs that do more harm than good, which some patients have been forced to take against their will and in excessive dosages. He collaborates with a very good psychiatrist on this. More commonly, however, his experience is that it can be difficult or impossible to

make psychiatrists understand that psychotropic drugs are like all other drugs, and that guidelines and recommendations must be respected.

Pushing antipsychotic drugs

Unfortunately, psychiatrists want the opposite of what Bertel wants; they want to loosen the guidelines. Danish psychiatrist Henrik Day Poulsen (see Chapter 2) recently argued that psychiatrists should be allowed to use three times the maximum dose of antipsychotics recommended by the drug agency.⁴⁴ Poulsen claimed that there is no risk in prescribing these higher doses and referred to safety data from Eli Lilly on olanzapine. But he forgot to tell the readers that he is on the Lilly payroll, and on many other companies' payrolls, too.

Olanzapine is the most used antipsychotic in Denmark and was at the centre of one of the biggest scandals about overmedication, which led to dismissal of several leading psychiatrists. Nonetheless, Poulsen argued that if psychiatrists couldn't use higher doses, it would be harmful for patients and increase violent situations in the wards, and in this he was supported by the Danish Psychiatric Association. However, several members of parliament and a number of patient organisations felt the psychiatrists already used too many antipsychotics and in too high doses.

What psychiatrists don't realise at all is that, most commonly, patients are violent because of the side effects of the antipsychotics they take (see Chapter 14); they should therefore come off them instead of having the dose increased.

Can it really be true that a tripling of the dose doesn't cause more harm? Of course not. As noted above, the increased mortality with antipsychotics is clearly related to dose and number of drugs.

But the silverbacks again displayed their organised professional denial. They referred to a 2006 report from the Danish National Board of Health produced by themselves, which claims that the use of several antipsychotics simultaneously doesn't increase the risk of death.³⁰ This cannot possibly be correct, and it turned out that the statistical method used in the report is totally faulty.⁴⁵ The report showed that those who got four antipsychotics had a higher mortality than those who got fewer drugs.

The Danish report also showed that half the patients were in treatment with more than one antipsychotic simultaneously, although there are no scientific data in support of this and although both national and international guidelines recommend against it. The record I have heard about was seven antipsychotics simultaneously. Half the patients were also in treatment with benzodiazepines and similar drugs

although the report mentioned that this combination increases mortality by 50-65%.³⁰ There were also more patients than the working group had expected – almost half – that were in treatment with both antipsychotics and antidepressants, and the report advised against this massive use of antidepressants.

What was most striking about the report was that it showed beyond a shadow of doubt that the use of psychiatric drugs was already out of control, and it was against this background that psychiatrists wanted to increase overtreatment even more. It's unbelievable.

A review of medication lists of 214 randomly selected citizens receiving residential care or home care (median age 84 years) in Copenhagen was similarly revealing.⁴⁶ An astounding 65% used one or more psychotropic drugs (antipsychotics 16%, antidepressants 44%, anxiolytics/hypnotics 27% and anti-dementia drugs 16%). Many citizens on antipsychotics were also on antidepressants (53%), anxiolytics/hypnotics (35%) and anti-dementia drugs (21%). A survey of 500 prescriptions for risperidone in France was also depressing.⁴⁷ The prevalence of co-prescription was 43% for antidepressants, 46% for benzodiazepines, 27% for other neuroleptics, 22% for “mood stabilisers” and 19% for anticholinergic drugs. The official Danish recommendations are that patients with dementia should not get antipsychotics, and that antipsychotics plus anxiolytics/hypnotics should be avoided.

The illegal marketing of antipsychotics is pervasive,³⁹ as the drugs are so expensive, and this is a main reason why the use of these drugs is totally out of control. In the UK, half the prescriptions by general practitioners are issued to people for a variety of non-psychotic problems including anxiety and sleep problems, and antipsychotics are particularly often used in people with dementia and in old people.⁴⁸ In the United States, the use of antipsychotics doubled in adults and went up eight-fold in children in just 11 years,⁴⁹ and in 2005, seven kids per 1,000 were in treatment with antipsychotics;⁵⁰ only 14% of prescriptions were for psychoses while most were for behaviour problems and mood disorders.³

Abilify is currently the most sold of all drugs in the United States, including statins. When I looked it up, 30 tablets of 15 mg cost \$800, which is obscene. Its maker, Bristol-Myers Squibb, agreed in 2007 to pay more than \$515 million to settle illegal marketing and fraudulent pricing practices involving payments to doctors to induce them to use the company's drugs, also for off-label use.³⁹ Several other companies also paid kickbacks to the doctors.³⁹

The history of antipsychotics has many similarities to that of the SSRIs. The

clinical research wasn't aimed at clarifying the role of the new drugs for clinicians and patients but was driven by marketing strategy, and new drugs were much hyped, both in sales pitches and in industry-sponsored research. When large, independent government-funded trials became available, it was easy to see that the new drugs aren't any better than the old ones.^{22, 23, 51-53} For example, a trial in 498 patients with a first episode schizophrenia found no difference in discontinuation rates between four newer drugs and haloperidol.⁵³ The study was funded by three drug companies but they were kept at arm's length.

Such independent trials, also in other areas of psychiatry and medicine, have taught us that the “best” drugs may simply be those with the most shamelessly biased data.¹⁰

Antipsychotics are standard treatment for bipolar disorder, which – as explained in earlier chapters – is mainly iatrogenic, caused by SSRIs and ADHD drugs, and they are also used for depression. We now see advertisements, e.g. for AstraZeneca, about combination therapy for depression, and there are preparations that combine the drugs in the same pill, e.g. Symbyax from Lilly, which contains fluoxetine and olanzapine, two of the worst psychotropic drugs ever invented.

It's remarkable that it has been possible to show in a meta-analysis of published trials that new drugs aren't better than old ones, as the research literature is so flawed. A 2009 meta-analysis of 150 trials with 21,533 patients showed that the psychiatrists had been duped for 20 years.^{54, 55} The drug industry has invented catchy terms such as “second generation antipsychotics” and “atypical antipsychotics,” but there is nothing special about the new drugs, and as antipsychotics – new and old – are widely heterogeneous, it's plain wrong to divide them into two classes. Haloperidol was the comparator in most of the trials, and, as noted above, it was often used in too large doses. Unsurprisingly, the flaws are introduced deliberately, e.g. an internal Pfizer memorandum explains that by increasing the dose of the comparator drug quickly, this will result in a high drop-out rate on that drug due to side effects.⁵⁶ Given all this, I strongly suspect the old drugs are better than the new ones.

Eli Lilly's crimes related to olanzapine

In 2009, Eli Lilly pleaded guilty to criminal charges and had to pay more than \$1.4 billion for illegal marketing as part of a settlement with the US Department of Justice.³⁹ The crimes concerned olanzapine, and as worldwide sales were nearly \$40 billion between 1996 and 2009, it's hard to believe that the fine will have any

deterrent effect.

The crimes were particularly aimed at pushing olanzapine hard to children and the elderly, and there were many other shady activities. Posing as physicians, Lilly salespeople asked “planted questions” during off-label lectures and audio conferences for physicians, and while knowing the substantial risk for weight gain and diabetes caused by olanzapine, the company minimised the problem in a widely disseminated videotape called *The Myth of Diabetes*. Lilly and their paid prostitutes among doctors also produced papers describing schizophrenia as a risk factor for diabetes!³ As usual, it’s never the drug that is the problem, it’s the disease.

Internal Lilly documents were leaked to the *New York Times* in 2006, which demonstrate the extent to which the company downplayed the harmful effects of olanzapine.^{39, 57, 58} However, Lilly instigated legal action against doctors, lawyers, journalists and activists to stop them from publishing the incriminating documents on the Internet, and they disappeared.

Even in 2007, Lilly maintained that “numerous studies ... have not found that Zyprexa causes diabetes,” but Lilly’s own studies showed that 30% of the patients gained at least 10 kg in weight after a year on the drug, and 16% gained 30 kg. Moreover, both psychiatrists and endocrinologists said that olanzapine caused diabetes in many more patients than other drugs,^{58, 59} and it is likely more harmful than many other antipsychotics.⁶⁰

But the company was in a precarious situation, just like it was before it launched fluoxetine in 1988, as fluoxetine would soon run out of patent. Lilly fooled people into believing that fluoxetine was a good drug, and now the company was desperate to fool people again with olanzapine. In relation to a lawsuit at the superior court of Alaska, details of the four trials Eli Lilly had submitted to the FDA to get olanzapine approved were revealed, and a physician wrote a disturbing report about this for the court.⁶¹

The FDA’s cover-up of Lilly’s manipulations were just as disgraceful as when the FDA protected fluoxetine. The FDA rejected two of the studies, but the other two were also unacceptable. There was a placebo lead-in of four to nine days, which means that withdrawal symptoms were inflicted on the placebo group; a benzodiazepine was allowed – just like in the fluoxetine trials – but there was no information about how many patients took it in the placebo and drug groups; more than half the patients dropped out quickly although the trials lasted only six weeks; there were lots of missing data; and patients could be switched to open treatment with olanzapine after only two to three weeks if they had responded poorly. *It’s impossible to get anything reliable out of trials like these.* In one trial,

haloperidol was the comparator drug, but it was overdosed up to 20-fold in comparison with olanzapine.

Despite all this, olanzapine wasn't better than placebo in many of the analyses. One of the rejected trials was very large, 431 patients, but olanzapine doses of 5, 10 and 15 mg weren't any more effective than 1 mg. The FDA seemed to have panicked over this study, which it buried. The dose was so low that very few dopamine receptors would be occupied by olanzapine. Thus, this study not only proved the "chemical imbalance" dopamine theory wrong, it also demonstrated once again that what we see in trials of psychiatric drugs are not real effects but mainly biased evaluations because of poor blinding.

Lilly wanted to make doctors use olanzapine also for mood disorders and had the audacity to call the drug a mood stabiliser, although it doesn't stabilise mood. Like other antipsychotics, it dampens wild thoughts – in fact, any thoughts. It was a challenge for Lilly that general practitioners were worried about the harms caused by antipsychotics, but Lilly was determined to "change their paradigm," as they euphemistically called it in an internal document. Lilly also prepared fictitious patient stories for use by the sales force.⁶⁰

Lilly's many tricks of the trade even ensured that the company got around a threatening patent problem. The patent was running out but Lilly got a new patent by showing that it produced less elevation of cholesterol in dogs than a never-marketed drug!⁶² I have seen many absurdities in relation to the patenting of drugs, but this one deserves first prize. Olanzapine raises cholesterol more than most other drugs, and it should therefore have been marketed as a cholesterol-raising drug, but that wouldn't have made it a blockbuster with sales of around \$5 billion per year for more than a decade,⁶² the most widely used antipsychotic drug in the world.

Lilly's huge commercial success with both fluoxetine and olanzapine illustrates that in psychiatry it doesn't really matter which drugs you have. Corruption, marketing and lies will ensure that doctors don't use drugs that are both better and cheaper. As an example of this information control, a Cochrane review from 2005 reported that the largest trial with olanzapine had been published *142 times* in papers and conference abstracts!⁶³ This carpet bombing with propaganda contributed to the fact that, in 2002 sales for olanzapine were 54 times larger than sales for haloperidol in Denmark. There was no excuse for this waste of money. At the time, olanzapine cost seven times as much as haloperidol per day, and two years earlier, a meta-analysis in the *BMJ* concluded that, "the new drugs have no unequivocal advantages."¹¹

Patient organisations contribute to the corruption. They often receive money

from the industry and know only what the drug firms have told them, or what the psychiatrists have told them, which is about the same, as they also get their knowledge from the industry. It was therefore not surprising when the chairman of an organisation for psychiatric patients in 2001 called it unethical that Danish psychiatrists in her view were too slow to use the newer antipsychotics such as olanzapine and risperidone.⁶⁴

Other companies also lied blatantly about their drugs. AstraZeneca presented data at international meetings indicating that quetiapine helped psychotic patients lose weight, while silencing a trial showing significant weight increases and while internal data showed that 18% of the patients had a weight gain of at least 7%.⁶⁰,⁶⁵ Astra-Zeneca propagated other lies.⁶⁰ It presented a meta-analysis of four trials showing that quetiapine had better effect than haloperidol, but internal documents released through litigation showed it was exactly the opposite: quetiapine was *less* effective than haloperidol.

Stigmatisation

It is often assumed that biological or genetic explanations of mental illness increase tolerance towards psychiatric patients by reducing notions of responsibility and blame.⁶⁶ The core assumption of anti-stigma programmes is that the public should be taught to recognise the problems as diseases, and to believe they are caused by biological factors like chemical imbalance, brain disease and genetic factors. However, studies have consistently found that this disease model *increases* stigmatisation and discrimination, e.g. a systematic review of 33 studies found that biogenetic causal attributions were generally not associated with more tolerant attitudes; they were related to stronger rejection in most studies examining schizophrenia.⁶⁶

The biological approach increases perceived dangerousness, fear and desire for distance from patients with schizophrenia because it makes people believe that the patients are unpredictable,⁶⁶⁻⁶⁹ and it also leads to reductions in clinicians' empathy and to social exclusion.⁷⁰

It furthermore generates undue pessimism about the chances of recovery and reduces efforts to change, compared to a psychosocial explanation. It is therefore not surprising that participants in a learning task increased the intensity of electric shocks more quickly if they understood their partner's difficulties in disease terms than if they believed they were a result of childhood events.⁶⁸

Many patients describe discrimination as more long-lasting and disabling than

the psychosis itself, and it is recognised as a major barrier to recovery.^{67, 68} Patients and their families experience more stigma and discrimination from mental health professionals than from any other sector of society, and there are good explanations for this. For example, over 80% of people with the schizophrenia label think that the diagnosis itself is damaging and dangerous, and therefore some psychiatrists avoid using the term schizophrenia.⁶⁸

In contrast to the psychiatric leaders, the public is firmly convinced that madness is caused more by bad things happening than by genetics or chemical imbalances.⁶⁸ This lucidity is remarkable, given that more than half the websites about schizophrenia are drug-company funded. The public also sees psychological interventions as highly effective for psychotic disorders (which is also pretty accurate, see Chapter 10), whereas psychiatrists opine that if the public's mental health literacy isn't improved, it may hinder acceptance of evidence-based mental healthcare (which means drugs!).

The spending of millions of dollars (largely by drug companies) to teach the public to think more like biologically oriented psychiatrists has had four outcomes: more discrimination, more drugs, more harms, and more deaths.

Hearing voices

One of the key features in the diagnosis of schizophrenia is that the patients hear voices. There is another approach to the voices than the disease-oriented understanding that psychiatry offers, which does not necessarily involve antipsychotic drugs. Many people begin to hear voices as a result of extreme stress or trauma, and an association called the Hearing Voices Network exists in several countries (e.g. <http://www.hearing-voices.org/>), which helps people who hear voices to live a normal life. Such networks want to give these people an opportunity to talk freely about the voices, e.g. in self-help groups, and support them to understand, learn and grow from them in their own way.

Hearing voices is called auditory hallucinations. It is not normal, but on the other hand these voices are real for the person who hears them. They are therefore not called delusions, which are thoughts about things that are plain wrong, e.g. when patients think they are Jesus or Napoleon. But the line is not sharp. What should we say about people who consider themselves normal but believe they have lived before, or the many who believe that there is life after death?

References

- 1 Whitaker R. Mad in America. Cambridge: Perseus Books Group; 2002.

- 2 Whitaker R. *Anatomy of an Epidemic*. New York: Broadway Paperbacks; 2010.
- 3 Moncrieff J. *The bitterest pills*. Basingstoke: Palgrave Macmillan; 2013.
- 4 Kirk SA, Gomory T, Cohen D. *Mad science: psychiatric coercion, diagnosis and drugs*. New Brunswick: Transaction Publishers; 2013.
- 5 Cole JO. Phenothiazine treatment in acute schizophrenia; effectiveness: the National Institute of Mental Health Psychopharmacology Service Center Collaborative Study Group. *Arch Gen Psychiatry* 1964;10:246-61.
- 6 Breggin PR. The rights of children and parents in regard to children receiving psychiatric diagnoses and drugs. *Children & Society* 2014;28:231-41.
- 7 Breggin P. *Psychiatric drug withdrawal: a guide for prescribers, therapists, patients, and their families*. New York: Springer Publishing Company; 2013.
- 8 Belmaker RH, Wald D. Haloperidol in normals. *Br J Psychiatry* 1977;131:222-3.
- 9 Thornley B, Adams C. Content and quality of 2000 controlled trials in schizophrenia over 50 years. *BMJ* 1998;317:1181-4.
- 10 Safer DJ. Design and reporting modifications in industry-sponsored comparative psychopharmacology trials. *J Nerv Ment Dis* 2002;190:583-92.
- 11 Geddes J, Freemantle N, Harrison P, et al. Atypical antipsychotics in the treatment of schizophrenia: systematic overview and meta-regression analysis. *BMJ* 2000;321:1371-6.
- 12 Moncrieff J, Cohen D, Mason JP. The subjective experience of taking antipsychotic medication: a content analysis of Internet data. *Acta Psychiatr Scand* 2009;120:102-11.
- 13 Prien RF, Levine J, Switalski RW. Discontinuation of chemotherapy for chronic schizophrenics. *Hospital and Community Psychiatry* 1971;22:20-3.
- 14 Bola J, Kao D, Soydan H, et al. Antipsychotic medication for early episode schizophrenia. *Cochrane Database Syst Rev* 2011;6:CD006374.
- 15 Read J. A history of madness. In: Read J, Dillon J, eds. *Models of madness*, 2nd ed. London: Routledge; 2013.
- 16 Harrow M, Jobe TH. Does long-term treatment of schizophrenia with antipsychotic medications facilitate recovery? *Schizophr Bull* 2013;39:962-5.
- 17 Adams CE, Awad GA, Rathbone J, et al. Chlorpromazine versus placebo for schizophrenia. *Cochrane Database Syst Rev* 2014;1:CD000284.
- 18 Melander H, Ahlqvist-Rastad J, Meijer G, et al. Evidence b(i)ased medicine – selective reporting from studies sponsored by pharmaceutical industry: review of studies in new drug applications. *BMJ* 2003;326:1171-3.
- 19 Melander H. [Selective reporting – greater problem than selective publishing]? *Läkartidningen* 2005;102:224-5.
- 20 Leucht S, Kane JM, Etschel E, et al. Linking the PANSS, BPRS, and CGI: clinical implications. *Neuropsychopharmacology* 2006;31:2318-25.
- 21 Khin NA, Chen YF, Yang Y, et al. Exploratory analyses of efficacy data from schizophrenia trials in support of new drug applications submitted to the US Food and Drug Administration. *J Clin Psychiatry* 2012;73:856-64.
- 22 Lieberman JA, Stroup TS, McEvoy, et al. Effectiveness of antipsychotic drugs in patients with chronic schizophrenia. *N Engl J Med* 2005;353:1209-23.
- 23 Jones PB, Barnes TR, Davies L, et al. Randomized controlled trial of the effect on quality of life of secondvs first-generation antipsychotic drugs in schizophrenia: Cost Utility of the Latest Antipsychotic

- Drugs in Schizophrenia Study (CUtLASS 1). *Arch Gen Psychiatry* 2006;63:1079-87.
- 24 Harrow M, Jobe TH, Faull RN. Do all schizophrenia patients need antipsychotic treatment continuously throughout their lifetime? A 20-year longitudinal study. *Psychol Med* 2012;42:2145-55.
 - 25 Harrow M, Jobe TH. Factors involved in outcome and recovery in schizophrenia patients not on antipsychotic medications: a 15-year multifollow-up study. *J Nerv Ment Dis* 2007;195:406-14.
 - 26 Haro JM, Novick D, Bertsch J, et al. Cross-national clinical and functional remission rates: Worldwide Schizophrenia Outpatient Health Outcomes (W-SOHO) study. *Br J Psychiatry* 2011;199:194-201.
 - 27 Timimi S. Children's mental health in the era of globalisation: neo-liberalism, commodification, Mcdonaldisation, and the new challenges they pose. In: Victor Olisah (ed), *Essential Notes in Psychiatry*; 2012. Available from: <http://www.intechopen.com/books>.
 - 28 Dold M, Li C, Tardy M, et al. Benzodiazepines for schizophrenia. *Cochrane Database Syst Rev* 2012;11:CD006391.
 - 29 Tiihonen J, Lönnqvist J, Wahlbeck K, et al. 11-year follow-up of mortality in patients with schizophrenia: a population-based cohort study (FIN11 study). *Lancet* 2009;374:620-7.
 - 30 [Usage of antipsychotics among 18-64-year old patients with schizophrenia, mania or bipolar affective disorder]. København: Sundhedsstyrelsen; 2006.
 - 31 Schneider LS, Dagerman KS, Insel P. Risk of death with atypical antipsychotic drug treatment for dementia: meta-analysis of randomized placebo-controlled trials. *JAMA* 2005;294:1934-43.
 - 32 Ray WA, Meredith S, Thapa PB, et al. Antipsychotics and the risk of sudden cardiac death. *Arch Gen Psychiatry* 2001;58:1161-7.
 - 33 Ray WA, Chung CP, Murray KT, et al. Atypical antipsychotic drugs and the risk of sudden cardiac death. *N Engl J Med* 2009;360:225-35.
 - 34 Healy D, Howea G, Mangin D, et al. Sudden cardiac death & the reverse dodo verdict. *Int J Risk Safety Med* 2014;26:71-9.
 - 35 Saha S, Chant D, McGrath J. A systematic review of mortality in schizophrenia: is the differential mortality gap worsening over time? *Arch Gen Psychiatry* 2007;64:1123-31.
 - 36 Joukamaa M, Heliövaara M, Knekt P. Schizophrenia, neuroleptic medication and mortality. *Br J Psychiatry* 2006;188:122-7.
 - 37 Tenback D, Pijl B, Smeets H. All-cause mortality and medication risk factors in schizophrenia. *J Clin Psychopharmacol* 2012;32:31-5.
 - 38 Waddington JL, Youssef HA, Kinsella A. Mortality in schizophrenia. Antipsychotic polypharmacy and absence of adjunctive anticholinergics over the course of a 10-year prospective study. *Br J Psychiatry* 1998;173:325-9.
 - 39 Gøtzsche PC. *Deadly medicines and organised crime: How big pharma has corrupted health care*. London: Radcliffe Publishing; 2013.
 - 40 De Hert M, Correll CU, Cohen D. Do antipsychotic medications reduce or increase mortality in schizophrenia? A critical appraisal of the FIN-11 study. *Schizophr Res* 2010;117:68-74.
 - 41 Gøtzsche PC. [More harm than good]. *Klassekampen* 2015 March 4:25.
 - 42 Torniainen M, Mittendorfer-Rutz E, Tanskanen A, et al. Antipsychotic treatment and mortality in schizophrenia. *Schizophr Bull* 2014 Nov 24. pii: sbu164. [Epub ahead of print]
 - 43 Khan A, Faucett J, Morrison S, et al. Comparative mortality risk in adult patients with schizophrenia, depression, bipolar disorder, anxiety disorders, and attention-deficit/hyperactivity disorder participating in psychopharmacology clinical trials. *JAMA Psychiatry* 2013;70:1091-9.
 - 44 Schmidt AL. [Well-known psychiatrist: trebled dose of medicine is quite okay]. *Politiken* 2014 Sept 9.

- 45 Hallas J. [Use of several antipsychotics simultaneously and the risk of death]. *Ugeskr Læger* 2007;169:618.
- 46 Futtrup TB, Helnæs AK, Schultz H, et al. Psychotropic medication in a randomly selected group of citizens receiving residential or home care. *Dan Med J* 2014;61:A4965.
- 47 Martin K, Bégau B, Verdoux H, et al. Patterns of risperidone prescription: a utilization study in south-west France. *Acta Psychiatr Scand* 2004;109:202-6.
- 48 Marston L, Nazareth I, Petersen I, et al. Prescribing of antipsychotics in UK primary care: a cohort study. *BMJ Open* 2014;4:e006135.
- 49 Olfson M, Blanco C, Liu SM, et al. National trends in the office-based treatment of children, adolescents, and adults with antipsychotics. *Arch Gen Psychiatry* 2012;69:1247-5.
- 50 Agovino T. Antipsychotic drug use among kids soars. 2006 May 2. <http://www.drugs.com/forum/latest-drug-related-news/antipsychotic-drug-use-among-kidsssoars-34792.html> (accessed 2 Jan 2015).
- 51 Tyrer P, Kendall T. The spurious advance of antipsychotic drug therapy. *Lancet* 2009;373:4-5.
- 52 Rosenheck RA. Pharmacotherapy of first-episode schizophrenia. *Lancet* 2008; 371:1048-9.
- 53 Kahn RS, Fleischhacker WW, Boter H, et al. Effectiveness of antipsychotic drugs in first-episode schizophrenia and schizophreniform disorder: an open randomised clinical trial. *Lancet* 2008;371:1085-97.
- 54 Leucht S, Corves C, Arbter D, et al. Second-generation versus first-generation antipsychotic drugs for schizophrenia: a meta-analysis. *Lancet* 2009;373:31-41.
- 55 Tyrer P, Kendall T. The spurious advance of antipsychotic drug therapy. *Lancet* 2009;373:4-5.
- 56 Pfizer memorandum. 1989 April 26.
- 57 Lenzer J. Drug company tries to suppress internal memos. *BMJ* 2007;334:59.
- 58 Berenson A. Eli Lilly said to play down risk of top pill. *New York Times* 2006 Dec 17.
- 59 Berenson A. Lilly settles with 18,000 over Zyprexa. *New York Times* 2007 Jan 5.
- 60 Spielmans GI, Parry PI. From evidence-based medicine to marketing-based medicine: evidence from internal industry documents. *Bioethical Inquiry* 2010. DOI 10.1007/s11673-010-9208-8.
- 61 Jackson GE. An analysis of the olanzapine clinical trials – dangerous drug, dubious efficacy. 2003 March 3. <http://psychrights.org/states/Alaska/CaseOne/30-Day/ExhibitD-Olanzapine.htm> (accessed 2 Feb 2015).
- 62 Healy D. *Pharmageddon*. Berkeley: University of California Press; 2012.
- 63 Duggan L, Fenton M, Rathbone J, et al. Olanzapine for schizophrenia. *Cochrane Database Syst Rev* 2005;2:CD001359.
- 64 Larsen N-E. [New medicine has considerable adverse effects]. *Dagens Medicin* 2001 Sept 27.
- 65 McGauran N, Wieseler B, Kreis J, et al. Reporting bias in medical research – a narrative review. *Trials* 2010;11:37.
- 66 Angermeyer MC, Holzinger A, Carta MG, et al. Biogenetic explanations and public acceptance of mental illness: systematic review of population studies. *Br J Psychiatry* 2011;199:367–72.
- 67 Read J, Haslam N, Magliano L. Prejudice, stigma and “schizophrenia.” the role of bio-genetic ideology. In: *Models of Madness*, 2nd Ed. (John Read and Jacqui Dillon, eds.). London: Routledge, 2013.
- 68 Read J, Haslam N, Sayce L, et al. Prejudice and schizophrenia: a review of the “mental illness is an illness like any other” approach. *Acta Psychiatr Scand* 2006;114:303-18.
- 69 Kvaale EP, Haslam N, Gottdiener WH. The ‘side effects’ of medicalization: a meta-analytic review of how biogenetic explanations affect stigma. *Clin Psychol Rev* 2013;33:782–94.

Lebowitz MS, Ahn WK. Effects of biological explanations for mental disorders on clinicians' empathy. *Proc Natl Acad Sci USA* 2014;111:17786-90.



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Jake McGill Lynch.



Shane Clancy.



Stewart Dolin.



Candace Downing.



Cecily Bostock.



Woody Witzak and his wife Kim.



Four brave American women who fight to get the truth out about how dangerous SSRIs are. From left to right: Mathy Downing, Wendy Dolin, Sara Bostock and Kim Witzak. Los Angeles, November 2014.

Bipolar disorder

The prevalence of bipolar disorder, previously called manic depression, has increased dramatically. This epidemic has hit children particularly, where the prevalence rose 35-fold in just 20 years in the United States.¹ The fact that doctors in America make this diagnosis in children 100 times more often than in the United Kingdom² illustrates that it is usually a fake diagnosis in the United States. True mania in children is extremely rare and the explosion in bipolar is mainly caused by three things:

- 1) The diagnostic criteria have become much broader and any child with temper tantrums runs a risk of becoming diagnosed.
- 2) The US healthcare system often mandates more serious diagnoses in order to provide reimbursement, which fosters diagnostic upcoding.²
- 3) Increased use of psychiatric drugs and illegal drugs.
- 4) Mood swings are common in normal people, and the drug industry has skilfully used this fact to convince doctors that many people with depression are bipolar.³

This is a tragedy. Bipolar is often treated with antipsychotics, and, as noted in the last chapter, the rate of development of tardive dyskinesia is an alarming 5% per year.⁴ Even “mild” cases of eye blinking or grimacing can humiliate, stigmatise and isolate a child, and more severe cases may disable children with painful spasms in the neck and shoulders, abnormal posture and gait, or constant agitated body movements and a need to frantically pace.

Studies have found that between one-third and two-thirds of first episode bipolar patients had become emotionally unstable after they had used illegal drugs such as cocaine, marijuana and hallucinogens.¹ Even the American Psychiatric Association has admitted that all antidepressant treatments, including electroshock, may provoke manic episodes. A US study of nearly 90,000 patients aged five to 29 years showed that those treated with antidepressants converted to bipolar at a rate of 7.7% per year, three times greater than for those not exposed to drugs, i.e. a drug-induced conversion rate of about 5% a year.⁵ This is not only an American phenomenon. A systematic review of trials in children and adolescents

showed that 8% of people treated with antidepressants developed mania or hypomania on drug and only 0.2% on placebo.⁶ A systematic review including all ages also found an 8% rate.⁷ ADHD drugs can also induce bipolar disorder, as they are stimulants.

These studies show that there is only one important reason for the huge increase in the prevalence of bipolar disorder: doctors. It is an iatrogenic epidemic, and Whitaker has estimated that its prevalence is 250 times more common now than before the drug era.¹ Iatrogenic mania leads to a lot of misery, including extramarital sexual affairs in people who would not normally have these impulses.⁸

Lithium became the magic bullet for mania and bipolar after a physician had reported his successful treatment of ten manic patients in 1949.¹ But he forgot to mention that he killed one of them and that two others became severely ill. Lithium is highly toxic and its serum concentration needs to be monitored. Furthermore, this metal is similar to antipsychotics in its effects, which include emotional blunting, apathy, a decline in cognitive functioning and impoverished lives with little social contact.^{1,9} Patients who come off lithium may end up worse than ever before, and one study showed that the time between recurrent episodes following lithium withdrawal was seven times shorter than it was naturally.¹ Withdrawal effects include mania and depression, which mislead people into believing that lithium has been helpful.

In contrast to other psychotropic drugs, lithium perhaps reduces suicides. However, this effect is uncertain. In a meta-analysis of four small trials in patients with unipolar or bipolar mood disorders, there were unusually many suicides; six on placebo and none on lithium among only 241 and 244 patients, respectively.¹⁰ As the authors wrote themselves, the existence of only one or two moderately sized trials with neutral or negative results could materially change this finding. Moreover, the placebo group could have an artificially increased risk of suicide because of withdrawal symptoms (a cold turkey design).

Just like depression and schizophrenia, bipolar disorder also appears to have taken a more chronic course because of the drugs. Earlier, about one-third of manic patients suffered three or more episodes in their lives, but now it is two-thirds, and antidepressants and ADHD drugs may cause rapid cycling between ups and downs.¹

Another artificial psychiatric epidemic is bipolar II.¹¹ Unlike bipolar I, it has no mania or psychotic features, and the diagnostic criteria are very lenient. There only needs to be one episode of depression, and one episode of hypomania lasting

more than four days. This opens up the floodgates for treating vast numbers of patients with antipsychotic drugs causing tremendous harm at a huge cost; even the old drug quetiapine cost a staggering £2,000 a year in the UK in 2011.

The diagnosis of hypomania builds on simplistic questions like, “I drink more coffee.” Adding insult to injury, bipolar I and II are mixed together in the industry’s clinical trials so that one cannot see whether antipsychotics have any effect in bipolar II, which is supposed to be milder. A really smart marketing trick where some patients pay with their lives to increase the income for an already copiously wealthy industry.

Abilify (aripiprazole), the currently the most sold drug in the United States, is not only approved for acute treatment but also for maintenance treatment of bipolar disorder. However, in 2011, researchers could find only one trial,¹² which suffered from the usual problems – cold turkey in the placebo group, too short a duration, and low completion rate. The trial was cited by 80 publications, of which only 24 mentioned adverse events reported and only four mentioned the study’s evident limitations.

In 2001, GSK published a ghost-written clinical trial report on the use of paroxetine in bipolar adults.¹³ It was so manipulated that one of the researchers, who in addition complained that the data from his study were effectively stolen from him,¹⁴ filed a complaint of scientific misconduct. There was no effect of the drug over placebo but the published paper led people to believe there was.¹³ Furthermore, although it is dangerous to use SSRIs in such patients because of the high risk of induction of mania, and although manic symptoms was an outcome in the protocol, the published paper said nothing about this! The evidence indicated that the GSK-assigned prestigious authors on the published article never reviewed or even saw preliminary drafts of the paper, but only saw the final edited manuscript just prior to final acceptance by the *American Journal of Psychiatry*.

Like other doctors,¹⁵ psychiatrists have great difficulty facing the damaging results of their actions. They may hail the increase in bipolar as “better” diagnosis,⁸ and they will almost always postulate that the drug unmasked the diagnosis,⁸ e.g. when 12 of 60 children became bipolar on antidepressants.¹ I have heard professors of psychiatry say this at public meetings. What a perfect way of burying the problems:

We psychiatrists are good, the drugs we use are good, and our benefactors, the drug industry, are good, so anything untoward that happens to our patients are entirely due to themselves or their disease.

“Mood stabilisers”

I only use this term in inverted commas, as it is so misleading. “Mood stabilisers” are anti-epileptic drugs that don’t stabilise the mood; they suppress emotional responsiveness by numbing and sedating people.⁹ Psychiatrists have never made the precise meaning of this term clear.³ It is not surprising that doctors think anti-epileptics work for mania, as everything that knocks people down “works” for mania. Like other psychotropic drugs, anti-epileptics have many harmful effects, e.g. one in 14 on gabapentin develops ataxia, which is lack of voluntary coordination of muscle movements.¹⁶

These drugs increase the risk of suicidal thoughts and behaviour. The package insert for gabapentin (Neurontin) states that the risk of suicidality is doubled, and that, “There were four suicides in drug-treated patients in the trials and none in placebo-treated patients, but the number is too small to allow any conclusion about drug effect on suicide.” I think I can offer Pfizer a more reasonable interpretation of these data.

The trial literature in this area has been distorted to such a degree that – even with psychiatric measures – it is extreme. Gabapentin is a notorious example. The drug was only approved for very few people, those with treatment-resistant epilepsy, but Warner-Lambert, later bought by Pfizer, promoted it illegally and sold it for virtually everything, including ADHD and bipolar disorder.¹⁵

There was huge corruption and doctors willingly participated in the company’s illegal activities. Almost 90% of influential thought leaders were willing to tout gabapentin at meetings after having been updated on the company’s promotional strategies. They were handsomely rewarded for harming patients, and Warner-Lambert tracked high-volume prescribers and also rewarded them. A company executive told a salesperson about “Neurontin for everything ... I don’t want to hear that safety crap.”¹⁵

There was also huge corruption of the trial data, which involved selective reporting of trials, statistical analyses and outcomes that happened to turn out positive.^{15, 17, 18} Patients were inappropriately excluded or included in the analyses, and spin made negative results appear positive. Bias was already introduced at the design stage, highly likely deliberately, e.g. high doses were used that led to unblinding, although Pfizer recognised that unblinding due to adverse events could corrupt the study’s validity. The final layer of corruption was accomplished by ghostwriters: “We would need to have ‘editorial’ control.”

The company insisted on pressing doctors to use much higher doses of Neurontin than those approved, which meant a higher income for more harm. I

have often wondered how many people Warner-Lambert and Pfizer killed with Neurontin.

In 2010, a jury found Pfizer guilty of organised crime and a racketeering conspiracy. Six years earlier, Pfizer had paid \$430 million to settle charges that it fraudulently promoted Neurontin for unapproved uses, but the size of the fine showed that crime pays. The sales were \$2,700 million in 2003 alone, and about 90% was for off-label use.

We see similar problems with other drugs. For lamotrigine, for example, seven large, negative trials remained unpublished and invisible for the public, whereas two positive trials were published.¹⁹ Two positive trials are enough for FDA approval, and the FDA regards the rest as failed trials, though it is actually a failed drug.

In my view, anti-epileptic drugs shouldn't be used for mental disorders.

A young man's experience

Australian child psychiatrist Peter Parry has described how a young American man was destroyed by biological psychiatry.²⁰ Adam came from a screwed-up family, was physically abused by a sibling, his parents divorced young, and his mother had a lot of issues. He was 12 when first diagnosed, and had been branded with depression, anxiety and severe OCD, which had since disappeared. Unfortunately for Adam, his psychiatrist apparently worked at one of the two US centres that had gained fame by inventing and marketing a new diagnosis, childhood bipolar disorder. The psychiatrist could have asked Adam about a lot, but didn't, and within three months, Adam was on many drugs, including several anticonvulsants, several antipsychotics, a couple of antidepressants and lithium.

The documents expressed concern that Adam was suffering a degenerative neurological disorder, apparently without any consideration of the cognitive impairing effects of the heavy pharmacotherapy.

Adam's parents tended to interpret every solitary behaviour as part of the "disease" and his mother gained a lot of collateral from it. Adam began to realise that he only had this "disease" at home in the presence of two or three people who happened to be a part of his life, but he also realised that if he questioned his craziness, that was considered part of the "illness." So he really felt trapped. Many of his arguments with his mother that landed him in hospital began several hours earlier as an argument solely about wanting to stop his medicines. There was always context.

The "mitochondrial disorder thing" was a disaster. The testing and consultation

dragged out for months and at one point his mother told him they didn't know if his brain would keep "degenerating" and said: "You're gonna die".

In only four months, he gained over 25 kg and felt like a cow, in contrast to having almost qualified for the national swimming championships a year before his diagnosis. Every SSRI he was put on completely obliterated his sex drive. Early on, an SSRI caused akathisia and agitation with insomnia and intense frustration but – *although there were no core symptoms of mania such as euphoria, flight of ideas or grandiosity* – the famous psychiatrist diagnosed this as "mania."

A thousand articles about the "new disease" contained very little and generally only in passing about trauma and child abuse, and rates of physical and sexual abuse in cohorts from the two child psychiatric centres that pioneered the "disease" were far below rates in community surveys, and emotional abuse appeared to have not been considered at all.

The sessions with Adam's psychiatrist involved his mother and the psychiatrist discussing his symptoms, with little space for him to talk about the physical and emotional abuse by his brother, or the background to the conflict with his mother.

The treatment Adam received could trigger a Medical Board investigation in Australia, yet Adam's legal inquiries indicated that his treatment would be deemed "standard practice" in the United States!

Inevitably, Adam's diagnoses had an impact upon his sense of identity and familial relationships. Identity development can be severely damaged by a misdiagnosis of bipolar, where one's every thought and feeling can cause doubt as to whether it is a part of the self or some "disease."

As noted in Chapter 6, a biomedical explanation is likely to foster greater rather than less stigma and induce prognostic pessimism.

References

- 1 Whitaker R. *Anatomy of an Epidemic*. New York: Broadway Paperbacks; 2010.
- 2 Parry P, Allison S, Bastiampillai T. Reification of the paediatric bipolar hypothesis in the USA. *Lancet Psychiatry* (Published online December 1, 2014).
- 3 Moncrieff J. *The bitterest pills*. Basingstoke: Palgrave Macmillan; 2013.
- 4 Breggin PR. The rights of children and parents in regard to children receiving psychiatric diagnoses and drugs. *Children & Society* 2014;28:231-41.
- 5 Martin A, Young C, Leckman JF, et al. Age effects on antidepressant-induced manic conversion. *Arch Pediatr Adolesc Med* 2004;158:773–80.
- 6 Offidani E, Fava GA, Tomba E, et al. Excessive mood elevation and behavioral activation with antidepressant treatment of juvenile depressive and anxiety disorders: a systematic review. *Psychother Psychosom* 2013;82:132-41.

- 7 Baldessarini RJ, Faedda GL, Offidani E, et al. Antidepressant-associated mood-switching and transition from unipolar major depression to bipolar disorder: a review. *J Affect Disord* 2013;148:129-35.
- 8 Breggin P. Medication madness. New York: St. Martin's Griffin; 2008.
- 9 Breggin P. Psychiatric drug withdrawal: a guide for prescribers, therapists, patients, and their families. New York: Springer Publishing Company; 2013.
- 10 Cipriani A, Hawton K, Stockton S, et al. Lithium in the prevention of suicide in mood disorders: updated systematic review and meta-analysis. *BMJ* 2013;346:f3646.
- 11 Spence D. Bad medicine: bipolar II disorder. *BMJ* 2011;342:d2767.
- 12 Tsai AC, Rosenlicht NZ, Jureidini JN, et al. Aripiprazole in the maintenance treatment of bipolar disorder: a critical review of the evidence and its dissemination into the scientific literature. *PLoS Med* 2011;8:e1000434.
- 13 Amsterdam JD, McHenry LB. The paroxetine 352 bipolar trial: a study in medical ghostwriting. *Int J Risk Saf Med* 2012;24:221-31.
- 14 Roehr B. Professor files complaint of scientific misconduct over allegation of ghostwriting. *BMJ*. 2011;343:d4458.
- 15 Gøtzsche PC. Deadly medicines and organised crime: How big pharma has corrupted health care. London: Radcliffe Publishing; 2013.
- 16 Neurontin prescribing information. <http://labeling.pfizer.com/ShowLabeling.aspx?id=630> (accessed 9 Jan 2015).
- 17 Dickersin K. Reporting and other biases in studies of Neurontin for migraine, psychiatric/bipolar disorders, nociceptive pain, and neuropathic pain. Available online at: www.pharmalot.com/wp-content/uploads/2008/10/neurontin-dickersin-2.pdf (accessed 10 December 2008).
- 18 Vedula SS, Bero L, Scherer RW, et al. Outcome reporting in industry-sponsored trials of gabapentin for off-label use. *N Engl J Med* 2009;361:1963-71.
- 19 Ghaemi SN. The failure to know what isn't known: negative publication bias with lamotrigine and a glimpse inside peer review. *Evid Based Ment Health* 2009;12:65-8.
- 20 Parry P. Biologism in psychiatry: a young man's experience of being diagnosed with "pediatric bipolar disorder." *J Clin Med* 2014;3:334-47.

Dementia

There was a public outcry in England in 2014 when its National Health Service had decided to pay the general practitioners £55 for each case of dementia they diagnosed. The official aim was to “increase the identification of patients with dementia and to ensure that they and their families and carers get the support that they need.”¹ No one really promised too much with this political mumbo jumbo.

Dementia is not an emergency, however, so there is time to find out what care these people need. This can be done discreetly, without stigmatising people with a diagnosis many fear so much that they consider severe dementia worse than death. Sometimes, however, a diagnosis can be useful, as it may help the families understand what is going on; may relieve the demented persons from being held responsible for their actions and behaviours; and gives them an opportunity to put their legal affairs in order before they have lost their capability to think clearly or are declared mentally incapable.

One doctor talked about crossing an ethical line that has never been crossed before – cash for diagnoses – and argued that if he saw someone with memory impairment and called it dementia, he would be rewarded, but if he called it mild cognitive impairment or depression, he wouldn't get paid. He added that the diagnostic process depended on trusting that the doctor was acting in the patient's best interests, which must not be contaminated by such financial incentives.

So, is this blowing for a diagnosis hunt among the unsuspecting elderly who come to see their doctor for other reasons based on good evidence? Absolutely not! Hunting for diagnoses and rewarding people for saddling their horses and lassoing whatever they come across that looks like a demented person is screening. But no trials that could tell us whether screening for dementia does more good than harm have ever been performed.²

The UK National Screening Committee issued an exemplary report six months before the reward for each demented head was introduced. The report contains everything we need to know to make an informed decision.² After having gone through the available tests for dementia and Cochrane and other systematic reviews about types of support we might consider offering, the report concludes,

as did also the US Preventive Services Task Force, that we should not screen for dementia or mild cognitive impairment.²

I cannot recall ever having seen an official report that kills a public health initiative as completely as this one. I am sure this reflects that it wasn't written by geriatricians, but by two public health people not on industry payroll, as geriatricians recommend and use drugs that don't work for dementia.

The report leaves no doubt about how foolish the whole thing is. It dutifully mentions the possibility that "Early diagnosis of dementia could potentially allow people with dementia and their carers to plan for the future whilst the patient still retains the capacity to participate in decision making, and to start any potential treatment earlier." But what treatment? This looks like a carefully crafted pharma friendly statement, but "treatment" can be so much else than drugs, and indeed it should be.

The report says by the end that screening may harm more people by falsely alarming them than it might help. The facts are these:²

- it is only in people aged 85 and above that a positive test is likely to indicate that a person has dementia;
- drugs do not reduce the risk of progression to Alzheimer's disease;
- the effects of acetylcholinesterase inhibitors and memantine are so small that they may not be clinically meaningful;
- other types of drugs do not provide any benefits either;
- there is no evidence that any pharmacological intervention results in improvements in quality of life or well-being;
- cognitive stimulation leads to improvements in global cognitive function, self-reported quality of life and well-being, and in staff ratings of communication and social interaction;
- physical exercise, massage/touch therapies and music therapy could perhaps be helpful.

What this tells us is that we should care for our elderly, give them something meaningful and stimulating to do, and encourage them to be physically active. We have always known that.

The small effects registered in drug trials are likely spurious, as they can easily have been caused by unblinding bias because of the drugs' conspicuous side effects.

A Cochrane review of three acetylcholine esterase inhibitors, donepezil (Aricept), galantamine and rivastigmine, didn't pay attention to this problem and

concluded that, “The three cholinesterase inhibitors are efficacious for mild to moderate Alzheimer’s disease.”³

Even without considering the unblinding problem, I would dispute this conclusion. The improvement in cognitive function was 2.7 points, in the midrange of a 70-point scale. This is less than the 4 points the FDA considers the minimally relevant clinical change.⁴ We can also compare with the smallest effect that can be perceived on the Hamilton scale for depression, which is 5-6, although the maximum on this scale is only 52 (Chapter 3).

Perhaps it plays a role that the Cochrane review was written by a statistician who might not know much about drugs, e.g. she wrote that “donepezil appears to have no serious or common side effects.” This looks like a sentence in a glossy advertisement for the drug or a trial report written by Pfizer, the vendor of Aricept, and it is highly misleading. The harms are both common and serious, and 29% of the patients left the drug group on account of adverse events, as compared to only 18% in the placebo groups.³ The most common side effects of donepezil are nausea, diarrhoea, not sleeping well, vomiting, muscle cramps, feeling tired, and not wanting to eat. This is not what we would want for an old person who might already have problems with not sleeping well, feeling tired, and not wanting to eat.

The list of frequent side effects in Pfizer’s product information for Aricept is very long.⁵ Hypotension and syncope occurs in more than 1% and when old people fall, there is a considerable risk that they break their hip and die. A large Canadian cohort study showed that people who took anti-dementia drugs had almost a doubled risk of hospitalisation for syncope compared to demented people who didn’t take these drugs, and they had more pacemakers inserted and more hip fractures.⁶ Most astonishingly, more than half the patients who were admitted to hospital for bradycardia were retreated with the same type of drug after discharge!⁶ This is yet another illustration that doctors cannot handle psychotropic drugs safely. Another study, of 5,406 nursing home residents in the United States with *advanced* dementia, found that one third received cholinesterase inhibitors and one fourth memantine, also a drug used for dementia.⁷ *None* of these patients should have received these drugs.

It is particularly interesting that no benefits for society could be found for any intervention,² as we often hear about the economic burden of dementia and how important it is to intervene. These political sales pitches – which tend to coincide with general elections – are vacuous.

A long-term trial of 565 patients with mild to moderate Alzheimer’s disease that compared donepezil with placebo found no meaningful effects whatsoever,

and the authors concluded that donepezil isn't cost-effective, with benefits below minimally relevant thresholds.⁸ In contrast to other trials, it was publicly funded. This trial was excluded from the Cochrane review,³ for no good reason, as far as I can see. The outcomes after three years were similar on drug and placebo with respect to institutionalisation, progression of disability, and behavioural and psychological symptoms.⁸ The trial was published in 2004, but six years later, Eisai Medical Research's TV commercials for Aricept implied that the patients' cognitive and daily functioning, including attention, focus, orientation, communication, social interaction and engagement, will be restored to normal.⁹ "Don't wait. Talk to your doctor about Aricept," as the ads said. The FDA told the company that it had broken the law.

The English initiative is unethical, costly and harmful. It impacts negatively on their quality of life to give people a diagnosis of dementia several years before they or their loved ones would have detected anything themselves. If left alone, many of them would have died peacefully without ever having thought about the possible terror of their brain slowly becoming dissolved. They might also have preserved their dignity and independence if left undiagnosed. Above all, few of us would want to get pitied and seen upon as someone who "needs help."

We should not disrupt the lives of our elderly with useless diagnoses of dementia. Our prescription drugs are the third leading cause of death,¹⁰ and those who have been lucky enough to have survived till old age shouldn't be lured into taking drugs for dementia. We should remove the drugs from the market to ensure doctors cannot use them.

A 2007 report described what it called the gap between the number of people diagnosed with dementia and the estimated prevalence. It found for the 65-69 age group that five people per 1,000 were diagnosed, against an expected 13.² I wonder why the Goodness Industry always sees such gaps as a problem? It's not necessarily a problem. It's a gift to people that so many of them don't "need help." But of course there will always be some who could be helped by their families, but aren't, as they and their families have not yet realised that they are demented.

The criteria for the diagnosis have been broadened, and DSM-5 doesn't even use the term dementia, but speaks about mild neurocognitive disorder, which means "Evidence of cognitive decline from previous higher level of functioning in one or more cognitive domains."¹¹ I think everyone above 50 would qualify for these criteria.

Guess where the foolish idea of rewarding doctors for head-hunting demented brains came from? Yes, it was pushed by the UK's Alzheimer's Society, which is

funded by Eli Lilly,¹² one of the big pharmaceutical companies committed to the field.¹³ The society says that, “Over 800,000 people in the UK are living with dementia but only 43% of these people currently receive a diagnosis ... This prevents people from accessing the medicine and support that they not only so desperately need but deserve.” The Society also calls Lilly a “fantastic partner, and their contribution to the Alzheimer’s Society Early Diagnosis campaign is highly valued.” I am sure Lilly values the symbiotic relationship even more.

The *Financial Times* has also enlightened us:¹⁴ “The high risks involved in Alzheimer’s drug development have deterred investment, prompting David Cameron, UK prime minister, to launch a push last year through the G8 group of leading economies to find ways of incentivising research.”

Okay, I got it. A sure way of incentivising research is to give us all a diagnosis of dementia or mild cognitive impairment the first time we forget something. We might even invent childhood mild cognitive impairment. The political tactics also involved calling the initiative “case finding,” to avoid it from being shut down by the National Screening Committee. However, there is no difference between case finding and screening. Case finding *is* screening.¹⁵

The Alzheimer Society of Canada also receives industry money and it launched an awareness month campaign in 2012 with the central message, “The need for an early diagnosis.”¹⁶ According to the society’s head, “The earlier you have access to the drugs that are available ... the more likely these drugs are to help manage your symptoms and potentially even slow down the progression of the disease.” Industry speak, and utterly wrong. In Quebec they say: Cod always begins to rot from the head down. But perhaps we should invent a greyish ribbon and arrange runs for the cure, hoping we won’t get lost on the way because we have forgotten not only who we are but where we are.

The industry-sponsored trials are extremely hyped. A rhetorical analysis of 13 such trials of donepezil showed that 7 encouraged off-label use and that phrases such as “well tolerated and efficacious” were common.¹⁷ The authors also found that the average reported benefit was equivalent to a few months’ change in the progression of Alzheimer’s. And then we have not even factored in the unblinding bias in the trials.

The sales of these totally useless drugs bring in billions of dollars annually, and useless diagnostic tests can likely do the same. The FDA has approved a first drug/tracer to light up amyloid, for use with positron emission tomographic (PET) scans, which have a price tag of \$3000-\$4000.¹⁸ Eli Lilly, the manufacturer, specifies on the label that the scan does not establish a diagnosis of Alzheimer’s disease or other cognitive disorders.

So what is the scam for? It is a gross failure in this area of research that drug development is based on the hypothesis that Alzheimer's disease is caused by the formation of amyloid plaques in the brain. At least four companies, including Eli Lilly, have developed antibodies, and they substantially reduced beta-amyloid in phase 2 trials.¹⁹ However, the phase 3 trials have shown uniformly negative results for patients, but instead of giving up on a wrong hypothesis cherished for more than two decades, its acolytes have questioned everything but the hypothesis. This looks like professional hara-kiri to me, but in mental health, people can make fools of themselves and have excellent careers at the same time. It is really odd.

I have spoken to Peter Whitehouse, whose original neuroscience research led to the development of the major anti-dementia drugs. He has written a book about the myth of Alzheimer's disease where he explains that it's not a brain disease or a mental illness.²⁰ He now fights for the de-medicalisation of memory dysfunction and to make people understand that, contrary to what we have learned, the symptoms we associate with Alzheimer's are not simply a brain's molecular breakdown occurring in old age, and the much touted plaques in the brain are not related in any simple way to declining memory. Many people with plaques do not develop Alzheimer's. I thought we were on safer ground here than with the myth about the chemical imbalance for mental disorders, but alas, there is also a heavy mythology around Alzheimer's that benefits clinicians, researchers and the drug industry while harming patients. No different from psychiatry, really.

Pfizer has provided a most bizarre example of a me-again drug with donepezil.¹⁰ It was the biggest player in the lucrative market for Alzheimer's disease with over \$2 billion in annual sales in the United States alone.²¹ Four months before the patent expired, the FDA approved a new dose, donepezil 23 mg, which would be patent-protected for three more years, whereas the old doses of 5 and 10 mg were not.

The advertising was directed towards patients and contained untrue statements, but the scam worked. One would have thought that doctors, patients and relatives would have been smart enough to use either 20 or 25 mg of the drug to save money, but no. And the FDA failed us again of course. Its own medical reviewers and statisticians recommended against approval, as the 23 mg dose didn't produce a clinically meaningful benefit, whereas it caused significantly more adverse events, particularly protracted vomiting. The reviewers even added that the adverse events could lead to pneumonia, massive gastrointestinal bleeding, oesophageal rupture and death,²² but this didn't impress the director of the FDA's neurology division, Russel Katz, who overruled his scientists.

What should we then do for demented people? Take care of them! A systematic

review of 33 trials of agitated demented people showed pretty large effects of care,²³ which could be communication skills training, activities, music, touch, massage and talking to people.

We make people demented with psychotropic drugs

It's likely that all psychotropic drugs can cause chronic brain damage, which is often permanent, and the hallmark of which is impaired cognitive function (see Chapter 11). Since psychotropic drugs are so commonly used, a great deal of the dementia we see today is iatrogenic, i.e. caused by doctors. A 17-year follow up of the Framingham Heart Study found that use of antidepressants increased the risk of developing dementia by about 50%.²⁴

Benzodiazepines seem to double the risk of dementia.²⁵ At the peak of their use, 10% of the entire Danish population could be in treatment.²⁶ In 2007, it was estimated in a National Audit Report that 560,000 people in England have dementia,²⁷ which suggests that around 50,000 people in England suffer from iatrogenic dementia. I think it is safe to say that the magnitude of the problem is about this big, as the other psychotropic drugs also cause dementia, and as the usage of psychotropic drugs increases with age. The report also said that the main risk factor for dementia is age, and that cardiovascular factors are also important.²⁷ Not a single word about drugs causing dementia, of course.

I have problems accepting that age is a risk factor for anything. We cannot lower our risk by changing our birth certificate, so it's not a modifiable risk factor but an inevitable consequence of continuing to survive. If anybody insists on calling age a risk factor, I shall insist that birth is a risk factor, which with 100% certainty leads to death at some point.

Imagine if we didn't have drugs and didn't even know what they were, and a novelist wrote about a planet where people took so many drugs that every citizen could be in treatment with 1.5 drugs every day, from when they were born till they died (which is the drug consumption in Denmark). Further, that the doctors on this planet made many people demented with drugs they said would make them happy by correcting a chemical imbalance in their brain, but which harmed the citizens in many other ways than just taking away their memories. And that these doctors were then praised and received a premium for bringing in every patient they had harmed in this way with their brain-active chemicals.

Would you then not say that this plot was just too fanciful, too unrealistic and too much science fiction, and that you wouldn't be interested in reading the novel? I bet you would. But this is what we have today on planet Earth.

References

- 1 Kmietowicz Z. Doctors condemn “unethical” £55 payment for every new dementia diagnosis. *BMJ* 2014;349:g6424.
- 2 Screening for dementia. UK National Screening Committee. 2014 April.
- 3 Birks J. Cholinesterase inhibitors for Alzheimer’s disease. *Cochrane Database Syst Rev* 2006;1:CD005593.
- 4 Molnar FJ, Man-Son-Hing M, Fergusson D. Systematic review of measures of clinical significance employed in randomized controlled trials of drugs for dementia. *J Am Geriatr Soc* 2009;57:536-46.
- 5 ARICEPT® (donepezil hydrochloride) tablets. <http://labeling.pfizer.com/ShowLabeling.aspx?id=510> (accessed 31 Dec 2014).
- 6 Syncope with cholinesterase inhibitors. *Rev Prescrire* 2011;31:434.
- 7 Tjia J, Briesacher BA, Peterson D, et al. Use of medications of questionable benefit in advanced dementia. *JAMA Intern Med* 2014;174:1763-71.
- 8 Courtney C, Farrell D, Gray R, et al. Long-term donepezil treatment in 565 patients with Alzheimer’s disease (AD2000): randomised double-blind trial. *Lancet* 2004;363:2105-15.
- 9 Food and Drug Administration. Letter from Sharon M. Watson to Eisai Medical Research Inc. 2010 Feb 3.
- 10 Gøtzsche PC. *Deadly medicines and organised crime: How big pharma has corrupted health care.* London: Radcliffe Publishing; 2013.
- 11 Walter C, Edwards NE, Griggs R. Differentiating Alzheimer disease, Lewy body, and Parkinson dementia using DSM-5. *J Nurse Practitioners* 2014;10:262–70.
- 12 Alzheimer’s Society. Annual Review 2013/14.
- 13 Alzheimer’s Society. Our partnership with Lilly UK. http://www.alzheimers.org.uk/site/scripts/documents_info.php?documentID=1997 (accessed 20 Nov 2014).
- 14 Ward A. AstraZeneca and Eli Lilly to unite for Alzheimer’s drug challenge. *Financial Times* 2014 Sept 16.
- 15 McCartney M. “Case finding” in dementia is simply screening with no evidence of benefit. *BMJ* 2014;349:g4791.
- 16 Furlini L. Messing with the message: Alzheimer Society of Canada’s advice and Big Pharma’s self-interest. <http://pharmawatchcanada.wordpress.com/2012/02/06/messing-with-the-message-alzheimer-society-of-canadas-advice-and-big-pharmas-self-interest/> (accessed 1 Jan 2015).
- 17 Gilstad JR, Finucane TE. Results, rhetoric, and randomized trials: the case of donepezil. *J Am Geriatr Soc* 2008;56:1556-62.
- 18 Molchan S. A tale of two conferences. *JAMA Intern Med* 2014;174:856-7.
- 19 Granger D. Beta-amyloid: the emperor really does have no clothes. *Forbes* 2014 Dec 20.
- 20 Whitehouse P, George D. The myth of Alzheimer’s: What you aren’t being told about today’s most

dreaded diagnosis. New York: St. Martin's Press; 2008.

- 21 Schwartz LM, Woloshin S. How the FDA forgot the evidence: the case of donepezil 23 mg. *BMJ* 2012;344:e1086.
- 22 Lenzer J. FDA is criticised for licensing high dose donepezil. *BMJ* 2011;342:d3270.
- 23 Livingston G, Kelly L, Lewis-Holmes E, et al. Non-pharmacological interventions for agitation in dementia: systematic review of randomised controlled trials. *Br J Psychiatry* 2014;205:436-42.
- 24 Saczynski JS, Beiser A, Seshadri S, et al. Depressive symptoms and risk of dementia: the Framingham Heart Study. *Neurology* 2010;75:35-41.
- 25 Billioti de Gage S, Moride Y, Ducruet T, et al. Benzodiazepine use and risk of Alzheimer's disease: case-control study. *BMJ* 2014;349:g5205.
- 26 Nielsen M, Gøtzsche P. An analysis of psychotropic drug sales. Increasing sales of selective serotonin reuptake inhibitors are closely related to number of products. *Int J Risk Saf Med* 2011;23:125–32.
- 27 National Audit Office. Improving services and support for people with dementia. 2007 July 4.

Electroshock

Electroconvulsive therapy (ECT) has a long history.¹ Like its predecessors, insulin coma therapy and metrazole, which also cause convulsions, it could have a short-term effect on some psychiatric disorders, but the more I read, the more uncertain I become.

The Cochrane review on schizophrenia is from 2005.² More people improved with ECT than with placebo or sham ECT, relative risk 0.76 (95% CI 0.59 to 0.98), but this finding is uncertain. It is barely statistically significant, the trials were small (only 392 patients in 10 trials), and the authors noted that the larger the trial, the smaller the effect, which suggests that negative trials exist that haven't been published.

The review authors tried to reduce bias, e.g. they only used scores if provided by patients or independent raters or relatives, not the therapist. But there were many problems with the trials, and the authors were too generous in my view, as they only excluded trials if more than 50% of people were lost to follow-up. The authors reported that ECT was better than sham ECT for the Brief Psychiatric Rating Scale, but there were only 52 patients in the analysis, and we have no idea for how many patients data were missing or why. Further, the difference was only 6 on a scale that goes to 126, which is a good deal below what is a clinically relevant difference (see Chapter 6).

ECT was considerably less effective than antipsychotics, e.g. twice as many patients weren't improved in the ECT group, relative risk 2.18 (CI 1.31 to 3.63).

The authors didn't draw firm conclusions about any short-term benefit, and there was no evidence for any long-term benefit. Other systematic reviews have also failed to find benefits beyond the treatment period, for either schizophrenia or depression.^{3,4} In my view, it cannot be justified to use ECT for schizophrenia.

As for depression, a 2003 review found that ECT was more effective than simulated ECT (6 trials, 256 patients, effect size -0.91, 95% CI -1.27 to -0.54), corresponding to a Hamilton score difference of 10, and ECT was also better than drugs (18 trials, 1,144 participants, effect size -0.80, 95% CI -1.29 to -0.29).⁴

However, the quality of the trials was poor; most trials were small; the results would likely change materially if a few neutral studies were identified; the trials rarely used primary outcomes that were relevant for clinical practice; and the data suggested that ECT caused cortical atrophy in the brain.⁴ The authors advised that the trade-off between making ECT optimally effective in terms of amelioration of depressive symptoms and limiting the cognitive impairment should be considered. Researchers often have difficulty using plain language that readers understand. I think that what they meant was that it's uncertain whether ECT for depression does more good than harm.

Psychiatrists believe ECT can be life-saving for some people, but there are no convincing data in support of this belief,^{3, 4} whereas we know that ECT can be deadly. The UK review included four observational studies of total mortality but the results were unclear.⁴ Another, more comprehensive systematic review found a death rate of about 1 per 1000,³ which is 10 times higher than what the American Psychiatric Association says.³ As it may seem surprising that ECT can kill people, I shall tell you what a mother conveyed to me before a lecture I gave in Brisbane. The psychiatrists killed her son with ECT but the doctors succeeded in resuscitating him. When he woke up, he had severe burns after the procedure and during the next two to three months he couldn't say anything people could understand. He is permanently brain damaged and his social skills are very poor; he cannot live on his own.

Patients do not share the psychiatrists' views on ECT, particularly not in relation to its long-term harms. In 2003, the UK Royal College of psychiatrists' fact sheet stated that more than eight out of 10 depressed patients who receive ECT respond well and that memory loss is not clinically important.⁵ However, in a systematic review, the patients gave an affirmative response to the statement "electroconvulsive therapy is helpful" in only between 29% and 83% of the various studies,⁵ and the lowest satisfaction levels were obtained in studies led by patients rather than by psychiatrists.

Studies of ECT using routine neuropsychological tests have concluded that there is no evidence of persistent memory loss, but what is measured is typically the ability to form new memories after treatment (anterograde memory). Reports by patients of memory loss are about the erasing of autobiographical memories, or retrograde amnesia, and they are pretty damning.⁵ With a strict definition of memory loss, between 29% and 55% of the patients are affected, and with looser criteria, the range goes from 51% to 79%. Other studies also suggest that ECT may cause permanent brain damage.³ In the 1940s, it was acknowledged that ECT

works because it causes brain damage and memory deficits, and autopsy studies consistently found brain damage, including necrosis.³

To say, as the psychiatrists who authored a Cochrane review of depressed elderly did,⁶ that, “Currently there is no evidence to suggest that ECT causes any kind of brain damage, although temporary cognitive impairment is frequently reported” and that “ECT seems to be a safe procedure” is plainly wrong. The official guidance for general practitioners in Denmark on depression is even worse. It states that, “Many have an unfounded fear of ECT treatment, although there is no evidence that the treatment causes brain damage; in fact, there is strong evidence that new nerve cells are formed in response to treatment.”⁷ What the guidance really says is that ECT causes brain damage, as new nerve cells form in response to brain damage!

Some leading psychiatrists admit that ECT is one of the most controversial treatments in medicine,⁸ and the UK National Institute for Health and Care Excellence (NICE) recommends that ECT only be used to achieve rapid and short-term improvement of severe symptoms, after an adequate trial of other treatments has proven ineffective, or when the condition is considered to be potentially life threatening, in individuals with severe depressive disorders, catatonia, and a prolonged or severe manic episode. The Royal College of Psychiatrists appealed this decision, as the college found it would prevent patients from receiving ECT who might benefit, but the appeal was rejected.⁵

ECT doesn't seem to have long-lasting beneficial effects, whereas it causes permanent and serious harm. It “works” by making people confused and it destroys people's memories, which are what define us as humans.

Repeated audits by the Royal College of Psychiatrists showed that many hospital trusts failed to adhere to the college's standards,⁵ e.g. one audit found that only a third of ECT clinics met the standards.⁴ There are also wide variations in clinical practice and in rates of usage.³⁻⁵ In Denmark, forced treatment with ECT quadrupled in just seven years in the 1990s, but forced treatment is immensely unpleasant, the patients are very scared, it often elicits colossal bitterness and anger, and it is perceived by the patients as a breach of trust.⁹

In my view, ECT should be forbidden, as it destroys people and is widely abused as forced treatment. As long as this hasn't happened, we must ensure at the very least that no one can be forced to get it (see Chapter 15).

I saw a very moving documentary in the Danish Parliament about Mette, a nurse who had heard voices since she was eight years old and had been a psychiatric patient for 15 years.¹⁰ The film won the best foreign film award at

Mad in America's International Film Festival in 2014. Mette had been diagnosed with paranoid schizophrenia and had received vast amounts of medicine, 150 electroshock treatments and a disability living allowance. Mette was stigmatised and surrounded by prejudice but after she decided to reclaim her own life and leave psychiatry, she achieved some of her greatest goals. Mette and the filmmaker, and many current and previous patients, psychologists and psychiatrists were in the audience and I asked why on earth her psychiatrists had continued exposing her to all these electroshocks when they so obviously didn't help her. No one was able to give me a satisfactory reply.

Mette's story illustrates what I mean by psychiatrists' abuse of forced treatments. Even when they so clearly don't work, psychiatrists continue out of despair to try them in an endless progression, which is harmful for the patients' brains and personalities.

Some psychiatrists have never used electroshock. One is Ivor Browne from Ireland who reserved the therapy for patients in lifethreatening situations, which he never encountered in his long career.¹¹ The fact that ECT is never used in Trieste in Italy also demonstrates that ECT can be dispensed with.

ECT is about as primitive and unspecific a "therapy" as one can think of. No one who experiences computer problems would dare send electricity into the computer that changes what is stored there and how the programmes function. Our brain is the most extraordinary "computer" we can imagine and ECT surely induces changes, which is the very rationale for its use. I therefore wonder why anyone would dare to use ECT.

You many think it's too easy for me to dismiss both ECT and drugs as treatments for depression, as I am not a psychiatrist and not depressed. So let me tell you this. I have known many people with depression, including some very close to me, and have seen how damaging the treatments have been. I have also studied the scientific literature. Therefore, should I one day suffer from serious depression, the only treatment I would accept is psychotherapy.

References

- 1 Whitaker R. Mad in America. Cambridge: Perseus Books Group; 2002.
- 2 Tharyan P, Adams CE. Electroconvulsive therapy for schizophrenia. Cochrane Database Syst Rev 2005;2:CD000076.
- 3 Read J, Bentall R. The effectiveness of electroconvulsive therapy: a literature review. Epidemiol Psychiatr Soc 2010 Oct-Dec;19:333-47.
- 4 UK ECT Review Group. Efficacy and safety of electroconvulsive therapy in depressive disorders: a systematic review and meta-analysis. Lancet 2003;361:799-808.
- 5 Rose D, Fleischmann P, Wykes T, et al. Patients' perspectives on electroconvulsive therapy: systematic

- review. BMJ 2003;326:1363.
- 6 Stek ML, Wurff van der FFB, Hoogendijk WJG, et al. Electroconvulsive therapy for the depressed elderly. Cochrane Database Syst Rev 2003;2:CD003593.
 - 7 [Clinical guidance for general practice: unipolar depression, diagnosis and treatment]. Danish Society for General Practice 2010.
 - 8 Carney S, Geddes J. Electroconvulsive therapy. BMJ 2003;326:1343-4.
 - 9 Frich M. [Use of electroshocks quadrupled]. Jyllands-Posten 1998 May 19.
 - 10 Mettes Stemme (Mette's Voice). <http://madinamericainternationalfilmfestival.com/mettes-stemme-mettes-voice/>(accessed 9 Dec 2014).
 - 11 Browne I. The writings of Ivor Browne. Cork: Atrium; 2013.

Psychotherapy and exercise

Do as much as possible for the patient, and as little as possible to the patient

BERNARD LOWN, NOBLE PEACE PRIZE WINNER

Psychotherapy is a very broad concept. It comes in many varieties, there are many schools of thought, and there are thousands of trials. To start with, I shall point out some key issues.

The underlying theories and the applied methods are rarely crucial. It is more important that the therapist is intelligent and empathic and is able to establish a good relationship with the patient, which makes it possible to arrive at a mutual understanding of what the problems are, what caused them, and what the best way forward would be. When we respect the patients and treat them as reasonable beings, they will respect themselves, which is the first important step towards healing.

Psychiatric drugs keep patients locked in the patient role and change their personality in ways that are often highly undesirable for the healing process and also disliked by the patients.¹⁻⁵ Many patients describe how the drugs take their feelings away and that they care less about others, which makes it more difficult for them to learn to cope with life's challenges. Psychotherapy is the opposite of this. It is about teaching people to overcome the challenges they face rather than numbing them with drugs.

Psychiatrists often assume that they should be the leaders of multidisciplinary teams, as they are the only ones who can prescribe drugs. However, considering the harms they create with their many diagnoses and drugs, they should generally not lead such teams. The focus should be on avoiding psychiatric diagnoses and drugs as much as possible and on understanding the patients. Psychologists have this expertise, whereas few psychiatrists get trained in psychotherapy today.

In the long term, the harmful effects of psychiatric drugs exceed their benefits. This is not so for psychotherapy. A good psychotherapist can sometimes achieve remarkable results that may last for a lifetime with no side effects, and although

there are bad therapists and it can go wrong, it is clear to me that the benefits of psychotherapy outweigh any harms. Furthermore, therapy can often be given in groups, and it therefore need not be expensive, either for the individual or for society, in contrast to drugs.

Politicians traditionally argue that there are so many people with mental disorders that they cannot afford to provide psychotherapy to the extent they would like, but why don't we hear the same politicians criticise the waste of so much money on far too expensive drugs that are no better than other, off-patent drugs? Psychiatry's current tunnel vision with its sole focus on drugs is immensely expensive for our national economies, as this strategy has dramatically increased the number of chronically ill people and the number of people on disability pensions.⁴ It would be far cheaper and lead to considerable improvements in mental health, if we used drugs very little and provided people with the psychotherapy they need.

We should remove all reimbursements for psychiatric drugs and use the vast amounts of money saved to offer psychotherapy for a small fee, at no additional cost to society.

There is a shortage of professionals who can provide psychotherapy. However, many people are helped by paraprofessionals who can be experienced patients (recovery mentors), residents in the area, or students.⁶ They ground their therapeutic relationship not so much in established theory or empirical research but in day-to-day experience and common sense. They have usually had some degree of training, and are connected to and supervised by professionals.⁶ Self-help programmes (see below) and the help of relatives and friends can also have beneficial effects on mental disorders and they don't cost anything.

Since psychiatrists who prescribe pills earn much more money than those who practise psychotherapy, we will need to remove this incentive by paying handsomely for psychotherapy.

Some interventions are so simple that we don't need to prove in randomised trials that they work. Much of what we can do to help people with mental problems is in this category, at least when we deal with relatively mild cases. In more serious cases, we need trials to guide us.

Psychotherapy for anxiety and depression

Anxiety is a psychological problem and all parents have experienced that they can

lessen their children's anxiety quite easily. It doesn't require a psychotherapeutic education or any knowledge of receptors and transmitters in the brain to do this, it only requires that you take an interest in other people.

Shyness is a good example. People have always drunk alcohol to lessen their shyness in social situations, particularly before attempting to make successful contact with the opposite sex. In psychiatry, this common human condition is called social phobia. It was a rare disease until the drug companies dubbed it social anxiety disorder, which sounded better for marketing purposes, and invented a market for it. They boosted sales tremendously, aided by PR firms and their paid allies among psychiatrists and patient organisations.⁷ The pool of patients went up from about 2% to 13% – one in every eight people – handsomely helped by the ridiculous criteria in DSM that broadened over time.

In this way, shyness became a disease to be treated with a drug every day. I don't deny that some people are disabled by their shyness, but when industry marketing and corruption take over, it becomes the many that are treated and not the few.

Exposure therapy can be highly effective for people with anxiety, including those with obsessive compulsive disorder. It puts participants in direct and prolonged contact with the feared situations and I shall give an astounding example, which I experienced as a young student at the University of Uppsala in Sweden. I lived in a hostel and the student across the corridor, Bengt, was very shy. I couldn't recall I had ever seen him with a woman. But one day Bengt started to attend a course with a fine name I have forgotten. He explained that they went through various exercises, one of which involved taking off their shirts while sitting on the floor and moving close together so that their bare backs touched each other. When we heard about these bodily exercises, the corridor's most witty guy invented the name "the hugging course."

We found it quite amusing and a bit laughable, but that was before we saw the remarkable result of this group-based psychotherapy. Sometimes, when I met Bengt in the kitchen in the morning at weekends, he prepared breakfast for two and smiled in a subtle way as he walked back to that night's trophy. Now and then, I caught a glimpse of the woman and noted that it was a new one each time. When I asked him why he didn't stick to the one he already had, he just smiled again. Our shy friend had developed into a formidable Don Juan, which I appropriately nicknamed him.

There is no doubt that shyness should be treated with psychotherapy and not drugs. A 24-week trial randomised 375 patients with social phobia to sertraline or to gradual exposure to the feared symptoms.⁸ It found a similar effect of exposure

and sertraline, but during an additional six-month follow-up the exposure group continued to improve, whereas the patients from the sertraline group did not. This is what we would expect. Put people on drugs and they don't learn anything about how to cope with their anxiety. In contrast, psychotherapy usually has lasting effects.

A Cochrane review of 41 trials in children and adolescents with mild to moderate anxiety showed very large effects from cognitive behavioural therapy.⁹ The odds ratio for remission, compared with waiting-list controls, was 0.13 (95% CI 0.09 to 0.19), and the reduction in anxiety symptoms had an effect size of -0.98 (95% CI -1.21 to -0.74). There was no difference between individual, group and family/parental formats, and cognitive behavioural therapy wasn't better than other psychological therapies or treatment as usual. The outcome was assessed blindly in 32 of the 41 trials. The effect was smaller in large trials but the largest trials also showed large effects.

A Cochrane review of psychological therapies for generalised anxiety disorder in adults also found positive effects.¹⁰

A systematic review found a substantial effect on depression from psychotherapy, effect size 0.67, but the effect decreased with an increasing number of patients and after adjustment for this it was 0.42.¹¹ A 2013 trial of 469 patients with depression in general practice that had not responded to drugs found that the addition of cognitive behavioural therapy had a reasonable benefit, effect size 0.53.¹²

A Cochrane review of anxiety and depressive disorders did not find a difference between the results obtained by paraprofessionals and professionals (psychiatrists or psychotherapists), effect size 0.09, 95% CI -0.23 to 0.40.⁶ The paraprofessionals performed far better than a control condition, odds ratio 0.34, 95% CI 0.13 to 0.88. These results agree with those from numerous other studies.^{6, 13} Even people without any professional education or supervision can be very helpful for patients with mental disorders,¹³ which begs the question: Can patients also help themselves? Indeed they can.

An intervention with the funny name bibliotherapy relies on written texts, computer programs, or audio/video-recorded material.⁶ Meta-analyses have found effect sizes ranging from 0.53 to 0.96 for various problems, including difficulties with sleep, sexual problems, depression, anxiety, and other mood disturbances. Psychoeducation is part of many treatment strategies, and it might also be considered a kind of bibliotherapy.⁶ A Cochrane review of self-help where printed materials, audio or video recordings, computers or the Internet were used to teach adult patients behavioural or cognitive behavioural therapy for anxiety

found a clear effect compared with no intervention, effect size 0.67, 95% CI 0.55 to 0.80 (72 studies and 4,537 participants), but it seemed to be somewhat less effective than face-to-face therapy.¹⁴

A systematic review that compared sleeping pills of the benzodiazepine type with behavioural therapy found only one randomised trial where people had used a sleeping diary.¹⁵ The authors therefore included 20 before-and-after studies in their review and the results were pretty convincing despite the weak design. Both types of treatment had large effects, with mean effect sizes greater than 0.80, and the only difference was that people fell asleep faster on behavioural therapy than on drugs.

Simply being kind and empathic may also help people fall asleep. I once knew a nurse who – when she had time – didn't give old people at the hospital ward sleeping pills but caressed them gently on the neck, whereupon they relaxed and fell asleep. When I was young, I attended a course where we learned how to relax and fall asleep while resting our head on our arms across the table. I was convinced it would fail, but I fell asleep very quickly. Later, I demonstrated the technique to a friend who had trouble falling asleep. My demonstration of how to relax in all muscles and breathe slowly and deeply while listening to your own, calm hypnotic voice was so effective that I fell asleep while I was still talking!

Psychotherapy for obsessive compulsive disorder

Obsessions are often related to thoughts about microbial contamination, and typical compulsions are cleaning, washing, praying, counting or checking the same things many times in a pathological way.¹⁶ The quality of life is often severely affected, also for the family that can suffer immensely from the person's neurotic behaviour, as it was called not so long ago. It is therefore fortunate that behavioural and cognitive behavioural therapy are highly effective.

A Cochrane review of trials in adults found that psychotherapy resulted in far fewer symptoms than if the patients had received treatment as usual (waiting list controls), effect size -1.24, 95% CI -1.61 to -0.87 (seven trials and 241 patients).¹⁶ This effect was obtained despite the fact that, in all but one trial, some participants in both groups were concurrently receiving drugs.

Another Cochrane review, also in adults, found an even larger effect compared with a waiting list condition, effect size -1.65, 95% CI -2.62 to -0.67 (calculated by me), but this result was based on only three small trials with 87 patients in

total.¹⁷ The review furthermore showed that psychotherapy was better than antidepressants (clomipramine or sertraline), effect size -0.36, 95% CI -0.72 to 0.00 (calculated by me) (three trials with 118 patients).

In contrast to psychotherapy, the effect of SSRIs is substantially smaller for obsessive compulsive disorder. A Cochrane review of 17 trials and 3,087 adults found an effect size of -0.46, 95% CI -0.55 to -0.37 (calculated by me).¹⁸

Thus, there is no doubt that obsessive compulsive disorder should be treated with psychotherapy and not with drugs. However, in 2014 the chairwoman for the Danish OCD association argued that patients with OCD, including children, should take antidepressants and should ignore the tragic stories about people who had committed suicide while on SSRIs.¹⁹ She claimed that SSRIs protect against suicide, also when used in children, and argued that to ask a patient with OCD not to take the drug would be the same as asking a diabetes patient not to take insulin. She said that therapy and follow-up by the doctor was needed to “support the drug effect the pills have.”

Her article might as well have been written by Lundbeck. I therefore asked her whether her association accepted industry money, which wasn't the case. The association allowed me to publish an article in their journal where I explained why antidepressant drugs should be avoided in children and young people.²⁰

My reply caused a former patient to write her story, which is all too typical.²¹ Aged 16, with severe OCD, her psychiatrist had given her a pill saying it stabilised serotonin in the brain. Six months later, she got suicidal thoughts for the first time. Six years later, she was still on drugs but her psychiatrists were only interested in renewing her prescriptions. She persuaded her fourth psychiatrist to taper off the drug, and then one evening she suddenly noticed the beautiful and joyful song of the birds, for the first time in years. The happiness she felt was indescribable. She hadn't felt any progress before she dropped the pills and declared war on OCD, helped by her psychologist. Another psychologist told me that his name had been deleted from the list of therapists at the OCD association and that he suspected it was because he was against drugs. He also suspected that industry money was involved, but it seems that the harmful myths the industry and psychiatrists have created together are so powerful that it is not always necessary to corrupt patient organisations with money. Words are enough.

The former patient ended her story with a brilliant idea. She asked if it was fair that pills were subsidised when psychotherapy wasn't and suggested:

Couldn't we introduce an arrangement where we got money by saying

no to happy pills, which we could then use to pay the psychologist?

Psychotherapy for schizophrenia

The good results obtained by psychiatrist Loren Mosher by avoiding using antipsychotics (see Chapter 6) were threatening to other psychiatrists.²² His staff treated people with empathy and respect, with as little medicine as possible, and they had fewer relapses and functioned better in society in terms of holding a job and attending school than those receiving antipsychotics. None of his staff were psychiatrists and it was offensive to these professionals to suggest that ordinary people could help crazy people more than psychiatrists with their “wonder” drugs. But Mosher was the chief of the Center for Studies of Schizophrenia at the US National Institute of Mental Health, so it wasn’t obvious how he could be stopped. The NIMH clinical project committee therefore raised doubts about the scientific rigour of his research team and reduced the funding for Mosher’s project to such a low level that it was a financial kiss of death.²²

This is the standard method used in healthcare by the silverbacks when the results of a project threatens the status quo and their carefully pruned self-image. Mosher tried to get around the obstacle by applying for funding from the NIMH division that dealt with social services, and the peer review committee was very enthusiastic. However, the clinical projects committee killed his project right off, as it threatened the very credibility of academic psychiatry with its medical model of drug therapy. This was done with derogatory remarks about the study’s postulated “serious flaws” and with the fatal blow that further funding would only come forward if Mosher stepped down so that the committee could redesign the project with another investigator!

This is one of the ugliest manoeuvres I have ever seen being used against a high-ranked investigator, and a bitter Mosher said 25 years later that, “If we were getting outcomes this good, then I must not be an honest scientist.”²² When the committee had killed the project for good, and it was no longer risky to be a little honest, it came with a remarkable admission:

“This project has probably demonstrated that a flexible, community based, non-drug residential psychosocial program manned by non-professional staff can do as well as a more conventional community mental health program.”²²

They made Mosher an outcast and threw him out of the NIMH three years later. Others in America who dared question the merits of neuroleptics learned quickly that this would not advance their career, and NIMH did not allot any more funds to this type of project.⁴

However, Mosher's approach was adopted in several European countries, and the physicians reported similarly good outcomes as Mosher's, with minimal use of drugs.²² Many years later, psychiatrist John Bola analysed the follow-up data from Mosher's study that had gathered dust in the archives and discovered that they were even more positive than what Mosher had published.²²

Mosher's results have been confirmed in Finland.⁴ It began in 1969, when psychiatrist Yrjö Alanen instructed his staff to listen to patients, who despite paranoid utterances told meaningful stories, often about their difficult past. The core treatment was group family therapy where the focus was not on psychotic symptoms but on preserving hope by talking about the patient's past successes to help strengthen the patient's grip on life. Unfortunately, today the Finnish guidelines are rather mainstream in the sense that they call for the patients to be kept on drugs for at least five years after a first episode, which is a prescription for disaster.

In Lapland, Jaakko Seikkula continued and the method became known as Open Dialogue therapy. A study of 75 patients, 30 of whom had schizophrenia and 45 other psychoses, showed that two-thirds were never exposed to antipsychotics, and after five years, 80% were working, in school, or looking for work.⁴ Seikkula has explained to Whitaker that if people are put on medication, they lose their grip on life and can no longer take care of themselves. The idea is therefore to limit the use of psychotropic drugs by having open meetings where participants share their thoughts freely with each other. The language used at these meetings is very different from the language therapists usually use, and they listen more to the patients' experiences and ideas and also to the family.⁴ As Danish philosopher Søren Kierkegaard wrote in the 1800s, we should meet our fellow human beings where they are, and this also applies to psychiatry.

An important part of the method is that the team organises a meeting within 24 hours if a psychosis is on its way. Only two to three new cases of schizophrenia appear each year in western Lapland, a 90% drop since the early 1980s, which is because the duration of the psychotic episodes rarely exceeds the six months required for the diagnosis. Spending on psychiatric services also dropped.

Psychotherapy for schizophrenia seems to be cost-effective. According to a NICE guideline from 2012, a systematic review of the economic evidence showed that cognitive behavioural therapy improved clinical outcomes at no additional cost, and economic modelling suggested that it might result in cost savings because of fewer hospital admissions.²³

There have also been remarkable results in the United States.⁴ The most severely disturbed children in California, with histories of sexual and physical abuse and horrible neglect, which all other institutions had given up on, were assigned a mentor in a shelter who gave the kids their personalities back by withdrawing the often multiple drugs they were on and by establishing emotional relationships with them, which isn't possible to do with a heavily drugged person. When a child arrived, the staff didn't ask what was wrong with it but what had happened to it. Their behaviour had often worsened after they were put on drugs.

In randomised trials of getting people back to life, the Individual Placement and Support model, which is a highly defined form of supported employment, has had dramatic effects.²⁴ A small trial of a novel seven-month psychosocial treatment designed to prevent a second episode of psychosis in first episode remitted patients is also interesting.²⁵ After 12 months, there were fewer relapses, compared to usual care. This effect was not sustained long-term, perhaps because the patients in the psychosocial group adhered more to their medication than those in the usual care group.

It wasn't until 2014 that the first trial of psychotherapy in people with schizophrenia who were not on antipsychotic drugs was published.²⁶ The authors had chosen patients who had all declined to be treated with drugs. The effect size was 0.46 compared to treatment as usual, which is about the same of that seen in trials comparing antipsychotics with placebo, which is a median of 0.44.^{26, 27} This drug effect is much exaggerated, however, because of unblinding bias and because serious harms were inflicted on patients in the placebo groups who got exposed to a cold turkey (see Chapter 6). This means:

The effect of psychotherapy is likely better than the effect of antipsychotics.

Peter Breggin has described what a remarkable effect empathy, caring and understanding can have in patients with severe schizophrenia.²⁸ As an 18-year old college freshman without any mental health training, he volunteered at a state mental hospital and approached the patients as he would want himself to be approached, with care and concern, and with a desire to get to know the patients and finding out what they needed and wanted. He was immediately appalled by how abused and humiliated the patients were by the authoritarian and sometimes violent staff, and by the brain-damaging treatments they used, including insulin coma therapy, electroshock and lobotomy, all the while he was told that these treatments “killed bad brain cells,” which he found unlikely of course.

Breggin developed an aide programme in which 15 students were assigned their own patient among those who were chronic inmates considered beyond help – burnt out schizophrenics – who had not yet been subdued by chlorpromazine. He and his colleagues were able to help 11 of the 15 patients to return home or to find improved placements in the community. During the next one to two years only three returned to the hospital. The programme drew national headlines and was praised as an important innovation by the Joint Commission on Mental Illness and Health in 1961. This was the last psychosocially oriented document to be issued by NIMH. Ever since, the focus has been on co-operative efforts with the drug industry to promote biochemical explanations and drugs.

By taking an interest in the patients in his private practice, instead of destroying them with drugs and electroshock and pigeonholing them with diagnostic labels, Breggin never burnt out but has continued to enjoy his work long after normal retirement age. In my opinion, this is the recipe for becoming a successful psychiatrist. Since 1968, when Breggin started his practice, he has never put a patient on a psychiatric drug except for occasional sleeping pills during a crisis or withdrawal.

In the study from the early 1970s, where Rosenhan and seven other normal people were admitted to a psychiatric hospital because they said they heard voices (see Chapter 2), interesting observations were made about contacts between staff and patients.²⁹ The staff generally avoided continuing conversations patients had initiated, and by far their most common response consisted either of a brief reply to the question, offered while they were “on the move” and with the head averted, or no reply at all. The encounter frequently took the following bizarre form:

Pseudopatient: “Pardon me, Dr X, could you tell me when I am eligible for grounds privileges?”

Physician: “Good morning, Dave. How are you today?” (moves off without waiting for a response).

Physicians, especially psychiatrists, were even less available than the rest of the staff. They were rarely seen on the wards. Quite commonly, they would be seen only when they arrived and departed, with the remaining time being spent in their offices or in the cage.

This happened a long time ago and it might not be representative of today’s psychiatry, but the problem Rosenhan described is still with us. Psychiatrists have found out recently that if they talk more with their patients with schizophrenia, there is less need of forced treatment. Professor Merete Nordentoft conveyed this

positive experience in a TV debate with me. I wonder, however, why this is something that psychiatrists should rediscover. Shouldn't they have known this all along? I also wonder why it is extremely rare that anyone uses the principle of Open Dialogue.

Exercise

Exercise is good for many things, including self-esteem, so it could be viewed as a type of psychotherapy. Whatever its effects on mental disorders it is better to encourage people to exercise than to take psychiatric drugs. Exercise is recommended in the United Kingdom for mild depression, and GPs may write a prescription for it, typically for half a year.⁴ The patients have found that exercise helps them focus on their health rather than on their sadness and to stop thinking of themselves as “victims.”

Schoolteachers have good experiences with exercise, e.g. sending the kids for exercise with music between classes, which they say can have dramatic effects on those who are at risk of getting an ADHD diagnosis. It makes them calmer and more attentive in class afterwards, which is an experience many of us have had.

Exercise also has an effect on depression. There are few longterm comparisons between SSRIs and exercise, but those that exist are interesting. In a four-month trial of 156 patients with major depression, the effect was similar for exercise and sertraline, but six months later only 30% of the patients in the exercise group were depressed, as compared with 52% in the sertraline group and 55% in a group that was randomised to both exercise and sertraline.³⁰ The poor result in the combined group was expected, as it is difficult to perform psychotherapy in drugged people. The differences were seen despite a low treatment contrast: 64% of patients in the exercise group and 66% in the combination group reported that they continued to exercise, but 48% of the sertraline patients also initiated an exercise programme.

A Cochrane review of exercise found an effect on depression that was very similar to that reported for SSRIs and for psychological therapy.³¹ A systematic review of exercise in old people with depression found a similar effect (effect size of 0.34).³²

There are also trials of exercise as prophylaxis in general populations of children. In a Cochrane review of trials that compared vigorous exercise with no intervention, six studies reporting anxiety scores showed an effect of exercise (effect size -0.48, 95% CI -0.97 to 0.01).³³ Five studies reporting depression scores were also positive (effect size -0.66, 95% CI -1.25 to -0.08). However, the

trials were generally of low methodological quality and highly heterogeneous with regard to the population, intervention and measurement instruments used.

References

- 1 Kessing L, Hansen HV, Demyttenaere K, et al. Depressive and bipolar disorders: patients' attitudes and beliefs towards depression and antidepressants. *Psychological Medicine* 2005;35:1205-13.
- 2 Healy D. *Let them eat Prozac*. New York: New York University Press; 2004.
- 3 Breggin P. *Medication madness*. New York: St. Martin's Griffin; 2008.
- 4 Whitaker R. *Anatomy of an Epidemic*. New York: Broadway Paperbacks; 2010.
- 5 Read J, Cartwright C, Gibson K. Adverse emotional and interpersonal effects reported by 1829 New Zealanders while taking antidepressants. *Psychiatry Res* 2014;216:67-73.
- 6 Boer PCAM, Wiersma D, Russo S, et al. Paraprofessionals for anxiety and depressive disorders. *Cochrane Database Syst Rev* 2005;2:CD004688.
- 7 Moynihan R, Cassels A. *Selling Sickness: how the world's biggest pharmaceutical companies are turning us all into patients*. New York: Nation Books; 2005.
- 8 Haug TT, Blomhoff S, Hellström K, et al. Exposure therapy and sertraline in social phobia: 1-year follow-up of a randomised controlled trial. *Br J Psychiatry* 2003;182:312-8.
- 9 James AC, James G, Cowdrey FA, et al. Cognitive behavioural therapy for anxiety disorders in children and adolescents. *Cochrane Database Syst Rev* 2013;6:CD004690.
- 10 Hunot V, Churchill R, Teixeira V, et al. Psychological therapies for generalised anxiety disorder. *Cochrane Database Syst Rev* 2007;1:CD001848.
- 11 Cuijpers P, Smit F, Bohlmeijer E, et al. Efficacy of cognitive-behavioural therapy and other psychological treatments for adult depression: meta-analytic study of publication bias. *Br J Psychiatry* 2010;196:173-8.
- 12 Wiles N, Thomas L, Abel A, et al. Cognitive behavioural therapy as an adjunct to pharmacotherapy for primary care based patients with treatment resistant depression: results of the CoBaIT randomised controlled trial. *Lancet* 2013;381:375-84.
- 13 Kahneman D. *Thinking, fast and slow*. London: Penguin Books; 2012.
- 14 Mayo-Wilson E, Montgomery P. Media-delivered cognitive behavioural therapy and behavioural therapy (selfhelp) for anxiety disorders in adults. *Cochrane Database Syst Rev* 2013;9:CD005330.
- 15 Smith MT, Perlis ML, Park A, et al. Comparative meta-analysis of pharmacotherapy and behavior therapy for persistent insomnia. *Am J Psychiatry* 2002;159:5-11.
- 16 Gava I, Barbui C, Aguglia E, et al. Psychological treatments versus treatment as usual for obsessive compulsive disorder (OCD). *Cochrane Database Syst Rev* 2007;2:CD005333.
- 17 O'Kearney RT, Anstey KJ, von Sanden C. Behavioural and cognitive behavioural therapy for obsessive compulsive disorder in children and adolescents. *Cochrane Database Syst Rev* 2006;4:CD004856.
- 18 Soomro GM, Altman DG, Rajagopal S, et al. Selective serotonin re-uptake inhibitors (SSRIs) versus placebo for obsessive compulsive disorder (OCD). *Cochrane Database Syst Rev* 2008;1:CD001765.
- 19 Broni B. [Another view on antidepressant drugs]. 2014 Jan 28. <http://www.ocd-foreningen.dk/stot-os/bliv-frivillig/7-artikler/243-en-anden-vinkel> (accessed 4 Feb 2014).
- 20 Gøtzsche PC. [Antidepressant drugs should be avoided in children and young people]. *OCD-nyt* 2014;1:7.

- 21 Mikkelsen LG. [Happy pills as Müsli sprinkle]. <http://ocd-inderstinde.dk/debat.2.html>. 2014 Feb 1 (accessed 4 Feb 2014).
- 22 Whitaker R. Mad in America. Cambridge: Perseus Books Group; 2002.
- 23 Freeman D, Freeman J. At last, a promising alternative to antipsychotics for schizophrenia. The Guardian 2014 March 7.
- 24 Baksheev GN, Allott K, Jackson HJ, et al. Predictors of vocational recovery among young people with first-episode psychosis: findings from a randomized controlled trial. Psychiatr Rehabil J 2012;35:421-7.
- 25 Gleeson JF, Cotton SM, Alvarez-Jimenez M, et al. A randomized controlled trial of relapse prevention therapy for first-episode psychosis patients: outcome at 30-month follow-up. Schizophr Bull 2013;39:436-48.
- 26 Morrison AP, Turkington D, Pyle M, et al. Cognitive therapy for people with schizophrenia spectrum disorders not taking antipsychotic drugs: a single-blind randomised controlled trial. Lancet 2014;383:1395-403.
- 27 Leucht S, Cipriani A, Spineli L, et al. Comparative efficacy and tolerability of 15 antipsychotic drugs in schizophrenia: a multiple treatments meta-analysis. Lancet 2013;382:951–62.
- 28 Breggin P. Psychiatric drug withdrawal: a guide for prescribers, therapists, patients, and their families. New York: Springer Publishing Company; 2013.
- 29 Rosenhan DL. On being sane in insane places. Science 1973;179:250-8.
- 30 Babyak M, Blumenthal JA, Herman S, et al. Exercise treatment for major depression: maintenance of therapeutic benefit at 10 months. Psychosom Med 2000;62:633–8.
- 31 Cooney GM, Dwan K, Greig CA, et al. Exercise for depression. Cochrane Database Syst Rev 2013;9:CD004366.
- 32 Bridle C, Spanjers K, Patel S, et al. Effect of exercise on depression severity in older people: systematic review and meta-analysis of randomised controlled trials. Br J Psychiatry 2012;201:180-5.
- 33 Larun L, Nordheim LV, Ekeland E, et al. Exercise in prevention and treatment of anxiety and depression among children and young people. Cochrane Database Syst Rev 2006;3:CD004691.

What happens in the brain?

Calling psychiatric drugs “anti”-something is a misnomer

The deceptions in psychiatry also extend to drug names. It makes sense to talk about antibiotics, as these drugs cure infections by killing or inhibiting the growth of microorganisms. In contrast, a chemical cure for mental diseases doesn't exist. Antipsychotics don't cure psychosis, antidepressants don't cure depression, and anti-anxiety drugs don't cure anxiety; in fact, these drugs can *cause* psychosis, depression and anxiety, particularly if used longterm and if people try to get off them.

These drugs are not “anti” some disease. They don't cure us, they simply change us by causing a wide array of effects in people,¹ just like street drugs do. And they are not in any way targeted, although the drug names suggest they are, for example, there is nothing selective about selective serotonin reuptake inhibitors (SSRIs). This term was invented by SmithKline Beecham to give paroxetine an advantage over other SSRIs, but it was adopted by all companies.² There are serotonin receptors throughout the body, and the drugs have many other effects than increasing serotonin, e.g. they can affect dopamine and noradrenaline transmission and can have anticholinergic effects.³ The drugs don't even target depression; they have similar effects to many other brain-active substances, including alcohol and benzodiazepines. It is therefore not surprising that a Cochrane review found that alprazolam, an old benzodiazepine, performed better than placebo for depression and similarly to tricyclic antidepressants.⁴ Whether any of these drugs have a true effect on depression is another matter, and they likely haven't (see Chapter 3).

Some of the many unspecific effects are called beneficial by the drug companies, although it can be disputed whether the patients really benefit, and the rest they call side effects. “Side effect” is a marketing term used to imply that a problem is minor, although these effects are often the main ones, seen in most patients, e.g. sexual disturbances with SSRIs and excessive sedation with

antipsychotics. The distinction is wholly arbitrary, e.g. delayed ejaculation caused by SSRIs can be beneficial for those bothered by premature ejaculation but detrimental for others, as SSRIs can prevent ejaculation from occurring altogether.

We know a good deal about how the drugs interact with receptors in the brain and influence neurotransmitters, but despite quite different biochemical effects at the molecular level, they work more or less in the same way, either by suppressing emotional reactions so that people get numbed and pay less attention to significant disruptions in their lives or by stimulating them.^{2, 5-7} It is noteworthy that psychotropic drugs are developed based on rat experiments and selected if they disrupt the rat's normally functioning brain.⁶

Since psychiatric drugs – just like alcohol, marijuana, heroin and other addictive substances – alter people's personality in significant ways and make it substantially more difficult for them to live a normal social life and learn how to cope with life's difficulties, it would be more adequate, if we insist on using the prefix "anti," to call them antipersonality pills or antisocial pills, as many patients get socially isolated and care less about themselves and others. SSRIs, for example, reduce the identification of negative facial expressions of anger and fear in human volunteers,⁸ and some patients say they feel like living inside a cheese-dish cover.

My preferred "anti"-term for antidepressants would be antisexuals, as their main effect is to ruin people's sex lives. As other psychiatric drugs, e.g. ADHD drugs and antipsychotics, may also impair people's sex lives, we might call the whole lot antisexuals, or anti-life pills, as they prevent people from living a full life.

Antipsychotics I would call antihumans, as they impede the ability to live a normal enriching life and the ability to read, think, concentrate, be creative, feel and have sex. There isn't much life left for people on antipsychotics, and some describe it as being a vegetable or a zombie. It's no surprise that some patients commit suicide on antipsychotics and cannot see any hope. We often hear that schizophrenia carries a considerable risk of suicide but never hear about what this risk is for people who deliberately avoid taking antipsychotics. We cannot use the randomised trials to assess by how much antipsychotics increase suicides, as they are flawed by the cold-turkey design, which artificially increases suicides in the placebo group (see Chapter 6).

What I have just described illustrates how powerful drug marketing is. Merely by the language used, it fools us into believing in all sorts of things that just aren't true.

The way psychiatric drugs came into widespread use is also telling. None of the early drugs were developed in a rational fashion, based on a profound knowledge of biochemistry and receptors, but were discovered in a haphazard way and came into psychiatry because of their adverse effects, not because of some specific effect.

As noted earlier, the barbiturates came into human use after it was discovered that barbital was very effective in putting dogs to sleep.

The first antipsychotic drug, chlorpromazine, is a phenothiazine, which are compounds developed in the late 1800s for use as synthetic dyes. In the 1930s, they were used as insecticides and for swine parasites.⁹ In the 1940s, they were found to limit locomotion to such an extent that rats could no longer avoid electric shocks in escape experiments. Next, they were used in surgery for their numbing effect to enhance the effect of anaesthetics. Chlorpromazine was first used as an antihistamine for allergies but doctors observed that it made patients emotionally detached and disinterested in anything, which they described as a chemical lobotomy. When it was tried in patients with mania, the psychiatrists observed that it induced a profound numbness, an indifference where the patients didn't express their preoccupations, desires or preferences and rarely asked questions, like being separated from the world "as if by an invisible wall."

This is also what the patients feel. The predominant subjective effects reported by patients on the Internet when they take antipsychotics are sedation, cognitive impairment and emotional flattening or indifference.¹⁰

Chlorpromazine wasn't called an antipsychotic in the beginning, but a major tranquilliser or a neuroleptic, and it was acknowledged that its effects were highly unspecific. Right from the start, Smith, Kline & French promoted the idea of lifelong treatment with chlorpromazine.¹¹ It was hailed as a great advance, as it kept the patients docile and quiet, which was very popular with the staff in psychiatric wards. It was a formidable conflict of interest that the same staff evaluated whether patients had improved or not, and this conflict of interest clouds psychiatric research even today. People seem to have forgotten that the US Joint Commission on Mental Illness and Health stated in 1961 that the principal purpose of antipsychotics is to make highly disturbing persons more appealing to those who must work with them.¹²

The first benzodiazepine was chlordiazepoxide (Librium), which was synthesised in the mid-1950s based on work on chemical dyes, just like for chlorpromazine. It was discovered by accident in 1957 that the compound has hypnotic, anxiolytic and muscle relaxant effects.¹³

The first tricyclic antidepressant was imipramine, which is an analogue of

chlorpromazine.¹⁴ It was developed in an attempt to improve on the effectiveness of chlorpromazine and wasn't originally thought to be a treatment for depression. However, although the drug's tendency to induce manic effects was described as being quite disastrous in some patients, this paradoxical reaction to a sedative led to its testing in depressed patients.¹¹

SSRIs are said to have been developed in a rational fashion based on knowledge of receptors, but I have my doubts. The serotonin hypothesis is stone dead (see Chapter 3) and I don't see these drugs as any more rational than the others. What we have learned is that a detailed knowledge of receptors won't help us when we see patients because very different drugs may exhibit the same effects.

During my medical studies, I took great interest in clinical pharmacology and learned a lot about receptors, chemistry, and mode of action. But one thing really puzzled me, which I just couldn't figure out. Why was it that antihistamines, which were used against allergies, also worked for psychosis? Why was it that so many drugs, developed to fit with a certain receptor, also worked for completely different diseases that had nothing to do with that receptor? It took me many years to fully realise how deceptive the double-blind placebo controlled drug trial is when the outcome is subjective (see Chapter 3). Today, I have little interest in receptors and drug chemistry, as the drugs' side effects often explain why they seem to work for the most diverse diseases. Unfortunately, no one cares that what we measure in our trials is mostly bias. The drug regulators don't care the slightest bit, and the drug industry and their paid allies among doctors cash in. It is really foolish that new drugs can be approved if they happened to show an effect in two placebo controlled trials, no matter how bad they fared in many other trials, with no demands about avoiding unblinding bias, no demands about how large the effect should be to make any difference, and no demands about using clinically meaningful outcomes.

In the old days, antipsychotics were called major tranquillisers and benzodiazepines minor tranquillisers, which was more honest than current-day "anti"-disease nonsense, as it was the tranquillising effect that led to their human use. However, not even this terminology was okay, as it is a matter of dose how sedated people become.

Despite their humble history, antipsychotics are nonetheless at the heart of the fairy tale about progress in psychiatry.⁹ Antipsychotics decrease dopamine levels, and the number of dopamine receptors goes up to compensate for this. If the drugs are suddenly stopped, the response can very well be a psychosis, a phenomenon known as oppositional tolerance or supersensitivity psychosis. A psychosis can

even develop during continued treatment because of this, and may not respond to increased dosages.¹⁵

A similar phenomenon is seen with antidepressants. They increase serotonin levels, which results in a decrease in serotonin receptors, and sudden drug withdrawal can therefore cause depression. This rebound effect is also seen for benzodiazepines and lithium.^{6, 11} After a short while, people can experience worse symptoms than ever before, *even while they are still on the drugs*. And if they try to taper them off, it may become worse still.

Obviously, our brains tolerate poorly being forced into a new equilibrium with chemicals, and if the new homeostasis has lasted for more than a few weeks, people become dependent on them, just like they do on street drugs.

Genetic studies and transmitter research

Billions of dollars have been spent on finding genes predisposing to psychiatric diseases and on finding their biological causes, and this has resulted in thousands of studies of receptors and brain transmitters. In 2010 alone, NIMH spent \$400 million on brain and basic behavioural research,¹⁶ but the output of this enormity of research investment has been close to none.¹

The hype created by the researchers has given the public a totally wrong impression of what we know about genetics, transmitters and receptors in relation to mental illnesses. As an example, genetic association studies in ADHD have not found anything of interest. What has been reported has been weak associations,¹⁷ but the published literature isn't honest about this. A review of all articles asserting that polymorphisms of the gene coding for the D4 dopaminergic receptor are associated with ADHD found that only 25 summaries out of 159 mentioned that this association confers a small risk.¹⁸ I had heard several professors of psychiatry say in interviews that genetic factors were the most important causes for the development of ADHD, but I didn't believe it, among other things because ADHD is not a disease. It took some years before one of the ADHD professors revealed what the evidence was for this claim. She said there was 80% agreement between identical twins.¹⁹ So, people who are identical are pretty much identical also when it comes to branding them with the social construct we call ADHD. Surprise, surprise. This doesn't tell us anything about ADHD, it tells us more about that there is observer variation when labelling people with ADHD, as there wasn't 100% agreement.

The idea that ADHD is caused by a deficit of the dopaminergic system, the origin of which is mainly genetic, is also unfounded.¹⁷ There are no reliable

neurochemical, genetic, neuropharmacological or imaging data in support of the dopamine-deficit hypothesis of ADHD.¹⁷ Further, drugs that selectively inhibit the noradrenaline transporter and that do not affect the dopamine transporter are as efficient psychostimulants as those that affect the dopamine transporter, and L-dopa, which enhances dopamine release and effectively alleviates parkinsonian symptoms by correcting an overt dopamine deficit, is not effective in ADHD.¹⁷

I cannot go through the vast literature here, but everything I have looked at, also that which proponents of the theories wanted me to read because they found it convincing, has been a disappointment in terms of a genetic or biochemical explanation for the major psychiatric diseases. For example, a positron emission tomography study in *Science* that claimed that people with schizophrenia had more dopamine receptors than controls was later refuted, and also in this area, investigators have ignored inconvenient findings, e.g. when they found that receptor density was related to exposure to antipsychotics.¹¹

The believers have modified their dopamine hypotheses for ADHD and schizophrenia so much along the way to make them fit with the many contradictory data, that it looks like what science philosopher Karl Popper called pseudoscience. When a hypothesis is made immune towards rejection experiments, science ceases to exist.

It is similarly absurd to attribute a complex phenomenon like depression to one neurotransmitter when there are more than 200 such transmitters in the brain that interact in a very complex system we don't understand.¹⁷ But biological psychiatrists cannot afford to face the reality, as it would mean that their house of cards and their attractive funding opportunities would collapse. I would be thrilled if one day a true defect in the brain was found in people with a mental disorder that could be fixed with a drug, but it's not likely to happen.

Chronic brain damage

Brain imaging studies are central to the many attempts to make psychiatry look more scientific than it is. Clearly, if it could be shown that a disease leads to brain damage, this would be a strong argument for drug treatment, particularly if similar studies showed that drugs reduced the damage.

For rheumatoid arthritis, we have drugs that work, as judged by imaging studies. Disease-modifying agents slow down progressive joint destruction, but the drugs that accomplish this are dangerous and sometimes kill patients. In psychiatry, we only have the harmful effects of the drugs, which not only kill some patients but also damage their brains. Furthermore, it hasn't been documented that

psychiatric diseases can cause brain damage. It is absurd that psychiatrists treat millions of patients with psychiatric drugs for decades or for life under the pretence that they prevent brain damage.

It wouldn't be surprising if there were many brain imaging studies in the psychiatric literature, and if many of these were flawed, and this is indeed the case. One such study, which a psychiatrist professor sent me because he found it convincing, couldn't separate spontaneous remission of the depression from drug effects, which the authors themselves acknowledged;²⁰ a randomised trial is needed for this. A large study of 630 people found that use of antidepressant drugs, and not the depression, was related to smaller brain volumes and more white matter, but the differences were small and the study was cross-sectional.²¹

Brain imaging studies in patients with ADHD have not been revealing either.¹⁷

A 2012 systematic review surveyed the methodological state of the art in 241 functional magnetic resonance imaging (fMRI) studies.²² The review found that many of the studies didn't report on critical methodological details with regard to experimental design, data acquisition, and analysis, and many studies were underpowered. Data collection and analysis methods were highly flexible, with nearly as many unique analysis pipelines as there were studies. The review concluded that because the rate of false positive results increases with the flexibility of the design, the field of functional neuroimaging may be particularly vulnerable to false positives. Fewer than half the studies reported the number of people rejected from analysis and the reasons for rejection. Another review used meta-analysis and found that the false positive rate of neuroimaging studies lies between 10% and 40%.²³

A 38-page report from 2012 written for the American Psychiatric Association about neuroimaging biomarkers was totally negative, as it concluded that “no studies have been published in journals indexed by the National Library of Medicine examining the predictive ability of neuroimaging for psychiatric disorders for either adults or children.”²⁴

This means that researchers can get the result they want by manipulating their research. However, despite this huge potential for bias, studies and meta-analyses – performed by people who, judging from their papers, clearly didn't like what they found – have convincingly shown that antipsychotics shrink the brain.^{25, 26} They do this in a dose-dependent manner,^{9, 25} and they also shrink the brain in primates.²⁷ In contrast, the severity of illness had minimal or no effects.²⁵ There is no reliable evidence that the psychosis per se can damage the brain,²⁸ and although a large 2013 study claimed this,²⁹ it couldn't separate the effects of

treatment from any possible effect of the disease, which the authors acknowledged. A study that included patients with first episode psychosis found that short exposure to antipsychotics could lead to brain shrinkage of the gray matter, again with no relation to the severity of the illness.³⁰

It is wrong to tell patients they need to take antipsychotics to prevent brain damage; the fact is that antipsychotics cause brain damage, not the disease.

Schizophrenia is *not* a progressive brain disease, which many psychiatrists think it is.²⁷ They believe the disease leads to chronicity and social incapacity, but this perception is influenced by selection bias. The patients they see at their hospitals are the worst cases, not those that recover, and the truth is that about 40% achieve functional recovery.²⁸

I have not seen any convincing research showing that it is the disease that causes brain damage, whereas I have seen convincing research showing that the medication causes brain damage.^{25, 28, 31} In paper after paper I have read, the authors didn't even consider the obvious idea that it could be the medication and not the disease that caused the brain damage. This is inexcusable, given what we have known about these drugs for decades and given that virtually all patients have received medical treatment for their disease.

Chronic brain damage with persistent personality changes, e.g. with cognitive decline and emotional flatness long after the patients have come off the drugs, has been documented for virtually all psychiatric drugs.^{5, 31} Chronic brain damage is related to the length of drug exposure and often worsens when the dose is increased, whereas it will usually improve considerably when drugs are tapered off. If it had been the disease that caused the problems, patients should have become worse when the drugging was reduced.³¹ In addition to increasing memory problems, common symptoms for all the drugs include emotional instability with irritability and angry outbursts, which can be mistaken for Alzheimer's, terrifying the patient and the family.³¹

Antipsychotics kill nerve cells so effectively that their possible use against brain tumours has been explored.¹¹ The brain damage affects neurotransmission, including the number of receptors, and there is nothing strange about this. Hashish, LSD, alcohol and other brain-active substances may also lead to chronic brain damage and personality changes.

Although the science is convincing, psychiatrists rarely tell patients that their

drugs can cause brain damage. Leading psychiatrists often say the opposite, that it is important to take antipsychotics and antidepressants since untreated schizophrenia³² and depression^{33, 34} can cause brain damage, and that the sooner a person gets diagnosed and treated, the better the outcome.¹¹ I consider this lie to be similarly detrimental for patients as the lie about the chemical imbalance.

Recently, an influential depression researcher mentioned that depression doubles the risk of dementia and that antidepressants can help the brain regenerate.³⁴ He referred to a meta-analysis,³⁵ which is typical of the pseudoscience in psychiatry. It didn't mention anything about previous treatment, and there wasn't the slightest hint that the increased risk of dementia could be due to the medication, although this is far more likely (see Chapter 8).

Clinical observations confirm that antidepressants can cause chronic brain damage.³⁶⁻³⁸ Withdrawal symptoms and other SSRI-related harms can persist for years after patients have come off the drugs.^{1, 39} Further, there are many credible reports about persistent sexual dysfunction in humans,^{37, 40} which among other things involve genital anaesthesia and pleasureless orgasms.⁴¹ Rats can become permanently sexually impaired after having been exposed to SSRIs early in life,⁴² which we have confirmed in our systematic review of animal studies.⁴³ It is very likely that these effects are caused by an SSRI-induced inhibition of dopamine transmission. This can also explain why SSRIs can cause tardive dyskinesia and tardive dystonia, just like antipsychotics can.^{40, 44-47} Dechallenge and rechallenge experiments have confirmed that SSRIs cause these movement disorders.^{45, 47}

Benzodiazepines can also cause chronic brain damage,^{6, 31} and a carefully conducted study suggests that they double the risk of dementia.⁴⁸

ADHD drugs used early in life can also cause chronic brain damage. Animal studies have shown that this impairment includes anxiety, depression, less tolerance to stress, less response to natural rewards, less response to a novel environment and loss of sexual interest and capability.⁴⁹⁻⁵² Children treated with stimulants often develop atrophy of the brain,⁵³ but, as always, some researchers have argued that it is the disease that causes the atrophy, which is a pretty bizarre idea, as ADHD is not a disease but just confirmation that some kids are more active and irritating than others.

Psychiatrists have often used imaging studies to justify medicating children with ADHD,⁵⁴ but these studies are just as flawed as all the other studies that purport to show that the disease is the problem, not the drug. The researchers have failed to report whether the patients had received ADHD drugs, but other researchers have found out that this was indeed the case.¹⁷ The few recent studies

that studied unmedicated ADHD children have carefully avoided making straightforward comparisons of these patients with normal children, in contrast to all the studies where the researchers failed to report that the children were on drugs. This looks like large-scale scientific misconduct.

Addiction to psychiatric drugs

One of the best-kept secrets in psychiatry is that drug treatment leads to dependence, which makes it difficult for people to get off the drugs again because of abstinence symptoms.³¹ It is extraordinary that leading psychiatrists have denied this throughout so many decades⁵⁵ and that most of them even today fiercely deny that SSRIs can lead to dependence. Patients are not as easy to fool as the psychiatrists, however, and they know all too well that they become dependent on the drugs, including the SSRIs.^{56, 57}

There are several facts that should make it obvious to dispassionate observers that treatment with psychiatric drugs can lead to dependence.

As far as I know, all brain-active substances are addictive, including alcohol, opioids, barbiturates, benzodiazepines and street drugs, some of which are the same or very similar to prescription drugs. And withdrawal symptoms have been demonstrated empirically for all psychotropic drugs.

Furthermore, given the mechanism of action, we can predict that dependence must occur. Brain-active substances up- or down-regulate receptors and create a new homeostasis (equilibrium), which means that the brain no longer functions normally. Therefore, if the disturbing agent is suddenly removed, withdrawal symptoms occur in some people. We know about this also from other areas of clinical pharmacology, and we often call it a rebound effect. If a proton pump inhibitor prescribed because of heartburn is suddenly stopped, the dyspeptic symptoms may become even worse than before the drug was started. These mechanisms get many patients hooked on a drug for life, as the withdrawal symptoms make them believe that they still need the drug.

The deception about the dependence problem wouldn't have been possible if it wasn't for a massive cover-up to which the drug industry, drug regulators and doctors have all contributed. This unholy alliance has a long history. In the 1930s, addiction to barbiturates wasn't recognised and doctors pointing this out were ignored.⁵⁸ It took 40 years before the addiction problem was finally accepted by the UK Department of Health and it was realised that the reason people continued with barbiturates indefinitely wasn't that they were ill but because they couldn't

stop them without great suffering. In 1955, the United States produced so many barbiturate pills that 7% of the population could eat a pill every day. It looked like the ultimate realisation of the dream of a quick fix for life's pains, which Aldous Huxley described in 1932 in *Brave New World* where the citizens could take Soma pills every day to give them control over their lives and keep troubling thoughts away. In his time Soma pills were the barbiturates. Today TV commercials in the United States urge the public to do exactly the same. They depict unhappy characters that regain control and look happy as soon as they have taken a pill.⁵⁹

In the 1960s the benzodiazepines got their turn. Doctors believed they were harmless and prescribed them for almost anything, and Hoffman-La Roche pushed diazepam (Valium) to become the top-selling drug in the world.⁵⁸ Sales of benzodiazepines were so high that 10% of the Danish population could be on them,⁶⁰ which is extraordinary since the effect disappears after a few weeks because of development of tolerance. The companies denied for decades that benzodiazepines cause dependence, and although serious dependence was documented already in 1961, it wasn't generally accepted until more than 20 years later.⁶¹ The collective denial was huge. Even in 1980, the UK drug regulator concluded, based on submitted reports of adverse events, that only 28 people became dependent on benzodiazepines from 1960 to 1977. The true number is more likely to have been around half a million.

After the authorities, in the 1980s, at long last admitted that the huge consumption of benzodiazepines was a public health disaster and had started to warn against them, usage went down.⁶⁰ At the same time, the American Psychiatric Association tightened the criteria for substance dependence, very conveniently just before SSRIs appeared on the market.⁶¹ I have often wondered how much corruption was involved, as this change in criteria must have been worth billions for the companies.

The change was really major. Before 1987 dependence meant development of tolerance to a substance or withdrawal symptoms, which is how most people would define it. But from 1987 at least three criteria out of nine were needed and a time criterion was also added.⁶¹

It was now totally obscure whether a person was dependent or not and, as usual in the DSM manual, no one can remember all this or apply the criteria consistently from case to case. There is a lot of arbitrariness, and judgments and grades are involved. For example: "A great deal of time" (how much?); "substance often taken" (how often?); "Important social, occupational, or

recreational activities given up” (what is important and who decides on this?); “Frequent intoxication or withdrawal symptoms” (how frequent?); “Substance often taken to relieve or avoid withdrawal symptoms” (this criterion is meaningless; if a patient misses just one dose of paroxetine, it can elicit withdrawal symptoms – does “often” mean taking three paroxetine pills a day?).

The new criteria took the power of decision away from patients, as some of them require judgments by others, e.g. whether the patients “fulfil major role obligations at work, school or home.” I don’t dispute that judgments by others can be needed, e.g. if a heroin addict denies his dependence, but I firmly believe that patients in drug treatment should speak for themselves.

The time criterion is awfully foolish. Symptoms should have persisted for at least one month or should have occurred repeatedly over a longer time period. Very many patients who are dependent on drugs don’t fulfil the time criterion. They might have tried to stop a few times but quickly resumed treatment and decided never to try again because of the terrible abstinence symptoms they experienced. Such patients are not dependent according to the time criterion, although they are the ones who are the *most* dependent!

The new criteria are a smokescreen that serve to deflect attention away from the fact that SSRIs cause dependence. We found in our research that withdrawal symptoms were described with similar terms for benzodiazepines and SSRIs and were very similar for 37 of 42 identified symptoms,⁶¹ but when Lundbeck was interviewed about our findings, the company denied that people could become dependent on SSRIs.⁶²

The worst argument I have heard – also from professors of psychiatry – is that patients are not dependent because they don’t crave higher doses. If that were true, then smokers are not dependent on nicotine because they don’t increase their consumption of cigarettes!

To describe similar problems as dependence for benzodiazepines and withdrawal reactions for SSRIs is irrational, and for the patients it’s just the same. It can be very hard for them to stop either type of drug. In a survey, 57% of 500 Danish patients agreed to the sentence: “When you have taken antidepressants over a long period of time it is difficult to stop taking them,”⁵⁶ and in another survey, 55% of 1,829 patients in New Zealand taking antidepressants mentioned withdrawal effects, which 25% described as severe.⁵

Drug regulators, the extended arm of industry

It is difficult to see much difference between the regulators and the regulated. It

took more than a decade after drug agencies had the information about dependency before they warned about it, both for benzodiazepines and SSRIs, and the process was characterised by denial, downplaying of harms and even misrepresenting them.^{58, 61, 63} The UK regulator described withdrawal reactions after SSRIs as generally rare and mild, but independent researchers showed that the regulator had classified 60% of the “mild” reactions as moderate and 20% as severe!⁶³

The trick with the new DSM criteria for dependence was uncritically accepted by the authorities, which relied on them when they denied the dependence potential of SSRIs. For example, the UK regulator stated in 1998 that use of SSRIs doesn't lead to dose escalation and drug-seeking behaviour,⁶¹ as if this would prove they weren't addictive. Drugs can be addictive without having these properties, and it wasn't even correct that SSRIs don't lead to drug seeking behaviour. They do, as the same regulator acknowledged in 2004 when it even admitted that the SSRIs met the new, narrow criteria for dependence.

Studies conducted by Beecham on paroxetine in the mid-1980s before the drug came on the market showed that it could produce dependence in healthy volunteers but, despite being warned by senior figures in the field, the company did nothing and the studies weren't published.²

A few years after the introduction of SSRIs, concern was raised again about dependence. However, the drug companies and their paid allies among psychiatrists confused the issues and tended to interpret any withdrawal reactions as relapse.⁶¹ Paroxetine was even marketed directly to consumers as “non-habit forming” in the United States,⁶¹ and on the back of British packets of fluoxetine, this message appeared: “Don't worry about taking Prozac over a long period of time – Prozac is not addictive.”²

As recently as 2000, the European Medicines Agency firmly stated that, “SSRIs do not cause dependence.”^{58, 61} However, the agency also noted that SSRIs “have been shown to reduce intake of addictive substances like cocaine and ethanol. The interpretation of this aspect is difficult.”⁵⁸ The interpretation is only difficult for those who *will not* see. In 2003 the World Health Organization published a report noting that three SSRIs (fluoxetine, paroxetine and sertraline) were among the top 30 highest-ranking drugs for which drug dependence had ever been reported!⁵⁸

The drug companies also did what they could to obscure the issues,⁶¹ and the psychiatrist silverbacks on drug company payrolls have been immensely useful for the drug companies, as they have helped them hook more than one hundred million patients on drugs most of them didn't need. First the barbiturates, next the benzodiazepines, and now the SSRIs. And when problems arise, psychiatrists and

companies use the same tactic: Always blame the disease, never the pills,^{2, 5, 64} which the next section is about.

Drug dependence is often misinterpreted as relapse of the disease

As stated earlier, all psychotropic drugs can cause dependence.³¹ It can be useful to divide withdrawal symptoms into two phases: the immediate withdrawal phase consisting of new and rebound symptoms, occurring up to six weeks after drug withdrawal, depending on the drug elimination half-life, and the post-withdrawal phase, occurring after six weeks,^{6, 31, 39} and which may sometimes last for years.^{1, 39, 65}

Many people cannot get off SSRIs even when slow tapering is attempted. Out of 20 patients with panic disorder and agoraphobia who had been treated successfully with behavioural therapy, nine had withdrawal symptoms, which subsided within a month in six of them.⁶⁶ In the three other patients, who had all received paroxetine, the symptoms persisted and all three developed cyclic changes in mood that are characteristic for bipolar disorder but which they had not had before. Other studies have confirmed that about half the patients experience withdrawal symptoms.^{56, 67, 68}

It is pretty clear in this case that when patients treated successfully with behavioural therapy get symptoms when a drug dose is gradually reduced, it is likely to be withdrawal symptoms and not a return of their panic disorder. Unfortunately, psychiatrists and other doctors have a pronounced tendency to interpret withdrawal symptoms as disease symptoms, and the rating scales they use for grading the severity of the disease mislead them, as they often contain items that are withdrawal symptoms.¹¹ Doctors therefore put pressure on patients who try to stop taking drugs to continue with them.

I once explained at a large meeting for psychiatrists that many patients have difficulty stopping antidepressants, but to my big surprise a professor said he had no trouble withdrawing patients successfully. This might be because it is so common to interpret abstinence symptoms as relapse of the disease. If you always do that, you won't have any problems, as you simply put those patients back on full dose who cannot tolerate the withdrawal symptoms. But it's a tautology. Somewhat like: "This drug works for all patients with hysteria, and if it doesn't work for some patients, it's because they don't have hysteria."

Stuart Montgomery from the UK, who has numerous financial ties to drug makers, seems to interpret *all* withdrawal symptoms as relapse.⁶⁹ He studied 135

patients with depression who had responded to eight weeks of treatment with paroxetine and whom he randomised to drug or placebo for one year. *Although paroxetine was withdrawn abruptly – cold turkey – in the 67 patients randomised to placebo, he didn't mention a single withdrawal symptom in his paper!* After having inflicted tremendous harms on the placebo group patients, the authors concluded that they had confirmed “the reports from acute studies that the side-effects on paroxetine diminish with time until they become indistinguishable from placebo.” It's unbelievable. One of the patients committed suicide by hanging while on paroxetine during the first eight weeks.

Internal Pfizer documents show that Montgomery deliberately avoided to inform the drug regulator for which he worked that he also worked for Pfizer at the same time.⁵⁸ He informed Pfizer about how the regulator had reasoned in relation to its application for sertraline and advised the company about what it should do in order to get the drug approved.

Others are more thoughtful but may still lead their readers astray. A 2003 systematic review in the *Lancet* reported on 4,410 patients in 31 trials who had been randomised to continue on active drug or placebo after having responded to an antidepressant drug.⁷⁰ The relapse rate was 18% for patients who continued on active drug and 41% for those who continued on placebo. This seemingly impressive effect made the authors conclude in their abstract that “continued treatment with antidepressants would benefit many patients with recurrent depressive disorder.”

Most people only read the abstract so they won't know that the authors were more cautious in the main text. They explained that the increased risk of relapse in the placebo group might be due to a withdrawal reaction rather than a relapse. They also noted that this problem has been identified for lithium, for which acute withdrawal leads to manic relapse, and that trials with a withdrawal design (often called maintenance studies or relapse prevention studies) quite clearly inflated the apparent efficacy of lithium. In their own meta-analysis, they found that even after one to three years, there was a clear difference between the active group and the placebo group. However, some patients suffer from withdrawal symptoms for years, which would be expected to increase the risk of a new bout of depression, but it would then not be a true depression but a drug-induced withdrawal depression. Furthermore, there were rather few relapses in this time period and pure chance could therefore also have influenced the findings. What might be most important, a follow-up of one to three years is too short (see the schizophrenia withdrawal study below).

It's tricky that withdrawal symptoms and disease symptoms can be the same. If

a patient reduces the dose of an antidepressant and becomes depressed, it doesn't necessarily mean that the disease has come back. Two hallmarks of withdrawal-induced, depression-like symptoms are that they usually come rather quickly and usually disappear within hours when the full dose is resumed, whereas it takes weeks before the patients get any better if they have a true depression.

A trial of 242 patients with remitted depression illustrates these diagnostic difficulties.⁷¹ The patients had received open maintenance therapy with fluoxetine, sertraline, or paroxetine for four to 24 months, after they had become well. They then suddenly had their therapy changed to a double-blind placebo for five to eight days, but the timing of the treatment interruption was unknown to the patients and clinicians. The investigators had developed a 43-item list based on withdrawal symptoms reported in the literature, and after the placebo period patients were asked if they had experienced any of these. This checklist approach will tend to exaggerate withdrawal symptoms, and the study was funded by Eli Lilly, the maker of fluoxetine, which had an obvious interest in showing that fluoxetine causes fewer withdrawal symptoms than the two other drugs because of the very long half-life of its active metabolite, about one to two weeks.

But the results are nevertheless interesting. The three most common withdrawal symptoms were worsened mood, irritability and agitation (Table 11.1), which have nothing to do with relapse of the depression and, as expected, relatively few people had symptoms on fluoxetine. Out of 122 patients on sertraline or paroxetine, 40 had an increase in their Hamilton depression score of at least eight, which is a clinically relevant increase.

There would have been many more withdrawal symptoms if the drugs had been withdrawn for two to three weeks, but in fact 25 of the 122 patients fulfilled the authors' criteria for depression. Thus, this study shows why most doctors get it wrong when they think the disease has come back. Think about it. How many are likely to get a new depression in a random week in a group of 122 patients whose depression has been in remission for four to 24 months? Not 25 patients but perhaps one patient!

Table 11.1. Withdrawal symptoms in patients with remitted depression during a 5-8-day placebo period 4 to 24 months after remission.

	fluoxetine (n = 63)	sertraline (n = 63)	paroxetine (n = 59)
Worsened mood	22%	28%	45%
Irritability	17%	38%	35%
Agitation	16%	37%	31%
Hamilton increase ≥ 8	6%	30%	36%

In six short-term treatment trials, in which treatment was stopped abruptly and replaced by placebo at a time unknown to the investigators and patients, Eli Lilly reported withdrawal symptoms in 44% on duloxetine and 23% on placebo.⁶⁸

People may get terrible symptoms when they try to stop, both symptoms that resemble the disease and many others including some they have never experienced before and which can frighten them, e.g. electric shock sensations in the head after SSRIs.⁷²

Leading psychiatrists don't understand any of this, or they pretend they don't. Virtually all the silverbacks have interpreted the maintenance studies of antidepressants and antipsychotics as meaning that these drugs are very effective in preventing new depressions and psychoses,^{9, 73, 74} and that patients should therefore continue taking the drugs for years or even for life. In Denmark, it is a national goal that over 90% of patients with schizophrenia should be in drug treatment. This is a national tragedy.

Psychiatrists also say that depression has a more chronic course today than in the past. For example, the American Psychiatric Association's Textbook of Psychiatry from 1999 stated that not long ago most patients would recover from a major depressive episode, whereas now "depression is a highly recurrent and pernicious disorder."⁵

Psychiatrists overlook the fact that it is themselves that have created this epidemic by their systematic denial of the substantial role abstinence symptoms play and their reluctance to get patients off their drugs. Since there is no evidence that mental illness is chronic and lifelong, there is no scientific justification for the lifelong use of psychiatric medications. The apparent "chronicity" in mental disorders is an artefact of the medications themselves.¹ This was shown in a study of 172 patients with recurrent depression who had been in remission for at least 10 weeks since their last episode.⁷⁵ Of those who continued to take drugs, which they were supposed to do according to international guidelines, 60% relapsed in

two years. The relapse rate was similar for intermittent users (64%) whereas it was 46% in those who did not take drugs and only 8% in those who did not take drugs and received psychotherapy. Differences in disease severity could not explain these results, so they were not due to confounding by indication.

A careful analysis of 66 withdrawal studies of antipsychotics showed what the main problem is with such studies.^{9, 76} The relapse rate was three times higher in the groups with abrupt withdrawal than in the gradual-withdrawal groups!

As I have explained throughout this book, psychiatric drugs, if taken for more than a few weeks, create the diseases some of them have a short-term effect on, or other or even worse diseases, and acute conditions become chronic.^{2, 5, 6 36, 77, 78} This has been brought up time and again over the last 30-40 years, but no matter how strong the new evidence, leading psychiatrists every time swept it under the rug as quickly as possible.^{5, 9} It is too painful for them that, after they left the unscientific psychoanalysis behind, they must now accept that biological psychiatry, which on the surface made their speciality look as scientific as internal medicine, has not kept its promises.

Maintenance (withdrawal) studies were done for a good reason, to find out for how long patients need to be on drug. However, they are highly misleading if they are short-term. There are very few long-term studies, but one such study in 128 patients with schizophrenia is illuminating.⁷⁹ Remitted first episode patients were randomised to dose reduction or discontinuation, or to maintenance therapy, for two years, after which the clinicians were free to choose the treatments they felt the patients needed.

Seven years after the randomisation, 103 patients could be located. The short-term results showed that two years after randomisation, more patients had relapsed in the dose reduction/discontinuation group than in the maintenance group (43% versus 21%). However, after seven years, there was no difference (62% versus 69%). Furthermore, relapse was not the study's primary outcome. It is much more important that patients recover from their schizophrenia, and more patients had recovered in the dose reduction/discontinuation group than in the maintenance group after seven years (40% versus 18%). This happened despite the fact that the dose in last two years before the seven-year cut-off was 64% higher in the maintenance group, and that fewer patients had stopped taking their drug completely at seven years in this group (six versus 11 patients). Thus, the patients who had their dose decreased or discontinued fared much better in the long term than those who continued taking their antipsychotic drug.

A large meta-analysis of the placebo controlled trials showed that the apparent effect of continued treatment with antipsychotics on relapse prevention decreases

over time and is close to zero after three years.⁸⁰ Moreover, most of these trials are flawed, as patients on placebo were exposed to cold turkey withdrawal of their drug.

There is a Cochrane review of intermittent drug treatment for schizophrenia, but it isn't relevant, as the results from all included trials are short term.⁸¹

It is really bad medicine to keep patients on their drugs for years based on the false belief that this improves their prognosis.

The chemical imbalance nonsense

When I lecture for psychiatric patients and ask them whether they have been told that they need a drug to fix a “chemical imbalance” in the brain, roughly half of them confirm this. Quite often, they have also been told that this corresponds to being a patient with diabetes needing insulin. The fairy tale comparing antipsychotics and antidepressants with insulin was invented by psychiatrists in the 1950s,^{5, 11} and at the same time, the psychic energizers changed name to antidepressants.⁵ The “drug revolution” in psychiatry has even been likened to the introduction of penicillin but, as David Healy has dryly noted, in contrast to penicillin, there are more dead bodies in the drug groups than in the placebo groups of the trials.⁵

The chemical imbalance story is being told about all psychotropic drugs, also for benzodiazepines, but it is a blatant lie.^{1, 5, 9} It has never been documented that any of the large psychiatric diseases is caused by a biochemical defect and there is no biological test that can tell us whether someone has a particular mental disorder.

As an example, the idea that depressed patients lack serotonin has been convincingly rejected.^{2, 82, 83} Some drugs that *decrease* serotonin also seem to work for depression,^{2, 5} e.g. tianeptine, and the Irish drug regulator banned GSK from claiming that paroxetine corrects a chemical imbalance. Furthermore, mice genetically depleted of brain serotonin weren't depressed but behaved like wild-type mice in the wild.⁸⁴ There is much else that speaks against the chemical imbalance story, e.g. it takes weeks before antidepressants seem to work,⁸⁵ and the effect – if any – comes slowly and gradually, whereas monoamine levels in the brain increase in one to two days after the start of treatment.⁸³ Further, why should these drugs “work” in social phobia, which is not considered a lack-of-serotonin disease?⁸³

Nonetheless, until 2003, the UK drug regulator gave the industry a helping hand

by propagating in patient information leaflets⁶⁴ the false and totally undocumented idea about lack of serotonin as the cause of depression.

Dopamine metabolites are normal in patients with schizophrenia, but when they are medicated, their dopamine receptors increase by about 50% in response to the drugs' lowering of dopamine.⁹

In the beginning, when I explained to doctors that many patients had been told they had a chemical imbalance, I was often met with angry responses demanding that I documented my so-called allegations. They obviously didn't like to admit that they lied to their patients. I referred to what patients, caregivers and others had told me, and to websites where patients share their experiences, but this was taken to mean that I didn't know what I was talking about, as if it didn't have any value to listen to patients. When I argued that the documentation on the Internet is very convincing because patients rather consistently have had the same experiences, I was told that these were just anecdotes which, moreover, had not been published in a peer reviewed journal. As if that would make any difference!

The organised denial is deeply disturbing. In the Danish study of 500 depressed or bipolar patients I have quoted earlier, 80% agreed with the sentence: "Antidepressants correct the changes that occurred in my brain due to stress or problems."⁵⁶

When – pretty rarely, I must say – psychiatrists admitted that it has never been demonstrated that any psychiatric disease is caused by a chemical imbalance, they added that it's just a metaphor, as if it doesn't matter to use metaphors. It certainly does. Doctors use this metaphor to persuade patients, who feel badly about their medication and want to quit, to continue their suffering in the hope of obtaining some effect later. A Danish silverback, Professor Poul Videbech, illustrated this recently at a meeting where I argued that far too many people are in antidepressant treatment. He said in front of 600 people, "Who would take insulin from a diabetic?" and used the same allegory in an interview. So I suppose he means it.

People have told me about medical students who were put on an antidepressant the first time they consulted a doctor because they had difficulty with their studies, with the false messages about correcting a chemical imbalance like with insulin. When the students tried to stop and got abstinence symptoms, they were told it was their depression that had come back. In one case, the psychiatrist said bluntly at the very first visit that the student should take the pills for life! When we deal with cancer, we know it's important that patients don't lose hope, but in psychiatry, where hope is much more essential, as it's so important for recovery, some doctors take the hope away by saying the pills are for life.⁶ But there are no "rest-of-your-life" drug trials, so it's pure speculation, and wrong, too. An important

reason why most studies only last a few weeks is that many patients drop out of them early, as they don't like the drugs.

It is difficult not to get angry when confronted with such stories. In 2003, six US psychiatric survivors were also angry. They announced a "fast for freedom" and sent a letter to the American Psychiatric Association and other organisations stating that they would begin a hunger strike unless scientifically valid evidence was provided that the various stories the public had been told about mental disorders were true.⁵ They asked for evidence that major mental illnesses are biologically-based brain diseases and that any psychiatric drug can correct a chemical imbalance. They also required the organisations to publicly admit if they were unable to provide such evidence.

The medical director of the American Psychiatric Association tried to get off the hook by saying that, "The answers to your questions are widely available in the scientific literature." The hunger strike ended when people started getting health problems, but it was clear that the Association bluffed when it stated in a press release that it would not "be distracted by those who would deny that serious mental disorders are real medical conditions that can be diagnosed accurately and treated effectively." The Catholic Church couldn't have invented a better bluff, if people had required proof that God exists: "We priests and cardinals will not be distracted by those who would deny that God exists and knows about people's problems and can treat them effectively."

To a considerable extent, psychiatry is a pseudoscience, and the hoax about the chemical imbalance should be dealt with in the courts, as it looks like consumer fraud.

References

- 1 Unrecognised facts about modern psychiatric practice. Council for Evidencebased Psychiatry; 2014.
- 2 Healy D. Let them eat Prozac. New York: New York University Press; 2004.
- 3 Rosen RC, Lane RM, Menza M. Effects of SSRIs on sexual function: a critical review. *J Clin Psychopharmacol* 1999;19:67-85.
- 4 van Marwijk H, Allick G, Wegman F, et al. Alprazolam for depression. *Cochrane Database Syst Rev* 2012;7:CD007139.
- 5 Whitaker R. Anatomy of an Epidemic. New York: Broadway Paperbacks; 2010.
- 6 Breggin P. Medication madness. New York: St. Martin's Griffin; 2008.
- 7 Pamphlet on Informed Consent. International Society for Ethical Psychology & Psychiatry. http://www.psychintegrity.org/pamphlet_on_informed_consent.php (accessed 3 Dec 2014).
- 8 Harmer CJ, Shelley NC, Cowen PJ, et al. Increased positive versus negative affective perception and memory in healthy volunteers following selective serotonin and norepinephrine reuptake inhibition. *Am*

- J Psychiatry 2004;161:1256-63.
- 9 Whitaker R. Mad in America. Cambridge: Perseus Books Group; 2002.
 - 10 Moncrieff J, Cohen D, Mason JP. The subjective experience of taking antipsychotic medication: a content analysis of Internet data. *Acta Psychiatr Scand* 2009;120:102-11.
 - 11 Moncrieff J. The bitterest pills. Basingstoke: Palgrave Macmillan; 2013.
 - 12 Joint Commission on Mental Illness and Health. Action for Mental Health: Final Report. New York: Basic Books; 1961.
 - 13 Chlordiazepoxide. <http://en.wikipedia.org/wiki/Chlordiazepoxide> (accessed 10 Dec 2014).
 - 14 Tricyclic antidepressant. http://en.wikipedia.org/wiki/Tricyclic_antidepressant (accessed 10 Dec 2014).
 - 15 Chouinard G. Severe cases of neuroleptic-induced supersensitivity psychosis: diagnostic criteria for the disorder and its treatment. *Schizophrenia Research* 1990;5:21-33.
 - 16 Kirk SA, Gomory T, Cohen D. Mad science: psychiatric coercion, diagnosis and drugs. New Brunswick: Transaction Publishers; 2013.
 - 17 Gonon F. The dopaminergic hypothesis of attention-deficit/hyperactivity disorder needs re-examining. *Trends Neurosci* 2009;32:2-8.
 - 18 Gonon F, Bezdard E, Boraud T. Misrepresentation of neuroscience data might give rise to misleading conclusions in the media: the case of Attention Deficit Hyperactivity Disorder. *PLoS One* 2011;6:e14618.
 - 19 Gøtzsche PC. [Right or wrong about psychiatric drugs]. <http://www.deadlymedicines.dk/fup-eller-fakta-om-psykofarmaka/> (accessed 3 Feb 2015).
 - 20 Arnone D, McKie S, Elliott R, et al. State-dependent changes in hippocampal grey matter in depression. *Mol Psychiatry*; 2013;18:1265-72.
 - 21 Geerlings MI, Brickman AM, Schupf N, et al. Depressive symptoms, antidepressant use, and brain volumes on MRI in a population-based cohort of old persons without dementia. *J Alzheimers Dis* 2012;30:75-82.
 - 22 Carp J. The secret lives of experiments: methods reporting in the fMRI literature. *NeuroImage* 2012;63:289-300.
 - 23 Wager T, Lindquist M, Nichols T, et al. Evaluating the consistency and specificity of neuroimaging data using meta-analysis. *NeuroImage* 2009;45:S210–21.
 - 24 First M, Botteron K, Carter C, et al. Consensus Report of the APA Work Group on Neuroimaging Markers of Psychiatric Disorders. Approved by the Board of Trustees, July 2012.
 - 25 Ho BC, Andreasen NC, Ziebell S, et al. Long-term antipsychotic treatment and brain volumes: a longitudinal study of first-episode schizophrenia. *Arch Gen Psychiatry* 2011;68:128-37.
 - 26 Raduaa J, Borgwardt S, Crescinid A, et al. Multimodal meta-analysis of structural and functional brain changes in first episode psychosis and the effects of antipsychotic medication. *Neurosci Biobehav Rev* 2012;36:2325–33.
 - 27 Dorph-Petersen KA, Pierri JN, Perel JM, et al. The influence of chronic exposure to antipsychotic medications on brain size before and after tissue fixation: a comparison of haloperidol and olanzapine in macaque monkeys. *Neuropsychopharmacology* 2005;30:1649-61.
 - 28 Zipursky RB, Reilly TJ, Murray RM. The myth of schizophrenia as a progressive brain disease. *Schizophr Bull* 2013;39:1363-72.
 - 29 Andreasen NC, Liu D, Ziebell S, et al. Relapse duration, treatment intensity, and brain tissue loss in schizophrenia: a prospective longitudinal MRI study. *Am J Psychiatry* 2013;170:609-15.
 - 30 Pagsberg AK, Baaré WF, Raabjerg Christensen AM, et al. Structural brain abnormalities in early onset

- first-episode psychosis. *J Neural Transm* 2007;114:489-98.
- 31 Breggin P. *Psychiatric drug withdrawal: a guide for prescribers, therapists, patients, and their families*. New York: Springer Publishing Company; 2013.
 - 32 Lægemiddelindustriforeningen. [How do we ensure optimal medical treatment of patients with schizophrenia and other psychoses?]. 2013 June 4.
 - 33 Videbech P, Dalsgaard M. “[I have seen how people’s views on antidepressants lead to suicide]”. *Politiken* 2014 Sept 27:22.
 - 34 Videbech P. [The debate about antidepressants – do they work and does one become dependent]? *BestPractice Psykiatri/Neurologi* 2014 May:nr 25.
 - 35 Ownby RL, Crocco E, Acevedo A, et al. Depression and risk for Alzheimer disease: systematic review, meta-analysis, and metaregression analysis. *Arch Gen Psychiatry* 2006;63:530-8.
 - 36 El-Mallakh RS, Gao Y, Roberts RJ. Tardive dysphoria: the role of long term antidepressant use in inducing chronic depression. *Medical Hypotheses* 2011; 76:769–73.
 - 37 Hogan C, Nourya JL, Healy D, et al. One hundred and twenty cases of enduring sexual dysfunction following treatment. *Int J Risk Saf Med* 2014;26:109–16.
 - 38 Shipko S. Playing the odds, revisited. <http://www.madinamerica.com/2014/07/shooting-odds-revisited/> (accessed 10 Jan 2015).
 - 39 Belaise C, Gatti A, Chouinard VA, et al. Patient online report of selective serotonin reuptake inhibitor-induced persistent postwithdrawal anxiety and mood disorders. *Psychother Psychosom* 2012;81:386-8.
 - 40 Csoka AB, Bahrack A, Mehtonen OP. Persistent sexual dysfunction after discontinuation of selective serotonin reuptake inhibitors. *J Sex Med* 2008;5:227-33.
 - 41 Bahrack AS. Persistence of sexual dysfunction side effects after discontinuation of antidepressant medications: emerging evidence. *Open Psychol J* 2008;1:42-50.
 - 42 Maciag D, Simpson KL, Coppinger D, et al. Neonatal antidepressant exposure has lasting effects on behavior and serotonin circuitry. *Neuropsychopharmacology* 2006;31:47–57.
 - 43 Simonsen AL, Danborg P, Gøtzsche PC. Persistent sexual dysfunction after early exposure to SSRIs: systematic review of animal studies 2015 (submitted for publication).
 - 44 Caley CF. Extrapyramidal reactions and the selective serotonin-reuptake inhibitors. *Ann Pharmacother* 1997;31:1481-9.
 - 45 Gerber PE, Lynd LD. Selective serotonin-reuptake inhibitor-induced movement disorders. *Ann Pharmacother* 1998;32:692-8.
 - 46 Lee Y, Lin PY, Chang YY, et al. Antidepressant-induced tardive syndrome: a retrospective epidemiological study. *Pharmacopsychiatry* 2013;46:281-5.
 - 47 Lane RM. SSRI-induced extrapyramidal side-effects and akathisia: implications for treatment. *J Psychopharmacol* 1998;12:192-214.
 - 48 Billioti de Gage S, Moride Y, Ducruet T, et al. Benzodiazepine use and risk of Alzheimer’s disease: case-control study. *BMJ* 2014;349:g5205.
 - 49 Castner S. Long-lasting psychotomimetic consequences of repeated low-dose amphetamine exposure in rhesus monkeys. *Neuropsychopharmacology* 1999; 20:10-28.
 - 50 Marco E. Neurobehavioral adaptations to methylphenidate. *Neurosci Behav Rev* 2011;35:1722-39.
 - 51 Carlezon W. Enduring behavioral effects of early exposure to methylphenidate in rats. *Biological Psychiatry* 2003;54:1330-7.
 - 52 Bolanos C. Methylphenidate treatment during pre- and periadolescence alters behavioral responses to emotional stimuli at adulthood. *Biological Psychiatry* 2003;54:1317-29.

- 53 Breggin PR. The rights of children and parents in regard to children receiving psychiatric diagnoses and drugs. *Children & Society* 2014;28:231-41.
- 54 Leo J, Cohen D. Broken brains or flawed studies? A critical review of ADHD neuroimaging research. *J Mind Behav* 2003;24:29-56.
- 55 Nielsen M, Hansen EH, Gøtzsche PC. What is the difference between dependence and withdrawal reactions? A comparison of benzodiazepines and selective serotonin re-uptake inhibitors. *Addiction* 2012;107:900-8.
- 56 Kessing L, Hansen HV, Demyttenaere K, et al. Depressive and bipolar disorders: patients' attitudes and beliefs towards depression and antidepressants. *Psychological Medicine* 2005;35:1205-13.
- 57 Read J, Cartwright C, Gibson K. Adverse emotional and interpersonal effects reported by 1829 New Zealanders while taking antidepressants. *Psychiatry Res* 2014;216:67-73.
- 58 Gøtzsche PC. *Deadly medicines and organised crime: How big pharma has corrupted health care*. London: Radcliffe Publishing; 2013.
- 59 Tanne JH. Drug advertisements in US paint a "black and white scenario." *BMJ* 2007;334:279.
- 60 Nielsen M, Gøtzsche P. An analysis of psychotropic drug sales. Increasing sales of selective serotonin reuptake inhibitors are closely related to number of products. *Int J Risk Saf Med* 2011;23:125–32.
- 61 Nielsen M, Hansen EH, Gøtzsche PC. Dependence and withdrawal reactions to benzodiazepines and selective serotonin reuptake inhibitors. How did the health authorities react? *Int J Risk Saf Med* 2013;25:155-68.
- 62 Korsgaard P, Jensen JH. [Drug maker: nonsense! Denies dependence on happy pills: It isn't possible]. *Ekstra Bladet* 2012 Dec 11.
- 63 Medawar C, Herxheimer A. A comparison of adverse drug reaction reports from professionals and users, relating to risk of dependence and suicidal behaviour with paroxetine. *Int J Risk Saf Med* 2003/2004; 16:5–19.
- 64 Medawar C, Hardon A. *Medicines out of control? Antidepressants and the conspiracy of goodwill*. Netherlands: Aksant Academic Publishers; 2004.
- 65 Belaise C, Gatti A, Chouinard VA, et al. Persistent postwithdrawal disorders induced by paroxetine, a selective serotonin reuptake inhibitor, and treated with specific cognitive behavioral therapy. *Psychother Psychosom* 2014;83:247-8.
- 66 Fava GA, Bernardi M, Tomba E, et al. Effects of gradual discontinuation of selective serotonin reuptake inhibitors in panic disorder with agoraphobia. *Int J Neuropsychopharmacol* 2007;10:835–8.
- 67 Young A, Haddad P. Discontinuation symptoms and psychotropic drugs. *Lancet* 2000;355:1184.
- 68 Perahia DG, Kajdasz DK, Desai D, et al. Symptoms following abrupt discontinuation of duloxetine treatment in patients with major depressive disorder. *J Affect Disord* 2005;89:207-12.
- 69 Montgomery SA, Dunbar G. Paroxetine is better than placebo in relapse prevention and the prophylaxis of recurrent depression. *Int Clin Psychopharmacol* 1993;8:189-95.
- 70 Geddes JR, Carney SM, Davies C, et al. Relapse prevention with antidepressant drug treatment in depressive disorders: a systematic review. *Lancet* 2003; 361:653–61.
- 71 Rosenbaum JF, Fava M, Hoog SL, et al. Selective serotonin reuptake inhibitor discontinuation syndrome: a randomised clinical trial. *Biol Psychiatry* 1998;44:77-87.
- 72 Medawar C, Herxheimer A, Bell A, et al. Paroxetine, Panorama and user reporting of ADRs: Consumer intelligence matters in clinical practice and post-marketing drug surveillance. *Int J Risk Saf Med* 2002;15:161–9.
- 73 Raven M. *Depression and antidepressants in Australia and beyond: a critical public health analysis (PhD thesis)*. University of Wollongong, Australia; 2012. <http://ro.uow.edu.au/theses/3686/>

- 74 Kessing L, Nordentoft M, Middelboe T. [Right and wrong about psychotropic drugs]. *Politiken* 2014 Jan 17.
- 75 Bockting CL, ten Doesschate MC, Spijker J, et al. Continuation and maintenance use of antidepressants in recurrent depression. *Psychother Psychosom* 2008;77:17-26.
- 76 Baldessarini RJ, Viguera AC. Neuroleptic withdrawal in schizophrenic patients. *Arch Gen Psychiatry* 1995;52:189-92.
- 77 Andrews PW, Kornstein SG, Halberstadt LJ, et al. Blue again: perturbational effects of antidepressants suggest monoaminergic homeostasis in major depression. *Front Psychol* 2011;2:159.
- 78 Martin A, Young C, Leckman JF, et al. Age effects on antidepressant-induced manic conversion. *Arch Pediatr Adolesc Med* 2004;158:773–80.
- 79 Wunderink L, Nieboer RM, Wiersma D, et al. Recovery in remitted first-episode psychosis at 7 years of follow-up of an early dose reduction/discontinuation or maintenance treatment strategy: long-term follow-up of a 2-year randomized clinical trial. *JAMA Psychiatry* 2013;70:913-20.
- 80 Leucht S, Tardy M, Komossa K, et al. Antipsychotic drugs versus placebo for relapse prevention in schizophrenia: a systematic review and meta-analysis. *Lancet* 2012;379:2063-71.
- 81 Sampson S, Mansour M, Maayan N, et al. Intermittent drug techniques for schizophrenia. *Cochrane Database Syst Rev* 2013;7:CD006196.
- 82 Lacasse JR, Leo J. Serotonin and depression: a disconnect between the advertisements and the scientific literature. *PLoS Med* 2005;2:e392.
- 83 Hindmarch I. Expanding the horizons of depression: beyond the monoamine hypothesis. *Hum Psychopharmacol* 2001;16:203-218.
- 84 Angoa-Pérez M, Kane MJ, Briggs DI, et al. Mice genetically depleted of brain serotonin do not display a depression-like behavioral phenotype. *ACS Chem Neurosci* 2014;5:908–19.
- 85 Castrén E. Is mood chemistry? *Nat Rev Neurosci* 2005;6:241–6.

Withdrawing psychiatric drugs

It is not only dangerous to start taking psychiatric drugs, it can also be dangerous to stop them. Withdrawal from psychiatric drugs should be done carefully under experienced clinical supervision.

PETER BREGGIN¹

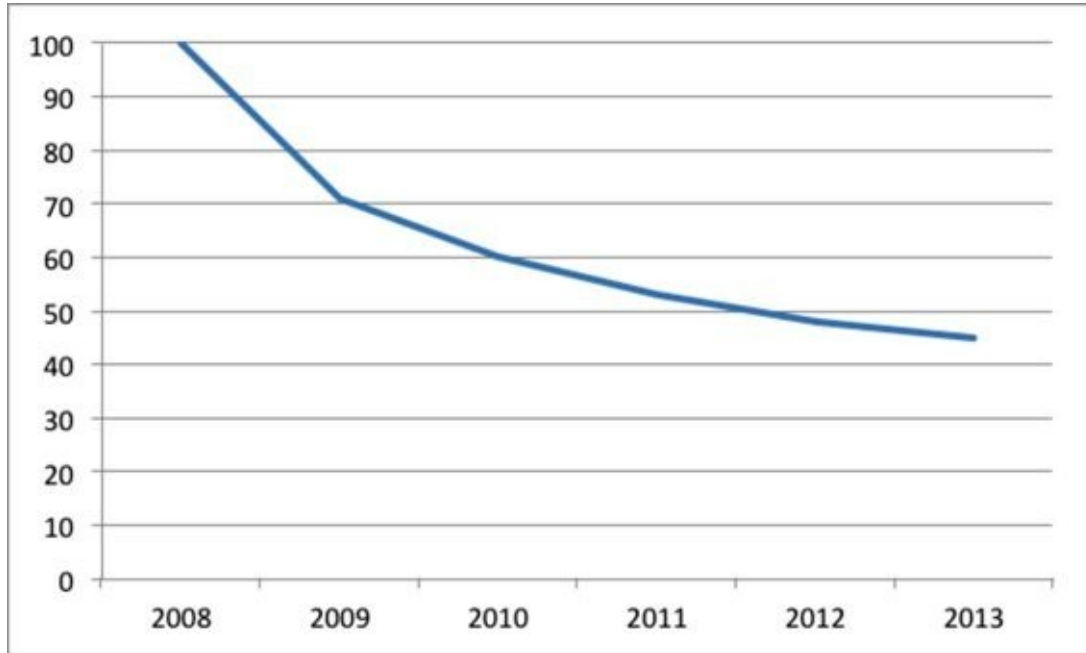
The worst drug epidemic ever

As explained in the last chapter, psychotropic drugs don't fix a chemical imbalance, they create one, which is why it is so difficult for many patients to come off the drugs again.

It is scary how many patients continue for years on end with SSRIs, particularly when considering that naturalistic studies have not found any benefit from long-term use.² In 2014 Finnish TV interviewed me for a documentary about depression, and the journalist had usage data from the Social Insurance Institution. Of 260,322 people who were on SSRIs in 2008, many were also on them the following years (some might have been temporarily off them). Even five years later, 45% still took them (Figure 12.1). What is most worrying is that the curve flattens out, which suggests that many patients are hooked on the drugs for life.

In New Zealand, 52% of patients reported having taken their antidepressants for more than three years,³ and in the United States, 60% were still taking their antidepressant after two years, just as in Finland, and 14% had taken the drug for at least 10 years.⁴ Overall usage for women aged 40-59 was a staggering 23%, and only 29% had seen a mental health professional within the last year. Such data indicate that the usage is out of control. If we conservatively assume that 30% of the patients take the drugs because they cannot get off them, it means that we have over 100,000 drug addicts in Denmark on SSRIs, which is about the same number of drug addicts we have on benzodiazepines.

Figure 12.1. Number of people out of 100 in Finland who were still taking an SSRI up to five years after starting a prescription.



General practitioners contribute the most to the misery. They deliver more than 90% of mental healthcare.⁵ In Australia, they prescribe about 90% of the antidepressants, most often for mild depression,⁶⁻⁸ despite the fact that there is international consensus that they don't work and shouldn't be used for mild depression, and 71% of the antipsychotics are also prescribed by them.⁷ I have heard an influential Danish general practitioner explain at a meeting that antidepressants are helpful as bridge therapy in mild depression before the patients can get an appointment for psychotherapy. This is very bad medicine.

To be on drugs year in and year out has little to do with having a disease and a lot to do with having a drug dependence. People with uncomplicated episodes of major depressive disorder (lasting no longer than two months and not including suicidal ideation, psychotic ideation, psychomotor retardation, or feelings of worthlessness) are hardly more likely to have a further episode within 12 months than people with no history of major depressive disorder, and the relapse rates are very low (3.7% versus 3.0%).⁹ Historical data are also revealing.¹⁰ Of Emil Kraepelin's 450 "depressed-only" patients from about a hundred years ago, 60% experienced only a single episode of depression, and only 13% had three or more episodes; similarly, more than half of 2,700 depressed patients admitted for a first episode from 1909 to 1920 in New York had only a single episode and only 17% had three or more episodes; and when 216 patients in Sweden were followed for 18 years, 49% never experienced a second episode, and an additional 21% had only one other episode.

Ages ago, before the huge abuse of psychotropic drugs started, depression was a self-limiting disease that in most cases was over in a few months, and even today the median duration of an untreated depression is only three months.¹¹

This is not what Figure 12.1 tells us. And it doesn't make it any better that the other major indication for SSRIs is anxiety, as it is similarly wrong to treat anxiety with addictive drugs for years. Treatment with antidepressants doesn't even seem to lower the risk of further bouts of depression; it seems to *increase* this risk.¹² The studies that show this suffer from various weaknesses, e.g. confounding by indication when treated and untreated patients are being compared, but even so, it's interesting that the median time to recovery in patients who suffered from a second episode of depression was 23 weeks when they received drugs and only 13 weeks when they didn't.¹¹

As explained in other chapters, it's clear that long-term treatment with psychotropic drugs is harmful. If it were true that psychiatric patients are comparable to patients with diabetes, the number of disabled mentally ill would have gone down after we introduced antipsychotics and antidepressants, but the number of people with psychiatric diagnoses forced into early retirement has exploded in all countries where the trend has been examined.¹⁰ I agree with Whitaker and Breggin that many, or likely even most, people on disability suffer from drug-induced harm, not from a mental illness. We have never seen such a gigantic catastrophe of iatrogenic disease production before.

The catastrophe has also hit our children hard. In 1987, just before the best-selling SSRIs came on the market and before the use of ADHD drugs skyrocketed, very few children were disabled by mental illness in the United States; 20 years later it was more than 500,000, a 35-fold increase.¹⁰ A 2002 survey of US child and adolescent psychiatrists showed that 91% of their patients were treated with psychiatric drugs.¹³ In only the remaining 9% was psychotherapy used without drugs. Overmedication is far worse in the United States than elsewhere, undoubtedly because of this country's ultraliberal traditions, also in healthcare. In 2000 psychotropic drugs were used in 6.6% of US children compared to 2.0% of German children.¹⁴ For stimulants, the rates were 4.3% vs. 0.7%, a six-time difference.

As Whitaker says, the huge overmedication would be impossible if psychiatry were honest,¹⁰ and *The Guardian* suggested what honest information might look like:¹⁵

Imagine that, after feeling unwell for a while, you visit your GP. "Ah," says the doctor decisively, "what you need is medication X. It's often pretty effective, though there can be side-effects. You may gain weight. Or feel drowsy. And you

may develop tremors reminiscent of Parkinson's disease." Warily, you glance at the prescription on the doctor's desk, but she hasn't finished. "Some patients find that sex becomes a problem. Diabetes and heart problems are a risk. And in the long term the drug may actually shrink your brain." Next comes a story that illustrates just how mad our societies have become with respect to biological psychiatry.

Imagine that a virus suddenly appears that makes people sleep 12-14 hours a day and move around slowly and become emotionally disengaged. Some gain 10, 20 or 40 kg of weight, their blood sugar and cholesterol go up, and they develop diabetes. People infected die substantially earlier than other people, some kill themselves, and parents panic over the thought that their children might also contract this horrible disease. Hundreds of millions of dollars are awarded to scientists to decipher the workings of the virus and they find out that it blocks a multitude of receptors in the brain – dopaminergic, serotonergic, muscarinic, adrenergic, and histaminergic – which lead to compromised brain function. MRI studies find that the virus shrinks the cerebral cortex, which is tied to cognitive decline. A terrified public clamours for a cure.

Such an illness has in fact hit millions of children and adults. What was just described are the effects of Eli Lilly's bestselling antipsychotic, olanzapine (Zyprexa).

I modified slightly a thought experiment invented by Whitaker.¹⁰ It is the same type of madness that keeps our societies totally indifferent to the fact that our prescription drugs are the third leading cause of death after heart disease and cancer.¹⁶

How can it be done?

My guess is that physicians do not stop the SSRIs because they have already had a few difficult experiences with what can happen. I suspect they want to think that the problems are not withdrawal-related, but a reappearance of the mythical chemical imbalance or a new onset of bipolar disorder. When you stop to think of how many patients the average family physician or psychiatrist puts on SSRIs, including themselves, family and friends, and the long-term results of these prescriptions, the cumulative misery effect is so large that if the physicians really became educated, they would be unable to live with themselves.

STUART SHIPKO, PSYCHIATRIST FROM CALIFORNIA¹⁷

Shipko describes his profession's organised denial. He is undoubtedly correct that many doctors shy away from taking their patients off their drugs because of bad experiences. It is also true that doctors know shockingly little about abstinence symptoms and about how to taper off drugs safely.¹ No one taught them how to stop drugs, whereas they have learned all too well from their silverbacks and the pharmaceutical industry how to start them and always to blame the disease for untoward symptoms, which helps them get out of their conundrum. It is much easier to renew a prescription than to stop an addictive drug, and it generates a much greater income, as more patients can be seen per hour.

People who want to stop drugs are mostly left to fend for themselves, sharing information on the Internet and through social media. A patient sent me her story about how she escaped the tyranny of life-long treatment and an incompetent psychiatrist:¹⁶

After a traumatic event, I was prescribed happy pills without adequate information about possible side effects. A year later, I asked the psychiatrist to help me stopping the drug, as I didn't feel it was helpful. She convinced me that I was undertreated and should have a higher dose and warned me against stopping the drug, as it could lead to chronic depression. During a time when the psychiatrist had long-term sick leave, I had the courage, supported by a psychologist, to taper off the drug. I had been on it for 3.5 years and had become more and more lethargic and indifferent to everything. It was like escaping from a cheese-dish cover. Tapering off the drug is not unproblematic, it gives you a lot of abstinence symptoms. When the psychiatrist returned after her illness, she was "insulted" about my decision to stop the drug. However, I was much better, and in reply to my question that I was no longer depressed, she said, "I don't know." "But if I don't want happy pills?" "Well, then I cannot help you!" was the answer. This psychiatrist had a close relationship to a manufacturer of happy pills.

Unfortunately, this story is pretty typical. I have received thousands of emails from patients and their relatives and they have inspired me to read more about patient experiences on websites. Scientific progress can be obtained in many ways and it is vitally important that we listen to patients' experiences with drugs. This is often far more reliable than what we can read in carefully manipulated reports of industry-sponsored research.

In a better world, psychiatrists would be eager to teach patients how to live without toxic substances in their brain, but in the real world many psychiatrists try to keep their patients on medications indefinitely.¹⁸ Doctors feel disrespected when patients ask to come off the drugs they have instituted, and a very common discharge notice in hospital patients' charts is: "The patient doesn't want drugs.

Discharged.”

It is therefore often psychologists, other therapists, pharmacists, friends and relatives that help patients come off their drugs. Some psychologists are worried about the possible legal implications, but given the disaster doctors have created and their unwillingness to help, I believe others must. When a system is harmful to people, we have a moral duty to fight it, and as long as the psychologists only help a patient psychologically who has decided herself to get off a drug, there shouldn't be any legal problems. I suspect the reluctance of some professionals is due to fear of being exposed in the media if something goes wrong (see below). For example, withdrawal psychosis is seen in 30-40% of long-term users of antipsychotics, and if a patient stops the drug and commits a horrendous act, everyone howls that patients must never stop taking their drugs.¹⁸ Few clinical challenges are more difficult and hazardous than removing antipsychotic drugs from a patient after years of exposure. Stopping lithium also involves a high risk of rebound mania.¹⁸

In 2012, the UK Royal College of Psychiatrists asked 817 patients what it was like for them to come off antidepressants, and 63% reported withdrawal symptoms.¹⁹ The most common symptom was anxiety (70%); other interesting symptoms were electric shocks/head zaps (48%), stomach upsets (33%), flu-like symptoms (32%), depression (7%) and suicidal thoughts (2%).

But the report ended with a false statement of course: “We would also like to reassure readers that despite some people having symptoms of withdrawal when stopping antidepressants, antidepressants are not addictive.”

Patients may get horrible symptoms when they try to stop the medication. A patient trying to get off olanzapine described it as pure hell, with terrible anxiety, severe panic attacks, paranoia, and horrible tremors.¹⁰ In contrast, it is usually easy to stop drugs in children, which is fortunate, as children should not receive psychotropic drugs.¹

Most patients are unable to judge themselves because the drugs have changed their brains. When the brain is deranged, it cannot detect that it is deranged; an outside observer is needed for this. The drugs reduce or cloud the highest human functions, including love, creativity and spirituality, and many people have no idea what they have been missing until they have come off their drugs.¹⁸ This is what Peter Breggin calls medication spellbinding.¹ While patients are on drugs, they often feel they have improved even when they have clearly become worse. It is important to know this. Doctors tend to believe when patients say they are well and will therefore be inclined to increase the dose to obtain further “improvement,” but the statement “I've never felt better in my entire life” may

simply mean that the patient is heading for a manic episode.¹ Antidepressants change people's lives for the worse while they think they are getting better.¹⁸

When patients are in the midst of painful psychiatric drug withdrawal, their brain is in a state of drug-induced crisis and it is truer than ever that they cannot believe what their mind tells them. Patients will usually feel they are themselves and will try to explain away their odd behaviour if confronted with it and during a tapering attempt they will often totally deny that they have become irritable, agitated, hostile or difficult in other ways and will react with anger over such "accusations."

This is one of the reasons it is so essential that patients are not alone, but that close relatives or friends observe them carefully. It can be downright dangerous if the family accepts the patient's false explanations. It is therefore preferable that the patient permits that friends and family can contact the therapist if they become concerned.¹

When patients have left suicide notes, only very rarely is there any indication that the drug was the problem; patients simply don't know this and think it is themselves that are the problem.¹

Abrupt withdrawal is particularly dangerous. With several classes of drugs, people may be struck by the extreme degree of restlessness known as akathisia, which predisposes to out-of-character violence, including suicide and homicide. A slow taper is necessary, which is best done with firm support from the family and close friends and in collaboration with a professional with considerable psychopharmacological knowledge. Unfortunately, few doctors have such knowledge and ever fewer are aware of the spellbinding phenomenon.

Spellbinding also works the other way round. When reducing drug dosage, patients may not notice any improvement or change in their condition, although everybody else can. Also in this case support is needed from significant others to prevent the patient from giving up the withdrawal attempt.

It is not surprising that many patients who have failed every time they tried have ultimately accepted their fate. It often requires strong determination, a lot of time, patience, and a long tapering period to get patients off the drugs while making the abstinence symptoms bearable. If patients have been on drugs for years, the tapering period may go up to more than a year. Danish psychiatrist Jens Frydenlund has told me that his record is eight years for an SSRI! He has worked with drug addicts for decades, and like other psychiatrists who have experience with both legal and illegal drugs,¹ he says that it is generally much easier to stop

heroin than to stop a benzodiazepine or an SSRI because the abstinence symptoms with heroin disappear rather quickly.

Frydenlund once listened to a new patient's story for two hours, which, according to her father, was the first time anyone had listened to her, and concluded by telling the patient that she didn't have schizophrenia but was a pill addict. He slowly took her off the monstrous amounts of drugs she was on, one by one, and she got well.

Half the patients with schizophrenia are in co-treatment with benzodiazepines, although this increases mortality (see Chapter 6), but Frydenlund has succeeded in getting almost all the patients off their benzodiazepines at two hospitals where he worked. The National Board of Health inspected him three times in 2014, as they are worried that he uses so few drugs! Another Dane, child psychiatrist Lisbeth Kortegaard, has stopped ADHD drugs in virtually all children that her colleagues had medicated and her experience is the same as Breggin's,¹ that the kids improve considerably and become normal kids again when she takes them off their drugs. Her colleagues became so hostile towards her going against the grain that I encouraged her to leave her post as chief physician and open her own practice, focusing on detoxifying children, which she has now done.

What we need more than anything else in psychiatry are detox clinics all over the country, in all countries, with easy and quick access free of charge, and education about the harmful effects of psychotropic drugs and how to stop them. Millions of people worldwide need help, and public investment in detox clinics would be highly profitable and beneficial in terms of fewer disability pensions, fewer drug deaths (see Chapter 14), much healthier citizens, and fewer serious crimes (see earlier chapters).¹⁸

It is of utmost importance to tell people how they can come off the drugs without consulting the doctor who started them on drugs, as he will often be against it. It is a sad testimony to this fact that most attempts to reduce or stop drugs are initiated by patients or their families.¹ Families often react to the chronic brain damage they have observed, with the patient becoming lethargic, apathetic, indifferent, caring less, suffering from memory lapses or doesn't seem like himself anymore.

Peter Breggin has written a most instructive book about drug withdrawal symptoms and how tapering should be carried out.¹ It should be obligatory reading for all doctors who use psychotropic drugs, as it will change forever the way they use them. The book is also very useful for patients and their relatives and friends, as it can help patients get safely off their drugs against their doctor's wishes. There are also useful websites, created by non-profit organisations or

psychiatrists.²⁰⁻²³ One should only taper off one drug at a time.

People who have succeeded in coming off psychiatric drugs and returning to a normal life often call themselves psychiatric survivors, which is a very apt term. One such person, lawyer Jim Gottstein, has given this advice:²⁴

You have to take responsibility for your own mental health and behaviour.

You have to learn to recognise your symptoms.

You have to learn what works for you.

Breggin also advises that the patient should be in charge of the pace of the taper and warns that, without the patient's own motivation and determination, withdrawal attempts are likely to fail.¹ Many patients report that the very last step, where they go from a very small dose to nothing, is the worst, so this is when strong determination and support is most needed.

It is important to reverse the medical disempowerment that is usually at play, and Breggin explains why:¹

Nurses, psychologists, social workers, teachers and other non-prescribing people have often been taught that their task is to push patients to comply with the prescribed medication. In this authoritarian model, the physician stands atop the professional hierarchy and prescribes pills much as one would expect an all-knowing judge to dispense justice. Like the patient, those lower down the hierarchy are supposed not to make any independent judgments or comments about the drugs. This model is not feasible. Patients and their families can look up information about drugs on the Internet, and so can non-prescribing professionals, and they can quickly learn more about a drug's harms than their doctor, who talks to salespeople, listens to industry-sponsored lectures, and relies on data from industry-run trials. Furthermore, psychotherapy requires an honest and open dialogue with the patient that cannot exclude discussing the patient's medication, particularly as the drugs are often the cause of the patient's problems.

A more egalitarian and respectful model of treatment is the norm in other areas of medicine, e.g. in diabetes, which the patients often control themselves. In the mental health field, however, where the patient's self-determination is much more important than in other areas of medicine, the authoritarian model is still alive and well. This must stop.

References

- 1 Breggin P. Psychiatric drug withdrawal: a guide for prescribers, therapists, patients, and their families. New York: Springer Publishing Company; 2013.

- 2 Hughes S, Cohen D. A systematic review of long-term studies of drug treated and non-drug-treated depression. *J Affect Dis* 2009;118:9–18.
- 3 Read J, Cartwright C, Gibson K. Adverse emotional and interpersonal effects reported by 1829 New Zealanders while taking antidepressants. *Psychiatry Res* 2014;216:67-73.
- 4 Pratt LA, Brody DJ, Gu Q. Antidepressant use in persons aged 12 and over: United States, 2005–2008. *NCHS Data Brief* 2011;76:October.
- 5 Jacob KS, Patel V. Classification of mental disorders: a global mental health perspective. *Lancet* 2014;383:1433-5.
- 6 Raven M. Depression and antidepressants in Australia and beyond: A critical public health analysis. PhD thesis, University of Wollongong, Australia; 2012. <http://ro.uow.edu.au/theses/3686/>
- 7 Karanges EA, Stephenson CP, McGregor IS. Longitudinal trends in the dispensing of psychotropic medications in Australia from 2009-2012: focus on children, adolescents and prescriber specialty. *Aust N Z J Psychiatry* 2014;48:917-31.
- 8 McManus P, Mant A, Mitchell P, et al. Use of antidepressants by general practitioners and psychiatrists in Australia. *Aust N Z J Psychiatry* 2003;37:184-9.
- 9 Dowrick C, Frances A. Medicalising unhappiness: new classification of depression risks more patients being put on drug treatment from which they will not benefit. *BMJ* 2013;347:f7140.
- 10 Whitaker R. *Anatomy of an Epidemic*. New York: Broadway Paperbacks; 2010.
- 11 Posternak MA, Solomon DA, Leon AC, et al. The naturalistic course of unipolar major depression in the absence of somatic therapy. *J Nerv Ment Dis* 2006;194:324-9.
- 12 Patten SB. The impact of antidepressant treatment on population health: synthesis of data from two national data sources in Canada. *Population Health Metrics* 2004;2:9.
- 13 Stubbe DE, Thomas WT. A survey of early-career child and adolescent psychiatrists: professional activities and perceptions. *J Am Acad Child Adolesc Psychiatry* 2002;41:123-30.
- 14 Zito JM, Safer DJ, de Jong-van den Berg LTW, et al. A three-country comparison of psychotropic medication prevalence in youth. *Child and Adolescent Psychiatry and Mental Health* 2008;2:26.
- 15 Freeman D, Freeman J. At last, a promising alternative to antipsychotics for schizophrenia. *The Guardian* 2014 March 7.
- 16 Gøtzsche PC. *Deadly medicines and organised crime: How big pharma has corrupted health care*. London: Radcliffe Publishing; 2013.
- 17 Shipko S. Playing the odds, revisited. 2014 July 21. <http://www.madinamerica.com/2014/07/shooting-odds-revisited/> (accessed 11 Oct 2014).
- 18 Breggin P. *Medication madness*. New York: St. Martin's Griffin; 2008.
- 19 Coming off antidepressants. Royal College of Psychiatry. <http://www.rcpsych.ac.uk/expertadvice/treatmentswellbeing/antidepressants/comingoffantidepressants.aspx> (accessed 16 Oct 2013).
- 20 Stopping psychiatric drugs. <http://www.mind.org.uk/information-support/drugs-and-treatments/medication-stopping-or-coming-off/making-the-decision-to-come-off/#.UsvmnuJkzNo> (accessed 21 Nov 2014)
- 21 Stopping antidepressants. <http://www.btpinfo.org.uk/withdrawing-from-antidepressants> (accessed 21 Nov 2014).
- 22 <http://ww2.antidepressantsteps.com/?folio=9POR7JU99> (accessed 21 Nov 2014).
- 23 Stopping antidepressants. RxISK Guide to Stopping Antidepressants (accessed 21 Nov 2014).
- 24 Gottstein J. Mental health recovery stories. <http://akmhcweb.org/recovery/jgrec.htm>. 1999 Aug 27

(accessed 21 Nov 2014).

Organised crime, corruption of people and science, and other evils

We have allowed psychiatry's medico-industrial complex to grow like a malignant tumour, sending metastases in all directions in our societies, and we have allowed the psychiatric oligarchs to medicalise normality – even in our schools and in preschool children – and turning what were previously acute conditions into chronic ones. Another similarity to malignant tumours is that psychiatric drugs kill huge numbers of people (see next chapter).

Bribery and illegal marketing have to a substantial degree contributed to the drug epidemic. Although both practices are illegal, 52% of Danish general practitioners have experienced a salesperson saying the drug could be used outside the approved indication, and 34% have been exposed to information about drugs based on unpublished results.¹

We have allowed drug companies to commit habitual crime that involves pervasive corruption of leaders among doctors and other decision-makers and to lie to us routinely, both in their research and marketing and in their interactions with drug regulators.²

According to the definitions in US law, big pharma commits organised crime,² and the crimes have been particularly evil in psychiatry. A disproportionate number of the criminal activities have involved psychiatric drugs and have included illegal marketing, Medicare and Medicaid fraud, bribery of doctors, civil servants and politicians right up to the ministerial level, and disposal of evidence.

In 2012, I combined the names of the ten largest drug firms with “fraud” on Google and much of what I found on the first search page involved psychiatry.² The cases I described were often about illegal promotion to our most vulnerable citizens, children and the elderly, often accompanied by kickbacks to doctors to induce them to use particular drugs:

- ziprasidone, an antipsychotic, Pfizer
- pregabalin, an epilepsy drug, Pfizer

- oxacarbazepine, an epilepsy drug, Novartis
- bupropion, an antidepressant, GSK
- paroxetine, an antidepressant, GSK
- lamotrigine, an epilepsy drug, GSK
- quetiapine, an antipsychotic, AstraZeneca
- risperidone, an antipsychotic, Johnson & Johnson
- olanzapine, an antipsychotic, Eli Lilly
- valproate, an epilepsy drug, Abbott

Psychiatry is a goldmine for big pharma.² The diagnoses are vague and easy to manipulate, and many drugs are hilariously expensive, although they are not any better than off-patent drugs, but that's no hindrance for their widespread use.

Leading psychiatrists are at high risk of corruption, both financially and of their academic integrity and objectivity, and in fact psychiatrists collect more money from drug makers than doctors in any other specialty; they are also “educated” with industry’s hospitality more often than any other specialty.³ Leading psychiatrists are highly effective drug pushers, also for uses that are illegal for the companies to advertise, and they are often very well paid for their “assistance,” even when they publish textbooks and papers that ghostwriters have written for them,² which is considered scientific dishonesty.

Until 2015, it was little known that Allen Frances, chairman of DSM-IV, which has earned the American Psychiatric Association more than \$100 million, and two other psychiatrists, John P. Docherty and David A. Kahn, had been very active in promoting risperidone in return for cash.⁴ They received about one million dollars for helping Johnson & Johnson market risperidone partly illegally, for non-approved indications. It started in 1995, one year after publication of the DSM-IV, and involved what an ethics specialist has called serious deception, corruption and distortion of the scientific evidence. Frances and his colleagues wrote guidelines designed specifically to persuade physicians to prescribe risperidone as first-line treatment for schizophrenia. These guidelines were not independent, and they were developed in close collaboration with the company. When they were done, the three psychiatrists established Expert Knowledge Systems for the purpose of creating and helping implement a risperidone marketing plan, which included to “influence state governments and providers” and to identify key opinion leaders who could tout risperidone at meetings advertised as Continuing Medical Education lectures. Johnson & Johnson later had to pay more than \$1.5 billion for its unlawful and deceptive marketing that included lies about the life-threatening harms of risperidone.²

In 2006, the drug industry accounted for about 30% of the American Psychiatric Association's \$62.5 million in financing.⁵ Psychiatrists who received at least \$5,000 from makers of antipsychotics wrote three times as many prescriptions to children as other psychiatrists although the drugs were not approved for most uses in children. Joseph Biederman and other Harvard psychiatrists, who more than anybody else have pushed drugs heavily to children, had underreported their earnings to university officials; each of them had made more than a million dollars from drugs makers during eight years. As is well known, studies have shown that researchers who are paid by a company are more likely to report positive findings when evaluating that company's drugs.²

It is very lucrative for doctors to participate in industry-sponsored trials, as they get publications, fame and other benefits.² Above all, they get money, which can be used for other research at the clinic or to supplement the doctor's private economy. Specialists may receive as much as \$42,000 for enrolment of one patient in a trial, and patients may therefore be coerced into participating, or coerced into continuing taking a drug that gives them unpleasant side effects or even increases their risk of dying, as payment is sometimes only provided for patients that stay in the trial to its planned finish date.

Top psychiatrists can earn millions of dollars for themselves and may attract much larger sums for their institutions, which explains why they cover up for the psychiatrists' misdeeds. As an example, the corruption at Emory University where Charles Nemeroff worked was kept secret for more than a decade.² One reason why the scam could continue for so long was that at least 15 whistleblowers were ordered psychiatric evaluations by Emory's psychiatrists who reportedly wrote up such exams without even examining the targeted doctors or gathering factual evidence, where after several of them were fired. Some of these "evaluations" were done by Nemeroff himself.

Doctors who take money from many companies usually argue that they are not in the pocket of industry because they are not dependent on any particular company. But company people don't see it that way; they call them drug whores.⁶ What the doctors really say is that it's okay to be a prostitute as long as you make sure you have many customers every day so that you aren't dependent on any particular one.

Our academic institutions have also allowed themselves to become corrupted. They grant ownership to the collected data to the sponsor and often accept that the doctors will have little influence on any publications.⁷ The competition for research funds means that companies can shop among the various academic centres and choose those who are least willing to raise uncomfortable questions.

Patients know perfectly well they cannot trust the pharmaceutical industry. In large surveys, it ended up at the bottom, along with automobile repair shops and tobacco companies, in terms of the confidence people had in the industries.² The reason patients trust their medicines is that they extrapolate the trust they have in their doctors into the medicines they prescribe. Patients don't realise that virtually all of their doctors' knowledge about drugs comes from the industry they don't trust. Furthermore, they don't know that their doctors may have self-serving motives for choosing certain drugs for them, such as a well-paid second job in a company, which many doctors have,² and that many of the drug industry's crimes are only possible because corrupt doctors contribute to them.

The health professionals' main task is to find out whether the treatments they might consider using have beneficial effects that exceed the harmful ones. When it comes to drugs, we have completely failed in this task. We have left it to the industry to carry out the pivotal placebo controlled trials, although we know that companies often cheat with their studies,² which they can do behind closed doors because they don't allow others to get access to the raw data for re-analysis.

Not even the doctors who provided the data are allowed access to the full data set, but it is the patients who suffer the consequences of this corruption of the evidence base.² When the FDA in 2003 was reviewing unpublished data from trials of SSRIs in children and adolescents to see if the drugs increased suicide risk, the academics who had published positive results of these drugs were worried and issued a report disputing evidence that their use increased suicidal behaviour. They had contacted the companies to get access to the data they had themselves generated, but some drug companies refused to provide them.⁸

It is really scary and totally unacceptable that the only people in the world who have seen the entire dataset in industry trials are company employees.

If independent investigators want to do their own trials and ask a company for matching placebos, this request will often be refused, or the company will demand a ludicrous sum for the placebos knowing that this will stop the trial.²

Industry-sponsored randomised trials aren't science. Medical science aims at finding the truth and at improving the treatment of patients, but when trial data are secret, it cannot be considered science. It is marketing dressed up as science, and the trials are often flawed.² Rigorous science should put itself at risk of being falsified but the industry protects its hypotheses by *ad hoc* modifications, or by designing trials that make them immune to refutation.² Thereby, the industry puts

its hypotheses in the same category as pseudoscience.⁹

The industry also often changes the analysis plan once the sponsor has seen the data.^{2, 10, 11} Until recently, it was difficult to detect such cheating, as trial protocols were regarded as confidential. About 15 years ago, however, our research group succeeded in getting access to a cohort of protocols submitted to a research ethics committee in Copenhagen.¹⁰ We included 102 trial protocols and their corresponding publications; three-quarters of these trials were industry funded. To our great surprise, at least one protocol-defined primary outcome had been changed in 63% of the trials. And in 33% of the trials, a new primary outcome was introduced in the published report that didn't exist in the protocol. *Not a single publication acknowledged that primary outcomes had been changed!*

The reason this is so devastating for the trustworthiness of trials is that there are often many outcomes, which may be further divided or combined, creating even more chances of hitting the bull's eye. Roughly just half of all trials are ever published, and in those that see the light of day only half of the outcomes are included.^{2, 10} What we are left with is therefore only a quarter of the studied outcomes, and this quarter may have been subjected to data torture until they confessed.

Psychiatry provides many examples of the fact that we cannot trust industry-sponsored research at all.² For example, what predicted the effect of fluoxetine in head-to-head trials against other antidepressants was who the sponsor was.¹² When fluoxetine was the experimental drug, fluoxetine was best, and when it was the control drug, it was worst. As another example, a study of 142 trials of six antipsychotics or antidepressants showed that most deaths (62%) and suicides (53%) were not reported in articles when compared with summaries of the same trials on websites.¹³

Randomised trials were introduced to protect us from the many useless treatments on the market, but oddly enough, they have given the ultimate power of knowledge production to big pharma that now uses them for getting approval for treatments of little or no value, and which are often harmful.² It's very strange that we have accepted a system where the industry is both judge and defendant, as one of the most firm rules in laws of public administration is that no one is allowed to be in a position where they evaluate themselves.

Drummond Rennie, deputy editor at JAMA, has explained how corrupt industry-supported drug trials are by comparing them with court trials.¹⁶ In a court trial, the various parties, judge, jury, opposing counsels, witnesses and police, are independent of one another. In a clinical trial, it is very much in the interest of the drug's sponsor to make everyone in the process its dependent, fostering as many

conflicts of interest as possible. The sponsor designs the trial so that it will likely have an outcome that pleases the sponsor; the sponsor pays those who collect the evidence, often doctors or nurses, and pays those who analyse the evidence, drops what is inconvenient, and keeps it all secret – even from the trial physicians. The sponsor deals out to the FDA bits of evidence and pays the FDA (the judge) to keep it secret. Panels (the jury), usually paid consultant fees by the sponsors, decide on FDA approval, often lobbied for by paid grass-roots patient organisations who pack the court. If the trial is positive for the sponsor, the sponsor pays subcontractors to write up the research and impart whatever spin they may; they pay “distinguished” academics to add their names as “authors” to give the enterprise credibility, and often publish in journals dependent on the sponsors for their existence. If the drug seems no good or harmful, the trial is buried and everyone reminded of their confidentiality agreements. Unless the trial is set up in this way, the sponsor will refuse to back the trial, but even if it is set up as they wish, those same sponsors may suddenly walk away from it, leaving patients and their physicians high and dry. In short, we have a system where defendant, developers of evidence, police, judge, jury, and even court reporters are all induced to arrive at one conclusion in favour of the new drug.

Our drug regulators should be impartial judges, but they are part of the problem. They are firmly against providing independent researchers access to unpublished study reports, trial protocols and other data in their possession, constantly arguing that they need to protect the commercial interests of the drug industry.¹⁵ They seem to have forgotten that their job is to protect the patients. They are keen to protect themselves and keep their work away from public scrutiny. Corruption at the FDA is common, and when the higherups overrule the agency’s own scientists and make obviously harmful decisions, it smells of corruption.² I don’t think patients are aware that the drug regulatory authorities expose them to many ineffective and harmful drugs that kill them in huge numbers.²

In matters of health, there should be no tolerance for deception,¹⁶ and yet, that is exactly what we often see in industry-sponsored research and marketing. Lundbeck has illustrated this with escitalopram, and I have previously described in detail how Lundbeck convinced the world that a molecule can be better than itself.²

Lundbeck’s evergreening of citalopram

Lundbeck’s drug citalopram (Cipramil or Celexa) was one of the most widely used SSRIs, and when the patent ran out, Lundbeck had a new patent ready for the

same substance. Citalopram consists of two halves, which are mirror images of each other, but only one of them is active. Lundbeck threw out the inactive half, patented the active half, and called the rejuvenated me-again drug escitalopram (Cipralext or Lexapro).

Lundbeck delayed market entry of cheap, generic citalopram by paying the manufacturers to stay away and was fined €94 million by the European Commission for this violation of EU antitrust rules.¹⁷ Lundbeck produced some trials comparing the two versions of the molecule and claimed that me-again was better than old me,¹⁸ but even if we take the results at face value, they showed that there was no meaningful difference between the two versions of the drug; after eight weeks the difference was 1 on a scale that goes up to 60, which is irrelevant (see Chapter 3).

Interestingly, this happened in the country where Hans Christian Andersen wrote *The Emperor's New Clothes*. Independent researchers found that the efficacy appeared to be better for escitalopram than citalopram (odds ratio 1.60; 95% confidence interval 1.05 to 2.46) in head-to-head trials, but when they did an indirect comparison of the two drugs based on 10 citalopram and 12 escitalopram placebo controlled trials, the efficacy was similar (odds ratio 1.03; 0.82 to 1.30).¹⁹ Usually, direct comparisons are more reliable than indirect comparisons, but the drug industry distorts its research to such an extent that the indirect comparisons are sometimes the most reliable ones.

Lundbeck's partner in America, Forest, was fined more than \$313 million and faced numerous lawsuits from parents of children who had either committed or attempted suicide, and Forest pleaded guilty to charges relating to obstruction of justice and illegal promotion of citalopram and escitalopram for use in children and adolescents.^{2, 20} Six years earlier, a Forest executive lied before Congress saying that Forest followed the law and had not promoted any of the drugs to children.

Forest engaged in widespread corruption of doctors to promote Lundbeck's drugs,² and in 2009 the US Senate released some really nauseating documents it had requested from Forest.²¹ Forest would communicate that escitalopram offers superior efficacy and tolerability over all SSRIs, which is totally surreal. I have never heard of any drug that is both more effective and safer than all other similar drugs. We are told that sales mirror the promotional effort, which is true. Sales of SSRIs are closely related to the number of drugs on the market ($r=0.97$),²² and in the United States each new agent added to the aggregate use without a concomitant decrease in previously introduced agents,²³ which shows that the use of these drugs doesn't reflect a genuine need; it's about marketing.

Furthermore, the Forest documents speak about producing ghostwritten articles for “thought leaders,” and the company recruited about 2,000 doctors as drug pushers to tout Lexapro at meetings, using the slide kit prepared by Forest.

There was also a huge programme of “trials,” where the results seemed to have been determined beforehand, before the trials had even started, and of course there were “unrestricted grants” to help the American Psychiatric Association and others to develop “reasonable practice” guidelines, which was about improving “the percent of patients who adhere to the full duration of therapy.” Total corruption of academic medicine resulting in immense harms to the many patients who cannot get off the drug once they have adhered to “the full duration of therapy.”

So what was Lundbeck’s reaction to the partner’s crimes? “We know Forest is a decent and ethically responsible firm and we are therefore certain that this is an isolated error,” said the sales director of Lundbeck.²⁴ Perhaps this confidence in Forest’s business ethics is related to the fact that Lexapro had sales of \$2.3 billion in 2008.² At any rate, it’s perverse that anyone calls a company like Forest decent and ethically responsible.

Lundbeck’s CEO Ulf Wiinberg also had an interesting sense for business ethics. In 2014 he had to leave his job because he had received shares as a “gift” from a company in which Lundbeck bought shares one month later.²⁵ The value of the shares was about \$1 million, and in addition to this, Wiinberg received about \$3 million from Lundbeck when he left.²⁶ He likely won’t need social benefits. He has already taken them himself.

Psychiatry’s fantasy world

The leading psychiatrists have created a pseudo-world of their own, full of erroneous ideas based on poor science and pseudoscience, particularly in relation to the validity of diagnoses, the effects of diseases on the brain, and the effects of drugs on patients. In this pseudo-universe, they have been heavily supported and seduced by a criminal drug industry that has earned billions on the lies while killing millions of patients, many of whom should not have received the drug that killed them.

Psychiatrists are supposed to be experts on psychiatric drugs, but they aren’t – they are surprisingly ignorant. In 2014, the American Psychiatric Association wrote on its homepage about depression that:²⁷

“Antidepressants may be prescribed to correct imbalances in the levels of chemicals in the brain. These medications are not sedatives, ‘uppers’ or

tranquilizers. Neither are they habit-forming. Generally antidepressant medications have no stimulating effect on those not experiencing depression.”

All of this is wrong, and even healthy people can develop numbness or mania on an antidepressant. The association furthermore noted that, “If a patient feels little or no improvement after several weeks, his or her psychiatrist will alter the dose of the medication or will add or substitute another antidepressant.” There is no good evidence that it’s helpful to increase the dose or switch between drugs, and if it seems that a drug is better than another it’s very likely just because the patient would have improved anyway at this point in time, even without treatment.

In 2008, John Ioannidis from Stanford remarked that the assumed effectiveness of antidepressants perhaps was a myth constructed from small randomised trials with non-relevant outcomes, improper interpretation of statistical significance, manipulated study design, biased selection of study populations, short follow-up, and selective and distorted reporting of the results.²⁸ He also asked for very large long-term trials.

Three years later, a group of prominent psychiatrists responded:²⁹

“Persistent, untreated depression produces a type of neurodegenerative disorder, associated with synaptic changes ... Similar to poor control of blood sugar in diabetics, poor control of symptoms in Major Depression is associated with worse long-term outcome and greater overall disability ... antidepressants prevent relapses ... 53% of the placebo patients relapsed, whereas only 27% of drug-treated patients relapsed ... After the FDA issued a black warning [sic] against antidepressants, antidepressant prescriptions for this population diminished and there has been a concomitant increase in actual suicide ... There have been concerns regarding whether certain antidepressants may cause suicides. We now know this is a myth largely fuelled by the media ... Newer studies of children do not confirm an increase in suicidal ideation ... Naturalistic studies show that the incidence of suicide rate tends to go down as the incidence of antidepressant treatment goes up.”

All of this is seriously wrong or misleading. These authors quoted Robert Gibbons three times and Göran Isacson once (see Chapter 3 about their research) to “prove” their point that antidepressants protect children against suicide, although the truth is that they double the suicide risk. Their paper is from 2011. I fail to understand how Stefan Leucht, who has published much good research and is an editor in the Cochrane Schizophrenia Group, could coauthor all this harmful nonsense, but it says something about how deep the collective delusions and denial in psychiatry go. They hit even the best of psychiatrists. It is very, very tragic.

A 2012 newspaper article called “Behind the myths about antipsychotics” was similarly tragic and also deeply ironic because its authors, four leading Danish psychiatrists, propagated what they warned about, myths about antipsychotics.³⁰ They wrote that:

Most patients suffering from schizophrenia have disturbances in the dopamine system; the genes are by far most important (about 70-80%); large international registry studies show that patients with schizophrenia who are not treated with antipsychotics are at higher risk of dying prematurely than patients who are in treatment; numerous studies have documented that the risk of new psychotic episodes and a more severe course of the disease is increased if patients stop taking the antipsychotic medicine; in our large study, we found no indications that polypharmacy with antipsychotics increases mortality; and large register-based studies in Denmark and Finland show that concomitant treatment with several antipsychotics is not associated with increased mortality.

All of this is totally wrong or seriously misleading (see Chapter 6). I shall describe why these myths are so harmful for patients based on a patient story that comes from one of the four authors’ university hospital in Copenhagen. The patient had been admitted with mania, and although he had asked not to be treated with drugs, he received forced treatment with olanzapine, and in his own words: At discharge, when I had been declared cured after my first episode diagnosis, I tried to behave well, fearing that I might not be released after all. The psychiatrist forcefully urged me to continue with olanzapine. I didn’t dare tell her that I had spat out most of the pills in the washbasin and therefore asked, for the sake of appearances, for how long she thought I should take the drug? For the rest of my life, she replied, because I had a chronic disease, with a great risk of relapse, and I need not be afraid of the side effects.

The patient didn’t take the drug because he had read a newspaper article I published in January 2014 about ten myths in psychiatry,³¹ which also exists in English,³² and he has been well ever since without drugs.

Is there any hope for a specialty like this? I have heard critical psychiatrists say that leading psychiatrists seem to suffer from cognitive dissonance, as what they see and hear doesn’t influence them. Many patients who have managed to get out of the medicine hell by their own efforts say that the drugs have stolen many years of their lives.

A Danish witch hunt

The same day my article about the ten myths appeared, the chairman of the Danish

Psychiatric Association declared in the same newspaper, on its website: “Antidepressant drugs protect against suicide.” A month later, 16 Danish professors in psychiatry responded to my article, but without mentioning my name, just like one was not supposed to mention the evil Voldemort’s name in Harry Potter.³³ They wrote that a number of studies show that treatment with antipsychotics increase longevity, compared with no treatment. This isn’t true; antipsychotics shorten life (see Chapter 6).

My first article was “Psychiatry gone astray.”³¹ I responded to the professors in a new article, which I called “Leading psychiatrists have still gone astray.”³⁴

Two months later, the Danish Psychiatric Association attempted character assassination of me and they almost succeeded. They wrote a one-page letter to the Cochrane Schizophrenia Group and to the Cochrane Depression, Anxiety and Neurosis Group in which they complained about my first article.³¹ They focused on a sentence, which they quoted out of context: “the citizens of Denmark would be better off, if all psychotropic drugs were withdrawn from the market.” In actual fact, my article ended thus:³¹

“Psychotropic drugs can be useful sometimes for some patients, particularly in short-term use, in the acute situations. But after my studies in this area, I have arrived at a very uncomfortable conclusion: Our citizens would be far better off if we removed all the psychotropic drugs from the market, as doctors are unable to handle them. It is inescapable that their availability causes more harm than good. The doctors cannot handle the paradox that drugs that can be useful in short-term treatment are very harmful when used for years and create those diseases they were meant to alleviate and even worse diseases. In the coming years, psychiatry should therefore do everything it can to treat as little as possible, in as short time as possible, or not at all, with psychotropic drugs.”

The psychiatrists mentioned in their letter that I had been criticised by the Minister of Health, the director of the National Board of Health, the director of the Danish Patients Association, the president of the Cancer Society, the president of the Danish Psychiatric Association and the president of the Organisation of Danish Medical Societies. The criticism by the last-mentioned president actually came six months earlier, when my book about the drug industry’s crimes was published.² His criticism was erroneous and he appeared not to have read my book, but that didn’t affect the psychiatrists the slightest bit, as they were now on a

witch hunt. They enclosed a translation of his criticism in their letter, but not my published rebuttal of his criticism. They ended their letter by asking: “How do you, with the specific knowledge you have on antipsychotics and antidepressants, respectively, evaluate Peter Gøtzsche’s statements as presented in his article. We would be very pleased if you would take up the task of making such an evaluation.”

It wasn’t the editors in the two review groups who responded to the letter, but Cochrane’s CEO, deputy CEO and two other people in Cochrane’s leadership. I wasn’t consulted on the Cochrane leadership’s response to the psychiatrists. I knew that Cochrane had received various complaints from people and organisations about whether Cochrane supported my views on the matter as expressed in my book,² but not that the Danish Psychiatric Association had contacted Cochrane.

While I was on holiday in a jungle in Panama, surrounded by birds, tarantulas, monkeys, butterflies and sloths, with little contact to the outside world, and therefore with no chance of defending myself, the news that my own organisation had denounced me ran amok in the Danish media, and the psychiatrists celebrated their kill by reading aloud Cochrane’s letter at the Danish Psychiatric Association’s annual meeting.

The letter from Cochrane’s leadership, which was only one page, said that, “Cochrane is treating very seriously the points you raise concerning comments made by Professor Gøtzsche on the use of psychotropic medication. I want to state explicitly that these are not the views of The Cochrane Collaboration on this issue and we do not endorse them.” The letter furthermore noted that I was speaking on my own behalf, which was correct, and as “part of the promotional work” I conducted surrounding publication of my book, which wasn’t correct. I wrote my newspaper article in order to draw attention to some major problems in psychiatry and it started thus:

“I have researched antidepressants for several years and I have long wondered why leading Danish psychiatrists, including several professors, base their practice on a number of erroneous myths, which are unfounded. These myths are harmful to the patients, particularly since Danish psychiatry is extremely top-down controlled. Many psychiatrists are well aware that the myths don’t hold and have told me so, but they don’t dare deviate from the official positions because of career concerns. Being a specialist in internal medicine, I don’t risk ruining my career by incurring the professors’ wrath and I shall try here to come to the rescue of the many conscientious but oppressed psychiatrists and patients by listing the worst myths and explain why they are harmful.

There was only one reference to my book in the article, and it was necessary,

as I wrote that I had estimated in my book that just one of the many antipsychotics, olanzapine (Zyprexa), has killed 200,000 patients worldwide.

The Cochrane letter also stated that, “The views contained in this book are also not the views of Cochrane.” This comment was unnecessary, as it is evident that what people write in their books are their own views, not those of any organisation, and as the psychiatrists had not referred to my book. Furthermore, my book is not about my personal views. It’s about facts, and with its more than 900 references, it’s unusually well documented.

There was a sentence in the Cochrane letter, which was not a response to an issue the psychiatrists had raised but stood on its own and was misunderstood: “We will be asking Professor Gøtzsche to share with Cochrane colleagues any unpublished data that is not yet publicly available, so that it can be incorporated objectively into new or existing Cochrane Systematic Reviews as appropriate; and then be seen and evaluated by you [the Danish psychiatrists] and other specialists in the field.”

It came as no surprise to me that Danish journalists interpreted this as meaning that I had now come under Cochrane censorship and wouldn’t be allowed to publish anything unless it had been approved by Cochrane. This was of course not the intention of the letter. We do research on unpublished data we have obtained from the European Medicines Agency about antidepressants, but we are evidently under no obligation to share these data with anyone before we have finished our own research and decide to do so of our own free will. Our first papers have come out and they show that the clinical study reports contain extensive data on major harms that don’t exist in published journal articles or in trial registry reports.^{35, 36}

The letter from Cochrane’s leadership proved to be a threat to what I had built up over 30 years, including my centre, which is on government funding. The Minister of Health declared publicly that my person and the centre wasn’t the same thing, which I and my senior researchers interpreted as meaning that I could be fired. Very weird indeed, as I had done nothing wrong. I simply pointed out what I have detailed in this book and what many others have pointed out before me.

Cochrane’s leadership sent a letter to the newspaper that broke the story explaining that there had been misunderstandings. But it was too late. The damage had been done and not a single journalist admitted they had misrepresented the first letter, even though some of what they had written was demonstrably untrue. I published various rebuttals, including an article with a similar title as one of H. C. Andersen’s famous novels, “The Cochrane feather that became five hens,” which is about how rumours become established truths when they are repeated often

enough.

The newspaper that broke the story seriously misrepresented the first Cochrane letter,³⁷ which the second letter pointed out. For example, the subheading stated that I did not have support “for a number of controversial statements about the drug industry and the use of psychiatric medicine.” Some people interpreted this as an acquittal of the drugs industry’s dirty methods and a verdict about my book, but Cochrane now clarified that it neither supported nor refuted my interpretation of the evidence. The newspaper wrote that, “the organisation doesn’t agree either with the views Peter Gøtzsche describes in his book where he compares the business model of the drug companies with criminal organisations.” This was free fantasy, which Cochrane rejected: “We have not at any time expressed any opinion about Gøtzsche’s views about drug companies.”

It was scary to see the extent to which some journalists can sometimes distort their stories when they smell blood. This newspaper article gave people the impression that what I had documented so carefully in my book, e.g. that the drug industry engages in organised crime, wasn’t correct.

The second Cochrane letter contained this very important information: “The Cochrane Collaboration currently has nearly 34,000 members in over 100 countries. Every member, including Professor Gøtzsche, is entitled to express their personal opinions and do work that is independent of The Cochrane Collaboration.”

After this experience I felt like the senator in ancient Rome who said that people wouldn’t succeed stabbing him in the back, as he had so many scars already that the knife wouldn’t get through.

Allow me to say at this point that I am a member of several networks of critical psychiatrists and that I get invitations to hold talks for psychiatrists worldwide. This would hardly be the case if there was no substance in what I say. My article on psychiatry’s ten myths³² has been translated into English, Spanish, Norwegian and Finnish and can be found on several websites, including those of David Healy and Robert Whitaker who know a good deal more about the harm done by psychotropic drugs than those who criticise me.

Ten months after the witch hunt, a *BMJ* paper with views very similar to mine appeared.³⁸ The paper addressed strategies in low and middle-income countries but its suggested solutions should be adopted everywhere. It noted that wealthy countries have created expensive and inefficient mental healthcare and that government, industry, and experts make decisions at the top for people at the bottom who are left out of the decision-making process and often out of the care

system entirely. Even with the exorbitant healthcare spending in the United States, the mental health system fails to reach more than half the people with the most serious mental disorders.

The paper also mentioned that we should start by listening to people and empowering them so that they can define their needs and design the systems they want.

We should train lay health workers and generalists rather than specialists.

Lay health workers, backed up by medical generalists (primary care nurses and doctors), currently provide over 90% of mental health-care worldwide. They can learn to manage depression, anxiety, psychosis, and substance misuse, just as they learn to manage malaria, HIV, and tuberculosis. Specialists tend to develop a selective inattention to matters outside their expertise, thereby missing context and creating silos of care, overdiagnosis, and overtreatment. Wealthy countries are now spending billions of dollars trying to convert systems that are based on specialists back into integrated models of care so that they can control excessive treatments.

Community based psychosocial interventions should be emphasised, rather than drug treatments.

Peer and family support, meditation, employment, and technology tools are generally effective, have few side effects, and are more durable than psychiatric drugs. Wealthy countries spend huge resources on medications, mainly because of advertising and lobbying rather than because they are effective; a rational mental health system would rely on judicious use of generic drugs.

It's difficult to argue against this paper. An ancient practice of dealing with the mentally ill was to throw them into a pit of snakes. The theory was that if something like that would make a normal person insane, then it must work in reverse as well.³⁹ But hold on; isn't that what we are still doing? Treating the mentally ill with drugs that can make normal people crazy, hoping that the opposite will miraculously occur?³⁹

Lecture tour in Australia

In February 2014, I received an email from a Victorian farmer in Australia whose

only son took his life at age 19 a year earlier when he was on venlafaxine. He wanted to inform people about how dangerous SSRIs are and asked if I would be willing to go on a lecture tour, which he offered to arrange. After having purchased over 20 different books on malpractice in big pharma, he said that my book² “shone the strongest light on the issues.”

I wish to do what I can to reduce the harms caused by psychiatric drugs and felt it would also be a good opportunity to strengthen our network in Australia. The farmer was a superb organiser and in just 11 days, I gave 17 lectures on four different subjects including Cochrane and mammography screening at public venues, hospitals and universities in Australia and was also interviewed for radio, TV and newspapers.

People were very interested and I learned a lot from those I met – psychiatrists, other doctors, patients, relatives of patients, politicians, civil servants in the Ministry of Health, and filmmakers. I found the power structure in Australian psychiatry very disturbing, as there are many examples of how it had prevented an open debate about issues of crucial public health importance. Two psychiatric professors stand out: Ian Hickie and Patrick McGorry, once the Australian of the Year, and both with huge influence on national policies.

In 2011 psychiatric epidemiologist Melissa Raven and four academic colleagues including psychiatrist Jon Jureidini and two ethicists lodged a complaint to the University of Sydney about a clinical trial called The Beyond Ageing Project led by Ian Hickie. They had serious concerns about the ethics and the methodology of the trial, which investigates whether sertraline can prevent depression in older people who are not depressed. The University sought the advice of two expert reviewers and claimed that they had now addressed the problems. The University refused on several occasions to share the reviewers’ report and other relevant documents with the lame excuse that there was an overriding public interest against disclosure.

In contrast to this Australian closed-mindedness, my research group has been granted access to current trial protocols in Denmark via our research ethics system. Raven appealed this decision to an outside body, under the New South Wales freedom of information legislation, and it found that the University had not established an overriding public interest against disclosure. Of course it hadn’t, as it cannot be done! The public *always* has an overriding interest in knowing what goes on in clinical trials and why. However, the University still refused to hand over the documents, and the matter still hasn’t been resolved but is now dealt with by the judicial system. It’s unbelievable. In my opinion, if Hickie or others have anything against disclosing what they do and why, they shouldn’t be allowed to conduct or approve trials.

There surely was something of interest to reveal. The Human Research Ethics Committee at the University of Sydney, to which Raven and her colleagues first complained, found major problems with the trial. A 50 mg dose of sertraline was abruptly stopped and this was justified as common practice! Furthermore, the Committee noted that a telephone call two weeks after cessation of therapy was rather late to pick up withdrawal effects and requested a telephone call three to five days after withdrawal. The Committee also found that the Participant Information Statement should be modified to clearly state the known risks of the study and that current participants should be re-consented, after being informed of these risks.

Given what we know about antidepressant drugs, I find the idea of trying to prevent depression in older people totally absurd. Patrick McGorry has spearheaded equally absurd trials about using antipsychotic drugs in order to prevent people who have never had a psychosis from developing a psychosis. There is no good reason whatsoever to believe that these drugs can prevent psychosis; in fact, they *cause* psychosis in the long run and when people try to get off them. But McGorry has published at least one such trial,⁴⁰ while another trial, of quetiapine in children as young as 15 “at risk” of psychosis, was halted after international protests.⁴¹ Some of McGorry’s peers said that his youth early intervention model had been “massively oversold” and Allen Frances was particularly harsh in his criticism.⁴² Frances attacked the Australian Government’s plan to spend \$222 million expanding McGorry’s programme by funding another 16 Early Psychosis Prevention and Intervention Centres and called it a “vast untried public-health experiment.” He also noted that the false positive rate in selecting pre-psychosis as a precursor for psychosis is at least 60-70% in the very best hands and may be as high as 90% in usual practice. It’s like performing bilateral mastectomy on all women to prevent breast cancer, as also in this case about 90% would never have developed the disease.

So, these are the two silverbacks the Australians have to guide them in psychiatry, both with numerous conflicts of interest in relation to the drug industry. Other views than theirs are not welcome. Maryanne Demasi from the Australian Broadcasting Corporation (ABC) worked on a documentary about antidepressants and interviewed among others David Healy and me. We used a lot of time refuting Ian Hickie’s arguments and explaining to Demasi why he was wrong. Ten months later, the ABC’s leadership cancelled the documentary. Hickie had teamed up with McGorry and they refused to appear on camera, but that’s not a valid excuse for dropping a highly relevant programme; journalists routinely say that someone has refused to comment. Demasi had worked hard to get the scientific facts right,

which meant that I saw a good deal of Hickie's emails. Hickie's denial of the facts was otherworldly:

Hickie denied that antidepressants increase the suicide risk in children and adults and recommended Demasi read Gibbons' work, noting that suicide rates increase when antidepressant use decreases (Hickie sent 10 of Gibbons' papers to Demasi); he asked what evidence there was that actual suicide is a side effect; he claimed that FDA's black box warning wasn't justified and might have caused harm; he noted that antidepressants are not over-prescribed; he said that suicidal thoughts are not the same as completed suicides; he evaded the question about the chemical imbalance but said that antidepressants do not cause a chemical imbalance; he rejected the idea that general practitioners don't have time for full mental health histories and follow ups; he claimed that a very extensive literature showed that antidepressants can prevent relapse; and he opined that there was no wide debate about psychiatry, as the critique comes from fringe groups.

What a mouthful. Australians are not supposed to know the truth about antidepressants and they are not given the opportunity to hear views other than those cherished by the silverbacks. But this censorship has cracks. Some of my talks were filmed and the one from Melbourne is publicly available (see my website for links, www.deadlymedicines.dk).

By refusing to appear in the TV programme, Hickie also got off the hook in another matter. He knew that Demasi would ask him about his conflicts of interest in relation to a highly dubious, unsystematic review he published in the *Lancet* as first author.⁴³ It was about melatonin-based drugs for depression, but "In particular, we highlight agomelatine," which got four pages, whereas four other drugs only got one page in total. Both authors had numerous ties to Servier, which sells agomelatine, and there were possibly honoraria that weren't declared in the paper. The second author, Naomi Rogers, had received an "unrestricted educational grant" from Servier, which in my view is a euphemism for corruption, as the industry doesn't just give its money away.² Hickie and Rogers claimed that fewer patients on agomelatine relapsed (24%) than do those on placebo (50%), but a systematic review by other psychiatrists found no effect on relapse prevention, no effect as evaluated on the Hamilton depression scale, and that none of the negative trials had been published.⁴⁴ Three pages of letters – which is extraordinarily much – to the editor in *Lancet* (21 January 2012) pointed out the many flaws in Hickie's review.

Psychiatry is not evidence-based medicine

Instead of a science of madness, we documented a mad science.

KLRK, GOMORY AND COHEN IN *MAD SCIENCE*⁴⁵

We all want to practice evidence-based medicine, which rests on three pillars: reliable research, clinical expertise and the patient's values and preferences. Psychiatry is *not* evidence-based medicine, as none of these apply:

1) The research is unreliable. The diagnoses are unscientific and arbitrary; the placebo controlled drug trials are unreliable, as they have not been effectively blinded; the placebo groups have been harmed by introduction of withdrawal effects; drug harms are being misinterpreted as disease symptoms, particularly when drugs are being withdrawn; harms are being vastly underreported; and almost all trials are controlled by the drug industry.

2) The clinical expertise is totally unreliable. Doctors almost always credit the drugs for any spontaneous improvement and puts the blame for any untoward symptoms on the disease, or they think they are caused by a new disease. This leads to additional psychiatric diagnoses and is a major reason why so many patients receive several types of psychotropic drugs.

3) Patient values and preferences are almost totally ignored. Patients and their relatives are rarely listened to, particularly when they complain about drug harms, and patients are subjected to forced treatment with drugs even when they know it will harm them.

Not even when the evidence is crystal clear is it being respected. In Denmark, SSRIs are recommended for children who are at risk of suicide⁴⁶ and for old people with depression,⁴⁷ although SSRIs increase mortality in these age groups (see Chapter 3). In just 14 years, from 1996 to 2010, there was a nine-fold increase in total dispensed psychotropic medication for children and adolescents in Denmark.⁴⁸ The two authors downplayed this horrible development by stating that it was only a two-fold increase after adjusting for increasing patient numbers. So, have the number of children in Denmark increased 4.5-fold? No. What they mean is that 4.5 times more children are sent to the mental health services, which in itself is wrong. It is therefore meaningless to adjust for this, but it could be related to the fact that one of the authors had ties to companies selling psychotropic drugs.

Considering the unblinding bias, it is not surprising that psychiatric drugs seem to work for virtually everything, e.g. antidepressants are used for all sorts of things including urinary incontinence, pain, premenstrual symptoms, premature ejaculation, hot flashes in menopause, and they even seem to work for shopaholics.²

Some effects are of course real, e.g. the sexual harms of antidepressants may explain why some men find them useful for premature ejaculation, but most measured benefits are non-existing. Consider, for example, a trial that showed that escitalopram reduced the number of hot flashes in menopausal women.⁴⁹ The number of hot flashes at baseline was 10 and after eight weeks there was a difference of 1 between drug and placebo. This tiny effect is likely just bias, as most women have guessed which drug they receive. And even if it were true, who cares? It cannot possibly be clinically relevant, particularly not considering all the harms that SSRIs inflict on women.

In 2014, the FDA approved paroxetine for hot flashes based also on one less flash a day.⁵⁰ The approval ran counter to the recommendation of the FDA's advisory committee, which concluded that the overall benefit-risk profile of paroxetine was unfavourable. The FDA opined that doses don't have to be tapered before use is discontinued, but the dose was 7.5 mg, close to the starting dose for depression, which is 10 mg. Due to its short half-life, paroxetine is one of the worst SSRIs for giving people abstinence symptoms! The FDA also allowed a new name, Brisdelle, which means that most patients won't know that Brisdelle is an antidepressant drug. The drug maker, Noven, doesn't exactly reveal this on its US home page:⁵¹

"If you're one of the millions of women dealing with hot flashes, you're not alone. They can be disruptive, uncomfortable, and embarrassing. And for some women, they can last for years. So why suffer more than you have to? You can do something different because there's a non-hormonal, prescription treatment option for moderate to severe hot flashes due to menopause called Brisdelle® (paroxetine)." *Nowhere does Noven tell the women that it is the same drug as Prozac and nowhere is it written explicitly that Brisdelle is an antidepressant drug.* I consider this consumer fraud. The homepage says: "Have a conversation with your doctor today." By all means, but tell your doctor not to use Brisdelle or any other antidepressant for hot flashes.

One of the new fads in psychiatry is the idea that some diseases are caused by inflammation, and a systematic review of 14 trials of celecoxib, a so-called nonsteroidal anti-inflammatory drug, showed an effect on depression, effect size 0.34.⁵² An effect of this magnitude can easily be caused by unblinding bias, and by the way, nonsteroidal anti-inflammatory drugs are not anti-inflammatory at all; it's just a misleading name.² And if the patients have pain some-where, which many depressed patients have, painkillers might seem to reduce the "depression." Many of the patients had arthritis, and in another study, of depressed patients treated with duloxetine, the patients also had substantial levels of pain.⁵³ Finally, we

should not treat depression with nonsteroidal anti-inflammatory drugs, some of the most deadly drugs we have.²

Psychiatrists are usually paid per patient enrolled in clinical trials and may not bother to go through all the items on the rating scales, such as Hamilton's depression scale, with the patient but may use their overall impression to score some of the items without having asked, or to score later based on memory.⁵⁴ Patients cheat, too. Some people participate in trials without being ill just to pocket the money, as a healthy person told a doctor on a train ride:⁵⁵

"I'm not depressed ... the trials are advertised, the best pay about £100 a day to volunteers. For a 20 day trial that's £2000 ... it's nice to see your regular friends."

Can we reform psychiatry or is a revolution needed?

When will psychiatrists finally accept that we are dealing with sensitive, delicately poised human beings, not machines to be tinkered with; that the very definition of life is one of self-organisation and self-management? The only real lasting change comes when we help a person to bring about the painful work of change within themselves.

IVOR BROWNE, IRISH RETIRED PSYCHIATRIST⁵⁶

There is a vast disconnect between what psychiatrists think about their treatments and what patients think about them. In a large survey of 2,031 people from 1995, people thought that antidepressants, antipsychotics, electroshock and admission to a psychiatric ward were more often harmful than beneficial.^{57, 58} This view concurs with the best evidence we have, but the social psychiatrists who had done the survey were dissatisfied with the answers, and argued that people should be trained to arrive at the "right opinion." In what way? More brainwashing?

In another survey from 1991, 91% of 2,003 lay people thought that people with depression should be offered counselling; only 16% thought they should be offered antidepressants; only 46% said antidepressants were effective; and 78% regarded antidepressants as addictive.⁵⁹ The psychiatrists' view on these responses was that, "Doctors have an important role in educating the public about depression and the rationale for antidepressant treatment. In particular, patients should know that dependence is not a problem with antidepressants." I fully understand why the survey also found that "the word psychiatrist carried

connotations of stigma and even fear.”

It's not the patients that need training, it's the psychiatrists and other doctors, but psychiatry is so much out of touch with reality that I am afraid that no amount of training will get us even close to where we want to be.

The self-delusion, denial and hype in psychiatry run really high. Just before fluoxetine reached the market in 1988, NIMH had surveyed the public about its views on depression, and only 12% wanted to take a pill to treat it.⁶⁰ However, the leaders of NIMH were determined to change this attitude and launched a totally misleading, so-called public awareness campaign: Depression is a serious disease that can be fatal if untreated; depression is underdiagnosed and undertreated; and 70-80% get better on drug and only 20-40% on placebo. This postulated 45% difference in effect is fraudulent; even the industry-friendly FDA found only 10%, and the true difference is close to zero (see Chapter 3). The campaign was immensely successful, and the media praised Prozac as the new wonder drug.

The hype was extreme even by American standards. A popular trick is to underline the seriousness of depression by saying that the suicide risk in major depression is 15%.⁶¹ Every major textbook quotes this high suicide risk, which comes from a 1970 study, but the true lifetime risk is only 2-3%.⁶¹

Many patients are called treatment-resistant. It isn't that the medication didn't work, the patient was treatment-resistant. This is very convenient for psychiatrists, as it puts the blame on the victim and not on the drug or on them, although they failed by endlessly trying new drug combinations and higher and higher doses, instead of taking patients off the drugs and talking to them, which might have led to recovery. The term treatment-resistant should be banned, as it is a cover-up for the system's own failures. If psychiatrists insist on using this term, they should accept that we might call them fact-resistant. Psychological research has shown that the more facts people are exposed to showing that their beliefs are wrong, the more steadfast they often become in their erroneous beliefs. This is where psychiatry is today. Thus, the fact-resistant psychiatrists are also treatment-resistant.

Psychiatry doesn't deliver what patients want and what they believe is most helpful to them. A meta-analysis of 34 studies showed that the patients, of which almost 90% had depression or anxiety, preferred psychological treatment three times as often as drugs.⁶² However, few patients get psychotherapy; almost all of them get drugs. David Healy is right when he says that psychiatric drugs are poisons that have been rebranded as fertilizers to be used as widely and early as possible.⁶³ The chemical pollution from psychotropic drugs is vastly more dangerous for our health than the chemical pollution of our food and environment.

General practitioners cannot handle psychiatric diseases either.⁶⁴ In relation to depression, for example, the chairman for the Danish Association for General Practitioners said in 2014 that they didn't have "oceans of time" and couldn't set aside a whole hour for one patient, as they also needed to think of their economy. But that is exactly the point. What is needed is plenty of time, in order to avoid drug treatment and to heal patients, not only for people with depression but for virtually everyone with a psychiatric disease or who is suffering from difficult life circumstances.

What should be done about this? Is there any hope that doctors can learn to handle psychiatric drugs in a way that creates more good than harm? Given the evidence we have, we will have to conclude that this cannot be done. Furthermore, all the initiatives I know about where pioneering psychiatrists or psychologists have tried to use drugs as little as possible – and rather consistently have gotten better results than mainstream psychiatrists – have been strangled. What people do in healthcare has little to do with what is right or wrong, but a lot to do with who holds power. There are strong guild interests to protect, and politicians and administrators loathe going against powerful specialist groups, as it gives them so much trouble. It is far more convenient to support psychiatric leaders when they say that initiatives for using drugs far less are unethical experiments on people with serious diseases and in great need of drugs. Specialists always say this when their guild interests come under threat.

In order to restructure psychiatry even a little bit, we need to work primarily with lawyers, journalists, patients and their relatives, and young psychiatrists in training who have not yet been spoiled by the silverbacks. We need nothing less than a revolution in psychiatry. Like in all revolutions, those in power must go and we must carefully construct a completely new curriculum for future psychiatrists.

References

- 1 Praktiserende Lægers Organisation. [Reporting the member survey about visits from the drug industry in general practice]. 2011 June 22.
- 2 Gøtzsche PC. Deadly medicines and organised crime: How big pharma has corrupted health care. London: Radcliffe Publishing; 2013.
- 3 Insel TR. Psychiatrists' relationships with pharmaceutical companies: part of the problem or part of the solution? *JAMA* 2010;303:1192–3.
- 4 Caplan P. Diagnosisgate: conflict of interest at the top of the psychiatric apparatus. *Aporia* 2015;7:30-41.
- 5 Carey B, Harris G. Psychiatric group faces scrutiny over drug industry ties. *New York Times* 2008 July 12.
- 6 Brownlee S. Overtreated: why too much medicine is making us sicker and poorer. New York:

- Bloomsbury; 2007.
- 7 Mello MM, Clarridge BR, Studdert DM. Academic medical centers standards for clinical-trial agreements with industry. *N Engl J Med* 2005;352:2202–10.
 - 8 Meier B. Contracts keep drug research out of reach. *New York Times* 2004 Nov 29.
 - 9 McHenry L. Biomedical research and corporate interests: a question of academic freedom. *Mens Sana Monographs* 2008 Jan 1.
 - 10 Chan A-W, Hróbjartsson A, Haahr MT, et al. Empirical evidence for selective reporting of outcomes in randomized trials: comparison of protocols to published articles. *JAMA* 2004;291:2457–65.
 - 11 Chan A-W, Hróbjartsson A, Jørgensen KJ, et al. Discrepancies in sample size calculations and data analyses reported in randomised trials: comparison of publications with protocols. *BMJ* 2008;337:a2299.
 - 12 Barbui C, Cipriani A, Brambilla P, et al. "Wish bias" in antidepressant drug trials? *J Clin Psychopharmacol* 2004;24:126-30.
 - 13 Hughes S, Cohen D, Jaggi R. Differences in reporting serious adverse events in industry sponsored clinical trial registries and journal articles on antidepressant and antipsychotic drugs: a cross-sectional study. *BMJ Open* 2014;4:e005535.
 - 14 Rennie D. When evidence isn't: trials, drug companies and the FDA. *J Law Policy* 2007 July:991–1012.
 - 15 Gøtzsche PC, Jørgensen AW. Opening up data at the European Medicines Agency. *BMJ* 2011;342:d2686.
 - 16 Gottstein J. Mental health recovery stories. <http://akmhweb.org/recovery/jgrec.htm>. 1999;Aug 27 (accessed 21 Nov 2014).
 - 17 European Commission. Antitrust: Commission fines Lundbeck and other pharma companies for delaying market entry of generic medicines. Press release. 2013 June 19.
 - 18 Gorman JM, Korotzer A, Su G. Efficacy comparison of escitalopram and citalopram in the treatment of major depressive disorder: pooled analysis of placebo-controlled trials. *CNS Spectr* 2002;7(4 Suppl. 1):40–4.
 - 19 Alkhafaji AA, Trinquart L, Baron G, et al. Impact of evergreening on patients and health insurance: a meta analysis and reimbursement cost analysis of citalopram/escitalopram antidepressants. *BMC Med* 2012;10:142.
 - 20 US Department of Justice. Drug Maker Forest Pleads Guilty; to pay more than \$313 million to resolve criminal charges and False Claims Act allegations. 2010 Sept 15.
 - 21 US Senate, Committee on Finance. Letter about Lexapro documents. 2009 Aug 12. Available online at: www.nytimes.com/packages/pdf/politics/20090831ME-DICARE/20090831_MEDICARE.pdf (accessed 2011).
 - 22 Nielsen M, Gøtzsche P. An analysis of psychotropic drug sales. Increasing sales of selective serotonin reuptake inhibitors are closely related to number of products. *Int J Risk Saf Med* 2011;23:125–32.
 - 23 Pirraglia PA, Stafford RS, Singer DE. Trends in prescribing of selective serotonin reuptake inhibitors and other newer antidepressant agents in adult primary care. *Prim Care Companion J Clin Psychiatry* 2003;5:153–7.
 - 24 Hytloft V. [Lundbeck partner in settlement about suicides]. *Berlingske* 2011 Feb 8.
 - 25 Wichmann S. [Lundbeck knew about violation of the rules]. *Berlingske* 2014 Nov 25.
 - 26 Hansen JC. [Pressured out – and rewarded]. *Berlingske* 2014 Nov 25.
 - 27 Depression. American Psychiatric Association. <http://www.psychiatry.org/depression> (accessed 14 Apr 2014).

- 28 Ioannidis JP. Effectiveness of antidepressants: an evidence myth constructed from a thousand randomized trials? *Philos Ethics Humanit Med* 2008;3:14.
- 29 Davis JM, Giakas WJ, Qu J, et al. Should we treat depression with drugs or psychological interventions? A reply to Ioannidis. *Philos Ethics Humanit Med* 2011;6:8.
- 30 Glenthøj B, Baandrup L, Ebdrup B, et al. [Behind the myths about antipsychotics]. *Politiken* 2012 Oct 19.
- 31 Gøtzsche PC. [Psychiatry gone astray]. *Politiken* 2014 Jan 6.
- 32 Gøtzsche PC. Psychiatry gone astray. 2014 Jan 28. <http://www.madinamerica.com/2014/01/psychiatry-gone-astray/> (accessed 30 Jan 2014).
- 33 Licht R, Nordentoft M, Bech P, et al. [Ten ways to better psychiatric treatment]. *Altinget* 2014 Feb 6.
- 34 Gøtzsche PC. [Leading psychiatrists have still gone astray]. *Politiken* 2014 Jan 21.
- 35 Maund E, Tendal B, Hróbjartsson A, et al. Coding of adverse events of suicidality in clinical study reports of duloxetine for the treatment of major depressive disorder: descriptive study. *BMJ* 2014;348:g3555.
- 36 Maund E, Tendal B, Hróbjartsson A, et al. Benefits and harms in clinical trials of duloxetine for treatment of major depressive disorder: comparison of clinical study reports, trial registries, and publications. *BMJ* 2014;348:g3510.
- 37 Toft ONM. [Gøtzsche's organisation denounces his statements about psychiatry]. *Altinget* 2014 March 25.
- 38 Drake RE, Binagwaho A, Martell HC, et al. Mental healthcare in low and middle income countries. *BMJ* 2014;349:g7086.
- 39 Epstein E. Independence from the therapeutic state. 2014 July 4. <http://www.madinamerica.com/2014/07/thinking-beyond-therapeutic-state/> (accessed 5 July 2014).
- 40 McGorry PD, Yung AR, Phillips LJ, et al. Randomized controlled trial of interventions designed to reduce the risk of progression to first-episode psychosis in a clinical sample with subthreshold symptoms. *Arch Gen Psychiatry* 2002;59:921-8.
- 41 Stark J. McGorry aborts teen drug trial. *Sydney Morning Herald*. 2011 Aug 21.
- 42 Dunley S. US expert slams Patrick McGorry's psychosis model. *The Australian* 2011 June 14.
- 43 Hickie IB, Rogers NL. Novel melatonin-based therapies: potential advances in the treatment of major depression. *Lancet* 2011;378:621-31.
- 44 Koesters M, Guaiana G, Cipriani A, et al. Agomelatine efficacy and acceptability revisited: systematic review and meta-analysis of published and unpublished randomised trials. *Br J Psychiatry* 2013;203:179-87.
- 45 Kirk SA, Gomory T, Cohen D. *Mad science: psychiatric coercion, diagnosis and drugs*. New Brunswick: Transaction Publishers; 2013.
- 46 Kessing L. [Depression in children and adolescents]. [Sundhed.dk](http://www.sundhed.dk) 2011 Mar 1.
- 47 Rønholt F. [Depression in the elderly]. [Sundhed.dk](http://www.sundhed.dk). 2011 Oct 25.
- 48 Steinhausen HC, Bisgaard C. Nationwide time trends in dispensed prescriptions of psychotropic medication for children and adolescents in Denmark. *Acta Psychiatr Scand* 2014;129:221-31.
- 49 Freeman EW, Guthrie KA, Caan B, et al. Efficacy of escitalopram for hot flashes in healthy menopausal women: a randomized controlled trial. *JAMA* 2011;305:267-74.
- 50 Orleans RJ, Li L, Kim MJ, et al. FDA approval of paroxetine for menopausal hot flashes. *N Engl J Med* 2014;370:1777-9.
- 51 Brisdelle (paroxetine). <http://www.brisdelle.com/about.php> (accessed 9 Jan 2015).

- 52 Köhler O, Benros ME, Nordentoft M, et al. Effect of anti-inflammatory treatment on depression, depressive symptoms, and adverse effects: a systematic review and meta-analysis of randomized clinical trials. *JAMA Psychiatry* 2014;71:1381-91.
- 53 Perahia DG, Quail D, Desai D, et al. Switching to duloxetine in selective serotonin reuptake inhibitor non- and partial-responders: effects on painful physical symptoms of depression. *J Psychiatr Res* 2009;43:512-8.
- 54 Healy D. *Let them eat Prozac*. New York: New York University Press; 2004.
- 55 Boyd R. A view from the man in the seat opposite. *BMJ* 1998;317:410.
- 56 Browne I. *The writings of Ivor Browne*. Cork: Atrium; 2013.
- 57 Raven M. *Depression and antidepressants in Australia and beyond: a critical public health analysis* (PhD thesis). University of Wollongong, Australia; 2012. <http://ro.uow.edu.au/theses/3686/>.
- 58 Jorm AF, Korten AE, Jacomb PA, et al. "Mental health literacy": a survey of the public's ability to recognise mental disorders and their beliefs about the effectiveness of treatment. *Med J Aus* 1997;166:182-6.
- 59 Priest RG, Vize C, Roberts A, et al. Lay people's attitudes to treatment of depression: results of opinion poll for Defeat Depression Campaign just before its launch. *BMJ* 1996;313:858-9.
- 60 Whitaker R. *Anatomy of an Epidemic*. New York: Broadway Paperbacks; 2010.
- 61 Raven M. Myth of 15% suicide rate was promulgated again. *BMJ* 2006;332:1154.
- 62 McHugh RK, Whitton SW, Peckham AD, et al. Patient preference for psychological vs pharmacologic treatment of psychiatric disorders: a meta-analytic review. *J Clin Psychiatry* 2013;74:595-602.
- 63 Wipond R. Where critical psychiatry meets community resilience. <http://www.madinamerica.com/2014/11/critical-psychiatry-meets-community-resilience/> (accessed 22 Nov 2014).
- 64 Brogaard M. [Depressed patients experiment with the medication]. 2014 Dec 28. www.videnskab.dk (accessed 28 Dec 2014).

Deadly psychiatry and dead ends

I've spent most of my professional life to evaluate the quality of clinical research, and I think it is particularly poor in psychiatry. Industry-sponsored studies ... are selectively published, are often transitory, are designed to favor the drug and demonstrate such small benefits that they probably do not outweigh the long-term damage.

MARCIA ANGELL, FORMER EDITOR OF
*NEW ENGLAND JOURNAL OF MEDICINE*¹

Psychiatry's almost manic obsession with ineffective, addictive drugs has led to a disaster in public health so big that nothing I have seen in other areas of medicine comes close.

Robert Whitaker is convinced that most psychiatric patients would be better off not receiving drugs at all.^{2,3} Whitaker once invited me to give a lecture at the Safra Center for Ethics at Harvard University in Boston, to which he belongs, and I have lectured with him on several occasions in different countries. Every time there have been psychiatrists in the audience who shared our views that the way we currently use psychiatric drugs causes far more harm than good.

On one such occasion, I gave an invited talk in Los Angeles at the annual conference of the International Society for Ethical Psychology and Psychiatry, which has been described as North America's leading organisation of critical thinkers in the mental health field.⁴ The title of the meeting was punchy, "Transforming Mad Science and Reimagining Mental Health Care," and the press release announced that the plenary speakers "shared the controversial belief that a 'medical model of care' – the idea that distress and misbehavior have physical causes that are best treated with physical means like medications – is causing more harm than good to individuals and to society." These speakers included leading psychiatrists like Allen Frances and David Healy, psychologists, psychotherapists, social workers, neuroscientists, and a previous patient. Peter Breggin, who was not at the meeting, has also concluded that psychiatric drugs do more harm than good.⁵

It was a fascinating meeting that made it clear that we need a revolution in psychiatry. Psychiatric survivor Laura Delano described how small groups of people gather to support each other in coming off psychiatric medications, deindoctrinating themselves from the biological model of mental illness and supporting each other through psychological crises and social change. When she read Whitaker's book, *Anatomy of an Epidemic*,² which won the 2010 Investigative Reporters and Editors book award for best investigative journalism, it suddenly dawned on her that she should reclaim her humanity and free herself from the prison of psychiatric "care." She had become dehumanised by psychiatry, she was called treatment-resistant, was on five drugs, and her drug-induced weight increase was even given a psychiatric diagnosis: binge eating! Whitaker's book saved her and helped her live with her pain more peacefully, until she had built up enough faith in herself to heal, so that she no longer felt the need to remind herself over and over again that she didn't need to believe everything her mind was telling her, as it was still under the influence of drugs.

Laura has connected with many practitioners who are slowly coming to understand the inefficacy and harm of the current "treatment" standard, but who feel powerless and afraid to do anything differently, fearing they could lose their licenses, face a lawsuit, get fired, or not get promoted. We must find ways to change this so that it becomes acceptable *not* to medicate people, which mainstream psychiatry considers "irresponsible," "dangerous," or even "life-threatening." We need to create a heightened consciousness around just how oppressed and harmed the patients have been by the "quick fix" mentality we have as a society, and to realise how false the "quick fix" story is in the first place, so that the demand for "psychiatric care" will lessen.

The organiser of the meeting, psychologist David Cohen, wasn't surprised to hear that people coming from different backgrounds independently had arrived at similar perspectives on the problems we're facing in psychiatry and how to go about solving them. He also reminded the audience that, over the last few years, mental health authorities have acknowledged an absence of findings from biological or genetic research that have translated into a difference in patient care. They have recognized that 50 years of increasingly sophisticated treatments have not reduced the burden of mental disorders; in fact they have increased it substantially.² At the same time, powerful conflicts of interest have been exposed that keep practitioners and patients uninformed about the true effects of drug treatments.

Usually, people who are extreme are few in number, but in this case it is the vast majority of psychiatrists that are extreme. It is truly extreme that psychiatrists

have built their specialty on a number of myths, lies and highly flawed research, which have harmed our nations to the extent we have seen. Marcia Angell has noted that psychiatrists should consider that other medical specialists, unlike psychiatrists, would be very reluctant to offer long-term symptomatic treatment without knowing what lies behind the symptoms, e.g. if a patient suffers from nausea or headache.¹ In my own specialty, internal medicine, we are on much safer ground when we intervene. Furthermore, apart from chemotherapy for cancer, it is difficult to identify a class of drugs in general use as toxic as antipsychotics.

In 2014, a senior psychiatrist at Rigshospitalet, the national university hospital in Denmark, which is where I work, underlined involuntarily just how necessary the revolution is. He was interviewed by a newspaper and said that SSRIs protect against suicide, with reference to observational studies. He also said we didn't overuse SSRIs, as the consumption reflects the number of ill patients. This is a sick system, which we must fight with all the means at our disposal.

Psychiatrists are slowly waking up to the tragedy they have created, and mainstream psychiatric journals, such as the *British Journal of Psychiatry*, now publish papers that are highly critical of the current model of biological psychiatry. For example, one paper stated that the research into biological mechanisms of mental and behavioural responses has failed to deliver anything of value to clinical psychiatrists and is very unlikely to do so in the future,⁶ and another predicted that the current biology-based model will be ruinous to the profession due to its consistent failure to deliver.⁷ It is noteworthy that these pessimistic statements come after more than 60 years of research in biological psychiatry.

It seems that many billions of research money have been wasted on false leads. Even Thomas Insel, the director of NIMH, is critical. He has pointed out that there is no evidence for reduced morbidity or mortality from any mental illness from new drugs developed over the last 20 years, in striking contrast to the steadily decreasing mortality rates for cardiovascular disease, stroke and cancer, and that there is little evidence that the prospects for recovery have changed substantially in the past century.⁸ That's a strong statement, but it's actually an understatement, as there is solid evidence that the prospects for recovery have *worsened* substantially because of the drugs we use. But what the public has heard about are reforms, revolutions, progress, innovations and paradigm shifts.⁸ Empty barrels make the loudest noise.

The connection between psychotropic drugs and homicide

Whether they are legal or illegal, it's unhealthy to perturb normal brain functions with drugs, and psychotropic drugs can lead to violence, including homicide.⁹⁻¹³ An analysis of adverse drug events submitted to the FDA between 2004 and 2009 identified 1,937 cases of violence, 387 of which were homicide.¹¹ The violence was particularly often reported for psychotropic drugs (antidepressants, sedatives/hypnotics, ADHD drugs and varenicline, a smoking cessation drug that also affects brain functions).

We know that antidepressants and ADHD drugs can lead to homicide,¹¹ but if we read the newest scientific literature, we are led to believe that it isn't clear whether antipsychotics increase or reduce violence. However, the observational studies in this area are just as problematic as the observational studies that claim that the use of antidepressants reduce the risk of suicide (see Chapter 3). We therefore shouldn't pay much attention to them, but I shall comment on a 2014 study from Sweden published in the *Lancet* that linked a crime register with a prescription register.¹⁴ The authors acknowledged that the evidence that drugs can reduce the risk of violence is weak. But they also said that in their own study, violent crime fell by 45% in patients receiving antipsychotics compared with periods when participants were not on medication.

Such studies are highly misleading. Patients might stop taking the drug because it gives them bad feelings that predispose to crime. Withdrawal effects also predispose to crime, and patients with severe psychopathology might have committed a crime and avoided taking drugs.

I debated with Norwegian psychiatrists in 2015 in a newspaper, and one of them wrote that it is the untreated patients that are dangerous. However, the study he referred to cannot be used to substantiate this claim.¹⁵ It showed that the risk for murder is greatest in first episode psychosis and decreases when patients are treated. But we don't know whether this risk would have been reduced equally, or perhaps even more, if the patients had *not* been treated with antipsychotics.

Curiously, our most prestigious journals have published some of the most misleading studies or commentaries I have ever found. An NIMH study reported that patients with serious mental illness – schizophrenia, major depression, or bipolar disorder – were two to three times more likely to be assaultive as people without such an illness. A professor of psychiatry who commented on the study in the *New England Journal of Medicine* mentioned that although it didn't specifically monitor the treatments, “it seems possible that treating psychiatric

illness does not just make patients feel better; it may also drastically reduce the risk of violent behavior.”¹⁶ This wishful thinking is contrafactual. Antipsychotics and antidepressants can cause violence and it will usually be the drugs, not the disease, that on rare occasions can make psychiatric patients commit horrendous acts. Studies that do not separate medicated from unmedicated patients are not worth the paper they are written on, and these patients were medicated!

In contrast to such flawed studies, it is pretty revealing to look at studies conducted before the advent of antipsychotics.³ *Before 1955, four studies found that patients discharged from mental hospitals committed crimes at the same or lower rate than the general population, whereas eight studies conducted between 1965 and 1979 found higher rates.*

Akathisia, the well-known causal factor for violent actions and crime, was given little attention in these years, and physicians generally interpreted the restless behaviour as a sign that patients needed a higher dose of the drug, which only increases the risk of crime. When the psychiatrists finally took an interest in their patients, the results were shocking. In one study, 79% of mentally ill patients who had tried to kill themselves suffered from akathisia.³ A 1990 study reported that half of all fights at a psychiatric ward were related to akathisia and another study found that moderate to high doses of haloperidol made half the patients markedly more aggressive, sometimes to the point of wanting to kill their torturers, the psychiatrists.³

Psychotropic drugs can cause people to lose some of their conscience, so that they lose control over their behaviour.¹⁰ Such people are at greatly increased risk of committing acts of crime and violence.

Several high-profile homicides have been committed by patients in a drug-withdrawal state, which also may cause akathisia,^{5, 10} and a clear sign that the psychiatrists generally don't know what they are doing and what they are causing is that they have virtually always interpreted such events as meaning that the patients need to be kept on their drug, rather than acknowledging the peril of using the drug in the first place.³ It is therefore their fault that the media have failed to write about it or investigate it. As David Healy says: “Violence and other potentially criminal behaviour caused by prescription drugs are medicine's best-kept secret. Never before in the fields of medicine and law have there been so many events with so much concealed data and so little focused expertise.” When one of the teenage shooters in the Columbine High School massacre, Eric Harris, was found to have an antidepressant in his blood, the American Psychiatric Association immediately denied a causal relation and added that undiagnosed and untreated mental illness exacts a heavy toll on those who suffer from these

disorders as well as those around them.¹⁷ This sickening marketing speak comes right from the drug industry, which provides generous funds to the association (see Chapter 13). Harris' partner, Dylan Klebold, had taken sertraline and paroxetine.

Adam Lanza killed 20 school children, six members of staff, his mother and himself in Newtown, Connecticut, in 2012. After this crime, the International Society for Ethical Psychology and Psychiatry called for an inquiry into the connection between such acts of mass murder and the use of psychotropic drugs.¹⁸ The media had noted that Lanza was taking prescription drugs to treat a neurological-development disorder, but nothing was revealed about the nature of these drugs. The society mentioned a number of other mass killings where psychotropic drugs might have had a causal role and noted that in 14 recent school shootings, the acts were committed by persons taking or withdrawing from psychiatric drugs, resulting in 58 killed and over 100 wounded.¹⁸ In other school shootings, information about the shooters' prescription drug use and other medical history was kept from public records.

It is difficult to know when psychotropic drugs are the major factor in these crimes, as the people who take them may suffer from severe personality disorders. But there is no doubt that these drugs can cause homicide, and the mass murders should therefore be routinely investigated for this possibility. There is enough evidence, for example, that antidepressants increase the risk of suicide and violence for the US Food and Drug Administration and its Canadian counterpart to require that drug companies include a black box warning to that effect on their packages. Antidepressants appear to more than double the risk of hostility events in adult and paediatric placebo controlled trials,¹⁸ and in our systematic review of studies in human volunteers, we found that antidepressants double the incidence of activating effects¹⁹ (see also Chapter 3).

How few drugs do we need?

We could have a much better psychiatry almost without drugs. Some psychiatrists hardly use any drugs at all. One is Lois Achimovich, Australia, a child psychiatrist for 40 years, who has never used stimulants or antipsychotics. He only uses diazepam, in low doses and only short-term, when a child cannot sleep in an acute situation, e.g. after the death of a parent. Peter Breggin once had a debate with a paediatrician who tried to look very judicious by stating that he only medicated a small number of children each year. He challenged Breggin to say what was wrong with that, and Breggin replied, "Doctor, I would not know which child to poison."

Several psychiatrists I have met have never used antidepressants, as they don't believe they work while they cause much harm. Like Achimovich, the only drugs Peter Breggin uses are benzodiazepines, and only temporarily, if people feel badly during drug withdrawal. Perhaps people like them don't see the worst cases, but they have nevertheless demonstrated that we very rarely need drugs.

One way to go, which David Healy and David Cohen have suggested, could be to make psychotropic drugs freely available over the counter. This is an interesting suggestion, provided that marketing to the public became forbidden of course. If there were no doctors as intermediaries, with all their false beliefs about chemical imbalance, targeted therapy and false reassurances about safety and drugs producing recovery and preventing relapses, many patients would give up taking psychotropic drugs very quickly, as their side effects are so horrible.

We could also take the opposite approach. More than 40 years ago, Archie Cochrane, whom the organisation I work for is named after, wrote:²⁰

“I would ban the prescription of amphetamines and put a large number of other psychotropic drugs on a list which could only be prescribed by psychiatric consultants. I do not suggest this because I think consultants know better than GPs which of these drugs will do more good than harm in the long run. I do not think anyone knows, but they may know more about side effects and, much more importantly, there are fewer consultants than GPs and it will make the prescriptions more difficult to get. Psychiatry, in my view, must be criticized as using a large number of therapies whose effectiveness has not been proven. It is basically inefficient.”

It's remarkable that Cochrane wrote this so long ago, as it's still the case today that psychiatric drugs are pretty inefficient.

Peter Breggin has suggested that we should prohibit giving psychiatric drugs to children, just like we have prohibited physical and sexual abuse.²¹ I agree completely that psychiatric drugging of children is a form of child abuse that should be prohibited, with very rare exceptions. We are not allowed to beat our children but are allowed to destroy their brains with drugs. We medicalise the inevitable conflicts that arise between parents and children, and methylphenidate (Ritalin) has become the modern version of the cane. This is a flagrant abuse of a faulty disease model and a serious violation of the children's human rights, which must be stopped.

The drugged child's brain cannot develop in its intended manner but develops in response to a toxic internal environment. Furthermore, the stigmatisation and loss of self-esteem, which often follows psychiatric diagnosis and treatment (see Chapter 6), is especially ominous in children who have yet to shape their

personalities, and it can hamper future opportunities even without considering the potential brain damage caused by the drugs. Children may learn to view themselves as physically or genetically disabled, with impaired self-determination and increased feelings of helplessness.²¹ It's horrible.

Also for adults, psychiatric drugs are a dangerous weapon that doctors cannot handle and most of them do far more harm than good. We could therefore take them off the market and spare a few for acute situations and for legitimate purposes outside psychiatry, e.g. for induction of anaesthesia and for treatment of epilepsy. This would mean tremendous progress for mental health, as far fewer people would be in treatment and far fewer would be harmed.

I shall try to estimate how little we need psychiatric drugs. I will leave out epilepsy drugs, as I don't know how much of the usage is for psychiatric purposes (at any rate, I believe these drugs shouldn't be used for psychiatric diseases). This leaves us with five drug groups: antidepressants, ADHD drugs, antipsychotics, anti-dementia drugs, and benzodiazepines and similar sedatives.

As antidepressants likely don't work, whereas they actually cause much harm, including deaths, personality changes, sexual disturbances and addiction, we shouldn't use them at all.

We shouldn't use ADHD drugs either. They might give some short-term relief but are clearly harmful when used long-term, which they almost always are.

Antipsychotics kill many people and destroy many more people's lives, and it's likely we could use benzodiazepines for the same indications. Whitaker has estimated that we could halve the two million adults disabled by schizophrenia in the United States if we used antipsychotics in a selective, cautious manner.² I have no doubt he is right. But it can be discussed whether we need this class of drugs at all.

Anti-dementia drugs shouldn't be used, as they don't work and are pretty harmful.

Benzodiazepines and similar drugs are also very harmful but we need drugs for sedation in acute situations and they are less harmful than antipsychotics.

I shall use Danish statistics (<http://medstat.dk/>) to illustrate how little we need psychiatric drugs. Currently, we use so many of these drugs that one out of seven Danes could be in treatment with a psychiatric drug every day for their entire life, from cradle to grave, if they took one drug each (Table 14.1).

Table 14.1. Usage of psychotropic drugs in Denmark in 2013. Defined daily doses per 1000 inhabitants per day; sales in million DKK.

	Usage current	Usage needed	Sales current	Sales needed
N05A Antipsychotics	14.3	1.2	591	34
N05B Anxiolytics	9.6	0.5	83	4
N05C Hypnotics and sedatives	19.9	1.0	88	4
N06A Antidepressants	80.0	0	367	0
N06B Psychostimulants	8.1	0	335	0
N06D Anti-dementia drugs	3.4	0	71	0
Total	135.3	2.7	1,535	42

Antipsychotics are used long-term although they are very harmful when used this way. We should only treat acute conditions, which is roughly about 5% of current usage, or less. The current usage is 14.3 defined daily doses per 1000 inhabitants per day (DDD), of which 1.1 is lithium. Lithium is perhaps an important drug, as it perhaps reduces suicides (see Chapter 7).²² On the other hand, most cases of bipolar disorder are caused by antidepressants and ADHD drugs, and if we stop using these, there wouldn't be much need for lithium; 0.5 DDD would seem more than enough. Thus, the 14.3 DDD could be reduced to 0.5 plus 5% of 13.2, which is 1.2 DDD.

It is not very often we would need a drug for acute anxiety or sleeping problems, and it should be short-term. Since most people on anxiolytics take them for years because they have become dependent on them, we could somewhat generously say that only 5% of current usage is needed.

If we used psychotropic drugs prudently, we would not need 135.3 DDD but only 2.7, which is 2% of current usage (see Table 14.1).

Our current usage of psychotropic drugs could be reduced by 98%.

In Denmark, 97% of all psychotropic drugs are used outside hospitals. We should therefore primarily target doctors who work in specialist practice, particularly general practitioners who prescribe most of the drugs by far. If we restricted psychotropic drug usage to hospitals, we could curb our drug epidemic. I am aware that this proposal seems radical but it actually isn't. We don't usually give chemotherapy outside hospital, and psychotropic drugs are also toxic and dangerous. This would be too restrictive, though, as psychiatrists in specialist

practice need the possibility to use drugs in acute situations.

The potential financial savings are even larger than 98%. Our costs would only need to be 3% of current expenditure (Table 14.1), but this is before we have taken into account that clinicians often use drugs that are five to ten times more expensive than equivalent drugs. We could therefore easily save 99% of our current expenditure. For Denmark, this would mean annual savings of around DKK 1.5 billion; for the United States it would mean annual savings of a good deal more than \$15 billion, as there is virtually no price control in that country.

Note that the contest is not between drugs and psychotherapy or any other specific mental health approach. The potentially earth-shaking contest takes place between drugs and real life, between an artificially distorted mental life and a clear mind and spirit.¹⁰ Peter Breggin has cautioned that the people most in need of help are the least likely to benefit from any form of help. Being drugged only pushes them deeper into helplessness, further crippling them psychologically and socially. Although he is himself a psychiatrist, Breggin advises that the most disturbed patients need to be protected from psychiatrists.¹⁰

How many people are killed by psychotropic drugs?

Psychiatric drugs are much, much more dangerous than you have ever, ever been led to believe by the doctors who are prescribing them. I genuinely believe that if most people knew how dangerous the psychiatric drugs really were, most people would never start on them, and I also believe that if most prescribers had even the faintest idea how dangerous they were, they would stop prescribing them. How is it that so many people can be ignorant about psychiatric drugs? Well, the truth is that's because they are all getting their information from the drug companies.

PETER BREGGIN²³

Likely all psychotropic drugs can lead to confusion and impaired coordination and balance, which can lead to falls and traffic accidents.²⁴⁻²⁹ Antidepressants are by far the most used psychotropic drugs (Table 14.1). They can cause orthostatic hypotension, sedation, and confusion and they double the risk of falls and hip fractures in a dose-dependent manner.^{28, 29} Hip fractures are often deadly, which makes psychotropic drugs a silent killer, as we will rarely suspect that it was the drug that caused the fall.

If we want to find out how many people psychiatric drugs kill, we might think

that placebo controlled randomised trials would be ideal, but that's not the case, and schizophrenia is a prime example. The cold-turkey design of most of these trials has caused some patients to commit suicide in the placebo group (see Chapter 6). We therefore need to find patients who were not already in treatment with antipsychotics before they were randomised.

In trials in dementia, pre-treatment is not so likely. A meta-analysis of such trials proved that antipsychotics kill people,³⁰ but the authors of a study about antipsychotic prescribing in UK primary care toned down the unwelcome news when they quoted this metaanalysis by saying that dementia “may be associated with” increased all-cause mortality.³¹ No “may be” and no “associated with” are appropriate here; the meta-analysis proved that antipsychotics kill people.

The meta-analysis included trials of newer antipsychotics, aripiprazole (Abilify), olanzapine (Zyprexa), quetiapine (Seroquel) and risperidone (Risperdal), in patients with Alzheimer's disease or dementia, and deaths were recorded up till 30 days after discontinuing the double-blind treatment. For every 100 patients treated, there was one additional death on the drug (3.5% versus 2.3% died, $P = 0.02$). Elderly patients are often treated with several drugs and are more vulnerable to their harmful effects, which means that the death rate is likely higher than in young patients. On the other hand, the trials generally ran for only 10-12 weeks although most patients in real life are treated for years. Furthermore, deaths on drugs are often underreported in industry-sponsored trial reports.¹³ I therefore believe a death rate of 1% is a reasonable estimate to use.

The authors of the meta-analysis also reported that 32% dropped out on the drug and 31% on placebo. Discontinuation rate is a good outcome, as it combines perceptions of benefits and harms from the drugs, and the result indicates that the drugs are pretty useless.³² So elderly patients are killed in huge numbers for no benefit, and yet, in the United States, a third of people in nursing homes take antipsychotics.³²

With regard to benzodiazepines and similar drugs, a cohort study of 34,727 patients found that increased doses increased mortality, and the drugs doubled the death rate, although the average age of the patients was only 55.³³ The excess death rate was about 1% per year. Another large cohort study of such drugs used for sleeping problems also found increased mortality with higher doses.²⁵ The authors did not report on absolute death rates but estimated that sleeping pills kill between 320,000 and 507,000 Americans every year.

With regard to SSRIs, a UK cohort study of 60,746 patients older than 65 showed that they led to falls more often than the older antidepressants or if the depression isn't treated, and that the drugs kill 3.6% of patients treated for one

year.³⁴ The study was done very well, e.g. the patients were their own control in one of the analyses. Some may argue that since it was an observational study, it hasn't been proved that antidepressants kill elderly people. But it's a strong message that even when the patients were their own control – which is a good way to remove the effect of confounders – the lethal effect was clear. Another cohort study, of 136,293 American postmenopausal women (age 50-79) participating in the Women's Health Initiative (WHI), found that antidepressants were associated with a 32% increase in all-cause mortality (hazard ratio 1.32, 95% CI 1.10 to 1.59) after adjustment for confounding factors.³⁵ This corresponded to 0.5% of people killed by SSRIs when treated for one year. Thus, the death rate was only one seventh of that found in the UK cohort but there are good explanations for this. The authors warned that their results should be interpreted with great caution, as the way exposure to antidepressant drugs was ascertained carried a high risk of misclassification, which would likely make it more difficult to find an increase in mortality. Further, the patients were much younger than in the UK study, and the death rate increased markedly with age (0.3% for 50-59 years, 0.6% for 60-69 and 1.4% for 70-79).³⁵ Finally, the women who were exposed and not exposed were different for many important risk factors for early death, whereas the people in the UK cohort were their own control.

Table 14.2. Usage of antipsychotics, benzodiazepines and similar, and antidepressants in Denmark in 2013 in people aged at least 65 years, and estimated number of drug-induced deaths. Defined daily doses per 1000 inhabitants per day. The estimated use at hospitals (1-3%) has been included.

	Usage	Population	Death rate	Deaths
Antipsychotics				
65-79 years	14.3	811,720	1.0%	116
80+ years	10.4	239,409	1.0%	25
Total				141
Benzodiazepines and similar				
65-79 years	61.1	811,720	1.0%	496
80+ years	94.2	239,409	1.0%	225
Total				721
Antidepressants				
65-79 years	119.3	811,720	2.0%	1937
80+ years	186.7	239,409	2.0%	894
Total				2831
Total, all three classes of drugs				3693

I therefore find that the 3.6% annual death rate is more reliable than the 0.5% rate but will use a conservative estimate of a 2% death rate.

We can now estimate how many patients are killed each year by antipsychotics, benzodiazepines and similar drugs, and antidepressants. I will use Danish data again, as they are pretty typical for psychotropic drug use in the western world, e.g. 12% of those aged 65 to 79 are in treatment with an antidepressant drug (Table 14.2); in the United States, usage is 14.5% in those at least 60 years of age.³⁶

Table 14.2 shows the estimated number of drug-induced deaths per year in those aged 65 and above caused by antipsychotics, benzodiazepines or similar, and antidepressants. The total number of deaths per year correspond to 209,000 deaths in the United States and to 539,000 deaths in the United States and the European Union combined.

Psychotropic drugs kill more than half a million people every year aged 65 and above in the western world.

There are some uncertainties related to this estimate. Some people are in treatment with two or even three different types of drugs and you can only die once. There is also survivorship bias, i.e. those who continue for years are those who tolerate the

drug. On the other hand, death can occur at any time, also in people who have taken a drug for years. For example, both antipsychotics and antidepressants prolong the QT interval on the ECG, and these drugs topped the list among all drugs in the FDA's Adverse Events Database for this side effect;³⁷ thus, a patient might die when another drug is added. We also know that benzodiazepines increase the mortality of antipsychotics,³⁸ so this combination is also risky. Furthermore, far more people are exposed to the dangers of these drugs than the data in the table shows, as I have assumed that all patients are treated for a full year.

Even focusing only on those aged 65 and above, the estimates show that psychotropic drugs are the third major killer after heart disease and cancer, which in 2010 killed 600,000 and 575,000 Americans, respectively.¹³ I have deliberately been conservative, and have not factored in deaths occurring in those under 65.

Based on studies in Europe and the United States, I previously estimated that our prescription drugs kill 200,000 people every year in the United States.¹³ This estimate now seems to be far too low, as psychotropic drugs alone kill more than this.

We could also look at the total sales figures for drugs, for example for Eli Lilly's best-seller, fluoxetine. In 2004, the company was under attack and sent this written statement: "Prozac has helped to significantly improve millions of lives. It is one of the most studied drugs in the history of medicine, and has been prescribed for more than 50 million people worldwide. The safety and efficacy of Prozac is well studied, well documented, and well established."³⁹ When drug companies face trouble, they often try to escape by using big numbers. Prozac has not improved millions of lives. Prozac has made millions of lives miserable, so let's estimate how many patients the drug has killed. In Denmark, 45% of total usage of antidepressants occurs in those aged 40 to 64, and 31% in those aged 65 and above, and using the same assumptions as above, Prozac has killed 311,000 people worldwide in the age group 65 and above up to 2004.

References

- 1 Angell M. "The illusions of psychiatry": an exchange. *New York Rev Books* 2011 Aug 18.
- 2 Whitaker R. *Anatomy of an Epidemic*. New York: Broadway Paperbacks; 2010.
- 3 Whitaker R. *Mad in America*. Cambridge: Perseus Books Group; 2002.
- 4 Wipond R. Where critical psychiatry meets community resilience. <http://www.madinamerica.com/2014/11/critical-psychiatry-meets-community-resilience/> (accessed 22 Nov 2014).

- 5 Breggin P. *Psychiatric drug withdrawal: a guide for prescribers, therapists, patients, and their families*. New York: Springer Publishing Company; 2013.
- 6 Kingdon D, Young A. Research into putative biological mechanisms of mental disorders has been of no value to clinical psychiatry. *Br J Psychiatry* 2007;191:285–90.
- 7 Kleinman A. Rebalancing academic psychiatry: why it needs to happen – and soon. *Br J Psychiatry* 2012;201:421–2.
- 8 Kirk SA, Gomory T, Cohen D. *Mad science: psychiatric coercion, diagnosis and drugs*. New Brunswick: Transaction Publishers; 2013.
- 9 Healy D. *Let them eat Prozac*. New York: New York University Press; 2004.
- 10 Breggin P. *Medication madness*. New York: St. Martin's Griffin; 2008.
- 11 Moore TJ, Glenmullen J, Furberg CD. Prescription drugs associated with reports of violence towards others. *PLoS One* 2010;5:e15337.
- 12 Lucire Y, Crotty C. Antidepressant-induced akathisia-related homicides associated with diminishing mutations in metabolizing genes of the CYP450 family. *Pharmgenomics Pers Med* 2011;4:65–81.
- 13 Gøtzsche PC. *Deadly medicines and organised crime: How big pharma has corrupted health care*. London: Radcliffe Publishing; 2013.
- 14 Fazel S, Zetterqvist J, Larsson H, et al. Antipsychotics, mood stabilisers, and risk of violent crime. *Lancet* 2014;384:1206-14.
- 15 Nielssen O, Large M. Rates of homicide during the first episode of psychosis and after treatment: a systematic review and meta-analysis. *Schizophr Bull* 2010;36:702-12.
- 16 Friedman RA. Violence and mental illness – how strong is the link? *N Engl J Med* 2006;16;355:2064-6.
- 17 Healy D, Herxheimer A, Menkes DB. Antidepressants and violence: problems at the interface of medicine and law. *PLoS Med* 2006;3:e372.
- 18 ISEPP Statement on the Connection Between Psychotropic Drugs and Mass Murder. http://www.psychintegrity.org/isepp_statement_on_the_connection_between_psychotropic_drugs_and (accessed 29 Dec 2014).
- 19 Bielefeldt AØ, Danborg P, Gøtzsche PC. Systematic review of adverse effects of antidepressants in healthy volunteer studies. *Proceedings of the 23rd Cochrane colloquium*. Vienna, Austria; 2015.
- 20 Cochrane AL. *Effectiveness and efficiency: random reflections on health services*. London: Nuffield Provincial Hospitals Trust; 1972.
- 21 Breggin PR. The rights of children and parents in regard to children receiving psychiatric diagnoses and drugs. *Children & Society* 2014;28:231-41.
- 22 Cipriani A, Hawton K, Stockton S, et al. Lithium in the prevention of suicide in mood disorders: updated systematic review and meta-analysis. *BMJ* 2013;346:f3646.
- 23 Breggin P. *Psychiatric drugs: more dangerous than you ever imagined*. Video, available from: <http://cepuk.org/>.
- 24 Leipzig RM, Cumming RG, Tinetti ME. Drugs and falls in older people: a systematic review and meta-analysis: I. Psychotropic drugs. *J Am Geriatr Soc* 1999;47:30-9.
- 25 Kripke DF, Langer RD, Kline LE. Hypnotics' association with mortality or cancer: a matched cohort study. *BMJ Open* 2012;2:e000850.
- 26 Glass J, Lanctôt KL, Herrmann N, et al. Sedative hypnotics in older people with insomnia: meta-analysis of risks and benefits. *BMJ* 2005;331:1169-73.
- 27 Hemmelgarn B, Suissa S, Huang A, et al. Benzodiazepine use and the risk of motor vehicle crash in the elderly. *JAMA* 1997;278:27-31.

- 28 Hubbard R, Farrington P, Smith C, et al. Exposure to tricyclic and selective serotonin reuptake inhibitor antidepressants and the risk of hip fracture. *Am J Epidemiol* 2003;158:77-84.
- 29 Thapa PB, Gideon P, Cost TW, et al. Antidepressants and the risk of falls among nursing home residents. *N Engl J Med* 1998;339:875-82.
- 30 Schneider LS, Dagerman KS, Insel P. Risk of death with atypical antipsychotic drug treatment for dementia: meta-analysis of randomized placebo-controlled trials. *JAMA* 2005;294:1934-43.
- 31 Marston L, Nazareth I, Petersen I, et al. Prescribing of antipsychotics in UK primary care: a cohort study. *BMJ Open* 2014;4:e006135.
- 32 Carey B. Drugs to curb agitation are said to be ineffective for Alzheimer's. *New York Times* 2006 Oct 12.
- 33 Weich S, Pearce HL, Croft P, et al. Effect of anxiolytic and hypnotic drug prescriptions on mortality hazards: retrospective cohort study. *BMJ* 2014;348:g1996.
- 34 Coupland C, Dhiman P, Morriss R, et al. Antidepressant use and risk of adverse outcomes in older people: population based cohort study. *BMJ* 2011;343:d4551.
- 35 Smoller JW, Allison M, Cochrane BB, et al. Antidepressant use and risk of incident cardiovascular morbidity and mortality among postmenopausal women in the Women's Health Initiative study. *Arch Intern Med* 2009;169:2128-39.
- 36 Pratt LA, Brody DJ, Gu Q. Antidepressant use in persons aged 12 and over: United States, 2005-2008. *NCHS Data Brief* 2011;76:October.
- 37 Healy D, Howea G, Mangin D, et al. Sudden cardiac death & the reverse dodo verdict. *Int J Risk Safety Med* 2014;26:71-9.
- 38 [Usage of antipsychotics among 18-64-year old patients with schizophrenia, mania or bipolar affective disorder]. København: Sundhedsstyrelsen; 2006.
- 39 Lenzer J. FDA to review "missing" drug company documents. *BMJ* 2005;330:7.

Forced treatment and involuntary detention should be banned

Of all tyrannies, a tyranny sincerely exercised for the good of its victims may be the most oppressive. It would be better to live under robber barons than under omnipotent moral busybodies. The robber baron's cruelty may sometimes sleep, his cupidity may at some point be satiated; but those who torment us for our own good will torment us without end for they do so with the approval of their own conscience.

C.S. LEWIS, NORTHERN IRELAND WRITER (1898–1963)

Forced treatment is the biggest ethical issue in psychiatry. Although it goes against our deepest instincts, it is commonly used, not only for severe cases of psychosis, but sometimes also for people who say they are thinking about suicide and for people who are not particularly psychotic. As free citizens, we vehemently oppose oppression and tyranny in all its forms. However, it is human nature to dominate and take control of others, which lies deep in our genes, as it carries a great evolutionary advantage. Striving for dominance is one of the strongest driving forces in nature, and to have power over others gives us emotional satisfaction and more offspring, which we can observe so clearly among our cousins, the great apes.

As power corrupts, there needs to be a power balance in human relations. In psychiatry, however, involuntarily admitted patients are totally powerless. This extreme power imbalance is a recipe for disaster, and there is nothing psychiatric patients fear more than forced treatment. They have been the victims of punitive measures for centuries without their consent, and the mere threat of such measures has often terrified patients to such an extent that they become docile in order to avoid them. Some psychiatrists have administered shocks to patients they disliked the most, and doctors have regularly prescribed shocks for those who were fighting, restless, noisy, quarrelsome, stubborn and obstinate.¹

Because of the extreme power imbalance, there is a high risk that forced treatment is being used to benefit staff rather than patients, in order to make their

work less stressful. Many patients have reported how the threat of mechanical restraints has been used to discipline them into taking drugs, which they didn't want because of their terrible side effects. And the threat continues when the patients have left hospital and live in a treatment home. If they refuse to take their medication, they might be kicked out of the facility and involuntarily re-admitted to hospital;¹ they might lose their social benefits; and they might even be denied access to a mental healthcare centre. This happened to a four-year-old child when a nurse practitioner in New York said she was bipolar and had a chemical imbalance, and suggested three drugs: valproate (an epilepsy drug), risperidone and lithium. Her parents refused this dangerous cocktail and she got well without drugs.²

Human rights in Europe

Patients often perceive forced treatment as torture, and in Europe the oversight of forced treatment comes under the convention prohibiting torture. Article 3 in the European Convention on Human Rights is very short but to the point: "Prohibition of torture. No one shall be subjected to torture or to inhuman or degrading treatment or punishment."³

What this means is detailed in another document,⁴ and to ensure that the convention is not just window dressing, a European Committee was set up, which, by means of visits, examines the treatment of persons deprived of their liberty with a view to strengthening, if necessary, the protection of such persons from torture and from inhuman or degrading treatment or punishment.⁵

Regarding psychiatry, the committee noted in its 2013 report⁵ that working with the mentally ill will always be a difficult task and that it has observed a dedication to patient care among the overwhelming majority of staff in most psychiatric establishments. However, the committee also observed that deliberate ill-treatment of patients in psychiatric establishments occurs.

I shall convey the key messages in the 2013 report in the next two sections and will thereafter give my comments on them.

Forced treatment

The admission of a person on an involuntary basis should not be construed as authorising treatment without his or her consent. The restraint of agitated or violent patients may on occasion be necessary, but this is an area of particular concern to the *Committee for the Prevention of Torture* (CPT), given the potential

for abuse and ill-treatment. The restraint of patients should be based on a clearly defined policy that should make clear that initial attempts to restrain patients should, as far as possible, be non-physical (e.g. verbal instruction) and that where physical restraint is necessary, “it should in principle be limited to manual control.” Talking to the patient to calm him or her down is the preferred technique.

Instruments of physical restraint (straps, straitjackets, etc.) should only very rarely be justified and must always be either expressly ordered by a doctor or immediately approved by a doctor. Such instruments should be removed at the earliest opportunity, and they should never be applied, or their application prolonged, as a punishment.

Every psychiatric establishment should have a comprehensive, carefully developed policy on restraint. The policy should also contain sections on issues such as staff training, complaints policy, internal and external reporting mechanisms, and debriefing.

Patients have repeatedly said they felt the whole ordeal to be humiliating, a feeling at times exacerbated by the manner in which the restraint was applied.

The CPT often finds that patients are restrained, usually with mechanical restraints, as a sanction for perceived misbehaviour or as a means to bring about a change of behaviour.

In many psychiatric establishments, the application of restraints is resorted to as a means of convenience for the staff. The usual justification is lack of staff but this reasoning is unsound. The means of restraint require more – not fewer – medical staff, as each case necessitates a member of staff to provide direct, personal and continuous supervision. Clearly, video surveillance cannot replace such a continuous staff presence.

A specific register should be established to record all instances of recourse to means of restraint. Reducing its use to a minimum often requires a change of culture.

In many psychiatric establishments, the use of restraint can be substantially reduced, and programmes set up in some countries for that purpose seem to have been successful, without this having led to an increased resorting to chemical restraint or manual control. The question therefore arises whether complete (or almost complete) eradication of mechanical restraint might not be a realistic goal in the longer term.

Patients' rights

The Committee for the Prevention of Torture states that psychiatric patients should be treated with respect and dignity, and in a safe, humane manner that respects their choices and self-determination. An introductory brochure setting out the establishment's routine and patients' rights should be issued to each patient on admission, as well as to their families. Any patients unable to understand this brochure should receive appropriate assistance.

Further, as in any place of deprivation of liberty, an effective complaints procedure is a basic safeguard against ill-treatment. Specific arrangements should exist enabling patients to lodge formal complaints with a clearly designated body, and to communicate on a confidential basis with an appropriate authority outside the establishment.

Patients should, as a matter of principle, be placed in a position to give their free and informed consent to treatment. Any derogation from this fundamental principle should be based upon law and only relate to clearly and strictly defined exceptional circumstances.

Of course, consent to treatment can only be qualified as free and informed if it is based on full, accurate and comprehensible information about the patient's condition and the treatment proposed; to describe ECT as "sleep therapy" is an example of less than full and accurate information. All patients should be provided systematically with relevant information about their condition and the treatment which it is proposed to prescribe for them.

The maintenance of contact with the outside world is essential, not only for the prevention of ill-treatment but also from a therapeutic standpoint. Patients should be able to send and receive correspondence, to have access to the telephone, and to receive visits from family and friends. Confidential access to a lawyer should also be guaranteed.

The patient should be able to consult his or her file, unless this is inadvisable from a therapeutic standpoint, and to request that the information it contains be made available to his or her family or lawyer. In the event of discharge, the file should be forwarded – with the patient's consent – to a treating doctor in the outside community.

Once means of restraint have been removed, it is essential that a debriefing of the patient take place. For the doctor, this will provide an opportunity to explain the rationale behind the measure, and thus reduce the psychological trauma of the experience as well as restore the doctor-patient relationship. For the patient, such

a debriefing is an occasion to explain his emotions prior to the restraint, which may improve both the patient's own and the staff's understanding of his behaviour.

Psychiatric treatment should involve a plan for each patient that includes a wide range of rehabilitative and therapeutic activities, including access to occupational therapy, group therapy, individual psychotherapy, art, drama, music and sports. Patients should have regular access to suitably equipped recreation rooms and have the possibility to take outdoor exercise on a daily basis; it is also desirable for them to be offered education and suitable work.

The CPT all too often finds that fundamental components of effective psychosocial rehabilitative treatment are underdeveloped or even totally lacking, and that the treatment provided to patients consists essentially of pharmacotherapy.

Regular reviews of a patient's state of health and of any medication prescribed is a basic requirement, which will enable informed decisions to be taken as regards a possible dehospitalisation or transfer to a less restrictive environment.

My comments

The European approach provides a blueprint for all nations to follow. The maintenance of contact with the outside world is absolutely essential but not always respected, e.g. a social worker in Norway told me about a patient who wasn't allowed to phone anyone outside the hospital. Such isolation increases the risk of abuse and of the Stockholm syndrome, where captives express empathy and sympathy with their captors. Such feelings are irrational in light of the danger endured by the victims, e.g. a Norwegian psychiatrist used forced treatment with olanzapine, arguing wrongly that untreated schizophrenia causes brain damage.

I oppose vehemently the European Committee's standpoint that a patient should not be able to consult his file if this is unadvisable from a therapeutic standpoint. The Committee doesn't explain what this means, and the exception to patients' right to their file is abused pervasively. US lawyer Jim Gottstein has told me that he doesn't know of a single patient who obtained the file by just asking for it. Therefore, people give an Authorisation for Release of Information to provide the file to PsychRights (see below) that then gives it to the patient. This works about half the time. One of the things providers do to try to prevent the patient from getting their own file is to charge for the copies, which can be prohibitive, as most patients are quite poverty stricken.

In the United States, many patients are unnecessarily imprisoned or are

homeless on the streets.⁶ Some US states still have the death penalty, and a mentally ill patient can be executed by the state, if a committee decides that the patient wasn't insane when he killed someone. This has happened but it is morally repugnant. A patient under the influence of a psychiatric drug may seemingly act in a rational fashion when he kills, but can nonetheless behave totally irrationally and out of character.⁷ Thus, by stating that a person wasn't insane in a forensic report to a court, which is a highly arbitrary decision, a psychiatrist may contribute to murder by the state. This is about as far from being a doctor as one can get, and psychiatrists should refuse to play kings or gods that decide over life and death.

I was involved as an expert witness in a much publicised court case where Graham Bishop, an Englishman, almost stabbed his two daughters to death in the hospital where I work. He was sentenced to 11 years in prison and permanent expulsion from Denmark, but the case was appealed. The forensic committee had acknowledged that methylphenidate (Ritalin) could lead to "increased irritability and emotional instability" and that they could not exclude the possibility that the drug could have influenced his psychological state when the act was committed. However, they considered this unlikely, arguing that he had previously taken similar doses without problems.

There were several issues with this argument. The fact was that he had never before taken such a high dose as he took just before he stabbed his daughters, but even if he had not increased the dose, he could still have reacted out of character under the influence of the drug because the events that led up to the misdeed were very stressful. Further, the harms of methylphenidate are far worse than the committee's euphemistic note about "increased irritability and emotional instability." Methylphenidate can cause violence, including homicide.⁸

I asked the forensic committee whether they considered it the standard of care that Bishop's psychiatrist had apparently said that Bishop could increase the dose without problems and with no upper limit. This question, and several others I had posed, was ignored by the committee, and their reply to my question: "Does the forensic committee think that intake of methylphenidate can increase the risk of violence, including homicide?" was: "The question is of a general character."

Yes, it was of a general character but relevant for the case. I was pretty uncomfortable about getting no answers and also about the committee being in a position where it was essentially asked to evaluate its own previous judgment. This constitutes an unacceptable conflict of interest, as few people are willing to admit their mistakes and overrule themselves. No one knows whether Bishop would have committed his hideous crime had he not been on methylphenidate.

I find the laws about forced treatment highly problematic. In many countries, a person considered insane, or in a similar condition, can be admitted to a psychiatric ward on an involuntary basis if the prospect of cure or substantial and significant improvement of the condition would otherwise be significantly impaired.

But is this ever the case? Are there any treatments that can cure insane patients, or which can lead to such substantial improvements that the patient's condition would be significantly impaired if she is not forced to go to hospital immediately? I don't think so, and, considering the abuse that takes place at psychiatric wards, this clause should be removed from the law of all nations. There is already a clause that, if patients present an obvious and substantial danger to themselves or others, they can be involuntarily admitted. We don't even need this. According to the National Italian Mental Health Law, a reason for involuntary treatment can no longer be that the patient is dangerous. If people are dangerous, it is a matter for the police.

Thus, we don't need forced treatment for patients under any circumstances. We don't need forced admission to hospital either, as patients in Italy can decide that they want treatment elsewhere.

Our physicians cannot give us insulin without our permission, not even if the lack of insulin might kill us, and they cannot force us to take any other drugs than psychiatric drugs. This discrepancy doesn't make sense.

There is a common law assumption of course, that if a person is unable to give consent, the health professional acts in accordance with what she herself would have preferred, e.g. by giving an unconscious person bleeding to death life-saving blood transfusions. But we cannot assume that a severely psychotic person would want psychotropic drugs, or that she is unable to understand what is being proposed or its consequences, e.g. she might decline drugs because of previous experiences of serious harm.

Our laws about forced drug treatment build on the terribly harmful misconception that antipsychotics have a specific effect on psychosis, which is good for people.¹ However, starting in 1975, patients took their fight to US state courts and battled for their human rights. At the same time, Soviet dissidents smuggled out manuscripts describing neuroleptics as the worst sort of torture, which made it tricky to explain how the same substance could be a poison in one country and a helpful remedy in another,¹ particularly as the poison was used as forced "treatment" in both countries.

The idea that it is permissible to drug incompetent people against their will ends up as being the justification to drug everyone who doesn't agree to it in those

kinds of settings. This cannot be defended from an ethical perspective, as it – quite objectively – usually is *not* in the person’s best interest. Furthermore, competence is about autonomy, which is not an all-or-nothing condition. People can be incompetent for some purposes and competent for others, and I firmly believe everyone is competent to decline psychotropic medication and electroshock, especially after they have had any experience with it. Thus, the key word is negotiation.

Psychiatric patients are the real experts and judges and it is only they who can provide a credible insight into the sometimes confusing chaos caused by injured feelings, just like only the sufferer knows what it feels like to have physical pain and can describe it. These are private feelings, and there can be great value in finding a meaning in the madness instead of rejecting it and knocking the patients down against their will with dangerous drugs and making zombies out of them. That won’t help them recover and tackle the symptoms of madness. It’s a slippery slope if psychiatrists assume that patients lack insight into their disease and the drugs used to treat it because of their psychiatric disorder, and that their judgments therefore shouldn’t count because they don’t know what is best for them. It opens the floodgate for health professionals to decide on everything, which short-circuits the good intentions of involving patients in their own treatment and increases the risk of abuse.

Psychologist David Rosenhan has drawn attention to the *Catch 22* position of psychiatric patients.⁹ Some patients have found that they should avoid mentioning certain things to their psychiatrist when hospitalised because it may lead to additional diagnoses and more medication, which the psychiatrist will rarely be interested in stopping again.

What should a patient then do when convinced that the drug and not the disease is the cause of her symptoms? If she says anything about having the dose reduced, she might end up having it increased, or having another drug prescribed on top of the current one, with the argument that she lacks insight into her disease.

Many of the emails I have received from patients and relatives describe exactly this. The power the psychiatric set-up gives to the health professionals is often abused in a way that makes patients helpless and deprives them of their dignity as a person; they are reduced to a “thing.” Here is what a former patient wrote to a psychologist I collaborate with:

I am a nurse but have experienced psychiatry from the patient’s side since 1999. I can “only” say that if I had ever treated any person like what I have seen and been exposed to myself, I would not have been able to live with the bad conscience this would have given me. I happened to sit next to someone whose son was admitted to a psychiatric ward and who said that he only saw the staff

when they came to tell him that he needed to take his medication. I escaped psychiatry's "captivating spiral" in 2004, and it makes me so sad to hear that there has been no significant evolution in the system. I know that psychologists have offered their help, but they have had to attend courses before they could be included in the care system. I fail to understand this, as what is most important is to show an interest in the patients, and if you do this, the patients will surely reveal the traumas that might have caused their mental disorder.

We don't help people by stigmatising them, locking them up, and drugging them, and it is noteworthy that it is patients that have demanded drug-free alternatives. For their doctors, becoming "stabilised" means using drugs to calm them down, which is very different to meeting the patients with their bewildered thoughts and allotting time to work through them without medication.

Psychiatrists have experienced that assertive communication, which involves taking a step back rather than running after the patient and intervening in turmoil, can considerably reduce the use of forced treatment. One such programme is *Basal Exposure Therapy*, which is used by Åse Lyngstad and her colleagues in Norway.¹⁰ It has similarities to the treatment of phobia, as it exposes patients to those factors that cause them to panic. The staff's role is to be on equal terms with the patients and it is the patients' own experience with drugs that is being discussed, instead of the usual top-down approach where the doctors ignore patients' complaints about side effects and their wish to stop the drugs. This approach enforces a different way of working, and a drug-free alternative is offered to the patient, always with a plan for tapering off drugs.

Why are there so few shining lights in psychiatry who understand that psychiatry is not so much about drugs as it is about human relationships?

Forced treatment must be banned

As for all interventions in healthcare, the overriding question is whether forced treatment does more good than harm. I have no doubt it does vastly more harm than good and that we will never be able to prevent widespread abuse if we keep it, and I shall explain why.

Not a single randomised trial has compared seclusion or mechanical restraint with no such intervention,^{11, 12} but these measures can be fatal.¹¹ Electroshock can also be fatal, but what is most worrying is that forced drug treatment kills many patients.

The fact that forced treatment can be fatal was recently underlined in a Danish register study of 2,429 suicides.¹³ It showed that the closer the contact with

psychiatric staff – which often involves forced treatment – the worse the outcome. Compared to people who had not received any psychiatric treatment in the preceding year, the adjusted rate ratio for suicide was six for people receiving only psychiatric medication, eight for people with psychiatric outpatient contact, 28 for people with psychiatric emergency room contacts, and 44 for people who had been admitted to a psychiatric hospital. Patients admitted to hospital would of course be expected to be at greatest risk of suicide because they were more ill than the others (confounding by indication), but the findings were robust and most of the potential biases in the study were actually conservative, i.e. favoured the null hypothesis of there being no relationship. An accompanying editorial noted that there is little doubt that suicide is related to both stigma and trauma and that it is entirely plausible that the stigma and trauma inherent in psychiatric treatment – particularly if involuntary – might cause suicide.¹⁴ The editorialists believed that a proportion of people who commit suicide during or after an admission to hospital do so because of conditions inherent in that hospitalisation.

A tragic case where a trial participant stabbed himself to death while on an antipsychotic drug at the University of Minnesota illustrates several of the ethical issues involved in the extreme power imbalance at psychiatric institutions.¹⁵ Dan Markingson agreed to enrol in a trial while committed involuntarily to hospital, raising questions about his ability to consent, and the lead researcher on the trial was also his treating psychiatrist. An independent review of the research practice at the university found only a single instance where consideration of the dual and potentially conflicting role of the treating psychiatrist/investigator was addressed. Faculty and staff in the Department of Psychiatry told the reviewers that they worked in a “culture of fear.” It took bioethicist Carl Elliott from the same university and others almost ten years of pressure before the university agreed to the investigation, but the review team was expressly forbidden to look into the Markingson case!¹⁶ It seems that fraud was involved, with photocopying of consent forms with identical answers supposedly given by different trial participants, fake signatures and incorrect diagnoses. Markingson’s mother had repeatedly raised concerns about his condition, questioning his involvement in the trial, but her pleas were ignored.

One of psychiatry’s many unfortunate fads is community treatment orders, often called assisted outpatient treatment in the United States, which are legal regimes making outpatient treatment compulsory. A 2014 Cochrane review (three trials, 752 patients) didn’t find any differences in service use, social functioning or quality of life compared with standard voluntary care.¹⁷ In clinical practice, this initiative has also failed. In the UK, it was hoped that these treatment orders,

which came into force in 2008-09, would lead to fewer hospital admissions but the admissions increased.¹⁸ Another problem has been the great variation in their use, with some areas discharging 45% of the patients with treatment orders and others none at all, which indicates a good deal of arbitrariness and uncertainty. Some psychiatrists find treatment orders unethical and, unsurprisingly, many patients find them stigmatising.

The UK mental health charity, Mind, has many concerns about community treatment orders.¹⁹ If a community patient's distress is manageable, the professionals may well argue that the set-up is working and should be continued, but at what point will it be stopped? Without the natural cap on hospital detention provided by the finite number of beds, these orders will undoubtedly be used for too long and for too many people, like a "lobster pot" – easy to get into but very difficult to ever get discharged from. Community treatment orders mean that many people who do not wish to take drugs for the rest of their lives are no longer able to make that decision. There is no escape from this *Catch 22*. If the patient remains well, this is taken to mean that the drugs are working, and if not, forced drugging is often increased, causing even more misery and more deaths. This is totally unacceptable.

When I lectured in Australia in 2015, I was told that only 3-5% of the patients come off the treatment orders again and I met with a doctor who had been on such an order on and off for 20 years. He gave me a copy of an evaluation by a psychiatrist who in 1995 deemed him insightful because he had alerted the community to the brain-damaging effect of antipsychotics! Another person I met was a psychiatrist who was also considered insane by her colleagues, also because she spoke out about the harms from psychotropic drugs. They tried to have her involuntarily confined to hospital but failed. Not much different from Stalin's incarceration of political opponents with the "help" of psychiatrists.

An increase in the use of compulsion in the community will inevitably result in an even greater reliance on drugs, in particular the dangerous use of depot injections of antipsychotics, which are commonly used for community patients, e.g. for the doctor I just mentioned.

In 2014, the Danish Ministry of Health issued what looks like a licence to kill. It allowed psychiatrists to use extraordinarily large doses for forced treatment and said that this applies especially to patients who have been in prolonged treatment and where smaller doses have been tried without a good therapeutic result.²⁰ It's unbelievable. These patients should have their drug withdrawn. Giving more of what was already not working doesn't help, it harms.

Forced drugging prevents people from making their own evaluations of the

benefits and harms of the drugs and from stopping medication, although this would often have been the most rational decision. According to Mind, people deemed fit to live in the community should be trusted to make such decisions for themselves, with support, and the approach to working with them should be based on gaining their trust; not on compelling them to take drugs, which will undermine the valuable therapeutic relationship with doctors, nurses and social workers that might otherwise be established. Many people consulted by Mind feel their relationships with professionals would be harmed by the increased threat of compulsion, with those professionals being turned into “Mental Health Act police officers.”

Forced treatment is very common. About 1% of all Americans are subjected to coercion in the name of mental health every year,²¹ which is a large-scale violation of the deeply treasured American freedom rights, and in Denmark 21% of the patients in psychiatric hospital departments were exposed to forced treatment in 2007.²² Forced drugging is far more common than any official statistics will tell us, however. Rule number one in psychiatric institutions is that patients must comply with the medication regimen, and the patients know that if they refuse they might not be discharged, or other unpleasant things might happen to them. This makes forced drugging look “voluntary.”

As I explained above, forced drugging isn’t needed. Only about 10% of patients refuse treatment, and most do so for only a short time, often because they don’t like the drugs or are afraid of their harms, which are very good reasons for refusing.²³

Extremely rare cases like forced feeding for life-threatening anorexia are already covered by other laws than those that apply specifically to psychiatry. We should therefore protest against forced treatment until it is banned by law and we can use the law to accomplish just that.

Professor Loren Mosher’s testimony in an Alaska court case about forced drugging is particularly lucid.²⁴ He stated that the therapeutic relationship is the single most important thing, and if you have been a cop and have used force, it becomes nearly impossible to change that role into the traditional role of the physician as a healer and advocate for the patient. This is why psychiatrists should stay out of the job of being police. Another reason is that violence breeds violence.

Mosher explained that if somebody is about to do himself or others grievous harm because of some altered state of consciousness, he would stop them in whatever way he needed to. He would prefer to do it with the police, and an

Icelandic psychiatrist told me that this is what the hospital staff would do in Iceland. It is important that the police are unarmed, which is the case in Iceland, and that there are very clear rules about their engagement (which can only be requested by the consultant), including that they cannot put a person in jail. In extreme cases, they would have to stay. This is a sign of respect. The police are called upon to deal with the risk of violence, whether people with mental health problems are involved or anyone else. Same laws for everyone, and in Iceland people trust the police as servants of the people. This is the natural way of handling a difficult situation, as it means that the staff doesn't get involved in serious fights with their patients.

Mosher reported that in his whole career he had never acted as a police officer. He formed the kind of relationship and an ongoing treatment plan, which was acceptable both to him and the patient, and which avoided their getting into a fight.

What makes Mosher's testimony so pertinent is that he is likely the person in the western world who has seen more acutely psychotic people without medication than anyone else. In his Soteria project, which he headed for 12 years, he sat for hours on end with psychotic but unmedicated patients, whom he found were among the most interesting of all people.

The Alaska Supreme Court decided that the government cannot drug someone against their will without first proving by clear and convincing evidence that it is in their best interests and there is no less intrusive alternative available.

The crucial point in this decision is what it means to be "available," and in another case, the court decided that if an alternative is "feasible," the state has to either provide it or let the person go.

I have met with Jim Gottstein, the lawyer who convinced the Supreme Court to rule as it did. He is currently president of The Law Project for Psychiatric Rights in Alaska (<http://psychrights.org/>), a public interest law firm that says on its homepage that it is:

"Devoted to the defense of people facing the horrors of forced psychiatric drugging and electroshock. We are further dedicated to exposing the truth about these drugs and the courts being misled into ordering people to be drugged and subjected to other brain and body damaging interventions against their will. Currently, due to massive growth in psychiatric drugging of children and youth and the current targeting of them for even

more psychiatric drugging, PsychRights has made attacking this problem a priority. Children are virtually always forced to take these drugs because it is the adults in their lives who are making the decision. This is an unfolding national tragedy of immense proportions.”

Gottstein has noted that the public’s opinion is that the drugs work, and that if people weren’t crazy, they would know that the drugs are good for them.² Accordingly, at court hearings, where hospitals apply for sanction of forced treatment, psychiatrists argue that no sound person would refuse medically sound treatment, and the courts comply with their wish.

It was therefore essential for Gottstein’s success to use scientific data to convince the Supreme Court that this isn’t true. The court ruled that, “Psychotropic medication can have profound and lasting negative effects on a patient’s mind and body” and “are known to cause a number of potentially devastating side effects.”

This was a stunning victory for human rights in psychiatry. It happened in 2003, and in 2009 Gottstein succeeded to persuade the authorities to fund a seven-bedroom Soteria home in Alaska where psychotic patients can recover with minimal or no use of drugs. But there is a long way to go. On the web page, “Psychiatry: Force of Law,” Gottstein explains that

psychiatrists, with the full understanding and tacit permission of the trial judges, regularly lie in court to obtain involuntary commitment and forced medication orders.

The experts frequently and openly subvert statutory and case law criteria that impose rigorous behavioural standards as predicates for commitment, and insurmountable barriers are raised to insure that the allegedly “therapeutically correct” social end is met.

Traditionally, lawyers assigned to represent state hospital patients have failed miserably in their mission. And the psychiatric profession explicitly acknowledges that psychiatrists regularly lie to the courts. Fuller Torrey, likely the most prominent proponent of involuntary psychiatric treatment, has said that it would probably be difficult to find any American psychiatrist who has not exaggerated the dangerousness of a mentally ill person’s behaviour to obtain a judicial order for commitment.

It is clear that the legal protections for people diagnosed as mentally ill are illusory and the court proceedings are fairly characterised as a sham. Indeed, our laws contribute to creating a *Catch 22* situation. For example, according to

Alaska's forced drugging statutes, "competent" means that the patient appreciates that he has a mental disorder or impairment, if the evidence so indicates; and denial of a significantly disabling disorder or impairment constitutes evidence that the patient lacks the capability to make mental health treatment decisions. In other words, denying that one is mentally ill is evidence that one is mentally ill!

The worst of all this is perhaps that very many of the patients, sometimes far more than half, are wrongly diagnosed with schizophrenia (see Chapter 6). This fact alone makes forced treatment totally reprehensible.

Psychiatrists usually say that it would be impossible to practice psychiatry safely without having the option of using forced drugging, restraints with belts and straps, and seclusion. But this is false. Studies have shown that, with adequate leadership and training of staff in de-escalation techniques, it *is* possible to practice psychiatry without using force.^{25, 26} Studies have also demonstrated that use of coercive measures is based much more on culture, traditions, and policies than on medical or safety requirements.²⁷ For example, in a 398-bed state psychiatric hospital in North Carolina, the use of mechanical restraints was reduced by 98% at the acute adult unit and it was eliminated at the community transition unit.²⁸ Overall, combining the two groups, the number of injuries was reduced and this was accomplished without an increase in the use of seclusion, manual holds, or drugs.

Another example is the Living Room in Arizona.²⁹ By welcoming involuntarily admitted patients in a living room where they could feel respected and at home, the staff abolished the use of seclusion and restraint almost entirely and far fewer patients needed to be sent to psychiatric hospitals. Initially, there was considerable resistance from the medical staff, but the experience from other peer-operated recovery programmes had shown that most of what the staff were saying would prove not to be true. The focus is almost exclusively on the person's problems and when the peers (recovery mentors) listen to them, they can say that they have had the same experiences and have recovered. So the focus is on recovery and on the patients' strengths instead of illness and of finding faults with them, and the patients value having a place they can come back to if they start to slip, without worrying about being locked up or subjected to restraint or forced drugging.

One of the Living Room programmes was eliminated when the regional health authority took over the crisis services. It wanted to redirect the focus to be more of a traditional medical model service and changed the name from Psychiatric Recovery Center to Urgent Psychiatric Care. I have seen this happen in many

countries. Whenever some clear-sighted pioneers have introduced a model that builds on respect for patients and preservation of their autonomy, and that model has demonstrated far better results than the traditional medical model, the “system” destroys it. It is unspeakably tragic. What does it take to wake people up to the fact that they do the wrong things in psychiatry? Why are people against a humane psychiatry?

A psychosis usually involves a devastating loss of confidence and trust in other human beings, and an acute psychotic break often responds to skilled human intervention, but instead of building rapport with their patients, psychiatrists reflexively resort to pressuring or forcing them into hospitals against their will, and drugging them, further humiliating and alienating them.³⁰ A few hours or days of disturbed behaviour are treated as a cause for a lifetime sentence to drug treatment, and psychiatrists in training will hardly ever see a patient who is not already snowed under with drugs and therefore get a wrong impression both of the patient and his strengths and of the potential for cure without drugs.

Psychiatrists should consider the fact that some patients don't tell them about their thoughts, how they feel, and what they experience, because they are afraid that if they are honest, it could lead to forced treatment. This is not a healthy therapeutic relationship. It is not laudable either that the staff often “justify” their actions by saying that, were it not for the forced treatment, the patient might have died. The evidence we have tells us the opposite. Forced drugging kills. A patient told me that she likened forced treatment to rape and said that there cannot be good rapes. This patient was raped by a man in her family when she was only nine years old with the remark: “Will you take off your pants yourself, or shall I do it?” She became terrified whenever the staff subjected her to forced treatment.

In Iceland, seclusion and restraint were abolished in 1932 and never used again.¹¹ That year, Helgi Tómasson, the first modern psychiatrist in Iceland, took the shackles, straightjackets and other physical restraints that existed in the mental hospital, Kleppur, and burnt them in a furnace – all except one set, which he sent to the Parliament where it is still on display.

The Icelandic psychiatrist who informed me about how difficult situations are handled in Iceland told me that he once worked in a hospital in England where seclusion was used rather a lot. He got a maintenance man to lock the room and put up a sign saying “Out of order!” which remained for about a month. When he took it down, the staff didn't use the seclusion room any more, as they had gotten used to not having this option.

He also said that when he worked at a psychosis ward in London, he and his colleagues waited on average about two weeks before starting antipsychotic

medication on newly admitted people, who had unfortunately nearly always been involuntarily admitted. They didn't want to force treatment on anyone, but most people did in the end, however, choose to take some medication, often in very small doses, so it is very well possible that it was respect, time and shelter that helped the patients, not the "sub-treatment threshold doses."

Practices vary enormously between countries. Involuntary hospital admissions in Europe range from 12 per 100,000 inhabitants in Italy to 233 in Finland.²⁵ Once admitted, rates of coercion also vary enormously. In the UK, mechanical restraint isn't allowed and seclusion is used rarely. In Austria, mechanical restraint is used 45 times more often than in the Netherlands, where forced drugging is also very little used, as the view is that involuntary medication is more invasive and threatens the personal integrity more than seclusion or mechanical restraint.¹¹ The Dutch mental health legislation is very restrictive regarding involuntary medication. It is allowed only in cases of acute emergency, and an emotional crisis is not a medical emergency. Psychiatrist Simon Wilkinson from Akershus University Hospital in Norway has told me that they don't have a regime for rapid tranquillisation and have never needed one in the last 20 years, which is in stark contrast to UK conditions. The staffing is better in Norway and difficult situations are foreseen and managed within the existing care culture.

Patients are of course against forced drugging, and if they are given the option of choosing between two evils, most patients prefer mechanical restraint for forced medication.³¹ But there should be no evil. According to psychiatrist Peter Breggin, forced medication is not therapy but coercion and should have no place in mental health practices.⁷ I shall also quote Jim Gottstein:³²

A commonly-held belief is that locking up and forcibly drugging people diagnosed with mental illness is in their best interests as well as society's as a whole. The truth is far different. Rather than protecting the public from harm, public safety is decreased. Rather than helping psychiatric respondents, many are greatly harmed.

I have explained throughout this book why drug treatment of psychiatric disorders increases violence instead of decreasing it, also in patients with schizophrenia. All the evidence we have tells us that forced treatment increases the harm done not only to patients but also to others.

Only soldiers at war and psychiatric patients are forced to run risks against their will that might kill them. This is perhaps the strongest argument against forced drugging. In rare cases force may be needed, e.g. if a patient is dangerous,

but restraint without belts, i.e. holding the patient firmly, will suffice.

Forced treatment can be avoided, and rather than claiming it would be impossible to practice psychiatry without it, psychiatrists should consider that it's impossible for some patients to *live* after having been exposed to this humiliating and dehumanising treatment. Some patients commit suicide after such an experience.¹³

Until we have outlawed forced treatment, we should monitor carefully the use of coercive interventions as an indicator of the quality of psychiatric inpatient treatment.¹¹ But we don't have to wait, in fact, we are obliged to stop these practices now. See next section.

United Nations forbids forced treatment and involuntary detention

The fundamental human right to equal recognition before the law applies to everyone, also to people with mental disorders. This is clear from the Universal Declaration of Human Rights, the International Covenant on Civil and Political Rights and the United Nations Convention on the Rights of Persons with Disabilities, which has been ratified by virtually all countries.³³

In 2014 the Convention specified that member states must immediately begin taking steps towards the realisation of the rights by developing laws and policies to replace regimes of substitute decision-making by supported decision-making, which respects the person's autonomy, will and preferences.³³

At all times, the individual autonomy and capacity of persons with disabilities to make decisions must be respected, which means that "mental health laws that permit forced treatment must be abolished." People have the right to be free from involuntary detention in a mental health facility and not to be forced to undergo mental health treatment; the right to respect for one's physical and mental integrity; the right to liberty of movement and to choose where and with whom to live; and the right to consent to medical treatment. "States parties have an obligation to require all health and medical professionals (including psychiatric professionals) to obtain the free and informed consent of persons with disabilities prior to any treatment."

"Forced treatment by psychiatric and other health and medical professionals is a violation of the right to equal recognition before the law and an infringement of the rights to personal integrity (art. 17); freedom from torture (art. 15); and freedom from violence, exploitation and abuse (art. 16). This practice denies the legal capacity of a person to choose medical treatment and is therefore a violation

of article 12 of the Convention.”

States parties must respect the legal capacity of persons with disabilities to make decisions at all times, including in crisis situations; must ensure that accurate and accessible information is provided about service options and that non-medical approaches are made available; and must provide access to independent support. Substitute decision-making regimes, in addition to being incompatible with article 12 of the Convention, also potentially violate the right to privacy of persons with disabilities, as substitute decision-makers usually gain access to a wide range of personal and other information regarding the person.

“States parties must abolish policies and legislative provisions that allow or perpetrate forced treatment, as it is an ongoing violation found in mental health laws across the globe, despite empirical evidence indicating its lack of effectiveness and the views of people using mental health systems who have experienced deep pain and trauma as a result of forced treatment.”

The Convention makes it clear that “unsoundness of mind” and other discriminatory labels are not legitimate reasons for the denial of legal capacity, and that the concept of mental capacity is highly controversial in and of itself.

“Mental capacity is not, as is commonly presented, an objective, scientific and naturally occurring phenomenon. Mental capacity is contingent on social and political contexts, as are the disciplines, professions and practices which play a dominant role in assessing mental capacity.”

“In most of the State party reports that the Committee has examined so far, the concepts of mental and legal capacity have been conflated so that where a person is considered to have impaired decision-making skills, often because of a cognitive or psychosocial disability, his or her legal capacity to make a particular decision is consequently removed. This is decided simply on the basis of the diagnosis of an impairment (status approach), or where a person makes a decision that is considered to have negative consequences (outcome approach), or where a person’s decision-making skills are considered to be deficient (functional approach). The functional approach attempts to assess mental capacity and deny legal capacity accordingly. It is often based on whether a person can understand the nature and consequences of a decision and/or whether he or she can use or weigh the relevant information. This approach is flawed for two key reasons: (a) it is discriminatorily applied to people with disabilities; and (b) it presumes to be able to accurately assess the inner-workings of the human mind and, when the person does not pass the assessment, it then denies him or her a core human right — the right to equal recognition before the law. In all of those approaches, a person’s disability and/or decisionmaking skills are taken as legitimate grounds for denying his or her legal capacity and lowering his or her status as a person

before the law. Article 12 does not permit such discriminatory denial of legal capacity, but, rather, requires that support be provided in the exercise of legal capacity.”

A person’s mode of communication must not be a barrier to obtaining support in decision-making, even where this communication is non-conventional, or understood by very few people. States must take measures to provide access to the support required and must ensure that support is available at nominal or no cost. The person must have the right to refuse support and terminate or change the support relationship at any time.

The ability to plan in advance is an important form of support, whereby persons with disabilities can state their will and preferences, which should be followed at a time when they may not be in a position to communicate their wishes to others. The point at which an advance directive enters into force (and ceases to have effect) should be decided by the person and included in the text of the directive; it should not be based on an assessment that the person lacks mental capacity. Where, after significant efforts have been made, it is not practicable to determine the will and preferences of an individual, the “best interpretation of will and preferences” must replace the “best interests” determinations.

All people risk being subject to “undue influence”, yet this may be exacerbated for those who rely on the support of others to make decisions. Undue influence is characterized as occurring, where the quality of the interaction between the support person and the person being supported includes signs of fear, aggression, threat, deception or manipulation. Safeguards for the exercise of legal capacity must include protection against undue influence; however, the protection must respect the rights, will and preferences of the person, including the right to take risks and make mistakes.

States have the ability to restrict the legal capacity of a person based on certain circumstances, such as bankruptcy or criminal conviction. However, the right to equal recognition before the law and freedom from discrimination requires that when the State denies legal capacity it must be on the same basis for all persons.

With respect to children, the best interests of the child must be a primary consideration and their views must be given due weight in accordance with their age and maturity, so that the will and preferences of children with disabilities are respected on an equal basis with other children.

Police officers, social workers and other first responders must be trained to recognise persons with disabilities as full persons before the law and to give the same weight to complaints and statements from persons with disabilities as they would to non-disabled persons.

The denial of the legal capacity of persons with disabilities and their detention

in institutions against their will, either without their consent or with the consent of a substitute decision-maker, is an ongoing problem. This practice constitutes arbitrary deprivation of liberty and violates articles 12 and 14 of the Convention. States parties must refrain from such practices and establish a mechanism to review cases where persons with disabilities have been placed in a residential setting without their specific consent.

My comment: If you still accept forced treatment and involuntary detention, I hope you will change your mind after having read the next section, which is a summary of a book that describes virtually everything that is wrong with psychiatry. It moved me so greatly that whenever I open it again, I get overwhelmed with sadness because I know that many psychiatric patients are abused and die under similar circumstances.

Dear Luise

In *Dear Luise*, Dorrit Cato Christensen writes about her daughter who was killed by psychiatry.³⁴ In his foreword, “You need to be strong in order to be vulnerable,” former Danish Prime Minister Poul Nyrup Rasmussen describes the book as heartbreaking. It truly is, and it should be obligatory reading for all doctors contemplating becoming psychiatrists. If they get through it without crying, they should find themselves another job.

Luise’s hospital admissions always involved troubleshooting, never finding out what strengths she had. It was only when she attended an alternative type of school that people were more interested in her strengths than her weaknesses, and she flourished while there. Her teachers had the necessary patience with her minor oddities, which quickly evaporated if she was given a little time. Psychiatry killed her because the psychiatrists didn’t listen to her, or her mother, or psychologists, or other health professionals, or even to their own staff that knew her much better than they did.

Most unfortunately, Luise’s story isn’t atypical at all. It started when she was seven and had socialising problems and displayed absentmindedness. This led to a series of wrong diagnoses and harmful treatments, starting with valproate for an assumed hidden epilepsy. The neurologist and psychiatrist both said she had improved on valproate, while the psychologist who knew her better said she had deteriorated. She gained 25 kg in weight, which became 6 kg in her chart.

Aged 11, Luise was admitted to a psychiatric ward for adjustment of valproate but ended up at a psychiatric treatment facility where she was raped by another

patient. The way she was treated was utterly dehumanising right from the beginning. She was accused of lying and living in a fantasy world, even though what she told was absolutely true. Her “fantasies” were reported to the social services as being a big problem and a sign of her disease. She was bullied by the staff. Dorrit overheard Luise saying: “I have a headache,” and a staff member said: “Listen, Luise has a headache, isn’t that funny? What are we going to do about it?” whereupon the staff and the inhabitants all laughed. Luise also attempted suicide while she was home on a weekend leave, as she did not want to go back to the institution.

Almost without exception, whenever Luise or Dorrit complained about side effects, the dosage of the suspect drug was increased, and in several instances what was written in the patient’s chart was plain wrong but made the staff’s actions look better. On several occasions, inconvenient correspondence with the authorities simply “disappeared.”

The staff noted that every time Luise had been home over the weekend, they experienced increased problems with her when she came back. It didn’t dawn on them that this was because she would rather stay with her mother. One day, a doctor stopped valproate cold turkey, although this is dangerous.

Reaching the age of 18, Luise was considered an adult who should now take care of her own matters. She was not prepared for that because of her former “protected life” in the treatment system. Her doctor suggested a psychiatric evaluation, so she could get the paperwork updated about what she needed help with.

When Luise kept on talking about some friends coming to visit, which wasn’t correct, Luise and her mother interpreted this as a delusion and sought admission to Rigshospitalet, Denmark’s National University Hospital, to have an evaluation done. Dorrit made sure that the receiving psychiatrist dictated into the tape recorder that Luise should not get psychiatric drugs but should merely be observed. The next day, however, Dorrit found Luise on the floor in her own urine, heavily intoxicated by drugs, and the staff refused to answer Dorrit’s question about which drugs Luise was on. There was nothing in Luise’s chart about observing her without drugs; instead, there was false information that she was already in treatment with an antipsychotic when she arrived, and the dose of this non-existing drug was then “increased.” This was a tremendous and dire error. Luise had never received an antipsychotic before. The psychiatrist asked Luise many leading questions and got answers that had nothing to do with her but which confirmed the doctor’s own prejudices. After eight days on that heavy medication, Luise developed a malignant neuroleptic syndrome, which carries a high mortality.

The psychiatrists apparently ignored this, as they continued the heavy drugging, which turned Luise into a helpless baby that put everything into her mouth. They interpreted these iatrogenic symptoms as signs of very poor intelligence and regression to childhood and started forced drugging her. Dorrit reluctantly accepted it, as she was told that otherwise Luise's condition could become permanent!

Luise fought against the huge doses, but as it ended with forced treatment every time, she stopped her resistance. After 12 days, she was broken: "Today, the patient offers no physical resistance but is anxious about being medicated and holds hands, and afterwards she is somewhat tearful."

When Luise was close to being discharged, a new psychiatrist came by and saw all sorts of problems and she was subjected to forced medication and belts for 22 days, during which she didn't breathe fresh air. After this, Luise was given a 30-minute leave but didn't return. A week later, the laconic note in her chart was: "Since the patient is discharged, mandatory treatment is discontinued." I really wonder why all this forced treatment was so important when Luise apparently could discharge herself, and after that nothing was important?

After six months without drugs, Luise was picked up by the police, while wandering around in Copenhagen airport one night. She often did so, because the airport reminded her of travelling, which she loved. When they said they would transport her to Rigshospitalet, she shouted and screamed. Why didn't they just drive her home?

The psychiatrists didn't believe Dorrit when she told them that Luise only hallucinated when she was on drugs. Luise didn't want drugs, but the psychiatrists noted in her chart that if she refused they would force her to take drugs. No true conversation seems to have taken place, and there was nothing in her files about how it had been for Luise during the six months she was not on drugs. It was totally absurd. The psychiatrists made her psychotic with antipsychotic drugs and then increased the dose because she was psychotic.

Three months later, after three weeks of forced drugs and belts at St. Hans Hospital, she escaped and went home to her mother. Three days later she got the ultimate punishment at Rigshospitalet after having been admitted for less than two hours: a diagnosis of paranoid schizophrenia, apparently without any kind of proper diagnostic process and with no questioning of why Luise had run away from St. Hans Hospital. The psychiatrist didn't know and didn't investigate which drugs Luise was on but gave her a tranquilliser and four different antipsychotics, including the drug that might have killed her earlier, as it caused a malignant neuroleptic syndrome.

Luise was sent back to St. Hans Hospital but to another department. They

didn't know which drugs she had received earlier, neither at Rigshospitalet, nor at their own hospital, and they started four new antipsychotics.

Two months later Luise fell asleep in her bed while smoking a cigarette after an extraordinarily large dose of an antipsychotic, probably as a result of a medical error. The bed caught fire but a member of staff quickly extinguished it. In court, she got a forced treatment order which, in principle, could be for life. No one asked the staff who knew her but a chief physician who didn't know her gave testimony, which sentenced her. Her sentence was used by the staff even many years later to overmedicate her, arguing how dangerous she was.

Luise moved to a treatment home, mostly inhabited by people with senile dementia. She often had nightmares about doctors holding her while they gave her injections, and she once dreamt that a particular doctor inserted the needle directly into her brain with an evil laugh. Due to the heavy drugging, she spent most of her time in bed. Tapering was sometimes discussed but the problem was that the psychiatrists changed all the time so a long-term plan was never made.

Dorrit asked to have the medication reduced and the nurse who was deputy chief at the treatment home also argued strongly for a dose reduction. Dorrit asked whether Luise could have been hallucinating because of the drugs, as she had never hallucinated without taking drugs, but the psychiatrist replied that this was unthinkable. Luise once told the psychiatrist that another patient had sent her evil thoughts, which was interpreted as insanity and led to an increase in drugs. However, that patient often screamed at night things like, "Get out of here – I'll kill you!" which Dorrit had heard herself and told the psychiatrist, but the psychiatrist didn't listen. Half an hour was set aside for this important conversation with Luise, Dorrit and the nurse, but the psychiatrist left after five minutes and there was nothing in the chart about the nurse having been present.

Luise now got a permanent psychiatrist, Sofus (not his real name), assigned to her but that didn't mean progress. He tried 11 different antipsychotics in just two years and changed the dose 26 times up or down. She sometimes got three times the highest recommended dose and the psychiatrists were puzzled that she had hallucinations. Luise vomited and this symptom was also ignored and explained as a sign of her illness. Dorrit was told that it was common that patients wouldn't accept that they were ill, and therefore vomited to avoid medical treatment.

When Dorrit asked for a second opinion with another psychiatrist, Luise's assigned psychiatrist wrote to her:

"I think it will be difficult to find a psychiatrists who would be willing to make such an evaluation ... If his evaluation were materially different from mine, I would of course take it into consideration, though I cannot promise to follow it – as long as I am responsible for the treatment, I also want a free hand. The situation

would be extremely inopportune for our future collaboration. And it would not really benefit Luise.”

Dorrit read about a test for slow metabolism, which might explain why Luise tolerated the drugs so poorly, as the drugs would then accumulate in her body. She contacted a chief of research, who agreed entirely that Luise should get tested because of her symptoms. However, he later said on the phone – speaking non-stop for an hour and 18 minutes – that someone like Luise could not take the test because, even though it might be positive, it was still the psychiatrist’s duty to prescribe this much medication considering how ill she was. He was careful to add that since Dorrit had now been informed, she would not receive a letter about the matter.

It was clear that the research leader must have taken advice from the psychiatrist, and Dorrit wrote a letter asking for a written response with the reasons for rejection of her request. She never got a reply.

One month later, Luise was admitted to Amager Hospital and after a few days, her psychiatrist wanted to transfer her to St. Hans Hospital. Luise was terrified at the thought of going back there and Dorrit therefore came to see her psychiatrist. She was shocked. Luise was running around wildly, trying to avoid the snakes and fictitious blood-soaked creatures that were coming out of the walls. A nurse who had been on duty the last two nights told Dorrit that this was the result of the last two drug dose increases.

But as always, the psychiatrists refused to face the consequences of their gross incompetence. There was now a new psychiatrist and she seemed somewhat uneasy about the situation. She was very unpleasant and didn’t even look at Dorrit but leafed through chart notes while throwing one accusation after another at her. It was Dorrit’s fault that Luise was not getting better because she was against drug treatment. It was Dorrit’s influence that set Luise against going to St. Hans Hospital. The psychiatrist recommended Dorrit stop visiting Luise since, according to the staff, she felt badly after her visits. The psychiatrist went on and on, her head buried in the chart. Dorrit never got the chance to ask questions since she had to defend herself against all the accusations.

The chart note from the same day said: “I speak with the patient who urgently asks for more Seroquel [quetiapine], which is also indicated.”

Luise did not request more Seroquel; she had no idea what was being talked about, being too busy dodging bloodthirsty monsters coming out of the walls. Dorrit wrote to the psychiatrist and pointed out that the staff would certainly not vouch for her claim that Luise felt worse after Dorrit’s visits – in fact, quite the opposite.

Two years before she died, Luise said to her mother: “You can write on my

tombstone that it was the medication that killed me.”

Now the reprisals began. Nobody on the ward would talk to Luise or Dorrit. The daytime staff was dismissive of Luise and made no positive contact. They were cold, as Luise put it, and Dorrit was also frozen out when she came to visit; the staff didn't even say hello but turned their backs on her. One day, Luise asked Dorrit to ask her contact person, a nurse, whether they had increased the dose the previous day. The nurse sat in the courtyard with her feet up on a chair, reading a magazine; she hardly looked up, replying that she really didn't know.

Luise was sent to St. Hans Hospital again, against her wishes. It was essential of course to provide a detailed description of her problems and her history, but this never happened. There was a short chart note speaking of a 30-year old woman with “schizophrenia and mental retardation” who was in treatment with two antipsychotics, three minor tranquillisers, and a drug against drug-induced Parkinsonism.

The first psychiatrist that turned up was shocked at the large doses of medication Luise had been getting at Amager Hospital and decreased the drugs. Later, another psychiatrist came by and increased the medication again.

A third psychiatrist who was going to be in charge of Luise, listened to Dorrit and was very surprised to hear that Luise could be a fully functioning girl when she wasn't heavily drugged. Dorrit told her that Luise would have been transferred to a home for the mentally retarded if Dorrit hadn't intervened. This psychiatrist was highly exceptional since she not only treated Luise as a human being, not some impersonal diagnosis, but also gradually reduced her medication, which clearly improved Luise's condition.

One day the staff phoned Dorrit and asked her to come, as they couldn't understand what had happened to Luise. She fought and raved, shouting “No, I won't.” For a very good reason. The ward she had just moved to was the same place where she was poisoned for two years while strapped down, subjected to forced injections, and where her bedding caught fire. These buried traumas now resurfaced.

The good psychiatrist took Luise seriously and arranged conferences with the psychologist. Luise was very happy about this and never missed a conference, no matter how tired or ill she was. She started to talk again about travelling, followed what was happening in the city's cultural milieu and wanted to see an exhibition of Turner's landscapes, which she and Dorrit had seen previously in London.

The reason for Luise's transferral to St. Hans Hospital was to adjust and stabilise her medication. After 18 months, she was – in Dorrit's words – sent back to Hell. The discharge letter says in part:

“During hospitalisation, we found no evidence of mental retardation. The patient appears of normal intelligence, well-oriented, and can problem-solve ... Our experience is that the patient is treatment-resistant to the medication. Treatment with antipsychotics has been reduced because of side effects ... There is still a mild form of tardive dyskinesia (involuntary grimacing) ... no change should be made for the next one to two years, and then further reduction since the patient should be regarded as treatment-resistant.”

This expert guidance wasn't respected at Amager Hospital. Luise returned to her treatment home, where she had the same psychiatrist as at the hospital, and in less than a year the hospital had killed her.

While at St. Hans Hospital Luise finally took the tests that were earlier denied her and they showed that she was a poor metaboliser and therefore accumulated drugs. She was still on drugs, as the good psychiatrists had had only six months to taper her heavy medication, and when Sofus, Luise's assigned psychiatrist at Amager Hospital, called for a meeting to discuss Luise's stay at St. Hans Hospital, Dorrit and her companion came armed to the teeth with good arguments for reducing the drugging further. Dorrit reminded Sofus about Luise's worrisome tendency to throw up, which she still did because of the drugs, but in the written record of the meeting, Sofus said that he could not remember the vomiting and that it was contrary to his position to reduce the medication. In actual fact, Sofus had himself mentioned this problem in her chart notes several times and had sent Luise for countless unpleasant gastrointestinal examinations.

The next day, the staff had a meeting with Sofus, which they had arranged because of Luise's vomiting. Luise's contact person, Dorte, was present and told Dorrit afterwards that Luise led the discussion herself and that she'd never seen Luise so strong and focused as in this conference. Dorte had declared at the meeting that, “Luise felt better and was more clear-headed than I've ever seen her before – one hundred per cent.”

However, the record of the meeting doesn't even indicate that Dorte was present or what she said:

“The patient says that she must stick a finger down her throat to induce vomiting. In addition, the patient is very talkative, digressive and somewhat disconnected. She reports that there are several people inside her room who are friendly, having a hard time and homeless. They are friendly but do not talk to her because they are invisible ... I [Sofus] ask about her orientation: The patient doesn't know what day of the week it is. When asked about the month, she replies the month before December, when Christmas is. I ask what year it is, and get no reply.”

What Sofus wrote about Luise's lack of orientation wasn't true. Luise always

knew the dates. Dorrit felt that Sofus depicted Luise as mentally retarded, as lack of orientation about time and place is one of the criteria for making the diagnosis.

A month later there was a new meeting and Dorte described that the staff had observed that Luise behaved very differently from other residents diagnosed with schizophrenia. She stressed that it was as if Luise was a complete outsider (which staff at the various wards where Luise had stayed over the years had also said). Dorte reported that the care team had observed how much better Luise felt after prolonged vomiting (i.e. in a less drugged condition). Sofus said that regardless of whether the diagnosis was right or wrong, it could never be erased. It was a very long meeting where Dorte argued well for the staff's position.

At the meeting, Dorrit and Dorte asked to see the record of Luise's conference with Sofus three weeks earlier. Dorrit was shocked to see Sofus's record stating that there had been a so-called urgent call from the staff: "The patient has been increasingly psychotic recently and has been vomiting. If the patient does not improve, she should be hospitalised." This was not at all correct, so Dorte brought out the chart and started to recite from the staff's daily patient notes, which indicated Luise had been very sociable and happy. She had helped with the daily chores and had not received any additional medication, as she normally did. Therefore, there was no evidence of increasing psychosis. Dorrit asked how it was possible to observe increasing psychosis at a single meeting since, if it were increasing, it would be happening gradually. Sofus said he had met Luise in the hallway and that she seemed psychotic.

Dorrit was very worried and didn't understand Sofus' motives. Why did Sofus write that Luise was increasingly psychotic when she was feeling better than she had for a long time? Was he perhaps setting Luise up for an imminent increase in her medication?

The minutes of this meeting included a great deal about the World Health Organization's diagnostic utterings about schizophrenia and little about Dorte's and the staff's views on Luise's diagnosis. It was all written rather vaguely. Dorrit got access to the record two weeks later, only to discover a whole new version of it. The key passage about the urgent call from the staff and Luise becoming increasingly psychotic had been deleted.

The new minutes now only dealt with Luise's vomiting and the text was more or less a copy of a letter Dorrit had written to the psychiatrist about her worries about Luise's vomiting and recent increase in medication. Dorrit's paragraph where she predicted that Luise would be hospitalised within a month if things went as usual wasn't included.

Luise was admitted to Amager Hospital after her vomiting ceased and she became intoxicated with drugs again. Sofus contributed to a dose increase despite

the fact that he had been involved in the discharge conference three months earlier at St. Hans Hospital where he learned that Luise should take as little antipsychotic medication as possible. Luise lost all hope and asked her mother at one of her visits: “Mom, do you think it’s better in Heaven?”

Luise’s best friend at the care home, who was also admitted to the hospital, and stayed in the room next to her, suddenly collapsed at the floor and died within a few minutes. Luise was completely shattered and all she said to her mother was: “I’ll be next.”

The staff didn’t offer any psychological help, arguing that Luise appeared completely untouched by the shocking death. Instead, Luise’s medication was increased because, as the nursing record said, “Rather troubled because of the commotion in the ward this morning. Seems everything is wrong with her and she’s asking us to take her blood pressure.”

So, the psychiatrists had just killed a patient with their drugs, but this was called “commotion,” and the staff apparently didn’t have even the most elementary knowledge about the drugs being used, e.g. that they take away people’s emotions so that they don’t respond like people who are not drugged. In addition, the lack of reaction from the other patients to the screams when Luise’s friend died was likely also related to their fear that they might be next to die from overmedication. They made every effort not to react emotionally to the situation, as they knew so well that the treatment offered for an intense reaction is rarely soothing conversation but rather restraints, possibly supplemented with a syringe. This reminds me of the ubiquitous fear that prevailed in Nazi concentration camps where everybody did their utmost not to show any emotions to avoid becoming the next corpse. A third reason that Luise didn’t react was that psychiatry had broken her. Initially, she fought back, which resulted in long-term coercive measures. Eventually, just the threat of forcible measures was enough to make Luise simply give in. On several occasions, Luise asked to be strapped down, which she preferred to the even worse alternative: extra medication or drugs by injection.

One would think that talking was a normal part of treatment at a psychiatric ward, but this wasn’t the case for Luise. When psychiatrists perceive the patients’ problems as misfiring neurons as a result of some brain defect, talking becomes irrelevant. It would have given her a chance to tell how she felt about her treatment and why she was afraid of the antipsychotic medication. Dorrit found it amazing how much better Luise felt when the good psychiatrist at St. Hans Hospital listened to her, and how things got even better in her sessions with a psychologist.

The Council of Europe’s Committee for the Prevention of Torture and Inhuman or Degrading Treatment or Punishment visited Denmark in 2002 and again in

2004, where it noted that the use of forced measures had increased. Four months after Luise's death, however, the practice of strapping patients had decreased significantly. Psychiatrists had realised that the "patients can take much more responsibility than we had assumed" and also that by talking to them and respecting them, far less restraint was needed and for far less time.

Over the years, Dorrit has written to several prominent psychiatrists asking if they could help her with a second opinion. None welcomed her request but asked her to talk to Luise's psychiatrist again. It is abundantly clear to me that doctors are very reluctant to put themselves in a position where their independent statements can be seen as a criticism of one of their colleagues. Dorrit finally found one, and he concluded that Luise suffered from Asperger's syndrome. It is quite common that such patients are misdiagnosed as having schizophrenia. He said he would contact Luise's psychiatrist (Sofus) and was sure that the two of them together could devise a long-term drug discontinuation plan for Luise. A month later, he phoned Dorrit and informed her that Luise's psychiatrist had refused to cooperate. Dorrit cried on the phone and said she might just as well wait for Luise to die. That came to pass four days later.

Early one morning, Dorrit got the phone call she had feared and had had nightmares about for years. Luise had fallen to the floor like her friend six months earlier, with convulsions, and died. Dorrit screamed and shouted: "No, no, no, it can't be. You've killed her with your medicine. That's what I always said. You were going to kill her."

Dorrit informed her sister Elsebeth who promised to come as quickly as possible. In the next call from the hospital, a cold female voice said that if they wanted to have flowers on Luise's deathbed, they had to bring them themselves.

Ninety minutes after the death call, Dorrit got a third call where a voice said that "if you want to see Luise before she's taken away, you have to come now." Dorrit yelled back tearfully: "I can't just come out there alone to see you people who have killed her, and say my final farewell to Luise, the person in my life I love the most!"

Luise's death was described as an "unintended event" by the doctor in the intensive care unit who was the one who had ordered the lethal injection. At his side sat Luise's regular consultant (Sofus). There was no offer of condolences. The doctors seemed angry, dismissive and insensitive. Elsebeth asked why they had not listened to Dorrit's warnings. A week earlier, Dorrit had told them that more drugs – and especially prolonged-release medication – would be Luise's death, because she couldn't throw them up. Dorrit had argued that no psychiatrist knew how much medication Luise could tolerate since she always vomited.

The answer to Elsebeth's question was: "We went by the book." But they had

not acted by the book. Luise was given a new antipsychotic drug on top of the three she was already taking, which contravened the guideline from the National Board of Health that says that doctors should avoid giving more than one drug at a time and that two drugs could be administered only in exceptional cases.

There was nothing in the record that showed what information Luise was given on prolonged-release injections, or what she said about it. On the day the injection was given, less than ten hours before Luise died, the chart said laconically:

“The patient was persuaded today to take prolonged-release medicine.” Then a few words about the dose and about her feeling well. The autopsy revealed marks around her body, which the coroner could not explain. Dorrit had no doubt these marks stemmed from what happened when Luise got the injection. Luise did not want medicine by syringe, which is crystal clear in the chart note from a week earlier, where she says no, never in my life do I want an injection.

Dorrit had called Luise in the afternoon on the day she received the lethal injection. Luise was angry and did not want a visit, which worried her mother who phoned the ward and was told that Luise was doing fine and just did not want a visit. When Dorrit then asked if there had been a change in her medication – she dreaded the injection the doctor had talked about and said it would be Luise’s death – they replied that they had decided to inform her about any medication changes only once a week, so she would find out a week later. Now Dorrit got really scared, but the next morning Luise was already dead.

There was nothing in the record about the injection, except the time, although it is required by law that a patient’s chart must record what information the patient has received about a new product and what the patient has articulated about it.

After Luise’s death, everyone advised Dorrit not to bother filing a complaint, as it is a degrading and exhausting process, and that she would never get anywhere with it, as psychiatrists are as thick as thieves.

Dorrit reported Luise’s death to the police, to the Patient Complaints Commission and to the Patient Insurance Association. She felt there surely was at least the possibility of a negligent homicide investigation. The police contacted her by letter nine months later stating they had concluded their investigation. By contrast, they had had regular contact with the Copenhagen Hospital Corporation and, through them, Amager Hospital. Three months after Luise’s death they had stated that they found no reason to interview the doctor Dorrit had reported to the police, but that they were awaiting the National Board of Health’s medical assessment of Luise’s treatment.

The Board of Health concluded that Luise had been treated in accordance with the standards of good specialist practice, which it certainly wasn’t, as it so clearly violated the Board’s own guideline.

Dorrit's complaint to the Patient Insurance Association, with the headline "Death from drug poisoning," led nowhere either. According to the Association's psychiatry expert, Luise had received the highest standard of specialist treatment. If that is really the case, I understand better why psychiatrists kill so many of their patients. The psychiatrist noted that, "the risk inherent in the medical treatment must be weighed against the suffering Luise Hjermin Christensen would have undergone without treatment." It's utterly unbelievable that the truth can be twisted in this way. Luise's suffering and death were caused by the drugs the psychiatrists had enforced upon her. She would have done well without the drugs, which amounted to three times the highest recommended dose, and on top of that, in a person who metabolised them poorly.

The Patient Complaints Board took three years to come to a decision. Again, Luise had been treated in accordance with the standards of good specialist practice.

The "licence to kill" in James Bond movies has a perverse meaning in psychiatry. It is considered the highest standard of specialist treatment to kill people after having tortured them for many years with the drugs that ultimately killed them, and which they begged their torturers not to use. Further, this "high standard" took no account of public statements by leading pharmacologists from Denmark and the Nordic countries affirming that the large dose of medicine without any doubt had been the cause of Luise's sudden death!

The absurdity of it all was total when it turned out that the same doctor Dorrit had filed the complaint against was hired by the Patient Complaints Board as a psychiatric expert seven months after Luise's death, i.e. while the case was still being considered. This means that the case might have been settled before it ever got started, as it would have been inconvenient for the Board to investigate a doctor they had just hired.

Several odd things happened during the proceedings. Luise's death certificate said "death from unknown causes" and as contributory causes of death it had "epilepsy and mental retardation." This was outrageous. There was nothing about drugs. And the hospital washed its hands. It disclaimed any responsibility for the two incorrect diagnoses and said it was a matter for the police and forensic experts.

Oddly enough, Luise's friend who had died six months earlier at the same hospital also had epilepsy listed as a contributing cause of death on her death certificate although she wasn't epileptic either. It's never the drug's fault or the psychiatrists' fault, it's you and your disease.

Dorrit's complaints led nowhere, but the massive media coverage helped launch a wide-ranging debate about the quality of mental healthcare in Denmark.

The system, however, congratulated itself for its first-class homicide where everyone seems to have been immunised beforehand against being found guilty. The officially accepted term for deaths such as Luise's is "natural death."

References

- 1 Whitaker R. *Mad in America*. Cambridge: Perseus Books Group; 2002.
- 2 Whitaker R. *Anatomy of an Epidemic*. New York: Broadway Paperbacks; 2010.
- 3 Council of Europe. European Convention on Human Rights. 2010 June 1. www.echr.coe.int (accessed 27 Dec 2014).
- 4 Council of Europe. European Convention for the Prevention of Torture and Inhuman or Degrading Treatment or Punishment. <http://www.cpt.coe.int/en/documents/ecpt.htm>. 2002 March 1 (accessed 27 Dec 2014).
- 5 Council of Europe. European Committee for the Prevention of Torture and Inhuman or Degrading Treatment or Punishment (CPT). CPT/Inf/E (2002) 1 - Rev. 2013. <http://www.cpt.coe.int/en/documents/eng-standards.pdf> (accessed 27 Dec 2014).
- 6 Frances A. Resuscitating the biopsychosocial model. *Lancet Psychiatry* 2014;1:496-7.
- 7 Breggin P. *Psychiatric drug withdrawal: a guide for prescribers, therapists, patients, and their families*. New York: Springer Publishing Company; 2013.
- 8 Moore TJ, Glenmullen J, Furberg CD. Prescription drugs associated with reports of violence towards others. *PLoS One* 2010;5:e15337.
- 9 Rosenhan DL. On being sane in insane places. *Science* 1973;179:250-8.
- 10 Høifødt BF. [Drug free units, for whom and how?]. 2014 Dec 5. <http://www.erfaringskompetanse.no/nyheter/medisinfrie-enheter-for-hvem-og-hvordan> (accessed 7 Dec 2014).
- 11 Steinert T, Lepping P, Bernhardsgrutter R, et al. Incidence of seclusion and restraint in psychiatric hospitals: a literature review and survey of international trends. *Soc Psychiatr Epidemiol* 2009. DOI 10.1007/s00127-009-0132-3.
- 12 Sailas EES, Fenton M. Seclusion and restraint for people with serious mental illnesses. *Cochrane Database Syst Rev* 2000;1:CD001163.
- 13 Hjorthøj CR, Madsen T, Agerbo E, et al. Risk of suicide according to level of psychiatric treatment: a nationwide nested case-control study. *Soc Psychiatry Psychiatr Epidemiol* 2014;49:1357–65.
- 14 Large MM, Ryan CJ. Disturbing findings about the risk of suicide and psychiatric hospitals. *Soc Psychiatry Psychiatr Epidemiol* 2014;49:1353–5.
- 15 Couzin-Frankel J. Human subjects protections under fire at the University of Minnesota. *Science* 2015 March 2.
- 16 Davies E. Investigating the fallout of a suicide. *BMJ* 2013;347:f6039.
- 17 Kisely SR, Campbell LA. Compulsory community and involuntary outpatient treatment for people with severe mental disorders. *Cochrane Database Syst Rev* 2014;12:CD004408.
- 18 Wollaston S. Community treatment orders are not helping people with mental illness. *The Guardian* 2013 Aug 14.
- 19 Mind. Community treatment orders. 2007 Dec. <http://www.mind.org.uk/information-support/legal-rights/supervised-community-treatment/community-treatment-orders/> (accessed 23 April 2014).

- 20 [Memorandum about dosage of drugs in psychiatry]. Ministeriet for Sundhed og Forebyggelse 2014 Oct 30.
- 21 Kirk SA, Gomory T, Cohen D. Mad science: psychiatric coercion, diagnosis and drugs. New Brunswick: Transaction Publishers; 2013.
- 22 [The use of forced treatment in psychiatry]. København: Sundhedsstyrelsen; 2008.
- 23 Mad in Australia. Justice Action 2013 Nov.
- 24 Superior court for the state of Alaska. Transcript of proceedings 3/5/03 & 3/10/03. <http://psychrights.org/States/Alaska/CaseOne/30-Day/3-5and10transcript.pdf> (accessed 21 Nov 2014).
- 25 Fiorillo A, De Rosa C, Del Vecchio V, et al. How to improve clinical practice on involuntary hospital admissions of psychiatric patients: Suggestions from the EUNOMIA study. *Eur Psychiat* 2011;26:201-7.
- 26 Scanlan JN. Interventions to reduce the use of seclusion and restraint in inpatient psychiatric settings: what we know so far, a review of the literature. *Int J Soc Psychiat* 2010;56:412–23.
- 27 Lay B, Nordt C, Rössler W. Variation in use of coercive measures in psychiatric hospitals. *Eur Psychiatry* 2011;26:244-51.
- 28 Godfrey JL, McGill AC, Jones NT, et al. Anatomy of a transformation: a systematic effort to reduce mechanical restraints at a state psychiatric hospital. *Psychiatr Serv* 2014;65:1277-80.
- 29 Ashcraft L. Peer services in a crisis setting; the Living Room. March 2006. <http://www.recoveryinnovations.org/pdf/LivingRoom.pdf> (accessed 22 Feb 2015).
- 30 Breggin P. Medication madness. New York: St. Martin's Griffin; 2008.
- 31 Schmied K, Ernst K. [Isolation and forced injection in the opinion of affected patients and patient care personnel]. *Arch Psychiatr Nervenkr* 1983;233:211-22.
- 32 Gottstein JB. Involuntary commitment and forced psychiatric drugging in the trial courts: rights violations as a matter of course. <http://psychrights.org/Research/Legal/25AkLRev51Gottstein2008.pdf> (accessed 22 Feb 2015).
- 33 United Nations Convention on the Rights of Persons with Disabilities. General comment No. 1 2014 May 19. [http://daccess-dds-ny.un.org/doc/UNDOC/GEN/G14/031/20/PDF/G1403120.pdf?](http://daccess-dds-ny.un.org/doc/UNDOC/GEN/G14/031/20/PDF/G1403120.pdf?OpenElement) OpenElement (accessed 1 April 2015).
- 34 Christensen DC. Dear Luise: a story of power and powerlessness in Denmark's psychiatric care system. Portland: Jorvik Press; 2012.

What can patients do?

There is a lot patients can do to help create a better and more humane psychiatry and to protect themselves against ill-treatment.

1) Try to avoid being treated with electroshock or psychotropic drugs. Remember that very few patients benefit from the drugs they take; that many more are harmed by them; and that we don't have safe drugs. There is widespread corruption at the FDA, and corruption also occurs at other drug agencies.¹ David Graham, who has spent more than 40 years working for the FDA's Office of Drug Safety, has illustrated the regulatory impotence and industry friendliness:²

“The way FDA approaches safety is to virtually disregard it ... The case of antidepressants and suicidality is a perfect example. How does the FDA handle this? With labelling changes. FDA knows that labelling changes don't change physician behavior ... what FDA says is: We can't be 95 percent certain this drug will kill you, therefore we will assume it doesn't – and they let it on the market ... if we wanted drugs that are safe, we could have it tomorrow. It is easy to design those studies. But FDA is not interested in that.”

In 2012 a former FDA scientist, Ronald Kavanagh, also spoke out:³

“Sometimes we were literally instructed to only read a 100- to 150-page summary and to accept drug company claims without examining the actual data, which on multiple occasions I found directly contradicted the summary document. Other times I was ordered not to review certain sections of the submission, but invariably that's where the safety issues would be ... I found evidence of insider trading of drug company stocks reflecting knowledge that likely only FDA management would have known. I believe I also have documentation of falsification of documents, fraud, perjury, and widespread racketeering, including witnesses tampering and witness retaliation.”

In contrast to drug agencies, as David Healy has pointed out, airline pilots are critically concerned with our safety because if we go down, they do too.⁴ If a patient goes down, doctors can blame it on the patient's illness rather than the drug and their own incompetence. When pilots report adverse events, they are taken

very seriously and it leads to change. Doctors rarely report adverse events and if they do, their reports are filed as anecdotal and don't lead to change.

2) If you have a mental health problem or other problems with your life, avoid seeing a psychiatrist unless you know that he or she tries to avoid drug treatment and is a good psychotherapist. If you go to a mainstream psychiatrist, you'll likely get harmed. Perhaps not immediately, but in the long run.

3) Ask if your doctor receives money or other benefits from the drug industry, has shares in a drug company, is visited by drug sales-people, or is being "educated" at industry-sponsored events. If any of this is the case, find yourself another doctor.

4) Don't go to the pharmacy at once if your doctor writes a prescription, but find the officially approved package insert on the Internet. This information can be pretty overwhelming with many unfamiliar medical terms, so you may wish to consult with a knowledgeable friend. It may take some time to digest all the information, but considering that many patients are treated for years, it is well worth the effort. There are also shorter summaries on the Internet that are easier to understand, but they are not necessarily accurate and may have been produced by drug companies, even if posted on seemingly neutral websites like those of patient organisations.

Do a Google search on the name of the drug or search on the drug regulators' homepages, where there can be separate information for patients and for doctors, e.g.:

<http://www.ema.europa.eu/ema/> (European Medicines Agency)

<http://www.fda.gov/> (Food and Drug Administration)

<http://www.mhra.gov.uk> (UK drug agency)

If you read the package insert, you'll likely know much more about the drug than your doctor does, and I am not joking. I can assure you that if doctors knew what is written in package inserts, they wouldn't prescribe so many drugs.

You may also look up independent information sources about drugs and other treatments, e.g. the Cochrane Library, www.cochrane.org. Abstracts of Cochrane reviews are freely available and the full reviews are freely available for half the world's population. You should realise, however, that almost all trials of psychotropic drugs summarised in Cochrane reviews are flawed. You therefore

need to be critical when you read reviews; in particular, the harms of the drugs are often downplayed or missing.

You are now in a better position to decide for yourself whether or not to take the drug. American TV commercials invariably end with something like, “Ask your doctor whether Prozac is right for you.” But since your doctor has very likely been influenced by the drug industry, it would be preferable to “Ask your doctor whether Prozac is wrong for you,” or even better, “Ask *yourself* whether Prozac is wrong for you.” If you ask me, no one should take antidepressants.

5) Avoid taking new drugs the first seven years they are on the market, as most drugs that are with-drawn for safety reasons get withdrawn within the first seven years.¹

6) If a drug is needed, ask your doctor if cheaper drugs are available. Take the drug for as short a time as possible and ask your doctor for a firm plan for tapering it off before you start. If your doctor doesn't think such a plan is needed, don't take the drug!

7) Always remember that our prescription drugs are the third major killer after heart disease and cancer, and that very many unnecessary deaths are caused by psychiatric drugs.¹ This is the result of widespread crime in the drug industry, widespread corruption of doctors, and impotent drug regulation. We have created a system that doesn't work as intended and which is dangerous for patients.

8) One of the most devilish problems in taking drugs is that, quite often, you don't realise that when you don't feel well, it could be a side effect of the drug. Although psychotropic drugs have many harmful effects, doctors tell their patients very little or nothing about the side effects when they prescribe them, or they say there are no side effects to worry about, which is never true.

9) If you have been trapped by psychiatry, reclaim control over your own life (see Chapter 14). You are the master of your life, the psychiatrists aren't and they usually make your life more miserable than it already was.

10) Ask yourself whether you really need the drugs you take and consider gradually reducing them, one by one, with professional assistance (not necessarily the doctor who prescribed them, who will usually be against stopping them). Remember it can be dangerous to stop drugs abruptly (see Chapter 12).

11) Remind yourself that we cannot believe a word of what drug companies tell us, neither in their research nor in their marketing or information to patients.

12) Withdraw your membership if your patient organisation accepts drug industry money or other favours. Patient organisations are often set up by drug companies, although they hide this. Between 1996 and 1999, the US National Alliance for the Mentally Ill, which calls itself “a grassroots organisation of individuals with brain disorders and their family members,” received almost \$12 million from 18 drug companies, led by Eli Lilly.⁵ It is hugely rewarding for companies to brainwash leaders of patient organisations, as they can allow themselves to be much more vocal and belligerent than the companies can.

13) Don't volunteer for trials unless the informed consent form contains a clause that the trial protocol, all analyses, and all the raw data (in an anonymised fashion that doesn't allow identification of individual patients) will be made publicly available.¹ Ensure – before you sign – that you have seen all agreements between the sponsor and the investigators, including monetary amounts and conditions. If doctors are uncomfortable about this, they have something to hide and you shouldn't participate in the trial.

Many industry-sponsored trials are marketing disguised as science that exploit patients for a monetary gain, not only for the company but also for the doctors or their institutions, and they often have a biased design that guarantees results that are useful for the company but which are misleading. Such trials are a breach of the implicit social contract between researchers and patients. To be truthful, patient consent forms should therefore often look somewhat like this:¹

I agree to participate in this trial, which I understand has no scientific value but will be helpful for the company in marketing their drug. I also understand that if the results do not please the company, they may be manipulated and distorted until they do, and that if this also fails, the results may be buried for no one to see outside the company. Finally, I understand and accept that should there be too many serious harms from the drug, these will either not be published, or they will be called something else in order not to raise concerns in patients or lower sales of the company's drug.

References

- 1 Göttsche PC. Deadly medicines and organised crime: How big pharma has corrupted health care. London: Radcliffe Publishing; 2013.
- 2 Blowing the whistle on the FDA: an interview with David Graham. *Multinational Monitor* 2004; 25(12).

- 3 Rosenberg M. Former FDA reviewer speaks out about intimidation, retaliation and marginalizing of safety. Truthout 2012 July 29.
- 4 Healy D. Medical partisans? Why doctors need conflicting interests. Aust N Z J Psychiatry 2012;46:704–7.
- 5 Herxheimer A. Relationships between the pharmaceutical industry and patients' organisations. BMJ 2003;326:1208–10.

What can doctors do?

There is a lot doctors can do to help create a better and more humane psychiatry.

1) We should work towards banning all forced treatment by law in all nations. We have other laws that allow us to save the lives of people, e.g. those with anorexia nervosa who are at risk of dying.

2) We should bury the DSM-5 and the corresponding parts of ICD-10 in psychiatry's grave and start all over again with the diagnostic system. NIMH has abandoned the use of the DSM as a research tool and in 2013, its president, Thomas Insel, explained why:¹

“Unlike our definitions of ischemic heart disease, lymphoma, or AIDS, the DSM diagnoses are based on a consensus about clusters of clinical symptoms, not any objective laboratory measure. In the rest of medicine, this would be equivalent to creating diagnostic systems based on the nature of chest pain or the quality of fever.”

No one with conflicts of interest in relation to the pharmaceutical industry should be allowed to participate in this work. We should narrow and restrict diagnoses so that the resources we already have would be sufficient to allow us to take care of the really sick people in a humane way and allow us the necessary time to listen and talk to them.

3) We need to refresh the art of being patient and wait and see without giving the patients sticky diagnoses and addictive drugs. “Watchful waiting over multiple visits can enable doctors to see if the problems will resolve without intervention.”² Good psychiatrists try to avoid prescribing drugs at the first visit but they are very rare. One of these psychiatrists wrote to me:

“I have been appalled by the state of psychiatry in my country. My professor is behind it all, from developing guidelines to postgraduate education. With experience, I have begun to see what a sham this recasting of mental distress as medical suffering really is. Reading critical psychiatry books has helped me make

sense of my clinical experience. I now strive to have discussions and relationships with patients that are non-coercive equal partnerships. A few patients have questioned if I am really a doctor because I treat them like humans, smile, laugh, and give them a sense of responsibility and ownership of their problems and treatments. I Google things in front of them if I am not sure, usually around side effects, drug interactions or odd withdrawal symptoms. It also made me understand my own history. In my twenties, I had a massive breakdown. At the time, I instinctively resisted all psychiatric labels and medical treatments. It took a long time to get back to normal but somehow I did. When I look back now I can easily see how, in the wrong hands, I could have been labelled schizophrenic, as I heard voices and had delusions and severe anxiety. Now I know my breakdown was no different to what my patients experience. Mental healthcare could be a lot better if it embraced treatments like Open Dialogue or Soteria and listened to the growing voice of psychiatric survivors, but the bio-maniacs have taken over everywhere, and they have money and power, so the hegemony prevails. Most people don't really know how it all works. They assume the drug companies are working for the good of humanity, so we are rather stuck.”

If someone becomes a psychiatrist after having had their own experience with psychosis, they usually retain a warm empathy for their patients, whereas without that experience, many psychiatrists seem almost dismissive of their patients as human beings. This is what so many patients complain about, that they are treated as a thing – a diagnosis – and not being respected.

4) We need psychiatric institutions where patients are guaranteed that psychiatric drugs will not be used under any circumstances, and patients should be free to choose such institutions.

5) We should constantly remind people that a life without drugs is possible and even desirable.

6) Doctors should insist on getting training in psychotherapy and should learn to see this as more important than anything else in psychiatry. A US psychiatrist said:³ “When I trained back in the 80s, we got 50 percent psychotherapy training and 50 percent biologic medication training. Today, the average psychiatric resident gets zero psychotherapy training. So all they have to offer is a pill.”

7) We need a major culture change where we see clinical trials as a public enterprise done for the public good, and performed by independent academic

institutions. We should stop the industry from being its own judges and therefore should no longer accept that the industry can conduct trials on patients. The industry could still pay for trials but should have nothing to do with them, and a public body with patient representatives should ensure that trials are relevant for patients and have relevant outcomes. This could break the vicious circle where drug companies choose investigators that have long-standing relations with the drug industry and don't ask uncomfortable questions. The arrangement would also be vastly cheaper for the industry. The European Society of Cardiology has estimated that university centres can perform drug trials for about 5-10% of the cost of industry trials where there are numerous for-profit middlemen (including doctors and hospitals) who take a hefty surcharge.⁴

8) Psychiatrists should embark on the long-term randomised trials that they have failed to carry out for decades that can inform us about the long-term effects of psychiatric drugs, including their permanent harms.⁵ Our public funders and governments should be more than happy with such trials, as the results will no doubt lead to huge savings for our societies, fewer deaths and healthier patients.

9) Doctors should avoid having financial conflicts of interest. The idea that, as long as they declare them, everything is all right, is silly. Financial conflicts of interest distort what people say and write,⁶ so how should readers handle a research report with authors on industry payroll? Should they ignore the report completely or downgrade it, and if so, in what way and by how much? The solution clearly is to avoid financial conflicts of interest entirely.

Doctors should therefore say no to industry money and favours of any kind, including meals and travel to congresses.⁷ If companies ask for advice, doctors can give it for free, and they should decline to be consultants or sit at companies' advisory boards, as they cannot be advocates for their patients and the drug industry at the same time. It is untenable that doctors won't accept a court case where the judge is paid by one of the sides whereas they willingly accept to be paid by the drug industry.

Unfortunately, there is a culture among doctors that allows acceptance of easy money, and companies may offer to transfer the money in ways that cannot be traced.⁶ But doctors and their organisations should consider whether they find it ethically acceptable to receive money that has been partly earned by organised crime that has harmed and killed many of their patients.⁶ We need to reverse this culture into one of professional ostracism so that a person on industry payroll would no longer show their face in places where their academic colleagues

gather. It's unbelievable that doctors cannot see that acceptance of easy money is corruption. This corruption has many euphemistic names, e.g. "discretionary funds," "no strings attached," or "unrestricted educational grants."

Doctors should also avoid accepting any surplus money generated by collaborative research with the industry, which they can use for their own research, as this impairs their judgment and critical sense and can lead to coercion of patients into trials with harmful drugs. The same applies to hospitals.

10) Institutions should not accept gifts from the industry, as such gifts distort the institutions' agendas.^{6, 8, 9} This will be hard to avoid, as the amounts can go up to one hundred million Euros for just one institution, which has happened in my country.

11) All countries should have publicly accessible and easily searchable websites of doctors' collaboration with industry, detailing the monetary amounts and other benefits, and there should be stiff penalties for missing information or incorrect amounts.

12) Doctors should not "educate" other doctors at industry-sponsored meetings. Such events have no genuine educational purpose but are just marketing, and the industry wouldn't sponsor these activities if they didn't increase sales. It carefully controls the content, although it tries to conceal this fact.⁶

13) Doctors should not add their names to ghost-written papers, which is fraud, as it gives them false credibility and misleads readers deliberately.⁶

14) Doctors should not meet with drug salespeople, as this leads to higher drug costs and irrational prescribing in other ways, and the more frequent the contacts with salespeople, the worse the outcome is for public health and our national economies.^{6, 10} As an example, general practitioners in France prescribed an antipsychotic three times as often if they had been visited by a salesperson in the last month touting such drugs.¹¹ I often wonder why any doctor is willing to believe anything drug companies tell them.

15) Medical journals should stop publishing drug trials. Instead, the protocols, results and the full dataset should be made available on publicly owned websites.^{6, 12} Our most prestigious journals earn a vast amount of money by

selling reprints to the companies of their trial reports, and the editors have a huge conflict of interest when they allow flawed research and flawed abstracts to be published, which often occurs.⁶ Instead of publishing trials, journals could concentrate on critically describing them.

I will end my book with a quote by biologist Richard Dawkins, Oxford, author of *The God Delusion*, who said: “Apparently, when you’ve become the establishment, it ceases to be funny when someone punctures the established bag of wind.”¹³ I hope that, with this book, I have punctured psychiatry’s established bag of wind effectively, as this will benefit patients and enable the coming generations of psychiatrists to do a more meaningful job.

References

- 1 Unrecognised facts about modern psychiatric practice. Council for Evidencebased Psychiatry. 2014 Apr 16.
- 2 Dowrick C, Frances A. Medicalising unhappiness: new classification of depression risks more patients being put on drug treatment from which they will not benefit. *BMJ* 2013;347:f7140.
- 3 Cooper M. Psychiatrist: Over diagnosis leads to strain on resources - KPLC 7 News, Lake Charles, Louisiana. 2014 Nov 27.
- 4 Bassand J-P, Martin J, Rydén L, et al. The need for resources for clinical research: The European Society of Cardiology calls for European, international collaboration. *Lancet* 2002;360:1866–9.
- 5 Wunderink L, Nieboer RM, Wiersma D, et al. Recovery in remitted first-episode psychosis at 7 years of follow-up of an early dose reduction/discontinuation or maintenance treatment strategy: long-term follow-up of a 2-year randomized clinical trial. *JAMA Psychiatry* 2013;70:913-20.
- 6 Gøtzsche PC. Dødelig medicin og organiseret kriminalitet: Hvordan medicinalindustrien har korrumpet sundhedsvæsenet. København: People’s Press; 2013.
- 7 Godlee F. Editor’s choice: say no to the free lunch. *BMJ* 2005 Apr 16.
- 8 Schafer A. Biomedical conflicts of interest: a defence of the sequestration thesis – learning from the cases of Nancy Olivieri and David Healy. *J Med Ethics* 2004;30:8–24.
- 9 Healy D. Academic stalking and brand fascism. In: Turk J (ed). *Universities at Risk. How politics, special interests and corporatization threaten academic integrity*. Toronto: Lorimer Press, 2008.
- 10 Wazana A. Physicians and the pharmaceutical industry: is a gift ever just a gift? *JAMA* 2000;283:373–80.
- 11 Verdoux H, Cougnard A, Grolleau S, et al. Impact of visits from pharmaceutical company representatives on antipsychotic prescription in primary care. *Schizophr Res* 2005;77:107-9.
- 12 Smith R, Roberts I. Patient safety requires a new way to publish clinical trials. *PLoS Clin Trials* 2006;1:e6.
- 13 Dawkins R. Postmodernism disrobed. *Nature* 1998;394:141-3.

Helpful websites

There are many websites about what psychiatry has done to people and about what people can do to reduce their risk of getting harmed by psychiatry and to come off psychiatric drugs. I have described some of them on my own website, www.deadlymedicines.dk, where there are also links to some of my lectures, patient stories and other information.

www.madinamerica.com Award winning science journalist Robert Whitaker's website about why psychiatry hasn't delivered what it promised. New posts appear almost every day.

<http://davidhealy.org/> Psychiatrist David Healy's website about the risks of taking prescription drugs and much else. RxISK.org is about research on and reporting of drug side effects, and about which drugs have been related to suicides.

<http://econsult.rxisk.org/> offers consultation with a medication specialist for a fee.

<http://www.breggin.com/> Psychiatrist Peter Breggin's website: What your doctor may not know: psychiatric drug facts. There is a lot about why and how to avoid psychiatric drugs.

<http://www.mindfreedom.org/> MindFreedom International unites grassroots groups to win human rights and alternatives for people labelled with psychiatric disabilities.

<http://psychrights.org/> is Jim Gottstein's homepage, with information about the force of the law, a search function with many pivotal research papers, and a long list of everyday horror stories from the mental health system.

<http://www.psychintegrity.org/> The aim of the International Society for Ethical Psychology and Psychiatry is to promote safe, humane, and life-enhancing approaches to those problems, which are often diagnosed as psychiatric

disorders.

<http://cepuk.org/> Council for Evidence-based Psychiatry communicates evidence of the harmful effects of psychiatric drugs and works to reduce psychiatric harm by informing policymakers and practitioners, by sharing the testimony of those who have been harmed, and by supporting research into areas where evidence is lacking.

<http://www.ssrstories.org/> is a collection of over 5,000 stories that have appeared in the media and where prescription drugs were mentioned as a possible cause for a variety of adverse outcomes including suicide and homicide.

<http://www.woodymatters.com/> is Kim Witczak's website with links to psychiatric drugs in relation to the FDA, big pharma, corruption, political reactions, and lawsuits (from 2003 to 2008).

<http://recoveringfrompsychiatry.com/> Psychiatric survivor Laura Delano's website with tips about how you can recover from psychiatry.

www.whocaresinsweden.com is a documentary by Jan Åkerblom in three parts about the risks of antidepressant drugs.

www.depression-heute.de is a website in German about depression.

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