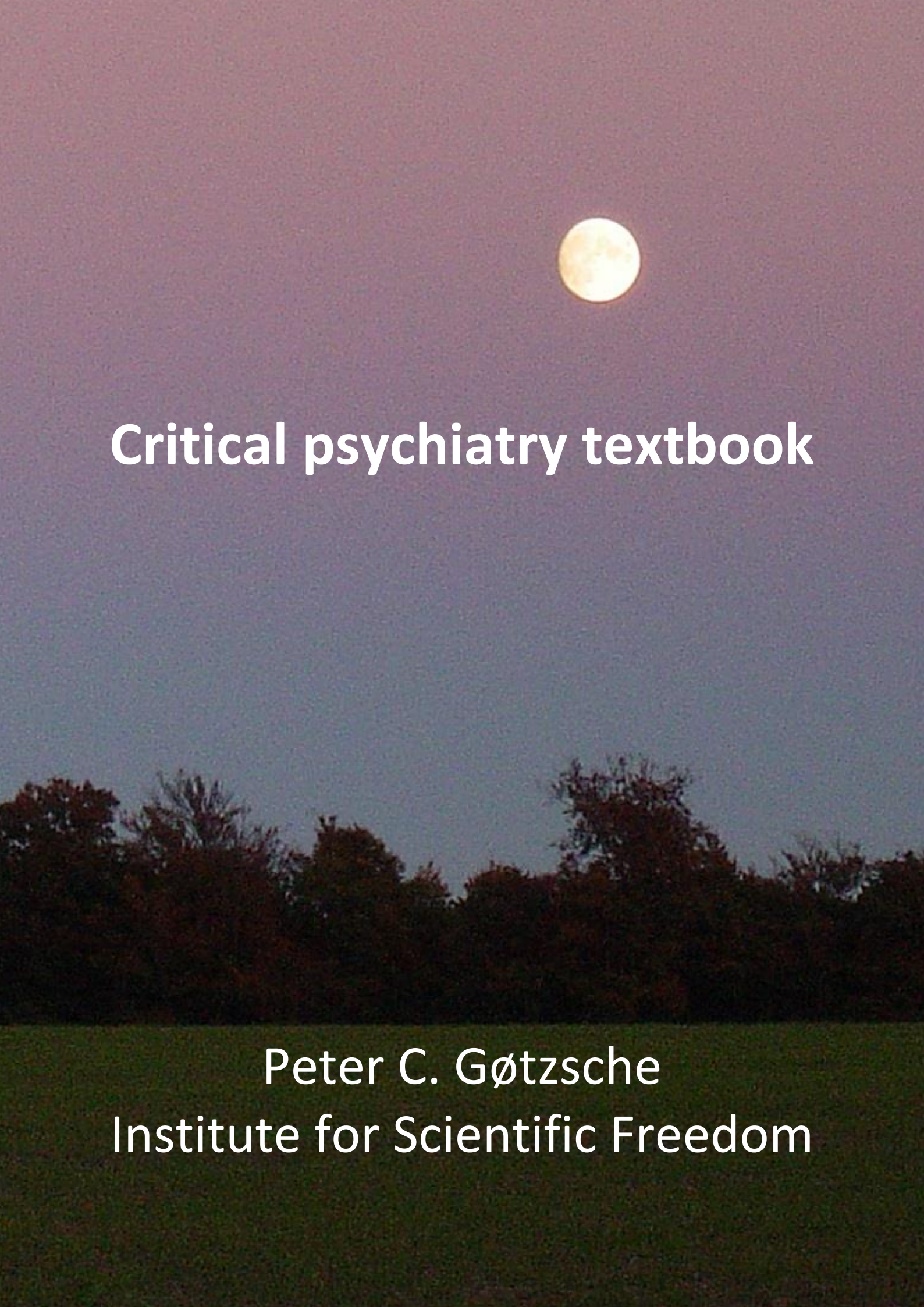

Critical psychiatry textbook

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1 Why a critical textbook of psychiatry?

Students of medicine, psychology and psychiatry, and allied health professions, learn about psychiatry by reading psychiatric textbooks. They generally believe what they read and reproduce it at their exams. It is therefore very important that the information conveyed in psychiatric textbooks is correct.

And that is the problem. There is a huge divide between the official psychiatric narrative and what the science shows. Much of what leading psychiatrists say and write about the reliability of psychiatric diagnoses; the causes of psychiatric disorders; if they can be seen in a brain scan or brain chemistry; and what the benefits and harms are of psychiatric drugs, electroshock and forced treatment is incorrect. This has been extensively documented by critical psychiatrists and others.[1-11]

The discrepancy between opinion and science is also prevalent in psychiatric textbooks. Coming generations of healthcare professionals will therefore learn a lot during their studies that is demonstrably incorrect to the detriment of their patients. This is why a critical textbook of psychiatry is needed.

More than in any other specialty, psychiatry is a discipline where it is of utmost importance to listen to the patients, which is the basis for the diagnostic system. But when the issue is their own practice, psychiatrists are rarely willing to listen even though the general public has experienced that psychiatry, as it is currently practised, does more harm than good.

A survey of 2,031 Australians showed that people thought that antidepressants, antipsychotics, electroshock and admission to a psychiatric ward were more often harmful than beneficial.[12] 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."

But were they wrong? I don't think so. As I shall show in this book, their views are in accordance with the most reliable scientific information we have.

We have a situation where the "customers," the patients and their relatives, do not agree with the "salespeople," the psychiatrists. When this is the case, the providers are usually quick to change their products or services, but this doesn't happen in psychiatry, which has a monopoly on treating patients with mental health issues, with family doctors as their complacent frontline sales staff that do not ask uncomfortable questions about what they are selling.

You might wonder who I am and why you should trust me rather than the psychiatrists who write textbooks. Well, it is not a question of trust but about who has the most valid arguments. That is up to you to decide. I have tried to help you by documenting carefully why I conclude that some statements in the textbooks are wrong and by dissecting research to explain why some research papers are more reliable than others.

Sound and unprejudiced debate about essential issues in psychiatry is rare. When defenders of the status quo do not have valid counterarguments against criticism of their practices, they do not respond to the criticism but attack their opponent's credibility instead.[7] If you ask questions to your teachers based on this book or other books[6-8] or scientific articles I have written, you might be fobbed off with replies like, "Gøtzsche? Never heard about him" (even though they know who I am), "Don't waste your time on him," "Is professor Gøtzsche a psychiatrist? Has he ever managed psychiatric patients? How can he judge what we do?" Or they will say that "Gøtzsche is an antipsychiatrist," which is the ultimate pseudo-argument psychiatrists use.[7 (page 16)]

You should not accept such replies but always ask for the evidence.

Apart from this, I think I have the necessary credentials for criticising psychiatry. I am likely the only Dane who has published more than 75 papers in "the big five" (*BMJ*, *Lancet*, *JAMA*, *Annals of Internal Medicine* and *New England Journal of Medicine*) and my scientific works have been cited over 150,000 times. I am a specialist in internal medicine and have worked in many specialties, including cardiology, endocrinology, haematology, hepatology, gastroenterology, infectious diseases, and rheumatology.

I have done research in psychiatry since 2007 and have uploaded my credentials in relation to this specialty on my website, scientificfreedom.dk (see under About, Staff). Briefly, I have had five PhD students in psychiatry; have been an expert witness in seven psychiatric court cases in seven countries; have received 12 awards or other academic honours; have published nine books or book chapters; have published 30 papers in medical journals with peer review and 128 other papers; and have given over 200 lectures at meetings and courses.

It took me years of close study to find out that the bottom line of psychiatry – which is also what the general public tells us[12] - is that it does more harm than good.[1,5-8] This makes the specialty unique, and the term "psychiatric survivor" says it all.[8] In no other medical specialty do some patients call themselves survivors in the sense that they survived *despite* being exposed to that specialty. They fought hard to find their way out of a system that is rarely helpful, and which many survivors have described as psychiatric imprisonment, or a facility where there is a door in, but not a door out.

In other medical specialties, the patients are grateful that they survived *because* of the treatments their doctors applied to them. We have never heard of a cardiology survivor or an infectious disease survivor. If you survive a heart attack, you are not tempted to do the opposite of what your doctor recommends but in psychiatry, as you will see in this book, you might die or get permanently disabled if you do what your doctor tells you to do.

Many psychiatric survivors have described how psychiatry, with its excessive use of harmful and ineffective drugs, has stolen 10 or 15 years of their life before they one day decided to take the responsibility for their life back from their psychiatrists and discovered that life is much better without drugs. They often say that what woke them up was that they read some of the books about psychiatry by psychiatrists David Healy,[2] Joanna Moncrieff,[3,4] or Peter Breggin,[11] or by science journalist Robert Whitaker[1,5] or me.[6-8]

In 2014, Norwegian psychiatrists wrote about what they called an "alarmingly high discontinuation" rate of psychosis pills in patients with schizophrenia, 74% in 18 months.[13] The psychiatrists argued it highlighted "the clinicians' need to be equipped with treatment strategies that optimize continuous antipsychotic drug treatment." If the psychiatrists had listened to their patients, they would have realised that these drugs should be avoided as long-term therapy.

When students have passed their exams, they will defend tooth and nail what they have learned. It is a curious trait of human psychology that once you have made up your mind, even when you were in serious doubt, you will vigorously defend your position when someone proves that the other option was the correct one.[14]

University textbooks are therefore a powerful tool for indoctrination – for arriving at the "right opinion" even when it is wrong. As an example, 21 out of 36 textbooks (58%) used by students in the Netherlands that discuss brain anatomy have sections on ADHD (attention deficit hyperactivity disorder) with inappropriate generalisations or ambiguous claims.[15]

Leading psychiatrists and their organisations rather consistently propagate misinformation in lectures, in the media, on websites, and in scientific articles.[1-8] You may wonder if this is really

true. Sadly, it is, but more and more critical psychiatrists have realised this and work on changing psychiatric practices. I am a member of the most important group, Critical Psychiatry Network founded by Joanna Moncrieff and based in the UK. We exchange ideas daily on an email list and discuss how we may contribute to reforming psychiatry.

In 2021, I got the idea that if I read and assessed the most commonly used textbooks in Denmark and wrote my own textbook explaining what was wrong with the other ones, this could be an eye opener for students everywhere. Danish textbooks would not be expected to be any different to those in other countries because mainstream psychiatry is the same in all countries. I hope other researchers will analyse the textbooks used in their country like I have done.

When reading books, it can be difficult to find out what is *not* there but should have been mentioned. Before I started reading, I therefore described in a protocol what I believe should be mentioned in psychiatric textbooks.

The pivotal issues I chose are those of obvious importance for the patients and those considered controversial, e.g. whether psychiatric disorders can be seen in a brain scan. The subheadings in my protocol were causes of psychiatric disorders, diagnoses, drug benefits, drug harms, withdrawal of psychiatric drugs, stigmatisation, informed consent, psychotherapy and other psychosocial interventions, and electroshock. As there are hundreds of psychiatric diagnoses, I focused on psychosis, depression, bipolar, ADHD, anxiety disorders, and dementia.

I identified the five psychiatric textbooks in Denmark most commonly used by medical and psychology students and evaluated if the information presented about causes, diagnosis, and treatment was adequate, correct, and based on reliable evidence. The textbooks were in Danish, had a total of 2969 pages, and were published between 2016 and 2021.[16-20]

The authors included some of the most prominent Danish professors of psychiatry, but the textbooks were far from being evidence-based. They often contradicted the most reliable evidence; various author groups sometimes provided contradictory messages even within the same book; and the way they used references was insufficient. It was my clear impression that the more implausible the claims, the less likely they were referenced.

The worst book in terms of the prevalence of seriously misleading or erroneous statements did not have a single literature reference, and all the editors and authors were psychiatrists.[18] The other four books had a bibliography at the end of each chapter, but often with no connection to the text. I therefore needed to guess which of the references that were relevant for the statements made, if any. Sometimes, there was just a name of a person and a year in the text, with no corresponding article or book in the bibliography. In such cases, I tried to find the relevant reference in a literature search on PubMed.

Two textbooks were more truthful than the other three. In one, a psychologist was one of the two editors,[17] and the other book had mostly psychologists as authors.[20]

I have added a page number to the textbook references and often also to references to other books to show where the information can be found. Thus,[17:919] means page 919 in that textbook (or, in a few cases, 1-2 pages further ahead, when the information appeared over several pages).

Psychotropic drugs were developed based on rat experiments and selected if they disrupt the rat's normally functioning brain.[7:229,21] The pills don't cure us, they simply change us by causing a wide array of effects in people, like all brain active substances do, including street drugs. And they are not in any way targeted. There is nothing particularly selective about selective serotonin reuptake inhibitors (SSRIs). This term was invented by SmithKline Beecham to give paroxetine an advantage over other drugs, 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.[22] The drugs don't even target depression. 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 depression pills.[23]

Psychiatric drugs 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.[2,5,21]

I shall therefore avoid the conventional nomenclature for drugs. It is misleading to call pills used for depression antidepressants and pills used for psychosis antipsychotics. These drugs are not "anti" some disease.[7:227] The "anti" also gives an association to antibiotics, which save lives, but psychiatric drugs do not save lives; they take many lives.[7:307] Furthermore, unlike antibiotics, they do not have disease specific properties.[3,4,7,24]

I therefore talk about depression pills and psychosis pills, which do not give any false promises. If we want to reform psychiatry, we will first of all need to change the psychiatric narrative and part of that narrative is the semantics. For the same reason, I shall speak about drug harms and not drug side effects, which is a euphemism, as side effects are sometimes pleasant.

2 Are psychiatric disorders mainly genetic or environmental?

The textbook authors were preoccupied with telling the students that psychiatric disorders are hereditary. Obviously, this gives the specialty prestige. It makes it look more scientific to claim that psychiatric disorders are in the genes and that they can be seen on a brain scan or in brain chemistry (see next chapter). But even if it were true, it would have no clinical consequences, as we cannot change our genes.

I shall explain in this chapter why the information in the textbooks about the causes of psychiatric disorders is generally highly misleading.

First, a sobering fact. Many billions of dollars have been spent by the US National Institute for Mental Health (NIMH) on finding genes predisposing to psychiatric diseases and on finding their biological causes. This has resulted in thousands of studies of receptors, brain volumes, brain activity, and brain transmitters.[7-231]

Nothing useful has come out of this enormous investment apart from misleading stories about what the research showed. This might have been expected from the outset. It is absurd, for example, to attribute a complex phenomenon like depression or psychosis or attention deficit and hyperactivity 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.[25]

The main purpose of psychiatric textbooks is to educate future clinicians. They will not become better clinicians by believing what the textbooks say about heredity. They might in fact become poorer clinicians. If they convey to the patients that their disorder is hereditary, they might take away the patients' hope of becoming normal again. The offspring could also be scared that they might one day come to suffer from a psychiatric disorder. When I was young, the narrative was that 10% of children with a parent with schizophrenia would become schizophrenic, and people were understandably worried that they might be next.

This is not a thing of the past. One of my colleagues, Danish filmmaker Anahi Testa Pedersen, got the erroneous diagnosis schizotypy when she became stressed over a difficult divorce. Many years later, she became enraged when she received a phone call from researchers who wanted to examine her daughter for any possible symptoms arguing that psychiatric disorders are hereditary.

If instead the psychiatrists focused on the environment the patients live in and the traumas they have experienced, there would be hope of recovery, as the environment can be changed and as the traumas can be treated with psychotherapy.

The textbooks did not pull any punches. They spoke of breakthroughs using genome wide association studies,[16:27,16:209,17:308] but there are none. For schizophrenia and similar disorders, each of the several hundred genes identified contribute very little,[18:94] and together, the many loci explain only about 5% of the so-called heritability.[16:210] For ADHD, it was the same. Many different genes have been found, each of which contributes very little.[18:229]

Nonetheless, the psychiatrists propagated the myth of heritability. They did this by quoting twin studies, which are a very soft type of science that has produced unreliable results. The psychiatrists used what I have called the UFO trick.[26] It is very common in science to mislead your readers this way, and it is all about not losing power and prestige and be forced to admit that you were wrong. If you use a fuzzy photo to "prove" you have seen a UFO when a photo taken with a strong telephoto lens has clearly shown that the object is an airplane or a bird, you are a cheat.

When genetic studies have come up empty handed, there is no reason to pollute psychiatric textbooks with fuzzy articles about twin studies, and no reason to read about them.

The fundamental problem with twin studies is that hereditary and environmental factors cannot be separated, not even when some of the twins have been adopted and grow up in another family. The "equal environment assumption" is simply not tenable.[27]

The 1990 Minnesota Study of Twins Reared Apart (MISTRA) illustrates the issues. It is an influential piece of heritability research.[28] Published in *Science*, it is heavily cited as one of five essential studies that examined monozygotic (MZA, or identical) twins who were considered to have been raised separately from each other. MISTRA focused on the intelligence quotient (IQ), and the researchers concluded that intelligence is highly heritable and that very little of it is due to upbringing or environment.

In 2022, 32 years later, this study was debunked.[29] The MISTRA publications had left out critical data. When these data were included, MISTRA failed to demonstrate that IQ is hereditary.

One of the main problems was that the control group - reared-apart dizygotic (DZA, or fraternal) twins - was omitted from the publication. Obviously, if MZA twins have similar IQs, but DZA twins have not, it will lend credence to the notion that IQ is hereditary. The researchers wrote themselves in *Science* that using MZA and DZA twin pairs "provide the simplest and most powerful method for disentangling the influence of environmental and genetic factors."

They even noted that this aspect of their research made it superior to previous research. So why did they not include the DZA data? They claimed that this was due to space limitations and the small sample size. None of this was correct and the sample size was very large for such studies and more than sufficient.

The likely reason for the omission is that when the data from both sets of twins are included, there are no significant differences between the groups, and the whole argument therefore falls apart.[29] If the average MZ correlation does not exceed the DZ correlation for a particular trait, a genetic influence hasn't been demonstrated.

Amazingly, later publications from the MISTRA group even found that the fraternal twins were more similar than the identical twins, but the researchers dismissed this finding in a footnote, calling it "sampling variability."[28] This is likely correct but the researchers prevented critics from reviewing their data, ensuring that no one would be able to test whether their conclusions were warranted.

This looks like fraud. Here is a telling table with the correlations from the 2022 re-analysis of the data that had become available:

	74 MZA pairs	52 DZA pairs	P-value
Wechsler (WAIS) IQ correlations	0.62	0.50	0.17
Raven's Progressive Matrices IQ correlations	0.55	0.42	0.18

There are many important limitations of twins reared apart studies, including:[29]

1) Twins aren't actually separated at birth. In these studies, 33% were separated after a year or more spent growing up together;

2) 75% of the pairs of twins still had contact with each other while growing up;

3) More than half (56%) were raised by a close family member;

4) In 23% of cases, the twins ended up being raised together again at some point or lived next door to each other.

One of the most serious limitations of such studies is that the twins were not randomly selected or followed from birth. Instead, the participants were adults who had already reconnected with each other, noticed similarities, and decided to participate in a study demonstrating heritability. In many cases, these twins ended up in the study after already being promoted in the media as being remarkably similar. This means that the participants were a self-selected group of people who had found themselves similar, who had been in contact with each other, and were usually not fully raised apart.

With a few exceptions, the psychiatry textbook authors swallowed it all, without any critical reflections. Here are some examples of what the textbooks say:

For schizophrenia and similar disorders, the risk ratio is 50 times higher for an identical twin than for other people;[16:207] the heritability is 80%[18:94,19:225] but the concordance rate in monozygotic twins is only 50%.[19:225] It defies reason how the heritability can be bigger than that found in monozygotic twins, which are 100% identical.

Another book mentioned that a Finnish study contradicted these results.[17:41] According to the book, it found that adopted children with a parent with schizophrenia only had an increased risk if they were adopted into a dysfunctional family. The Finnish paper is difficult to read,[30] but it clearly shows that it is important if there are mental health issues in the adopting family.

For affective disorders (depression and mania), the concordance was claimed to be 75% for monozygotic and 50% for dizygotic twins in one book,[18:113] but only 33% was reported for depression in another book.[16:261]

For bipolar, 80% of the cases were explained by genetics;[16:294] for autism and ADHD 60-90%;[20:11,20:467,18:229,17:612] and for obsessive compulsive disorder (OCD) 50%.[20:482]

I do not deny that, to some extent, the way we think and behave are in our genes. During evolution, natural selection has favoured the survival of people who, in situations of danger or stress, behaved in a way that increased their chance of survival. Thus, personality traits are partly hereditary, and it is unsurprising that if a boy in a family is energetic and impatient, the chance that his brother is also energetic and impatient is above average, and both of them might get a diagnosis of ADHD.

This does not make ADHD hereditary, however. ADHD is not something that exists in nature and can be photographed like a giraffe or a cancer can. It is a social construct, which people, including psychiatrists, usually forget. One textbook noted, for example, that women with ADHD are hit harder than men by ADHD in adulthood.[17:612] The ghost has come to life and is now a real thing that can hit people like a car can.

We should abandon such misconceptions. I therefore avoid using the expression “people with ADHD” and say “people with a diagnosis of ADHD.”

One of the times I lectured for the organisation *Better psychiatry*, a woman in the audience said: “I have ADHD,” to which I replied: “No, you haven’t. You can have a dog, a car, or a boyfriend, but you cannot have ADHD. It is a social construct.”

I explained it is just a label. People tend to think they get an explanation for their troubles when psychiatrists give them a name, but this is circular reasoning. Paul behaves in a certain way, and we will give this behaviour a name, ADHD. Poul behaves this way because he has ADHD. It is impossible to argue this way.

I often joked during my lectures that we also need a diagnosis for those children who are *too* good at sitting still and not make themselves seen or heard in class. This became true, with the invention of the diagnosis ADD, attention deficit disorder, without the hyperactivity.

From that day on, I have joked about how long we shall wait before we will also see a diagnosis for those in the middle. Then there will be a stimulant drug for everyone, and the drug industry will have reached its ultimate goal, that no one will escape being drugged.

Schizophrenia and related disorders

Since schizophrenia does not seem to be hereditary, I was interested in seeing what the textbooks said about environmental factors.

As causal factors, the textbooks noted prenatal complications, birth complications, neuroinfections,[18:94] hashish,[17:308] traumatic life events,[16:207,16:232,17:329] acute stress,[16:232] lithium poisoning, malignant neuroleptic syndrome, serotonin syndrome,[16:78] and abstinences after alcohol, benzodiazepines and gamma-hydroxybutyric acid (fantasy, a drug of abuse).[16:78]

What is more interesting is what the psychiatrists did not mention. Psychosis pills can cause psychosis, known as supersensitivity psychosis or oppositional tolerance.[4:45,31] The drugs decrease dopamine levels, and the number of dopamine receptors goes up to compensate for this. If the drugs are suddenly stopped, which patients often do because they tolerate them poorly, the response can be a psychosis. A psychosis can even develop during continued treatment because of this and may not respond to increased dosages.[32] Depression pills[33] and ADHD pills[34] can also cause psychosis (severe mania is a psychosis) but this was not mentioned either in the texbooks.

Traumas play a major role for the development of psychosis, but the textbooks generally ignored this. A typical example is a textbook that claimed 80% hereditability of schizophrenia while there was no numerical estimate for the role of traumas.[19:225] Only one textbook offered a risk estimate, which was a 4 times higher risk if the patient had suffered from physical or psychological abuse.[16:207]

The science is clear. A paper that analysed the 41 most rigorous studies found that people who had suffered childhood adversity were 2.8 times more likely to develop psychosis than those who had not ($P < 0.001$).[35] The P-value is the probability of getting such a result, or an even larger number than 2.8, if there is no relationship, which in this case is less than one in a thousand. Nine of the ten studies that tested for a dose-response relationship found it.[35]

Another study found that people who had experienced three types of trauma (e.g. sexual abuse, physical abuse and bullying) were 18 times more likely to become psychotic than non-abused people, and if they had experienced five types of trauma, they were 193 times more likely to become psychotic (95% confidence interval 51 to 736 times, which means that we are 95% confident that the true risk lies within this interval).[36]

Such data are very convincing unless you are a psychiatrist. A survey of 2813 UK psychiatrists showed that for every psychiatrist who thinks schizophrenia is caused primarily by social factors there are 115 who think it is caused primarily by biological factors.[37] Accordingly, one textbook noted that schizophrenia (and autism and ADHD) are neurodevelopmental disorders, characterised primarily by biological risk factors, and not primarily by psychosocial risk factors and stressful events in childhood.[19:51]

One textbook noted that the intelligence quotient (IQ) of patients with schizophrenia was about one standard deviation below normal, on average, and it attributed this to brain defects caused by the disease as well as sequelae in the form of impaired social contact and disturbed educational course.[18:84]

This is a considerable impairment of the intelligence. The normal quotient is 100 and one standard deviation below normal is 85. There were no references and no reflections if this result came from patients who had been treated with psychosis pills, in which case the low IQ could be a result of drugging the patients making it difficult for them to think and concentrate.

I therefore investigated this. I googled *IQ risk of schizophrenia*, and the top record was all I needed.[38] It was a study of 50,087 18-year-old males conscripted into the Swedish army who were followed up for 13-14 years. During this period, 195 of them had been admitted to hospital with schizophrenia. According to the abstract of the study, "The distribution of scores in those later diagnosed as suffering from schizophrenia was shifted in a downward direction, with a linear relationship between low IQ and risk. This remained after adjustment for potential confounders." The authors concluded that "The results confirm the importance of low intellectual ability as a risk factor for schizophrenia and other psychoses."

The abstract was dishonest and did not reflect what the study showed. In the main text, the authors wrote that "The positive predictive value for low IQ is poor with below average IQ (< 96) predicting only 3.1% of cases." I don't know where they got the 3.1% from, and in a table, the predictive values were much lower, e.g. 1.3% for those with an IQ below 74 and 0.6% for those with an IQ between 74 and 81, and also for those with an IQ between 82 and 89, and between 90 to 95.

The odds ratio for developing schizophrenia based on the IQ score was only 1.27 (1.19 to 1.36). This is a very small increase in risk, which, moreover, was inflated by confounders. The authors adjusted their analyses for socio-economic status, behavioural and school adjustment, drug abuse, urban upbringing, family history of psychiatric disorder and psychiatric disturbance at the time of testing. This led to notable reductions in the odds ratios for all four subscales of the IQ test, but the authors nonetheless claimed that the overall odds ratio was 1.28 after the adjustment. This seems to be a mathematical impossibility.

The authors did not report what the average IQ was for patients with schizophrenia but it was easy to calculate, as they showed a table with numbers in nine different IQ groups. The lowest was < 74 and the highest was > 126, but whether I used 70 and 130, respectively, for these extreme groups, or 65 and 135, I got the same result. The average IQ was 95, or very close to normal.

The textbook claimed that the average IQ was 85.[18:84] This supports my suspicion that these patients were likely incapacitated by psychiatric drugs when they were subjected to the IQ test.

A final question bothers me. What did the textbook authors want to achieve by claiming that people with schizophrenia were dumb? What is the relevance of this for future clinicians? None. It is likely that such information will worsen the stigma these patients are exposed to in psychiatry.[7:183]

It is often assumed that biological or genetic explanations of mental illness increase tolerance towards psychiatric patients by reducing notions of responsibility and blame.[39] 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 a chemical imbalance, brain disease and genetic factors. However, studies have consistently found that this disease model increases stigmatisation and discrimination. A systematic review of 33 studies found that biogenetic causal attributions were related to stronger rejection in most studies examining schizophrenia.[39]

The biological approach increases perceived dangerousness, and fear and desire for distance from patients diagnosed with schizophrenia because it makes people believe the patients are unpredictable.[39-42] It leads to reductions in clinicians' empathy and to social exclusion.[43] It also 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.[41]

Many patients describe discrimination as more long-lasting and disabling than the psychosis itself, and it is recognised as a major barrier to recovery.[40,41] 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 some psychiatrists therefore avoid using the term schizophrenia.[41]

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.[41] 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 they are, see Chapter 7), 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).

As I shall explain below, the spending of enormous amounts of money - largely by drug companies - to teach the public to think more like biologically oriented psychiatrists has had these outcomes: more discrimination, more drugs, more harms, more deaths, more people on disability pension, and greater costs for society.

Affective disorders

For affective disorders, some authors expressed less certainty than for schizophrenia. In one textbook, the authors claimed that the risk of affective disorder is increased 3-4 times if a parent is depressed,[19:210] and the risk of bipolar is increased 4-6 times if a first-degree relative is bipolar,[19:216] but they also admitted that it is very difficult to separate inheritance and environment and to investigate if the changes are a cause or consequence of the depressive condition.[19:210]

A major risk factor for becoming depressed has nothing to do with biological psychiatry but is simply living a depressing life you feel you cannot escape from. There was very little information in the textbooks about this. One book said that stress, living conditions and trauma can play a role for affective disorders but not how much, in contrast to its claims about the role of genes, which was 50%.[17:353] Another textbook mentioned trauma, especially in relation to the first manic episode,[18:113] and a third emotional abuse, neglect and physical abuse with odds ratios as high as 9 to 12.[16:263] It also noted that steroids, birth control pills and oestrogen blocking drugs increase the risk of depression but there was no mention that psychiatric drugs, e.g. benzodiazepines, depression pills and ADHD drugs, can also cause depression,[7,8,11,34,44,45] even though this is highly relevant, given their widespread use.

This was a general problem with the textbooks. I gave another example just above of the psychiatrists protecting their guild interests by not mentioning that the drugs they use can cause the very disorders they try to treat. This is dishonest and unhelpful.

ADHD and the fallibility of observational studies

For the ADHD diagnosis, risk factors included the mother's prenatal intake of tobacco, alcohol, or cocaine; decreased intrauterine growth; foetal exposure to insecticides, lead, or mercury; pre-eclampsia; premature birth; complicated births with hypoxia; low birth weight; postnatal infections; exposure to heavy metals; and possibly neuroinfections.[17:612,18:229]

It was claimed that even though environmental factors may contribute, they play a minor role.[18:229]

It should always be remembered that such claims about causality come from observational studies. They might therefore not be correct, but I did not notice any reservations in the textbooks.

In contrast, top researchers in epidemiology have strong reservations about what their colleagues publish. Observational studies are fraught with difficulties, which is easy to realise if we look at nutritional research.[46] People who eat little fruit and vegetables, or drink more than others, cannot be compared to vegetarians and teetotalers. They differ from them in all sorts of ways that could influence their longevity. Therefore, if nutritional advice is to be believed, it must come from carefully conducted randomised trials.

If we are to rely on observational evidence, high quality research is required, and the signal must be substantial because there is so much bias in these studies. Top epidemiologists have stated that, because it is so easy to be fooled, any less than stunning results are almost impossible to believe.[47] Some said that even a threefold risk increase is not persuasive, and that they can only be persuaded if the lower limit of the 95% confidence interval falls above a threefold increased risk.

When I examine claims made by psychiatrists by looking up the sources, I almost always find that the claims cannot be substantiated. To show you how this works, I examined one of the claimed risk factors for ADHD, low birth weight. I found a relevant article immediately by googling *low birth weight ADHD*, which mentioned that "Several studies have reported that children with a low or extremely low birth weight are as much as 3.8 times more likely to meet diagnostic criteria for ADHD."[48] This is bad science. If we describe several studies, we should not cherry-pick the one with the most extreme result but should say what they show on average, or what the median result was.

The authors quoted four studies, and I looked up the first one. It included 137 very low birth weight (VLBW) children that were compared at 12 years with a sample of matched peers for several psychiatric symptoms.[49] The main risk was ADHD, which was diagnosed in 31/136 (23%) of the VLBW children, compared to 9/148 (6%) of peers.

The risk ratio was 3.75, but I calculated that the 95% confidence interval went from 1.85 to 7.58. This means that the true risk of getting an ADHD diagnosis is likely to be between 2 and 8 times higher for VLBW children than for normal children.

Assuming the result is correct, which we cannot know, as positive results get published more often than negative ones (and I happened to select the most positive one), we may calculate how big the study should have been if the lower limit of the confidence interval should exceed 3. The lower limit becomes 3, if I multiply all numbers by 10. Thus, the study should have been 10 times bigger to arouse any interest among top epidemiologists.

This is a general problem with observational studies. They are usually far too small, and considering their inherent biases, with the additional risk of selective publication of results that

happened to be positive by chance, this means that most results from observational studies are misleading. Even if the studies are very large, they are often misleading, as we cannot eliminate the biases, no matter how we try to adjust for them statistically.

The VLBW study was biased. A table showed that parents of VLBW children were socioeconomically disadvantaged compared to the control group. Furthermore, the authors noted that parents with psychiatric disturbances were more likely to have children who were also vulnerable to psychological problems; that mothers of VLBW children were more depressed than mothers of other infants; and that most VLBW children had limited access to their mothers during the first six months of life. The authors found this of particular interest. So do I, as this could be the explanation for their findings rather than low birth weight.

It is not possible to adjust reliably for such differences with statistical methods. An ingenious study, in which a statistician used raw data from two randomised multicentre trials as the basis for observational studies that could have been carried out, showed that the more variables that are included in a logistic regression, the further we are likely to get from the truth.[50] The statistician also found that comparisons may sometimes be more biased when the groups appear comparable than when they do not; that adjustment methods rarely adjust adequately for difference in case-mix; and that all adjustment methods can on occasion increase systematic bias. He warned that no empirical studies have ever shown that adjustment, on average, reduces bias.

His study may be the most important one I have come across in my whole career. But I have not met a single researcher who did not know him personally, that are aware of his highly important results.

This is not to say that observational studies cannot be useful. Many things cannot be studied in randomised trials and we therefore have no other option than to do observational research. But it is unacceptable that the textbooks almost always described the results of such studies as if they represented the truth, with no caveats.

One textbook provided the sobering information that ADHD is defined arbitrarily as one end of a normal distribution curve, and that brain development is delayed but not qualitatively different from that in healthy children.[18:229]

If this is correct, 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 drug treatment increased pretty much linearly over the months from January to December,[51] and 50% more of those born in December were in treatment.

There are other studies that show the same. This means that if we approach the children with a little patience that allows them to grow up and mature, far fewer would get an ADHD diagnosis.

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.[7:138] It was clearly she who was the problem, not the kids, but as soon as the kids are branded with ADHD, it relieves everyone of any responsibility or incentive to redress the mess they have created, either at school or at home.

We have decided as a society that it is too laborious or expensive to modify the kids' environment, so we modify the kids' brain instead. This is cruel, as I shall explain in Chapter 9. The United States spend over 20 billion dollars a year drugging children for ADHD, which is enough for paying

the mid-career salaries of an extra 365,000 teachers.[52] And this goes up and up. The number of children with an ADHD diagnosis increased by 41% in just 8 years, from 2003 to 2011.[53]

Only one of the textbooks mentioned any of the important studies of the prevalence of the ADHD diagnosis in school classes according to age.[17:51] The belief in the false story about ADHD being a brain disease is so strong that it is close to impossible to correct the harmful narrative.

The indoctrination is very effective. In 2022, one of my colleagues gave a lecture in critical thinking for psychiatry residents. He asked them to review three studies.

One study showed that 16% of those with an ADHD diagnosis had genetic abnormalities (copy number variants), compared with 7% in the controls.[54] The researchers concluded that ADHD was a genetic disease. The residents were asked if this small difference was significant and could be applied to ADHD as a diagnostic category.

The second study looked for a genetic abnormality in neuropsychiatric disorders and is often cited for providing evidence of it.[55] The researchers reported that there was a common genetic component involved in the pathogenesis of five neuropsychiatric disorders. One of the disorders was ADHD. They found that those with ADHD were three times more likely to have this abnormality. But if you combine the data from two tables, you will find that only 0.3% had the genetic abnormality, so 99.7% didn't have it. But because only 0.1% of controls had it, the odds ratio was three.

The third study found that children with an ADHD diagnosis have smaller brains than other kids.[56] The effect size was 0.1, which means that patients with the diagnosis have a 47% chance of having a brain *bigger* than normal.[57] The effect size is also called the standardised effect size. It is the effect divided by the standard deviation of the measurements. This allows comparisons of measurements on different but similar scales. If, for example a scale has a 10-fold greater range than another scale, the standard deviation will also be 10-fold bigger, and the effect sizes can therefore be combined in meta-analyses.

The residents emphasised that genetic differences were highly significant and said that the brain volume study suggested that ADHD was a neurodevelopmental disease.

My colleague was flabbergasted. He told the residents that the data showed that nearly all the kids diagnosed with ADHD didn't have a genetic abnormality; that the odds ratio for the five-disorder study was meaningless; and that the brain volume study showed that there was a 96% overlap between kids with the diagnosis and kids without.[57]

The residents then got hostile. Didn't the lecturer understand that ADHD and the other disorders were biological disorders; that they were illnesses like diabetes or cancer?

My colleague had seen much insanity in psychiatry, but he told me that this was the most hopeless thing he had ever experienced. It is frightening that such people are supposed to take care of psychiatric patients in an evidence-based fashion. They are clearly not able to do this, as it requires that you have a minimum understanding of science.

The study that claimed that children with an ADHD diagnosis have small brains has been widely condemned. *Lancet Psychiatry* devoted an entire issue to criticisms of the study. Allen Frances, chair of the DSM-IV task force (DSM is the Diagnostic and Statistical Manual of Mental Disorders, issued by the American Psychiatric Association), and Keith Conners, one of the first and most famous researchers on ADHD, re-analysed the data and found no brain differences.[58]

The original researchers wrote in the discussion that "our results coming from highly powered analysis, confirm that ADHD patients truly have altered brains, i.e. that ADHD is a disorder of the brain. This is a clear message for clinicians to convey to parents and patients, which can help to

reduce the stigma that ADHD is just a label for difficult kids and caused by incompetent parenting."[56]

The stupidity in this message is heart-breaking. One of the critics wrote in *Lancet Psychiatry* that "there is no point in conveying that a child with ADHD has a brain disorder."[59] Of course not. It is not true, and it does not reduce stigma to tell such nonsense to clinicians, parents and children; it increases stigma.

The American Academy of Child and Adolescent Psychiatry writes on its homepage:[60] "ADHD is a brain disorder. Scientists have shown that there are differences in the brains of children with ADHD … some structures in the brain in children with ADHD can be smaller than those areas of the brain in children without ADHD."

In September 2021, The World Federation of ADHD International Consensus Statement was published.[61] It presented what the authors called "208 evidence-based conclusions about the disorder," but several of these were incorrect, e.g. "When made by a licensed clinician, the diagnosis of ADHD is well-defined and valid" and that treatment with ADHD medications reduces substance abuse, educational underachievement, and criminal activity (see Chapter 9).

There were 80 authors, so most of them cannot have contributed much to the paper. They did not specify which contributions they made but many of them had numerous conflicts of interest in relation to the drug industry. The paper asserted that there is a "polygenic cause for most cases of ADHD, meaning that many genetic variants, each having a very small effect, combine to increase risk for the disorder. The polygenic risk for ADHD is associated with general psychopathology … and several psychiatric disorders."

The great deception of doctors and the public occurs, among other reasons, because very small *group* differences compared to controls are represented as abnormalities found in *individuals* diagnosed with ADHD, even though the study data, when properly parsed, show that not to be true.[57] Once the data are reviewed, it becomes clear that decades of research into possible abnormalities in genes, brain volume, and brain chemicals all turned up negative.

3 Are psychiatric disorders detectable in a brain scan?

According to the psychiatric narrative, psychiatry is built on the biopsychosocial model of disease that takes biology, psychology, and socio-environmental factors into account when explaining why people fall ill.[8]

The reality is vastly different. Ever since the president of the US Society of Biological Psychiatry, Harold Himwich, in 1955 came up with the absurd idea that psychosis pills work like insulin for diabetes,[4:46] biological psychiatry has been the predominant disease model.

Despite 15 years of intense studying, I have been unable to find any important contribution of biological psychiatry to our understanding of the causes of psychiatric disorders and how they should best be treated.

The strong belief in biological psychiatry is also dominant in the textbooks. There is a lot about brain scan studies and brain chemistry and comparatively little about traumas, other psychosocial factors, poverty, discrimination, and other poor life conditions, even though they are important determinants for psychiatric disorders.[35,36,61]

One textbook was particularly misleading as it noted that social causal factors such as poverty, loneliness and housing shortages are of a more indirect nature and contribute to the maintenance of already established diseases.[18:27]

A little light shined through here and there. Elsewhere, in the same book, other psychiatrists contradicted this. They wrote that general improvements in housing standards, job opportunities and family support have great importance for primary prevention, and that traumas, like losses and physical and emotional abuse, are important factors for development of psychopathology.[18:293]

Another book noted, with a reference,[62] that childhood traumas are associated with elevated DNA methylation of brain-derived neurotrophic factor in patients with borderline personality disorder and that those who respond to psychotherapy have a decrease in DNA methylation.[17:41] However, the quoted paper showed that, for all patients, psychotherapy *significantly increased* methylation. Thus, the information in the textbook was misleading, as one obviously cannot separate those who will respond beforehand from those that won't respond. The authors of the paper even blamed the patients for the calamity: "Poor responders were mainly *responsible* [my emphasis] for the increase."

The textbook authors went to great lengths to convince their readers that the origin of psychiatric problems should not primarily be sought in people's living conditions but in the brain. Thereby they propagated the idea that psychiatric disorders are individual mishaps and not something that primarily comes from outside the individual and secondarily affects the brain.

We are told that biological psychiatry has created important results within genetics and psychopharmacology, and with imaging techniques,[17:919] and that imaging studies in depression have led to increased knowledge of hippocampus' role, which has produced clinically relevant results.[17:910] Pretty conveniently, the authors "forgot" to tell us in what way the imaging studies have been useful for clinicians.

One of the textbooks explained that neuropsychiatry is a further development of what was formerly called biological psychiatry.[17:207] But an erroneous idea does not become evidence-based or useful by giving it a new name, and to postulate that billions of people have wrong brains, which essentially is what biological psychiatry does, is as bad as it gets.

Schizophrenia and related disorders

The textbooks claimed it is indisputable that schizophrenia has a neurobiological background;[20:401] that schizophrenia[16:207,18:39,18:79] and affective disorders have an organic basis;[18:39] and that MR (magnetic resonance) and PET (positron emission tomography) scans have shown brain atrophy and disturbed brain metabolism in patients with schizophrenia and depression.[18:27]

When declaring schizophrenia an organic disease, the psychiatrists focused on brain imaging studies and brain chemistry, and the information in the textbooks was often very detailed. For example, one textbook noted that patients with schizophrenia have enlarged ventricles, smaller temporal lobes (superior gyrus temporalis), smaller medial temporal structures (hippocampus, amygdala and parahippocampus) and smaller frontal lobes.[19:227] In particular, the grey matter appeared to be affected. It was claimed that since several of these changes occur already at the onset of the disease, they are probably not a result of long-term medication.[19:227]

These claims are contradicted by studies that found that psychosis pills shrink the brain in a dose-related fashion and that the disease could not explain these changes,[63,64] but the textbook authors avoided commenting on these well-known studies.

One of the textbooks admitted that some of the reduction in grey matter seen with PET scans or fMRI (functional magnetic resonance imaging, which measures the small changes in blood flow that occur with brain activity) may be caused by the use of psychosis pills but added that several changes occur already at the onset of the disease and that there are also brain changes in those who later develop psychosis.[17:309] Another textbook noted that, although the brain changes were minor, they were also seen in people who have not received psychosis pills before.[16:221]

The problem with such statements is that brain scan studies are highly unreliable, as I shall explain in detail below. If any reliable studies had shown this, it would have been such a great triumph for biological psychiatry that we would have heard about them incessantly, but we do not, and in both cases, the authors did not give any references to their remarkable claims.

Another textbook claimed it was well substantiated that there are neuroanatomical changes; that psychotic patients have enlarged ventricles and 4% less grey matter than healthy people; and that first-episode patients also displayed this albeit to a lesser degree than in chronic patients.[20:405] On the other hand, the authors also noted that the findings were contradictory, with reference to a meta-analysis of over 18,000 subjects with schizophrenia,[65] and they noted that, although there is a progressive loss of brain tissue over time, it is very difficult to separate causal factors, e.g. drugs and drug abuse.[20:406]

This honesty did not last long. The same authors claimed that untreated psychosis increases the loss of brain volume and that it is likely that psychosis pills can offer some protection. This has never been shown, and it is extremely unlikely. Psychosis pills do not protect the brain; they harm the brain in numerous ways (see Chapter 7). Many studies have shown that psychosis pills kill nerve cells,[4:176,5:63] and they shrink the brain, too.[63,64]

Affective disorders

For affective disorders, the textbook authors' opinions were more divided than for psychoses. Some were highly confident that the diseases are biologic while others had reservations.

We are told that depressive conditions are associated with neurobiological changes; that there is nonspecific white matter change;[17:357] that cognitive difficulties in affective disorders may be

related to neurodegeneration;[17:358] that MRI and PET suggest a significant biological component;[18:113,18:122] that prolonged untreated depression may explain the brain atrophy that can be measured;[18:124] and that bipolar children have decreased amygdala volume and an altered connection between the prefrontal cortex, the basal ganglia, and the limbic system.[19:216]

One book one noted that recurrent or prolonged depression causes atrophy of the hippocampus.[16:267,16:557] In the same book, however, other authors wrote that it was not clear if white matter hyperintensities in bipolar were caused by the disease or the treatment or were present before any of these.[16:295]

This was one of the very rare admissions in the books that the changes observed on brain scans might be caused by the drugs. Usually, this possibility was totally ignored, as it also is in scientific articles. An editor of one of the textbooks,[18] professor Poul Videbech, published a meta-analysis in 2004 of imaging studies[66] that reported that depression causes a reduction of 9% in the size of the hippocampus, which one of the textbooks quoted.[20:433] Discussing the limitations of his study, Videbech noted that cross-sectional studies such as those he had included in the meta-analysis cannot conclude about causality. He asked: "Does the depression cause shrinkage of the hippocampus or are subjects with small hippocampi susceptible to depression?"

It did not occur to Videbech that people with depression are treated with depression pills, and that it could be the pills that caused brain atrophy. He did not mention this possibility, not even when discussing confounders where he included stress and alcohol abuse. He noted that, in three studies, a smaller volume in the right hippocampus or reduced density in the left "was linked to poor response to antidepressant medication," and that, if this result is confirmed, "it is clinically very interesting as a potential predictor of treatment response."

I cannot make any sense out of this sentence. It seems to me that Videbech suggested that, perhaps in future, all depressed people should have a brain scan. This won't happen.

ADHD

Strangely, ADHD - one of the most controversial diagnoses in all of medicine - was claimed to be one of the psychiatric disorders with the strongest evidence for a neurobiological etiology.[17:612] It was called a neurodevelopmental disorder,[16:462] or a neuropsychiatric developmental disorder,[17:610] characterised primarily by biological risk factors, and not primarily by exposure to psychosocial risk factors and stressful events in childhood.[19:51] It was claimed that ADHD represents a cerebral organ dysfunction and that clinical and neuroradiological studies have shown dysfunctional activity in the frontal lobes.[19:112]

Earlier, ADHD was called Minimal Brain Dysfunction where the focus was on a structural brain damage no one had ever seen.[17:610]

The fact is that ADHD is a social construct and that no reliable studies have shown any biological origin of this construct, or that the brains of people with this diagnosis are different to the brains of other people.[7,10] One textbook that noted that CT and MRI scans had shown less brain tissue and less white matter acknowledged that there are many methodological problems with imaging studies.[17:612]

In contrast, a chapter on ADHD written by two psychologists had no reservations.[20:469] It claimed, with references, that patients diagnosed with ADHD have smaller size of especially the right caudate nucleus, cerebellum and the total volume of the brain;[67] that they have less grey substance in the right caudate nucleus, ventromedial prefrontal cortex and rostral cingular gyrus,

which are not related to the use of ADHD medication;[68] and that fMRI scans have also shown differences to healthy people.[69]

It would be a waste of time to read these papers because the whole scanning literature is highly unreliable (see below on this page). But briefly, the first study was a meta-analysis of MRI studies that included all regions across all studies and found global reductions for ADHD subjects compared with control subjects, with an effect size of 0.41.[67] An effect size this big is a measure of the amount of bias in the reviewed studies and not of true differences. In other words: garbage in, garbage out.

The second study was also a meta-analysis, of predominantly very small studies, which we know are highly unreliable.[68] It included two datasets, and one had only 34 patients with ADHD in the studies, on average, the other only 16 patients.

The third study included 20 patients with ADHD.[69]

All three papers and similar ones should be ignored. The psychologists dressed themselves as serious scientists and then quoted pure garbage.

Anxiety disorders

A textbook noted that brain imaging studies had shown changes in amygdala in children with anxiety disorders but mentioned that it was not known if this was the cause of the disorder or a consequence of it.[19:146]

The other textbooks had no such reservations. Two psychologists wrote that patients with OCD have a dysfunction in the brain's frontostriatal circuit, which is the connection between the frontal lobes and the basal ganglia and thalamus, and that the metabolism in the right caudate nucleus was reduced if the patients had taken depression pills or had received cognitive behavioural therapy.[20:479]

Other authors wrote that patients with OCD had brain atrophy and increased grey matter but offered no references in support of this astonishing claim.[17:418]

We are told that the basal ganglia, thalamus and orbitofrontal part of the cortex are involved;[19:162] that some studies have shown normalisation of dopaminergic hyperactivity in striatum after treatment with depression pills or cognitive behavioural therapy;[17:419] that imaging studies have shown overactivity of the orbitofrontal cortex and caudate nucleus in patients with OCD that disappeared on successful treatment with drugs or psychotherapy;[16:364] and that effective drug or behavioural therapy can normalise the affected brain areas.[19:162]

The last two sentences are tautologies. They contain empty information like in the sentence: It will either rain tomorrow or it will not rain. If "effective" or "successful" treatment is used, the brain changes are normalised. If they are not normalised, the treatment was not effective, or the patient was treatment resistant. This is a win-win situation that seems to confirm something that is not correct, namely that there are brain changes in the first place.

Brain scan studies are highly unreliable

We should be highly sceptical towards the results of imaging studies. The textbooks did not convey much doubt but the one where all three editors were psychologists noted that they were aware of the limitations of the methods used in imaging studies and they questioned the findings that had been made.[20:10]

Another textbook noted that the findings obtained with structural and functional scans were inconsistent and varying, especially those obtained with functional MR scans that measure small changes in blood flow to various areas of the brain while the patient is given various tasks.[17:329]

This whole area is a mess of highly unreliable research.[7:233]

A 2009 meta-analysis found that the false positive rate of neuroimaging studies is between 10% and 40%.[70] And a 2012 report written for the American Psychiatric Association about neuroimaging biomarkers 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."[71]

One good research paper can sometimes make hundreds of poor studies redundant. This is the case for a 2012 systematic review by Joshua Carp that surveyed the methodological state of the art in a random sample of 241 functional magnetic resonance imaging (fMRI) studies.[72]

Carp found that many of the studies didn't report on critical methodological details about experimental design, data acquisition or analysis, and many studies were underpowered. Data collection and analysis methods were highly flexible. The researchers had used 32 unique software packages, and there were nearly as many unique analysis pipelines as there were studies. Carp 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 of the studies reported the number of people rejected from analysis and the reasons for rejection, and the median sample size per group was only 15, which generates an enormous risk of selective publication of those results that happened to agree with the investigators' prejudices. The order of processing procedures also permits substantial flexibility in the analyses.

Replication is essential for the trustworthiness of science, and scientific papers must report experimental procedures in sufficient detail that allows independent investigators to reproduce the experiments. This is far from the case in imaging studies.[72]

Carp published another important study in 2012.[73] He sought to estimate the flexibility of neuroimaging analysis by submitting a single fMRI experiment to the many unique analysis procedures described in the literature. Considering all possible combinations of these strategies, he came up with 6,912 unique analysis pipelines.

"Nearly every voxel in the brain showed significant activation under at least one analysis pipeline. In other words, a sufficiently persistent researcher determined to find significant activation in virtually any brain region is quite likely to succeed. By the same token, no voxels were significantly activated across all pipelines. Thus, a researcher who hopes not to find any activation in a particular region (e.g., to rebut a competing hypothesis) can surely find a methodological strategy that will yield the desired null result ... Selective analysis reporting may occur without the intention or even the awareness of the investigator. For example, if the results of a new experiment do not concord with prior studies, researchers may adjust analysis parameters until the 'correct' results are observed."

In a multiple observer study published in 2020, the researchers had asked 70 independent teams to analyse the same dataset, testing the same 9 ex-ante hypotheses.[74] The dataset included fMRI data from 108 individuals, each performing one of two versions of a task that was previously used to study decision-making under risk. The teams were asked whether each hypothesis was supported based on a whole-brain-corrected analysis (yes or no). On average across the 9 hypotheses, 20% of teams reported a result that differed from most teams, which was midway

between complete consistency across teams and completely random results. This study demonstrated that analytical choices have a major effect on the reported results.

In 2021, researchers reported that after they cautioned in 2016 that there are so many sources or error in imaging studies that findings should not be considered definitive but only suggestive, 24 MRI studies had appeared in *JAMA Psychiatry* and 22 in the *American Journal of Psychiatry* describing differences in such scans in samples of psychiatric patients.[75] All 46 studies concluded that their findings are evidence of changes in brain structure.

In 2022, other researchers used three of the largest neuroimaging datasets available including a total of around 50,000 individuals to quantify brain-wide association studies' (BWAS) effect sizes and reproducibility as a function of sample size.[76] The median sample size was only 23 people. The researchers found that BWAS reproducibility requires samples with thousands of people.

As a commentator wrote, the study showed that almost every person diagnosed with depression will have the same brain connectivity as someone without the diagnosis, and almost every person diagnosed with ADHD will have the same brain volume as someone without ADHD.[77] Yet, in the small studies, correlations were almost always greater than 0.2 and sometimes much larger, which, as the researchers wrote, should not be believed.

The conventional method for dealing with this problem is to increase the threshold for statistical significance. However, this will backfire in these small MRI studies because it inadvertently ensures that only the largest - and thus the least likely to be true - brain differences end up passing the significance test and being published.

The experience of the Editor-in-Chief of *Molecular Brain* is also relevant to consider when assessing the merits of brain scanning studies in psychiatry. In 2020, he described what happened when he requested to see the raw data in 41 of the 180 manuscripts he had handled.[78] Upon his requests, 21 of the 41 manuscripts were withdrawn by the authors, and he rejected a further 19 "because of insufficient raw data," which suggested that the raw data might not exist, at least for some of the cases. Thus, only 1 of 41 papers (2%) passed his reasonable test.

Unfortunately, brain scan studies have a psychological component. People are more prone to believe what they do not understand, which means that the more the result is imbedded in unintelligible but seemingly advanced statistics, the more likely it is that the readers will believe it.

Researchers have coined the term "seductive allure of neuroscience explanations" (SANE), which is a real phenomenon. Several studies have shown that people show greater trust in studies with neuroscience language and graphs, especially if there are brain images.[79,80]

4 Are psychiatric disorders caused by a chemical imbalance?

When I lecture for psychiatric patients, half or more say they have been told by their doctors that they are ill because they have a chemical imbalance in their brain.

My colleagues who work with the patients therapeutically have the same experience. But when confronted with this, leading psychiatrists are quick to deny that any psychiatrist ever said this to anyone, or they say they abandoned the idea decades ago. This is not correct. Even today, hospital based psychiatry in one of the five regions in Denmark mentions it on its home-page:[81]

“Schizophrenia is a disorder in the brain … People with schizophrenia have disorders in certain areas of the brain where the neurotransmitter dopamine is active. Other disturbances in the brain are also seen."

“Antidepressant medication acts on some of the chemical processes that are out of balance in the brain in depression. The medication normalises, among other things, the level of the stress hormone cortisol and the brain's neurotransmitters serotonin and norepinephrine.”

"Affective disorders are mental illnesses related to a chemical imbalance in the brain. It leads to mental health problems like depression, mania or a combination of both."

"Scans have shown that people with ADHD have changes in several places in the brain ... in the area that is responsible for planning, impulse control and attention. The cells of the brain use different neurotransmitters to communicate with each other. If you have ADHD, you will see disturbances in these substances ... the levels of the neurotransmitters dopamine and norepinephrine are low. Medical treatment of ADHD increases the amount of the two neurotransmitters in the brain. It improves brain function."

“The medicine acts on some of the chemical processes in the brain related to anxiety disorder ... antidepressant medication normalises the amount of the brain's neurotransmitter serotonin.”

The text about ADHD was particularly misleading. It indicated that we know exactly where in the brain the problems are and that they can be fixed like a key fits into a lock.

The drug industry also propagates the false narrative. A 2007 survey of US university students found that 92% had seen or heard that depression is caused by a chemical imbalance in the brain, and 89% of these had seen it on TV.[82] TV channels in USA are full of drug ads, also for prescription drugs, and this indoctrination is very effective.

Schizophrenia and related disorders

The information in the textbooks was often very detailed: The abnormalities in psychosis include changes in neurotransmission and hormonal signals;[18:27] they include neuron migration and synapse formation, which in turn lead to structural and functional changes in the brain, including enlarged ventral ventricles, as an expression of atrophy;[18:94] PET scans found dysfunction in the prefrontal cortex and in the hippocampus;[18:94] PET and SPECT scans have shown increased dopamine synthesis and liberation in many psychotic patients, primarily located to the associative striatum (the head of the caudate nucleus);[16:562] and symptom complexes are well correlated to dysfunction of certain cerebral areas on PET scans.[18:90]

We are also told that there is pathology of the synapses,[19:228] and that the findings are robust that there is increased synthesis and liberation of dopamine in the associative striatum.[16:215]

However, one book noted that not all patients have changes in the dopamine system.[16:221] This speaks against the hypothesis that people become psychotic because they have too much dopamine in their brains, and the truth is that it has never been documented that any of the large psychiatric diseases is caused by a biochemical defect in the brain. Furthermore, there is no biological test that can tell us whether someone has a particular mental disorder.

The dopamine hypothesis has been accepted as the basis for using psychosis drugs,[18:17] but it is the other way around. Psychosis drugs decrease dopamine and therefore the psychiatrists have claimed, heavily pushed by the drug industry, that the disease is caused by too much dopamine. They have published a huge array of poor studies that purportedly showed this. But the fact is that the studies that have claimed that a common mental disorder like psychosis or depression starts with a chemical imbalance in the brain are all unreliable.[7:247]

In 2003, the huge deception became too much for six psychiatric survivors. They were so angry about the stories they had been told by their psychiatrists that they 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 stories the public had been told about mental disorders were true.[5:331]

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." In his book, *The art of always being right*, philosopher Arthur Schopenhauer calls this deplorable trick "Postulate what has to be proven."[83]

The hunger strike ended when people started getting health problems, but the Association bluffed. 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."

Schopenhauer says about this trick: "If you are being worsted, you can make a diversion - that is, you can suddenly begin to talk of something else, as though it had a bearing on the matter in dispute and afforded an argument against your opponent … it is a piece of impudence if it has nothing to do with the case, and is only brought in by way of attacking your opponent."

This is one of many examples that psychiatry is more of a religion than a science. Religious leaders 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."

It is important to realise that a difference in dopamine levels between patients with a schizophrenia diagnosis and healthy people – even if it existed - cannot tell us anything about what started the psychosis.

If a house burns down and we find ashes, it doesn't mean that it was the ashes that set the house on fire. Similarly, if a lion attacks us, we get terribly frightened and produce stress hormones, but this doesn't prove that it was the stress hormones that made us scared. It was the lion.

People with psychoses have often suffered traumatic experiences in the past, so we should see these traumas as contributing causal factors and not reduce suffering to some biochemical imbalance that, if it exists at all, is more likely to be the result of the psychosis than its cause.

One textbook[16:238] listed a study showing that 9 people at ultra-high risk of psychosis who later developed psychosis had greater dopamine synthesis capacity in the striatum, with a huge effect size of 1.18, than did 29 healthy volunteers.[84] There was a positive correlation between dopamine synthesis capacity and symptom severity, but such studies cannot tell us what starts a psychosis. These people were already ill (they had already seen the lion) when they were recruited for the study even though they did not yet formally fulfill the criteria for what constitutes a psychosis.

Affective disorders

According to the textbooks, depressive conditions are associated with an influence on the hypothalamic-pituitary-adrenal cortex axis (HPA axis);[19:210] likely disturbances in the central nervous system and neurotransmitters;[17:357] and elevated cortisol.[17:357,18:122]

However, I also found alternative views. Three psychologists called it a hypothesis that depression should be due to a chemical imbalance - insufficient monoaminergic transmission - and that improvement was due to re-establishment of normal synaptic levels of serotonin and norepinephrine.[20:430] They noted, with references, that this does not agree with the observation that the effect comes after weeks of treatment, and that there are other reasons to consider the hypothesis insufficient.

The hypothesis that depressed patients lack serotonin has been convincingly rejected.[2,85,86] Some drugs that decrease serotonin, e.g. tianeptine, or does not increase serotonin, e.g. mirtazapine, also seem to work for depression,[2,5,87] and mice genetically depleted of brain serotonin are not depressed but behave like other mice.[88] Further, it would be difficult to explain why these drugs seem to work in social phobia, which is not considered a lack-of-serotonin disease.[86]

When I said in my lectures for psychiatrists and other doctors that many patients had been told they had a chemical imbalance, I was met with angry responses demanding that I documented my so-called allegations. My colleagues obviously didn't like to admit that they misinformed their patients. I referred to what patients, health professionals 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' testimonies.

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.

This organised denial is disturbing. In a Danish study of 493 depressed or bipolar patients from 2005, 80% agreed with the sentence: "Antidepressants correct the changes that occurred in my brain due to stress or problems."[89]

The myth about a chemical imbalance in the brain being the cause of depression and other psychiatric disorders won't go away. In 2018, my deputy director at the Institute for Scientific Freedom, Maryanne Demasi, and I collected information about depression from 39 popular websites in 10 countries (Australia, Canada, Denmark, Ireland, New Zealand, Norway, South Africa, Sweden, UK, and USA). We found that 29 websites (74%) attributed depression to a chemical imbalance or claimed that depression pills could fix or correct such an imbalance.[90]

The psychiatrists use this myth to convince their patients that they should continue taking drugs they would rather avoid because of their harmful effects. In 2013, the chairman of the

Danish Psychiatric Association, Thomas Middelboe, described the term chemical imbalance as a metaphor psychiatry had grasped to explain diseases whose causes are unknown.[91]

As illustrated above, cognitive dissonance also plays a role. In 2014, I debated with Poul Videbech – an editor of the textbook without references[18] - at a public meeting arranged by medical students. After I had documented that far too many people are in treatment with depression pills and had suggested that we tapered off the drugs, Videbech said, in front of 600 people including patients and their relatives: "Who would take insulin from a diabetic?"[7:249]

A year later, when I published my first book about psychiatry[7] and was interviewed in a newspaper,[92] Videbech said on the same page that he had known for 20 years that the theory of the chemical imbalance was too simple, and that it was outrageous that I had said that he and his colleagues still believed in it.

Well, the myth about the chemical imbalance is only a thing of the past when challenged. Psychiatry professor Birte Glenthøj was also interviewed and confirmed that the myth was alive and well: "We know from research that patients suffering from schizophrenia have, on average, increased formation and release of dopamine, and that this is linked to the development of the psychotic symptoms. Increased dopamine activity is also seen before patients are first given antipsychotic medication, so it has nothing to do with the medication."

In 2017, Videbech postulated again that when people are depressed, there is an imbalance in the brain.[93] Furthermore, he and another psychiatry professor, Lars Kessing, had written in their two contributions to the Handbook for Patients, which has official status in Denmark and is available on the Internet, that depression is caused by a chemical imbalance.[94,95]

I complained to the editor but got nowhere. Kessing and Videbech changed a few minor things and introduced new claims that made their articles even worse. I complained again, and again to no avail, and the misinformation about the chemical imbalance continued. In his update, Kessing added that, "it is known that antidepressant drugs stimulate the brain to make new nerve cells in certain areas." Videbech wrote the same, but there were no references. If this is correct, it means that depression pills are harmful to brain cells, as the brain forms new cells in response to a brain injury. This is well documented, for example for electroshock therapy and psychosis pills.[11]

Some leading psychiatrists, including Kessing,[89] consider their patients ignorant, but I must say that the level of ignorance among themselves about their own specialty is astounding. When a hypothesis has been rejected, again and again, no matter how much people have manipulated the research design and the data, it is time to bury it for good.

This won't happen. The chemical imbalance myth is not a question about science but about money, prestige, and guild interests. Can you imagine a cardiologist saying, "You have a chemical imbalance in your heart, so you need to take this drug for the rest of your life," when she doesn't have a clue what she is talking about?

The textbooks did not use the term chemical imbalance directly, but many statements were made about drugs correcting what was claimed to be over- or underproduction of chemical messengers in the brain.

The myth about the chemical imbalance might be the most harmful of the many myths in psychiatry. It tends to keep the patients locked in the role of passive receivers of harmful drugs for years or maybe for life. It is obviously more difficult for patients to opt out of drug therapy if they believe they get a drug that corrects something that is wrong with them. The patients often say that they are afraid of falling ill again if they stop taking their drug because of this myth.

In 2014, the American Psychiatric Association wrote on its website that "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."[7:276]

This is an amazing act of lying to the public. *All* of this is wrong, and healthy people can develop both numbness and mania and can become suicidal on depression pills.[2:179] Until January 2021, the website of the Association still claimed that psychiatric medications can help correct imbalances in brain chemistry.[96]

A 2022 article demonstrated the extent to which the psychiatrists still propagate the myth about chemical imbalances.[97] All six influential US and UK textbooks published from 1990 to 2010 that the authors examined supported the theory, at least in some sections, and devoted substantial coverage to it, and most of 30 highly cited reviews of the aetiology of depression supported it, as did most of 30 research papers on the serotonin system.

ADHD

The textbooks noted that the psychopathological development in ADHD is assumed to involve epigenetic changes and early acquired biochemical and hormonal dysregulation;[19:52] that a dysregulation of dopamine and noradrenaline in the brain is likely very important for the change in brain function;[19:113] and that disturbances of certain areas of the cortex and basal ganglia are in areas mainly controlled by dopamine.[18:229] None of this can be substantiated.

Anxiety disorders

A textbook mentioned that serotonin is important for the pathogenesis of OCD.[19:162] There were no references, but this has never been shown to be correct.

Inflammation, one of the latest fads in psychiatry

Inflammation is one of the latest fads in psychiatry.[7:289] A textbook noted the role of inflammation for the development of depression but did not explain what the significance of this was.[17:911]

Two of the editors of one of the textbooks[16] co-authored a 2014 systematic review of 14 trials of celecoxib, a so-called non-steroidal anti-inflammatory drug (NSAID), that showed an effect on depression, with an effect size of 0.34.[98]

However, many of the patients had arthritis.[98] It is not surprising that painkillers might seem to reduce the depression. Even if we ignore this, and tentatively assume that NSAIDs have an effect on depression, the effect size of 0.34 is so small that it is not clinically relevant (see Chapter 8).

There is another, little known reason why the meta-analysis cannot document that inflammation plays a role in depression. It is that, despite their name, non-steroidal, anti-inflammatory drugs do not have anti-inflammatory effects.

When the newly synthesised cortisone was first given to 14 patients with rheumatoid arthritis in 1948 at the Mayo Clinic in Rochester, Minnesota, the effect was miraculous.[99] The results were so striking that some people believed a cure for rheumatoid arthritis had been discovered, but the serious harms of corticosteroids quickly dampened the enthusiasm.

By calling the new pain-killers non-steroidal, anti-inflammatory drugs, the companies created the illusion that their effect was similar to that of steroids but without their serious harms. This marketing trick was highly effective and NSAIDs are used so much that they are one of the most important reasons why our prescription drugs are the third leading cause of death, after heart disease and cancer.[46:8]

I have asked many rheumatologists about the documentation that the drugs are anti-inflammatory but I received no useful answers. I therefore studied the issue myself.

With orthopaedic surgeons, I did a placebo-controlled trial in 173 patients with acute ankle distortions where we measured the oedema by volumetry, using the healthy foot as control for the displaced amount of water.[100] Using a factorial design, we randomised the patients twice: To a group that was instructed to immobilise the foot and was given crutches and to a group that was instructed to walk as normally as possible despite the pain; and to naproxen and placebo.

Mobilisation quickly reduced the oedema. After 2-4 days, the volume difference was 42 mL when the patients were mobilised compared to using crutches (P = 0.01). In contrast, there was no significant effect of naproxen (P = 0.42; difference 11 mL compared with placebo). Thus, mobilisation was anti-inflammatory, which naproxen wasn't, and it also led to much faster recovery.

The minor non-significant effect of naproxen could be real and simply a consequence of the drug's effect on pain, which would increase mobilisation. The company selling naproxen, Astra-Syntex, had provided the blinded trial medication but did not like our results, which were bad for marketing. Its statistician ensured that the most important results did not get published and that the trial report was unintelligible gobbledygook for the average doctor. But I spared a copy of the statistical report, which is why I am able to tell the true story.

I also did a meta-analysis of the placebo-controlled trials of NSAIDs. The drugs did not reduce the swelling of finger joints measured by jeweller rings in patients with rheumatoid arthritis.[101]

We should not treat depression with NSAIDs, some of the deadliest drugs we have.[6:155]

5 Psychiatric diagnoses are not reliable

> People are unlikely to question the underlying premises of their occupations, in which they often have a large financial and emotional stake.
>
> Judi Chamberlin, former mental patient[102]

In the protocol for my study of psychiatric textbooks, I noted that they should mention that psychiatric diagnoses are based on arbitrary criteria; that there is large interobserver variation when several psychiatrists assess the same patients independently; that psychiatric disorders can disappear again, without treatment; that psychiatrists are willing to change their diagnoses; and that patients can get their diagnoses removed based on a second opinion or longer follow-up.

I also noted that clinicians should not come up with additional diagnoses in people who receive psychoactive drugs because their adverse effects may mimic the criteria used for other diagnoses. It is therefore often impossible to say which is which, e.g. if a patient in treatment for depression or ADHD also come to suffer from bipolar disorder or if the observed symptoms are merely adverse effects of the drugs.[7,8]

Psychiatrists usually ignore this fundamental problem and may even say that the drug treatment has "unmasked" the new disorder, which is one of the reasons why contact with the psychiatric system often leads to several diagnoses and polypharmacy and why temporary problems with mental health often become chronic.

There was very little in the textbooks that even just hinted at any of these essential issues. One book noted that the psychiatrists had tried to make the diagnoses reliable and to ensure that the doctors agreed on how to use them.[18:24] But it did not explain that psychiatric diagnoses are highly unreliable and did not quote any studies on observer variability.

On the same page, this textbook noted that the diagnosis is affirmed or rejected based on the course of the disease and treatment results.[18:24] There are two obvious problems with this statement. First, the reality is that it is not possible to have an erroneous diagnosis removed. Numerous patients have tried and have been rejected. Second, it is circular evidence. If we give everyone a diagnosis of schizophrenia, and some become better when treated with psychosis pills, the diagnosis is confirmed for these patients and rejected for the rest. If we say it might rain tomorrow and it might not, and then let the "course of the weather" decide what was right, this doesn't prove anything about our capabilities as a meteorologist.

Further ahead, this textbook noted about the diagnostic criteria for depression that they are symptoms most people experience now and then: sadness, difficulty concentrating, sleep problems, etc., but that the important thing is, firstly, that the symptoms must exceed a certain clinical threshold before they can be considered a disorder, which requires clinical experience to determine; secondly, that they must have been present for more than 14 days.[18:119]

This boils down to psychiatrists' best friend, clinical experience, which is not reassuring for the patients they label and stigmatise for life with their diagnoses, which are often wrong.[7] If you are a patient, how do you object to a psychiatrist's clinical experience? You are bound to lose, with three arguments: You are not a psychiatrist; you do not have clinical experience; and since you have a mental health disorder, you might not be able to think clearly about yourself.

It is problematic to use a diagnosis like depression to explain an experience.[10:14] If I was asked why someone is feeling low and I answered that this is because she has depression, then a

legitimate question to ask is: "How do you know that this feeling low is caused by depression?" The only answer I can give is that I know it is depression because she is feeling low. If we try to use a classification that can only *describe* in order to *explain*, we end up with a tautology or circular thinking. A description cannot explain itself. Low mood and depression are synonymous; we cannot use one to explain the other.

The American Psychiatric Association proclaimed in 2021 that major depressive disorder is a common and serious medical illness that negatively affects how you feel, the way you think and how you act.[96] This is wrong. The Association has blown life into something that is just a name and therefore cannot cause anything. This is a very common error in psychiatry.

As the diagnostic criteria have been lowered, it is not surprising that studies have shown that more people are overdiagnosed than underdiagnosed for depression.[103] The term "major depressive disorder" has become contradictory in terms, as it now includes cases of mild depression even though such cases are neither major, nor depression, not even a disorder.[103]

One textbook described agitated depression, with hand-twisting restlessness, inner turmoil or persistent pacing around, and said that as the patients are unable to find rest, they are often highly suicidal.[18:119] What the authors described are the key symptoms of akathisia, one of the most dangerous harms of psychosis and depression pills. Akathisia is a state of extreme restlessness and inner turmoil. It literally means that you can't sit still. You may have the urge to tap your fingers, fidget, or jiggle your legs.

But the authors did not tell their readers about this or say how one may distinguish between the two conditions, which seems close to impossible. Is this also a matter of clinical experience? I am not joking. In 2015, I was invited to lecture at a hospital in Denmark by the psychiatric organisation in that region.[8-18] Rasmus Licht, professor of psychiatry and a specialist in bipolar disorder, also lectured. I asked him how he could know, when he made the diagnosis bipolar in a patient who received a drug for ADHD, that it was not just the drug harms he saw because they are very similar to the symptoms doctors use when diagnosing bipolar.

I was flabbergasted when he said that a psychiatrist was able to distinguish between these two possibilities.

Rasmus said a lot else that wasn't correct, which illustrated what psychiatry does to its own people. When I first met him, he was a bright young man who impressed me. I was one of the examiners when he defended his PhD about mania 17 years earlier and hadn't seen him in all those years. It was shocking to watch how he had assimilated psychiatry's wrong ideas. We corresponded a little afterwards, but I could not convince him he was wrong.

One of the things Rasmus wrote was that "it is mentioned in ICD-10 [ICD is WHO's International Classification of Diseases] and DSM-IV that if the mania only occurs when the patient has received an antidepressant at the same time, it speaks against bipolar disorder, as it is understood it could be drug induced mania. However, in contrast, the DSM-5 has taken the consequences of recent epidemiological studies and written that, even though a mania occurs during treatment with an antidepressant, this should be perceived as a true, i.e. primary, bipolar disorder. So, in this case, you speak against better knowledge.".

I wondered how it was possible for Rasmus to believe in such nonsense. It is total baloney to postulate that a mania that occurs during treatment with a depression pill is a new disorder when it might as well be a drug harm. It is a smart trick psychiatrists use to distance themselves from the harms they cause and from their accountability. It is always the patient who is to blame, never them or their drugs, is the message they send, also in their textbooks.

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 tentatively call it a drug-induced disorder.

In one of my books, I describe a patient, Stine Toft, who has never been manic, apart from the time when she received a depression pill, but she also got the diagnosis bipolar.[8:5] What psychiatry did to her was devastating, yet so typical, that I published her story on the Mad in America website.[104]

Stine was seriously harmed. She was told her condition would definitely last for the rest of her life; she was treated with depression pills, antiepileptics and a psychosis pill; put on 50 kg; lost about 14 years of her life to psychiatry; lost her husband; came close to suicide; and came on disability pension.

Stine's next husband saved her. He asked quite quickly "what the sickness was all about," because he couldn't see it. After a year and a half, she surrendered and agreed to withdraw the medication. She suffered an excruciating withdrawal phase because she did not receive the necessary guidance. It took two and a half years. This was when she came to know two of my books[7,46] and found out that everything she had experienced was well known and perfectly normal. It was shocking to her to read about how it is normal practice to be exposed to the hell she had been through, but also liberating to discover that she wasn't sick and that there was nothing wrong with her.

Stine is doing well today. She became a coach and a psychotherapist and has helped many patients taper off their depression pills, with great success. She no longer sees her family. They maintained the claim that she was ill and just needed to take her medication. Stine lectures but finds it difficult to get the message out. She has lectured for Psychiatry in the Capital Region about being bipolar, which was easy. People like to see a sick person and hear her story. But a psychiatric survivor's success story that calls the whole system into question is not considered interesting.

A special case of erroneous diagnoses is post-mortem diagnoses. Two textbooks claimed that 50% of suicides occur in people who are depressed,[17:358,18:129] and a third that by far most people who killed themselves had a treatment demanding psychiatric disorder.[16:534] However, a post-mortem diagnosis is highly bias-prone. 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. Furthermore, a depression diagnosis is made by questioning the patient, and one cannot talk to a dead person.

One textbook, which had a psychologist as one of its two editors, was markedly different to the others. It quoted the Canadian physician William Osler (died 1919): "It is much more important to know what sort of a patient that has a disease then what sort of a disease a patient has."[17:34] It also noted that to put the human being at the centre is to organise mental health with respect for the individual's integrity and self-determination, and that, in an evidence-based clinical practice, treatment must be adapted to the individual's and the relatives' personal perceptions, feelings, and expectations and not only to the diagnosis and the often meagre evidence associated with it.

The authors wrote that, "In the book we will see the person behind the diagnosis."[17:35] Later, the book repeated that it is the patient's perception of himself and his world that is at the centre.[17:136] This view is radically different to that in the other textbooks where the patient is the

passive receiver of drugs and is reprimanded if he doesn't want to take the drugs by being called non-compliant or treatment resistant, or as lacking insight into his disease.

This book noted that there are strong economic interests behind the diagnosis of new conditions, e.g. the use of ADHD medication has increased dramatically, and the ADHD diagnosis is being used increasingly, also about things that are not deviant or constitute a disorder, e.g. difficulty concentrating, restlessness, motor restlessness and impulsivity in children.[17:51]

This is correct. ADHD is an American construct, and with each revision of the DSM, a larger number of children are found to be above the threshold for diagnosis.[10:33] ADHD is the product of vested commercial, political and institutional interests. Nowhere in the story of this diagnosis has there been any significant scientific discovery.[10:35] It is the Emperor's new clothes.

The book mentioned that studies show that the youngest boy in the class has about a 30% greater risk of getting an ADHD diagnosis than the other children.[17:51] It is actually worse than 30%. As noted above, 50% more of children born in December were in drug treatment for ADHD than those born in January in the same class.[51]

The book noted that psychiatric diagnoses have poor validity and do not tell us much about the nature, course and treatment of the diseases.[17:212] The reliability of the diagnoses was also questioned: Will clinicians reach the same diagnosis? Both yes and no.[17:214] The diagnostic criteria are arbitrary, and there is great aversion in the population against psychiatry´s use of diagnoses, which are more stigmatising than they are a help for the doctor.[17:215]

This scepticism was repeated 703 pages later, in a chapter about psychiatry's history:[17:918] Can we trust the diagnoses and what do they really tell us about the patients' diseases? The anti-psychiatry movement after 1968 was in particular directed against the diagnoses, which were considered unreliable, stigmatising and alienating: "Who is it that are mad?"

Much of the book was traditional and full of errors. But the chapter about the history of psychiatry in Denmark was so bold that I suspected that the authors must be retired psychiatrists or close to retiring, or from another profession. I was right. Only one of the three authors was a psychiatrist, born in 1949. The other two were a psychologist and a medical historian.

The authors explained that slogans such as "the patient is an expert in his own life" have challenged psychiatry's traditional paternalistic attitude, and that the recovery movement – with the basic attitude that patients can recover and return to life outside the treatment system, like patients who are treated for non-psychiatric diseases - has been particularly important in social psychiatry.[17:910]

The authors even noted that there is no relation between the available evidence, national clinical guidelines and the content of treatment packages, and that psychiatry's image remains under pressure due to cases of overmedication and too much coercion.[17:919]

They ended their chapter by saying that one of the biggest problems is the high mortality among psychiatric patients.[17:920] This was not discussed in the other books or mentioned in the main text of this book, but by the end of a huge book, under the heading, *The history of psychiatry in Denmark,* starting on page 910. Students are very results oriented and prioritise what they read. Few students will ever read these 23 pages, even though they are among the most important ones in all five textbooks.

Two concepts are essential when discussing diagnostic tests, their validity and reliability. The validity of a diagnostic test refers to its ability to measure what it is purported to measure, which involves its ability to distinguish between people with and without a particular disease.

The two principal measures of test validity are sensitivity and specificity, which are the proportions of those who are ill that test positive, and the proportion of those who are healthy that test negative, respectively. Most people believe that the predictability of positive and negative diagnostic tests are constants, which they are not, as they depend on the prevalence of the disease that is being tested for.[105] The more uncommon a disease is, the more false positives will there be. This is why screening for depression is a bad idea. The screening test for depression recommended by the WHO is so poor that for every 100 healthy people screened, 36 will get a false diagnosis of depression.[7:46,106,107]

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.[108] This is expected, as about 90% of prescriptions for depression pills are written by general practitioners,[7:256,108,109] and they don't have much time.

The reliability of a diagnostic test depends on the accuracy and reproducibility of the test results. The accuracy is defined by comparing the test results with a final true diagnosis. There is no such final true diagnosis in psychiatry, and it is therefore not possible to determine the accuracy of a diagnostic test. But its reproducibility can be determined in observer variation studies where two or more psychiatrists suggest a diagnosis for the same patients.

Four of the five textbooks did not mention a single result from observer variation studies and gave the erroneous impression that psychiatric diagnoses are both valid and reliable. With rare exceptions, e.g. the admission that no questionnaires for diagnosing adult ADHD have been validated,[17:615] diagnoses were not doubted. One book claimed that the reliability of the diagnoses is good and noted that, to ensure that the criteria based diagnoses are sufficiently reliable, they were tested before usage in a big international study, and diagnoses that showed low reliability were either removed or the criteria were strengthened.[16:23]

It is obscure what the authors referred to, but what they wrote is wrong.[7:32,110,111] As one commentator put it after the appearance of DSM-5: "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."[112]

One of the books showed a figure demonstrating that the number of patients diagnosed with schizophrenia in Denmark had quadrupled from 2000 in 1971 to 7400 in 2010.[19:225] This should have woken up the authors but they did not comment on this stunning finding, even though something must be wrong with the validity of the diagnosis. On revision, this diagnosis can often not be sustained, e.g. it was rejected in 64% of 1023 people.[1:173]

A psychiatrist wrote to me:[7:360] In my twenties, I had a massive breakdown. At the time, I instinctively resisted all psychiatric labels and medical treatments. 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.

Diagnoses stick to the patient. Once made, everything the patient does or says during a hospital admission becomes suspect, as the patient is under observation, which means that the initial, perhaps tentative diagnosis, all too easily becomes a self-fulfilling prophecy.[7:30] The doctor's intuition and experience may suggest very quickly what the problem is, 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.

There is much overlap between the different diagnostic categories, often called high comorbidity, although the problem is not that the patient has several "diseases" but that the diseases are so vaguely defined, with overlapping symptoms, that many patients can get several diagnoses.

Even the book that was critical of psychiatric diagnoses, failed when it came to observer variation studies. When the two authors, a psychologist and a psychiatrist, discussed the validity and reliability of the diagnoses, they mentioned kappa, Cronbach's alpha, Hamilton's Depression Scale and a wealth of other scales and issues over 25 pages, but not a single result from inter-observer variation studies.[17:165]

Kappa values measure to which extent two observers agree beyond chance. If kappa is 0.60, it means that the agreement is only 60% of the difference between chance agreement and perfect agreement, which is pretty poor. There are many problems with kappa.[105] It presents statistical problems and does not tell us, for example, if the disagreement is important, which it surely is for psychiatric diagnoses because a diagnosis almost always lead to drugs, often for many years without interruption,[113,114] and also often to a downhill chronic course for the patient.[5:8,119:24]

The claim that the new diagnostic checklist system introduced by the American Psychiatric Association for its Diagnostic and Statistical Manual of Mental Disorders (DSM-III) in 1980 is reliable has been convincingly refuted in a book.[7:32,102,110] The disappointing results when two psychiatrists assess 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 two people did the work, which was a huge task.[110] Even the largest study, of 592 people, was disappointing despite the fact that the investigators took great care in training the assessors.[111] For bulimia nervosa, which is very 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 kappa values were only 0.64 and 0.65, respectively. This is frightening considering the devastating consequences of false positive diagnoses.

When researchers interviewed 463 people about 91 key symptoms for psychiatric disorders, 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.[1:168,115]

If the general population is exposed to just a few of the various diagnostic checklists that are being used, a large proportion will get one or more psychiatric diagnoses. When I lecture and try three diagnostic tests on the audience - for depression, ADHD and mania - about a quarter test positive for one or more diagnoses. Imagine if you tested people suspected of having cancer with a test that gave a quarter of them an erroneous cancer diagnosis. We wouldn't allow such a poor test to be used.

DSM-III from 1980 was replaced by DSM-IV in 1994, which was even worse than its predecessor and listed 26% more ways to be mentally ill. Allen Frances, chairman for the DSM-IV task force, has argued that the responsibility for defining psychiatric conditions needs to be taken away from the American Psychiatric Association because 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."[116] Frances noted that DSM-IV created three false epidemics because the diagnostic criteria were too wide: ADHD, autism and childhood bipolar disorder.

Psychiatric diagnoses are uncritically believed not only by psychiatrists but also by the media. Even websites that are critical towards overdiagnosis of diseases and overtreatment with drugs and advocate for a new biomedical and social model, convey information like, "One in four people in the world are prone to be affected by mental disorders at some point in life. These mental disorders are the leading causes of ill-health and disability worldwide."[117]

Several things are wrong with such commonly seen statements. First, many people are overdiagnosed. They do not suffer from a mental disorder but have problems in their lives. Second, they are not affected by a mental disorder. As already explained, to label people's problems does not create a being that attacks people. Third, mental disorders are not leading causes of ill health and disability. People suffering from deprivation, poverty, unemployment and abuse suffer ill health and disability; they are not attacked by some psychiatric monster.[96]

The bottom of journalism was reached when the United States established The Carter Center's Guide for Mental Health Journalism, which is the first of its kind.[8:162,118] This institution educates journalists to write flawed articles and to never question psychiatric diagnoses. Journalists should pin down exactly what a professional says is wrong with a patient and use that information to characterize a person's mental state. There is no encouragement for journalists to consider how people so diagnosed *see themselves*, or whether they accept their diagnostic label, or if the professional might be wrong.

According to the Carter Center, the DSM-5 is a reliable guide for making diagnoses. There is no mention of the fact that the diagnoses are arbitrary constructs created by consensus among a small group of psychiatrists, or that they lack validity, or that psychiatrists disagree a lot when asked to examine the same patients, or that most healthy people would get one or more diagnoses if tested enough.

Reporters are told to write that behavioural health conditions are common and that research into the causes of and treatments for these conditions has led to important discoveries over the past decade. They should also inform the public that prevention and intervention efforts – which mean drugs - are effective and helpful. This is the same message that the American Psychiatric Association and leading psychiatrists all over the world have been promoting for many years.

The guide prompts reporters to echo the message from the American Psychiatric Association that psychiatric conditions are often undiagnosed and undertreated, and that psychiatric treatment is effective. The guide avoids any discussion about how ineffective and harmful the drugs are and makes people believe that "treatment" also includes psychotherapy, even though this is rarely offered.

Nothing is mentioned about overdiagnosis. Reporters are not encouraged to explore why it is that the public health burden of mental disorders has grown dramatically in the past 35 years, at the same time as the use of psychiatric drugs has exploded.[5:8,119:24]

The guide states that between 70% and 90% of people with a mental health condition experience a significant reduction in symptoms and improvement in quality of life after receiving treatment. The source of this false information is the National Alliance on Mental Illness (NAMI), a corrupted patient organisation.[7:357] It is true that most people improve but that would have happened without treatment. Like many of the textbook authors, the Carter Center seems to have "forgotten" why we do placebo-controlled trials, and it has never been documented that psychiatric drugs improve quality of life; in fact, they worsen it (see Chapters 7 and 8).

Reporters are told to emphasize the positive and avoid focusing on the failures of psychiatric care. The guide does not provide any resources for obtaining the perspectives of people with lived experience, most of whom would speak critically of the conventional wisdom.

Unfortunately, the Carter Center is seen as a leader in training journalists on how to report on mental health. It encourages journalists to act as stenographers repeating conventional dogma.

It is difficult to see much hope for America. Journalists are told to convey the strongly misleading narratives created by the drug industry and US psychiatrists on industry payroll to the great harm of our patients and societies.[5-7]

It is very strange that there is such an institution in America. What the Carter Center does is like telling Chinese journalists that if they want to know what it is like for the Chinese people to live under a dictatorship, they should not ask the people but the Chinese leaders.

One book noted that a good rule of thumb is not to make a depression diagnosis in the first two weeks after stopping drug abuse or intake of medicine.[16:258]

This principle should apply to all patients. Diagnoses can make it difficult to get the education patients dream about, a job, certain pensions, to become approved for adoption, to get an insurance or child custody, or even just to keep a driver's licence.[120,121] Psychiatric diagnoses are often being abused in child custody cases when the parents get divorced.[120] Even when the diagnosis is obviously wrong and the psychiatrist herself seriously doubted it when she made it, it cannot be removed.[121] It sticks to the patients forever, as if they were branded cows.

Already on the next page, this book ventured in the opposite direction saying that older people are at risk for underdiagnosis of depression because relatives and sometimes the doctor accept and explain their sadness as understandable, based on the many losses of friends and perhaps the spouse and physical capacity.[16:259] The truth is the opposite. Old people are overdiagnosed to an unbelievable extent and sadness is a normal feeling, not a psychiatric diagnosis.

The book about child and adolescent psychiatry mentioned that diagnoses are designations for a condition, a kind of snapshot, and not designations for people.[19:36] It advises that diagnoses should be continuously assessed, re-evaluated and changed, and be considered dynamic tools with limited applicability outside of clinical and research contexts.[19:36]

This is brilliant, but why do psychiatrists not say the same about adults? They also change over time and a person in deep distress will not always be in deep distress. That person might be fine both before and after the visit to the doctor. Why is it then impossible to get a wrong diagnosis removed?

The authors warned that one must not indulge in uncritical use of diagnoses, e.g. they are often used as an admission ticket to social services. They claimed that if clinicians respect the limitations and scope of diagnoses and limit their use of diagnoses for administrative and official purposes, diagnoses do not in themselves imply a risk of stigmatisation.

This looks like a tautology. If diagnoses are used correctly, they do not lead to stigmatisation. If people are stigmatised, it is because diagnoses are not used correctly.

The reality is that diagnoses are *not* being used correctly, which leads to a lot of stigmatisation and misery.[7,8] Think about other issues. If people drove correctly, there would be no traffic deaths. If people drank alcohol correctly, there would be no alcoholics. If people ate correctly, no one would be overweight. What does this tell us? Nothing.

I shall end this chapter by praising Australian psychiatrist Niall McLaren whom I have met several times. He has written a very instructive book with many patient stories telling us that anxiety is a key symptom in psychiatry.[9] If a psychiatrist or family doctor doesn't take a very careful history, they might miss that the current episode of distress, which they diagnose as depression, started as anxiety many years earlier when the patient was a teenager. They should therefore have dealt with the anxiety with talk therapy instead of handing out pills.

Niall has developed a standard way with which he approaches all new patients in order not to overlook anything important. It takes time, but the time invested initially pays back many times over and leads to better outcomes for his patients than the standard approach in psychiatry.

Niall explains that "the value of biological psychiatry is that it isn't necessary to talk to a patient beyond asking a few standard questions to work out which disease he has, and that can easily be done by a nurse armed with a questionnaire. This will give a diagnosis which then dictates the drugs he should have." Sarcastic? Yes. True? Yes.

It doesn't seem to matter whether a diagnosis is correct or wrong. It will follow you for the rest of your life.

6 Psychiatric drug trials are not reliable

> There is probably no other area of medicine in which the academic literature is so at odds with the raw data.
>
> David Healy, professor of psychiatry[122]

> I have spent most of my professional life evaluating the quality of clinical research, and I believe it is especially poor in psychiatry. The industry-sponsored studies … are selectively published, tend to be short-term, designed to favor the drug, and show benefits so small that they are unlikely to outweigh the long-term harms.
>
> Marcia Angell, former editor, *New England Journal of Medicine*[123]

Before discussing the effect of psychiatric drugs, we need to realise that most placebo-controlled trials and head-to-head comparisons of two active drugs are heavily biased.[7,8] As an example, significantly more patients improved on fluoxetine when fluoxetine was the drug of interest than in trials where fluoxetine was the comparator drug.[124]

There are eight major reasons why placebo-controlled trials of psychiatric drugs are flawed, and most of them apply also to head-to-head trials.

By far most drug trials run for only a few weeks even though most patients are treated for many years. This is not evidence-based medicine, and drug effects can change over time due to development of tolerance.

Events after the trial stopped are ignored. The main effect of this is that the harms are underestimated (see Chapters 7 and 8).

By far most trials are conducted by the drug industry, which often manipulates with the design, analysis and reporting.[6,7]

About half of the deaths and half of the suicides occurring in trials of psychiatric drugs have been left out in published trial reports.[125]

The four additional reasons are the use of rating scales, lack of effective blinding, withdrawal effects in the placebo group, and manipulated data analyses and selective reporting.

Rating scales

Rating scales are used for measuring the reduction of symptoms. They do not say anything about whether the patients have been cured or can live a reasonably normal life.

The scores on such scales may easily improve even if the patients have not been helped, e.g. when someone is knocked down by a major tranquilliser (a psychosis drug) and expresses abnormal ideas less frequently.[4]

The effect of depression pills is measured on rating scales, e.g. the Hamilton Depression Rating Scale,[126] which contains items that are not specific to depression, including sleeping difficulties, anxiety, agitation, and somatic complaints. These symptoms are likely to respond to the non-specific sedative effects that occur not only with many depression pills but also with other substances, e.g. alcohol, opioids, psychosis pills and benzodiazepines, but we do not prescribe alcohol, opioids, or benzodiazepines for people with depression or call them depression pills.

Using the Hamilton scale, even stimulants like cocaine, Ecstasy, amphetamine and other ADHD drugs could be considered depression pills. Almost everything could. Indeed, many drugs that are not considered to be depression pills show comparable effects to them, e.g. benzodiazepines, opiates, buspirone, stimulants, reserpine, and other psychosis pills.[24]

Lack of effective blinding

Because of the conspicuous adverse effects of psychiatric drugs, placebo-controlled trials labeled double-blind are not double-blind. It takes very little unblinding before the small differences recorded can be explained by bias in the outcome assessment on a subjective rating scale.[7:51]

The blinding is broken for many patients in these trials, in some cases for all of them, as in a trial of alprazolam versus placebo.[127] Researchers had reviewed the blinding problems and 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."[127] This was in 1993, but the psychiatrists and the drug industry ignore this fundamental flaw in their research because it is useful for them to pretend the problem doesn't exist and that what they measure are true and beneficial drug effects.

The unblinding is a major reason why it is so much easier to invent new diseases than to invent new drugs.[128,129] When my research group reviewed the type of diagnoses investigated in placebo-controlled trials of depression pills, we counted 214 unique diagnoses, in addition to depression and anxiety.[130] The trials were driven by commercial interests, focusing on prevalent diseases and everyday problems to such an extent that no one can live a full life without experiencing several of the problems for which these drugs were tested. We concluded that depression pills are the modern version of Aldous Huxley's soma pill intended to keep everyone happy in *Brave New World*.

In 2001, Lundbeck's American partner Forest had performed a trial of citalopram for compulsive shopping disorder.[131] Another Lundbeck drug, escitalopram, reduced the daily incidence of hot flushes in menopausal women from 10 to 9.[132] This also looked like a joke but the trial was published in a US flagship journal, *JAMA*.

Helped by the lack of blinding, drug companies can show that their drugs "works" for virtually everything. Just think about the variety of drugs that are claimed to work for depression and schizophrenia.

Healthy volunteers who pretend they are ill can help, too. Some patients participate in depression trials without being depressed just to cash the money, as a healthy person told a doctor on a train ride:[133] "I'm not depressed … the trials are advertised … For a 20 day trial that's £2000 … it's nice to see your regular friends." When the fake volunteers notice they are on active drug because of its adverse effects, they can fake some improvement.

Withdrawal effects in the placebo group

The patients recruited for placebo-controlled trials are virtually always on a drug like the one being tested. This is because such patients are much easier to find than patients who are not on drugs.

After a short wash-out period without this drug, the patients are randomised to the new drug or placebo. The patients are likely to be those who have not reacted too negatively on getting such a drug before,[24] which means that the trials will underestimate the harms of the tested drug.

The patients might also react more negatively to placebo, e.g. because they miss the sedation or euphoria these drugs may cause (see, for example, the package insert for olanzapine[134]).

Some patients get withdrawal symptoms that are misinterpreted as a relapse of the disease because the symptoms can be the same as those that define a relapse. Introducing longer wash-out periods does not remove this cold turkey problem. If people have been permanently brain damaged before entering the trials, wash-out periods cannot compensate, and the symptoms that have been masked by ongoing treatment, e.g. tardive dyskinesia, might reappear. Even if that is not the case, the patients might suffer from withdrawal symptoms for months or years.[11,135,136]

Manipulated data analyses and selective reporting

When Joshua Carp criticised the brain imaging studies (see Chapter 3) noting that when analytic flexibility is high, investigators may elect to use methods that yield favourable outcomes and discard methods that yield null results,[73] he cited a study carried out by my research group.

We compared protocols for randomised trials we had obtained from ethics review committees with the trial publications.[137] Two-thirds of the trials had at least one primary outcome that was changed, introduced, or omitted while 86% of the trialists denied the existence of unreported outcomes (they did not know, of course, that we had access to their protocols when we asked). These serious manipulations were not described in any of the 51 publications.

This was the first time this phenomenon had been shown to be common, in a consecutive cohort of trials. Other attempts at getting access to trial protocols had failed, but I succeeded to get access in Denmark by guaranteeing that we would not describe the individual trials in our publication.

What we uncovered is known as the Texas sharpshooter trick. You fire a gun towards a target but miss it. Next, you wipe out your target and draw a new one around your bullet hole and present this to the public. You hit the bull's eye by committing fraud.

Based on our data, we did another study that is also relevant to know about when judging the trustworthiness of published trial reports.[138] In those 44 trials that were industry-initiated, the sponsor had access to accumulating data during 16 trials through interim analyses and participation in data and safety monitoring committees, but such access was disclosed in only one corresponding trial article. An additional 16 protocols noted that the sponsor had the right to stop the trial at any time, for any reason, which was not noted in any of the publications. The sponsor therefore had potential control over a trial in progress in 32 (73%) of these studies.

When the sponsor can peep repeatedly at the data as they accumulate, there is a risk that the trial will be stopped when it is favourable. Trials reported as having stopped early for benefit exaggerated the effect by 39% on average compared to trials of the same intervention that had not stopped early.[139]

We also found out that constraints on the publication rights were described in half of the protocols, which noted that the sponsor either owned the data, needed to approve the manuscript, or both. None of the constraints were stated in any of the publications.

Ghost authorship is also an important issue. It is the failure to name, as an author, an individual who has made a substantial contribution to an article. We found that none of the 44 protocols stated that clinical investigators were to be involved with data analysis.[140] There was evidence of ghost authorship for 33 trials (75%), which increased to 91% when we included cases where a person qualifying for authorship was acknowledged rather than appearing as an author. In 31

trials, the ghost authors we identified were statisticians. We likely overlooked some ghost authors, as we had very limited information to identify the possible omission of other people who would have qualified as authors.

A study by David Healy of papers on sertraline (Zoloft, Pfizer) showed that in a 3-year period, 55 papers had been written by a medical writing agency whereas only 41 papers had been written by other people.[141] Only two of the 55 papers acknowledged writing support from people not listed as authors, and all results were favourable for Pfizer. Healy has described how frank some companies are towards doctors: "We have had our ghostwriter produce a first draft based on your published work. I attach it here." When Healy was unhappy with the glowing review of a drug and suggested changes, the company replied that he had missed some "commercially important" points and published the paper in another academic's name.[142]

What we uncovered based on the protocols was new and it shocked the international research community. There were many comments about our results in scientific journals and the media.

I have been a kind of research detective all my life, and I once participated in a team with Richard Smith, Editor-in-Chief of the *BMJ*, that investigated a case of fraud in a trial committed by a researcher in Asia.

Fraud is much more common than people realise.[27] In 2021, Smith wrote about research fraud in the article, *Time to assume that health research is fraudulent until proven otherwise?*[143] He mentioned that a colleague had informed anaesthesiologist Ian Roberts that none of the trials he had included in a systematic review showing that mannitol halved deaths from head injury existed. All of them had a lead author who purported to come from an institution that didn't exist. The trials were published in prestigious neurosurgery journals and had multiple co-authors, some of whom didn't know they were authors until after the fake trials were published. When Roberts contacted one of the journals, the editor responded that, "I wouldn't trust the data." Roberts wondered why he then published the trial. None of the trials have been retracted.

Also in 2021, an analysis of individual patient data in 153 randomised trials submitted to *Anaesthesia* showed that 44% had untrustworthy data and 26% were fatally flawed, i.e. 70% were garbage.[144] When individual patient data were not available, it was more difficult to detect scientific misconduct, and now "only" 22% was garbage.

It is clear that we cannot take science on trust but must investigate every time if it is reliable. Roberts, an editor in a Cochrane group, has stated that it is a huge mistake that the motto for Cochrane reviews of trials is "Trusted evidence."[145] This motto was introduced by Cochrane's new CEO, journalist Mark Wilson, shortly after he took office in 2012. It sounds like self-praise from a drug company, which reflects that Wilson was marketing oriented and did not understand what science is about. He ruined the Cochrane Collaboration and suddenly left in a middle of April 2021 after its major funder had declared that it would cut its funding substantially.[146]

By far most Cochrane reviews of psychiatric drugs are unreliable because most of the included trials are unreliable, and the Cochrane authors and editors are not sufficiently critical towards the source material.

7 Psychosis

Psychosis drugs are the poster child of psychiatry and were highly praised in the textbooks. We are told that, before the advent of them, many patients needed to live the rest of their lives in hospitals and other institutions;[16:222] the discovery of the pills in the 1950s meant that many psychotic patients clearly improved their quality of life enabling their dismission from the institutions and reintegration into society;[20:416] patients who were previously tortured by their disease and were aggressive could now live alone or in protected housing;[18:307] psychosis pills led to a decrease in hospital beds;[16:616] chlorpromazine was a revolution in the treatment of psychotic disorders[16:560] and it contributed in particular to emptying psychiatric hospitals;[18:307] and - before chlorpromazine, lithium, depression pills and benzodiazepines, the seriously ill patients spent most of their lives in isolated institutions, behind locked doors, with barred windows, and physical force was used – but the development of psychiatric drugs in the 1950s revolutionised the treatment.[17:644]

Psychiatrists propagate this narrative all over the world to gain support for their specialty but *all* the above is wrong.[1,4-8] There were no references for the extravagant claims, but it has been documented that the pills had nothing to do with the emptying of the asylums.[1:155,3:53,147,148] Furthermore, it is impossible for drugs that – according to the standard scale for evaluating the effect on the psychosis - do not have clinically relevant effects (see just below) to produce such dramatic outcomes.

Since the "emptying of the asylums" is the core argument for the claimed revolution in psychiatric drug treatment that started with chlorpromazine in 1954, I shall explain why it is wrong. The misconception stems in particular from flawed studies in New York.[148] The authors noted that the populations in asylums fell after 1954 and ascribed this to drug treatment. Better studies were conducted in Michigan and California by other authors who compared treated and untreated patients. They found that the drugs did not increase discharge rates.

In 1985, a study debunked the myth totally.[148] It covered all US states and compared two nine-year trends in discharge rates, 1946 to 1954 with 1955 to 1963. The mean percentage change in discharge rates was 172 before chlorpromazine, a little higher than with chlorpromazine, 164.

There are no supportive studies of the myth from other countries either. In England, inpatient populations began to decline before the drugs were introduced; in France, inpatient populations increased for 20 years after the drugs were introduced;[148] and in Norway, inpatient numbers did not change with the introduction of the drugs.[3:54]

The Joint Commission on Mental Illness and Health, commissioned by the US Congress, wrote in 1961 that "Drugs have revolutionized the management of psychotic patients in American mental hospitals," quoting the misleading New York studies and avoiding mentioning the better designed Michigan study even though it was available.[148] It was politically expedient to dupe the population this way painting a false picture of huge progress in psychiatry.

Psychosis pills don't have clinically relevant effects on psychosis

One textbook noted that the strongest evidence in psychopharmacology is for the effect of psychosis pills in the acute phase of schizophrenia and for relapse prevention, as they markedly reduce the risk of relapse.[16:560] It claimed that the pills improve prognosis and survival in most

patients,[16:222] and that it is essential to know which biological processes in the brain the pills influence in order to offer the most optimal medical treatment.[16:216]

All of this is wrong. Robert Whitaker once wrote to me that it requires extraordinary mental gymnastics by the psychiatrists to conclude that these drugs, which cause obesity, metabolic dysfunction, diabetes, tardive dyskinesia, lethal cardiac arrhythmias, and so on, protect against death. They don't; they kill many people,[7:307] which I shall explain below.

It is impossible to offer a better treatment by knowing more about biological brain processes when the drugs do not have clinically relevant effects on the psychosis apart from tranquillising the patients, which is an unspecific effect.

Virtually all placebo-controlled trials of psychosis drugs are seriously biased by cold turkey effects in the placebo group, which occur when the psychosis drug the patient is already on gets withdrawn before randomisation. These iatrogenic harms are usually avoided in the actively treated group. The reason that Janssen could claim that its bestseller risperidone didn't cause more extrapyramidal (muscular) harms than placebo was the abrupt withdrawal of the previous psychosis drug, which inflicted these effects on the placebo group to such an extent that one in six patients got them.[1:276] The companies needed to show that their drugs reduced psychotic symptoms and they made some of the placebo patients psychotic by withdrawing their drug cold turkey.[4:45,31,149]

I have only found two trials where none of the patients had received a psychosis drug before. One was from China and appeared to be fraudulent.[150] It compared olanzapine with placebo in patients with first-episode schizophrenia.[151] The patients needed to have a score on the Positive and Negative Syndrome Scale (PANSS) of at least 60 to be included. However, the score before treatment was only about 9, even though by definition it must be at least 30 (the lowest score is 1 and there are 30 items). The score increased to 71.3 in the olanzapine group and to 29.4 in the placebo group. The authors reported that olanzapine was effective although patients on placebo fared much better. Furthermore, a difference of 42 in PANSS is implausibly large. In the placebo-controlled trials in submissions to the US Food and Drug Administration (FDA) of newer psychosis drugs, including olanzapine, the difference was only 6.[152]

The only trial that doesn't appear to be fraudulent and wasn't flawed by withdrawal effects was published in 2020, 70 years after the discovery of the first psychosis drug, chlorpromazine.[153] It randomised 90 patients with a first-episode psychosis with a duration of untreated psychosis (DUP) of less than 6 months to risperidone, paliperidone or placebo.

However, even after 70 years, the psychiatrists weren't yet ready to draw the obvious conclusions of their results. They wrote that the differences were "small and clinically trivial, indicating that treatment with placebo medication was no less effective than conventional antipsychotic treatment" (P = 0.95). They noted that "the immediate introduction of antipsychotic medication may not be required for all cases of first episode psychosis" with the reservation that "this finding can only be generalised to a very small proportion of FEP [first episode psychosis] cases at this stage, and a larger trial is required to clarify whether antipsychotic-free treatment can be recommended for specific subgroups of those with FEP."

What the authors should have written is something like this: "Our study was small, but it is unique because it only included patients who had not been treated with a psychosis drug before. We found that psychosis drugs are ineffective in patients with untreated psychosis. This is great progress for patients, as these drugs are highly toxic and make it difficult for them to come back to

a normal life. Based on the totality of the evidence we have, the use of psychosis drugs in psychosis cannot be justified."

The authors of a 2011 Cochrane review of psychosis pills for early episode schizophrenia pointed out that the available evidence doesn't show that the drugs are effective.[154] This is one of the few Cochrane reviews of psychiatric drugs that can be trusted. Apart from the cold turkey problem, Cochrane reviews in schizophrenia include trials in a meta-analysis where half of the data are missing.

This Cochrane review noted that double as many patients on chlorpromazine than on placebo were rehospitalised within three years, risk ratio 2.3 (1.3 to 4.0). There were also fewer rehospitalisations in the placebo group at the one-year follow-up in the famous trial funded by the US National Institute of Mental Health, which was published in 1964, but the difference wasn't quantified, and the original data appear to have been lost.[154] These data totally contradict the psychiatric narrative that psychosis pills emptied the asylums.

In trials supposed to be double-blind, but which are not blind in practice, investigators may report positive effects that only exist in their imagination. This occurred in the NIMH 1964 study, which is still highly cited as evidence that psychosis drugs are effective.

344 newly admitted patients with schizophrenia were randomised to phenothiazines such as chlorpromazine or to placebo.[155] The investigators reported, without offering any numerical data, that the drugs reduced apathy and made movements less retarded, the exact opposite of what these drugs do to people, which the psychiatrists had admitted a decade earlier.[5:49,5:61]

The investigators claimed a huge benefit for social participation (effect size 1.02) and that the drugs make the patients less indifferent to the environment (effect size 0.50). The drugs do the opposite. The authors also claimed, with no data, that 75% versus 23% were markedly or moderately improved and suggested that the drugs should no longer be called tranquillisers but antischizophrenic drugs.

Their study contributed to shaping the erroneous beliefs that schizophrenia can be cured with drugs and that psychosis pills should be taken indefinitely.[1]

The truth is that psychosis pills do not have clinically relevant effects on psychosis. Despite the formidable biases - cold turkey, lack of blinding, and industry funding that often involves torturing the data till they confess[6,7] - the published outcomes have been very poor.[4] The least clinically relevant effect corresponds to about 15 points on the PANSS scale[156] commonly used in the trials. Yet, what was reported in the placebo-controlled trials of recent drugs submitted to the FDA was only 6 points, or 3% of the maximum score of 210 on this scale.[152]

A textbook claimed that the effects on the dopamine system can restore homeostasis in brain signal transmission.[18:97] This assumes that there is a defect in the dopamine system to begin with, which has never been documented and is unlikely (see Chapter 4). We are also told that the treatment response is related to dopamine activity.[16:220] This is not possible for drugs that don't work.

There were case stories in one of the textbooks and they were always positive in relation to the drugs used, but most of them were misleading. Here are some examples.

A patient improved within a few weeks on a psychosis pill and no longer heard voices or experienced persecution.[18:87] The pills do not have such effects.

A patient improved a lot on a psychosis pill and had relapses when he did not want to continue with the drug.[18:89] It is highly likely that the psychiatrists confused withdrawal symptoms with relapse. And there is no reliable evidence that the pills can prevent relapse (see below).

A patient got a small dose of a psychosis pills and support, and improved.[18:89] It was more likely the support that helped the patient, or the patient would have improved anyway, without treatment or support.

An increased dose of a psychosis pill affected the time to relapse.[18:105] These pills do not have increased effect with increased dosage.[157]

It would be an eye-opener if the psychiatrists tried a psychosis pill on themselves. Two physicians have described how a single dose of haloperidol knocked them down.[158] 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.[4:116] Everyone felt anxious, restless, disengaged and demotivated to do anything; a 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. Peter Breggin calls this medication spellbinding.[135,159]

The predominant subjective effects reported by patients on the Internet when they take psychosis drugs are sedation, cognitive impairment and emotional flattening or indifference.[160] We also know from telephone help lines that what medicated persons miss the most are themselves.[1:179]

Psychosis pills were hailed as a great advance, but this was because they kept the patients docile and quiet, which was very popular with the staff in psychiatric wards.[148] It was a huge conflict of interest that the same staff evaluated whether patients had improved or not, and this conflict of interest clouds psychiatric practice and research even today.

Psychosis pills increase mortality substantially

The psychiatrists presented many arguments as to why it is important to use psychosis pills, but all of them were unsustainable. One of them was that patients with schizophrenia live 15-20 years less than other citizens,[18:288] and among the causes were mentioned suicide, accidents, cardiovascular diseases, metabolic syndrome, lifestyle, undertreatment of somatic diseases and drug harms.[16:628] Treatment with psychosis pills was not mentioned.[17:308]

One book noted that mortality is increased if the psychosis appears early in life,[19:239] but it didn't occur to the authors that the longer the duration of the psychosis, the longer the treatment with psychosis pills, and therefore also a higher mortality because the pills increase mortality.

Two textbooks raised the highly implausible claim that psychosis pills *reduce* mortality from psychotic disorders.[16:222,18:101,18:236] They don't; they *increase* mortality substantially.

It is not possible to use the placebo-controlled trials in schizophrenia to estimate the effect of psychosis pills on mortality because the drug withdrawal design increases mortality in the placebo group. The suicide rate in these unethical trials was 2-5 times higher than the norm.[1:269,161] One in every 145 patients who entered the trials for risperidone, olanzapine, quetiapine and sertindole died, but none of these deaths were mentioned in the scientific literature, and the FDA didn't require them to be mentioned.

When I decided to find out how deadly psychosis pills are, I focused on patients with dementia assuming that few of them would be in treatment before randomisation. A meta-analysis of placebo-controlled trials with 5,000 patients showed that after only ten weeks, 3.5% had died while receiving olanzapine, risperidone, quetiapine or aripiprazole, and 2.3% had died on placebo.[162]

Thus, for every 100 people treated for ten weeks, one patient was killed with a psychosis pill. This is an *extremely* high death rate for any drug.

Since half of the suicides and other deaths are missing, on average, in published psychiatric drug trials,[125] I looked up the corresponding FDA data based on the same drugs and trials. As expected, some deaths had been omitted from the publications, and the death rates were now 4.5% versus 2.6%, which means that psychosis pills kill two patients in a hundred in just ten weeks,[163] or double as many as the published trial reports indicate.

I also found a Finnish study of 70,718 community-dwellers newly diagnosed with Alzheimer's disease, which reported that psychosis pills kill 4-5 people per year compared to patients who are not treated.[164] If the patients received more than one drug, the risk of death was increased by 57%. As this was not a randomised trial, the results are not fully reliable, but they are plausible given the data from the randomised trials. Thus, the pills might kill 4 times as many patients as the published reports indicate, or even more, if we extend the observation period beyond one year.

One textbook noted that psychosis pills may increase mortality in patients with Alzheimer's disease.[18:49] This downplayed the problem. These pills not only *may* increase mortality, they *do* increase mortality, and to a substantial degree, which the textbook said nothing about.

This phenomenon is seen everywhere, in textbooks, scientific articles, on websites, in lectures and in interviews in the media. There is a huge asymmetry in the way psychiatrists describe benefits and harms. There are rarely any reservations when the benefits of drugs are commented upon and their effects are much exaggerated, which I shall exemplify throughout this book.

Another textbook was even worse. It noted that meta-analyses on large patient materials suggested a small excess mortality of patients with dementia treated with psychosis pills compared to placebo, but that it was uncertain what caused this excess mortality.[17:243]

This comes close to fraud. There was no reference, but the meta-analyses not only *suggested* but *proved* the excess mortality; it was not small but huge; and the FDA has explained what causes it: Most of the deaths in the demented patients were either cardiovascular (e.g., heart failure, sudden death) or infectious (e.g., pneumonia).[163]

The important question then is: Can we extrapolate these results to young people with schizophrenia?

We have no other choice. In evidence-based healthcare, we base our decisions on the best available evidence. This means the most reliable evidence, which are the data presented just above, two deaths per hundred people treated for ten weeks. Thus, absent other reliable evidence, we will need to assume that psychosis pills are also highly lethal for young people.

Young people on psychosis pills also often die from cardiovascular causes and suddenly,[8:40] and we would expect some of them to die from pneumonia. Psychosis drugs and forced admission to a closed ward make people inactive, and when they lie still in their bed, the risk of pneumonia and pulmonary emboli from a venous thrombosis increases, which might go unnoticed before it is too late. Psychosis pills also kill patients because of huge weight gains, hypertension and diabetes.

Considering that these drugs do not have a clinically relevant effect on psychosis, and that benzodiazepines are far less dangerous and even seem to work better for acutely disturbed patients,[165] the conclusion must be that psychosis pills should not be used for anyone. They should be taken off the market.

The psychiatrists do not blame their drugs or themselves for the considerably shorter lifespan patients with schizophrenia have, but the patients. It is true that the patients have unhealthy lifestyles and may abuse substances, in particular tobacco. But it is also true that some of this is a

consequence of the drugs they receive and the way they are treated. Some patients say they smoke because it counteracts some of the harms of psychosis pills, which is correct because tobacco increases dopamine while the drugs decrease it. And when people are locked up for weeks or months on end and have nothing to do, is it then strange that they smoke? Or drink? Or overeat? Or kill themselves? I don't think so.

When I tried to find out why young people with schizophrenia die, I faced a roadblock, carefully guarded by the psychiatric guild. It is one of the best kept secrets that psychiatrists kill many of their patients, also young ones, with psychosis drugs. I described my experiences with the roadblock in 2017, *Psychiatry ignores an elephant in the room*,[166] but subsequent events were even worse. This is a summary of a more comprehensive account.[8:40]

Large cohort studies of people with a first-episode psychosis provide a unique opportunity for finding out why people die. However, there is too little information in these studies, or no information at all, about the causes of death. In 2012, Wenche ten Velden Hegelstad and 16 colleagues published 10-year follow-up data for 281 patients with a first-episode psychosis (the TIPS study). Although their average age at entry into the study was only 29 years, 31 patients (12%) died in less than 10 years.[167] But the authors' detailed article was all about recovery and symptom scores. They took no interest whatsoever in all these deaths.

I wrote three times to Hegelstad but did not get the missing data. The third time she replied they would be published soon, but the new paper did not present the data I had requested.[168] Two months later, Robert Whitaker and I wrote to the editor of the journal, *World Psychiatry*, professor Mario Maj, asking for his help. He did not want to help us either in finding out why young people died so quickly.

We wrote again, explaining that people I had talked to in several countries about deaths in young people with schizophrenia - psychiatrists, forensic experts and patients - all agreed that we desperately need the kind of information we asked Maj to ensure became known. We called on him to make this happen as his ethical duty, both as a journal editor and as a doctor instead of telling us that he did not have space for our letter about this in his journal. We did not hear from Maj again.

In contrast to the authors of the TIPS study, Danish psychiatry professor Merete Nordentoft was forthcoming when I asked her about the causes of death for 33 patients after 10 years of follow-up in the OPUS study, also of patients with a first-episode psychosis.[169] I specifically mentioned that suicides, accidents and sudden deaths could be drug related.

Nordentoft sent a list of the deaths and explained that the reason cardiac deaths were not on the list was probably because the patients had died so young. But in the death certificates, she had seen some patients who had dropped dead, one of them while sitting in a chair, which is what we call cardiac deaths.

This is how it should be. Openness is needed if we want to reduce the many deaths that occur in young mental health patients, but very few psychiatrists are similarly open as Nordentoft.

Early intervention? Yes, but not with psychosis drugs

An argument for using psychosis drugs was that it is harmful not to intervene early, and the term "duration of untreated psychosis" (DUP) was often used. It was claimed that DUP worsens the prognosis for schizophrenia and similar disorders;[16:194,17:326,18:79,18:233 19:235,20:416] that it is harmful

for the brain to be psychotic;[18:98,20:416] and that with early intervention, a chronic course can be prevented for many patients[17:326] who can be taught to handle their vulnerability.[18:80]

These argument are not correct. When a drug doesn't work for a disease but only pacify the patients, it cannot be important to use it early in the course of a disease. Furthermore, the research – none of which was referenced - that claims that the duration of untreated psychosis is related to the prognosis is unreliable. People who are not treated early are not comparable to those treated early and they are in a worse condition, on average, with a host of prognostic factors that bode for a poor long-term outcome, e.g. homelessness and alcoholism.

It is not possible with statistical methods to adjust reliably for such differences. As already noted, the more variables you include in a logistic regression, the further you are likely to get from the truth[50] (see page 15).

One textbook noted that acute psychosis can be preceded by acute stress or trauma, and that full remission will usually be seen within a few months, often in a few weeks or even days.[16:232] This makes it even more unacceptable that the authors a few pages later recommended second generation psychosis pills and even said that "mood stabilisers" – likely antiepileptics - can be used, in addition.

Psychiatrists also claimed that psychosis drugs are often a prerequisite for psychotherapy and that drug free treatment has been tried for acute psychosis in some countries, but can be very dangerous, with a likely risk of brain damage and a high risk of suicide.[18:233]

If patients are very agitated, it may help getting in contact to sedate them but benzodiazepines are better at this than psychosis pills.[165] And it is usually easier to practice psychotherapy on a patient who is not sedated than on one who has difficulty concentrating and focusing.

It is outrageous to suggest that it can be very dangerous not to use psychosis pills. It is very dangerous to use them; they do not protect against brain damage but cause irreversible brain damage;[63,64] and they do not lower the risk of suicide, they likely increase it because of withdrawal effects, e.g. when the patients need a drug holiday, which increases the risk of akathisia,[134] and thereby of suicide and violence.[7]

Patient reports on the Internet show that suicidal thoughts when taking psychosis pills 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 ($P < 0.001$).[160] This harm would be expected to be related to the dose of the previous drug, which it clearly is.[170]

Akathisia was given little attention in many years, and physicians generally interpreted the restless behaviour as a sign that patients needed a higher dose of the drug, which aggravates the situation. When the psychiatrists finally took an interest in this, the results were shocking. In one study, 79% of mentally ill patients who had tried to kill themselves suffered from akathisia.[1:187] A 1990 study reported that half of all fights at a psychiatric ward were related to akathisia[171,172] 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. Psychotropic drugs can cause people to lose some of their conscience, losing control over their behaviour.[21] Such people are at greatly increased risk of committing acts of crime and violence.

A textbook claimed that clozapine seems to be able to reduce suicidal behaviour in patients with schizophrenia, and it mentioned that two small studies suggest that classic psychosis pills can be preventative across diagnoses.[17:811] This wishful thinking was cleverly manipulated by using the expression "seems to;" by referring to two small studies rather than telling us what all studies

showed; and by omitting the two studies in the reference list after the chapter leaving the reader in total darkness. This was the anti-thesis of evidence-based medicine.

Early intervention in schizophrenia is beneficial but provided it is not with psychosis pills but with psychosocial interventions.[7:170] In 1969, the WHO launched a study that showed that patients fared much better in poor countries – India, Nigeria and Colombia – than in the United States and four other developed countries.[1:226] 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. WHO therefore did another study, focusing on first-episode schizophrenia diagnosed with the same criteria in ten countries.[1:228] 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 big 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. People in poor countries couldn't afford psychosis pills, so only 16% of the patients were regularly maintained on them as compared with 61% in rich countries.

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 Lilly's drug, olanzapine, the other half with other psychosis pills.[173]

A 20-year study from Chicago by Martin Harrow showed that, among 70 patients with schizophrenia, those who were not on psychosis drugs after the first two years had far better outcomes than those who were on drugs.[174] This was not due to confounding by indication. The adjusted odds ratio of not being on drugs was 5.99 (3.59 to 9.99) for recovery and 0.13 (0.07 to 0.26) for rehospitalization.

Harrow was a prominent schizophrenia researcher at the National Institute of Mental Health, and other researchers arrived at similar results, but they all experienced that their funding dried out.[1,5]

Apart from avoiding the harmful effects of psychosis pills, there are other reasons why people with schizophrenia fared so well in poor countries.[175] 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. Some have asked me whether it would be more humane than using 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 and have killed hundreds of thousands of patients with psychosis pills.[6:232] This is not a humane system.

The famous Open Dialogue Family and Network Approach initiative in Lappland aims at treating psychotic patients in their homes.[8:91] The treatment involves the patient's social network and starts within 24 hours after contact.[176]

A comparison between Lappland and Stockholm illustrates the difference between an empathic approach and immediately forcing drugs on patients with a first-episode psychosis.[176,177] The patients in Lappland were closely comparable to those in Stockholm, but in Stockholm, 93% were treated with psychosis pills against only 33% in Lappland, and five years later, ongoing use was

75% versus 17%. After five years, 62% in Stockholm versus 19% in Lappland were on disability allowance or sick leave, and the use of hospital beds had also been much higher in Stockholm, 110 versus only 31 days, on average. It was not a randomised comparison, but the results are so strikingly different that it would be irresponsible to dismiss them. Furthermore, there are many other results supporting a non-drug approach to acute psychosis.[7:330]

The Open Dialogue model is gaining momentum in several countries and randomised trials are ongoing. It started 25 years ago,[176] and it was therefore surprising that the textbooks didn't mention it. Denmark has its own version of early intervention along similar principles, which started at about the same time. It is called OPUS because an orchestra consists of many different instruments, all working together to play a piece of music. The idea with OPUS is to create a partnership between the patient and all those who are part of the treatment including the family and social network.

The textbooks acknowledged that psychosocial interventions have a role in the treatment of schizophrenia,[16:615,20:418] and there were many remarks about the positive effects of these initiatives, e.g. of family involvement, outreach,[16:194,17:313] assertive community treatment on patient terms,[16:616,17:313] multidisciplinary teams, cognitive behavioural therapy,[16:224,17:318] and neurocognitive training.[16:624]

It was noted that the OPUS study in Denmark and the AESOP study in England showed that more than half of the patients no longer had psychotic symptoms after 10 years.[16:205] Studies have shown a reduction in readmissions, fewer hospital days, and an effect on psychotic symptoms, drug abuse, and negative symptoms.[16:617]

One book claimed, without references, that studies have shown that cognitive behavioural therapy can alleviate both psychotic and negative symptoms, and that randomised trials have shown that family intervention halves the risk of relapse and hospital days.[17:318] Another book referred to a systematic review,[16:620] which found that family psychosocial interventions halved the frequency of relapse of schizophrenia or schizoaffective disorder.[178] Hospital admissions were reduced by 32% whereas hospital days were only available in two small Chinese studies.

The authors noted that the treatment effects might be overestimated due to poor quality of the trials, e.g. insufficient blinding of the assessors. However, the effect on relapse was so large that it could hardly be caused by bias alone.

One book noted that supported employment made it three times more likely that the patients would find work.[16:625] The reference was to a Cochrane review of trials in severe mental illness and by far most of the patients were diagnosed with schizophrenia or schizoaffective disorder. The review noted that the evidence was of very low quality.[179] This was mainly because none of the 14 studies were blinded: "Participants could identify the given intervention by contents of the program."

Of course they could. Some interventions just cannot be blinded, but conclusions like these are produced when researchers slavishly follow the Cochrane cookbook approach, which down-grades the quality of the evidence for many useful interventions that cannot be blinded like a drug trial can.

It is unfortunate that Cochrane reviews routinely downgrade the results of psychosocial interventions, as they are so clearly superior to drugs. Another issue was that days in competitive employment, the review's primary outcome, was only reported in half of the 14 studies, which is more serious, as all the studies were about supported employment.

One of the books, which only had psychiatrists as authors, was even more focused on drugs than Cochrane reviews are. It claimed that environmental therapy and psychotherapeutic techniques can be used when the acute psychosis is under control with psychosis pills.[18:79] This is wrong. Psychotherapy can abolish the need for psychosis pills in many cases, as demonstrated by the experience with the Open Dialogue model and other approaches such as OPUS.

This book also contradicted itself. It noted that psychotherapy is recommended only in the stabilisation phase,[18:99] but on the next page, it said – when commenting on OPUS - that psychotherapy can also be used from the very beginning. Curiously, still on this page, the book claimed erroneously that psychosis pills are often a prerequisite for improvement and for making it possible to include the patient in other offers.[18:100]

The book also claimed that cognitive behavioural therapy is the only form of therapy for which there is evidence for an effect in psychosis.[18:102] This is also wrong. Family intervention, psychoeducation and mindfulness are also effective.[180]

Finally, the book noted that psychotherapy was not recommended for acute mania but was a well-documented supplement to medication as prevention.[18:117] We got it by now. Give them all drugs. Everything else is supplementary, if used at all. Even this recommendation was dubious. A network meta-analysis showed that psychoeducation plus cognitive behavioural therapy has a large effect on manic symptoms compared with treatment as usual, effect size −0.95 (−1.47 to −0.43).[181]

A much more reasonable book, which is the one that wrote the most about OPUS,[16] offered five references to Cochrane reviews in a literature list that was not directly linked to the statements about its effects. I have commented on two of them just above.[178,179] The other three were not particularly convincing.

One review was about intensive case management of severely mentally ill people in the community that included 40 trials, but most of them had a high risk of selective reporting of the outcomes, and not a single one provided data for relapse or important improvement in mental state.[182] Despite this, the authors wrote 273 pages for their Cochrane review – the size of a book - and concluded that the intervention is effective in ameliorating many outcomes and may reduce hospitalisation, increase retention in care, and globally improved social functioning. Lovely, but difficult to know if this is just wishful thinking given how poor the evidence was.

The second Cochrane review was about shared decision making but there were only two studies. The authors wrote 45 pages about them even if they could not conclude anything.[183] But we need not study shared decision making in randomised trials. We have an ethical obligation to respect the patients and involve them in our decisions. This ethical imperative cannot be suspended, not even when the patients are psychotic, according to the United Nations Convention on the Rights of Persons with Disabilities, which has been ratified by virtually all countries except the United States.[7:333,184] 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.[184]

The third Cochrane review was about early intervention for psychosis.[185] Even though there were 18 studies, they were diverse, mostly small, undertaken by pioneering researchers and had many methodological limitations, which generally made meta-analyses inappropriate. The authors found the evidence inconclusive but nonetheless wrote 134 pages about it. It is interesting that

they did not find convincing evidence for early intervention with drugs, as this was touted as being important in several textbooks (see above).

One textbook noted that psychosis pills dampen or remove positive symptoms such as hallucinations, delusions, thought disturbances and catatonia.[18:86,18:234] This gives the erroneous impression that the drugs are highly effective and have specific effects on psychosis. They work in the same way on patients, healthy volunteers and animals;[7] they are major tranquillisers, which was what they were called in the distant past; and they cannot remove hallucinations or delusions. 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 the psychiatrists noted that it didn't have any specific antipsychotic properties.[1-142]

It was recommended to treat pregnant women with schizophrenia because untreated psychosis can endanger the life of the mother and child.[17:669] There was no thoughts about that the pills just increase this risk further. This book noted that FDA in 2011 issued a general warning against using psychosis drugs because of extrapyramidal symptoms and withdrawal symptoms, which suggests that the drugs affect the brain in both the child and the mother.

Suggests that the drugs affect the brain? We have *known* for 70 years that the drugs hamper normal brain functions,[1:142] which is why they are being used. How can people supposed to be experts in psychopharmacology, which was the title of their chapter, write such nonsense? Well, they were all professors of psychiatry, which seems to be a *carte blanche* for people to write whatever they please.

In the package inserts for psychosis pills, e.g. for olanzapine,[134] the FDA warns that the drugs should be used during pregnancy only if the potential benefit justifies the potential risk to the foetus. This is not helpful advice. How should a doctor make such a judgment? FDA notes that neonates exposed to psychosis pills during the third trimester are at risk for extrapyramidal and withdrawal symptoms following delivery. There have been reports of agitation, hypertonia, hypotonia, tremor, somnolence, respiratory distress and feeding disorder in neonates, and in some cases, this has required intensive care unit support and prolonged hospitalisation. But according to Danish professors of psychiatry, it is only a *possibility* that psychosis pills affect the brain.

One book noted that patients with a diagnosis of schizotypy, which is a very dubious concept (see Chapter 15),[8:145] should be treated with psychosis pills if there are thought disorders, ruminations or psychotic episodes, as 25% develop schizophrenia.[18:106] There is no evidence for this, and many people have thought disturbances from time to time or ruminate.

In essence, this is a plea for prophylactic treatment of reasonably healthy people with toxic drugs, a horrible idea. The diagnostic test for this disorder is useless and bogus,[8:145] and it seems that most psychiatrists would test positive (see Chapter 15). Most psychiatrists should therefore be in prophylactic treatment with psychosis pills according to the advice in this textbook.[18:106]

Four books claimed that the pills work also for negative symptoms.[16:206,17:653,18:81,20:416] Negative symptoms include blunted affect, alogia (poverty of speech), asociality, avolition (lack of motivation or ability to do tasks or activities that have an end goal), and anhedonia (diminished capacity to experience pleasant emotions).[186] It was also claimed, in two textbooks, that psychosis pills have an effect on cognitive symptoms, [17:653,20:416] but two pages further ahead one of them noted that cognitive disturbances are largely unaffected.[20:418]

This information was confusing, contradictory, and wrong. The pills worsen negative symptoms and cognition, which has been known for 70 years,[1:142,5,7] and which was acknowledged in one of the books.[16:562]

One book mentioned that psychosis drugs can inhibit sensory inputs and psychological functions, which can increase negative symptoms and social isolation.[18:235] This contradicted directly claims in the same book, 154 pages earlier,[18:81] that psychosis pills have an effect on negative symptoms.

This textbook also noted that psychosis drugs can lead to drug abuse to stimulate the brain's reward system, which will worsen psychotic symptoms. It mentioned that direct sadness or depression occurs, but that it is often difficult to distinguish between a drug-induced depression from the understandable psychological reaction to having to live with a very severe disease, which has shaken one's self-perception.[18:235] This is the only time I came across an honest account of what psychosis pills really do to patients, and this is not beneficial for them.

One textbook claimed that several meta-analyses have shown that depression pills have an effect on negative symptoms.[18:101] There wasn't any reference to this remarkable statement. As I doubted it was correct, I looked up a couple of meta-analyses, which were both negative. One noted that "the quality of information is currently too limited to come to any firm conclusions;"[187] the other that "the literature was of poor quality" and that the results could "merely reflect selective reporting of statistically significant results and publication bias."[188]

This textbook noted that it can be difficult to distinguish between depressive symptoms, negative symptoms in psychosis and harms of psychosis pills.[18:101] Thus, two books admitted that psychosis pills worsen negative symptoms. Nonetheless, one book advised that, in case of persistent negative symptoms, some relief can be obtained by adding depression pills to the psychosis pills.[16:577]

This is a common theme in the textbooks. Instead of withdrawing the drug slowly that causes the problem, psychiatrists add additional drugs, which is an important reason for the massive overmedication of psychiatric patients that is well documented.[5,7,8,113,114]

No matter which psychiatric drugs people take – psychosis pills, depression pills, lithium, stimulants or benzodiazepines - or what their problem is, roughly one-third of the patients have their prescriptions renewed every year and are still in treatment with the same drug or a similar one ten years later. [113,114]

This tells a story of irresponsible doctors who don't know what they are doing or what they are causing. It also confirms what I wrote in a newspaper article in 2014 that our citizens would be far better off if we removed all psychotropic drugs from the market because it is clear that the doctors cannot handle them.[189]

Danish psychiatrists have admitted they have a problem. In a 2007 survey, 51% of 108 Danish psychiatrists said that they used too much medicine and only 4% that they used too little.[190] But usage of psychiatric drugs has continued to increase markedly in most countries, e.g. in the UK, psychosis pill prescriptions increased by 5% per year on average and depression pills by 10%, from 1998 to 2010.[191] We have not become more mentally ill to this degree. It is the effect of marketing and corruption.[6-8]

Psychiatry's main focus for the next decades should be on helping patients withdraw slowly and safely from the drugs they are on, instead of telling them that they need to stay on them and adding even more.

But this won't happen. Psychiatry's focus is on itself - a kind of eternal selfie it sends to the world all the time.

Psychosis pills do not prevent relapse

About length of treatment, one textbook noted that some patients will need lifelong drug treatment;[16:222] another that most patients with schizophrenia will need lifelong treatment.[17:657] This is clearly not true, which the results from Lappland demonstrate (see previous section).

The basis for this misconception is the so-called maintenance or continuation studies where patients in current treatment are randomised to continued treatment or placebo. Such studies cannot tell us if the patients still need the drug; they measure what the withdrawal effects are. But the psychiatrists conclude that the drugs reduce the risk of relapse[17:314,19:236] because they mistake withdrawal effects for relapse.

One textbook claimed the dramatic result that if patients stop treatment early, there is up to 85% risk of relapse while the risk is only 15% if the patients continue with the drug.[17:315] There was no reference and it is unscientific to write "up to." Evidence-based medicine is about what the effect is, on average, and one might as well write "down to," which doctors never do when they speak about drug effects they perceive as positive.

One page earlier, the authors were more modest saying that the risk of relapse is reduced by 60% but also that the risk is significantly less in studies of at least two years duration.[17:314] They quoted a 2012 meta-analysis for this result.[149]

I could not find the 85% versus 15% anywhere in the huge literature I have on my computer or by searching on the Internet. The meta-analysis reported 64% versus 27% with relapse after one year and 57% versus 22% independent of the duration of the study. Thus, the evidence-free claim of a 70% difference, which had Merete Nordentoft as first author, became only 37% and 35% in the meta-analysis.

The trials were flawed, as most patients on placebo were exposed to cold turkey withdrawal of their drug. The authors of the meta-analysis did a meta-regression with study duration as explanatory variable, which showed that the apparent effect of continued treatment with psychosis pills on relapse prevention decreased over time and was close to zero after three years.[149]

It is really bad medicine to keep the patients on their toxic drugs for years or lifelong based on the false belief that this improves their prognosis. When follow-up is longer than three years, it turns out that discontinuing psychosis pills is the best option. There is only one appropriately planned and conducted long-term maintenance trial, from Holland.[192] It has seven years of follow-up, and patients who had their dose decreased or discontinued fared much better than those who continued taking drugs: 21 of 52 (40%) versus 9 of 51 (18%) (P = 0.02) had recovered from their first episode of schizophrenia.

We have highly convincing evidence that psychosis pills prevent patients from becoming cured (see also previous section). And yet psychiatrists continue to recommend long-term treatment; many patients stay on the pills for many years; and many end up on disability pension. This is the upside down world of psychiatry and one of many signs that the whole specialty should be disbanded to protect the patients (see Chapter 16).

Danish researchers tried to repeat the Dutch study, but their study was abandoned because some patients were scared about what would happen if they did not continue with their drug while others wanted to come off it and did not want to be randomised to continuation. The key

investigator was Nordentoft who quoted the meta-analysis of the maintenance studies. Since the Dutch study is highly important and was published in 2013, it is curious that she didn't quote it.

There is another long-term study, from Hong Kong, published in 2018.[193] The researchers treated first-episode patients with quetiapine for two years; discontinued the treatment in half of them by introducing placebo; and reported the results at ten years. They found that a poor clinical outcome occurred in 35 (39%) of 89 patients in the discontinuation group and in only 19 (21%) of 89 patients in the maintenance treatment group.

I immediately suspected that the trial was flawed, as this result was the exact opposite of the Dutch result, and that the investigators had tapered off the drug too quickly. As there was nothing about their tapering scheme in the article, I looked up an earlier publication, of the results at three years.[194] They didn't taper at all; all patients randomised to placebo were exposed to a cold turkey.

The ten-year report was revealing: "A post-hoc analysis suggested that the adverse consequences of early discontinuation were mediated in part through early relapse during the 1-year period following medication discontinuation."[193] In plain language: We doctors harmed half of our patients by throwing them into the hell of a cold turkey.

The investigators defined a poor outcome as a composite of persistent psychotic symptoms, a requirement for clozapine treatment, or death by suicide. They called their trial double-blind, but it is impossible to maintain the blind in a trial with cold turkey symptoms, and it is highly subjective whether there are any persistent psychotic symptoms and whether clozapine should be given. It is much more relevant if the patients return to a normal life. A table showed that after ten years, 69% of those who continued taking their drug were employed versus 71% in the cold turkey group, a remarkable result considering the iatrogenic harms inflicted on the latter group. As noted earlier, trials like this one are highly unethical because some patients commit suicide when they experience cold turkey effects.

Please think about this: Why would drugs that have no clinically relevant effects when used for acute psychosis suddenly have dramatic effects on relapse when they are withdrawn after a considerable period of time? This makes no sense. But that is what psychiatrists want us to believe.

There was very little information in the textbooks about how to withdraw the drugs slowly and safely. One book explained that, because there was an upregulation in number of receptors, a too rapid dose decrease can elicit rebound psychosis, as downregulation is slow.[16:221] This was an admission that maintenance studies are fatally flawed. The book recommended gradual tapering over several months but did not advise how.[16:577]

Only one textbook gave advice about tapering. It advised a dose reduction of 20% every six months but did not explain what this means.[17:657] It could be 20% of the starting dose or 20% of the current dose. Thus, the dose reduction steps could be 80%, 60%, 40%, 20% and zero, which is a period of 2.5 years, or 80%, 64%, 51%, 41%, 33%, etc., in which case the withdrawal would take much longer. It is highly likely that what was advised was a linear taper, i.e. the first option, and not an exponential taper, as this is what psychiatrists routinely do.[8,195] In addition, an exponential taper would have required information about what to do when the dose became low, as otherwise the taper would never stop.

Very few people know that a linear taper is wrong. As the binding curves for drugs to receptors are hyperbolic, the taper must be exponential (see the receptor occupancy curve for the depression pill, citalopram, in Chapter 15).[8]

Since the binding curves are flat at the top and most patients are on a high dose, the initial dose reduction can often be relatively large without any ill effects. However, when the dose has become

low, the reductions often need to be small because the curve is very steep at low doses. Most importantly, tapering is a highly individual process, as patients react very differently to the same dose reduction. It is therefore a trial and error process in each case.[8:93]

Organised crime and fraud pay off

The psychiatrists used industry jargon when discussing the different psychosis drugs. All textbooks talked about first and second generation psychosis pills;[16:130,16:219,16:302,16:560,17:314,18:234,19:236,20:416] one also about third generation pills; [18:234] some drugs were called atypicals;[16:560] and some were called modern,[18:116] which suggests that you are outlandish and not up-to-date if you prefer other drugs.

As the drugs within these classes are widely different in their effects, it is meaningless to divide them into two or three classes. Academics should do better than echoing the misleading terms the industry has invented, which help them sell pills that are far more expensive than pills that are not worse, and in some cases even better, as they have fewer serious harms.[7]

As an example, one textbook mentioned that olanzapine, even though it was called a second generation drug, is not a first choice drug due to its metabolic harms.[20:418] But marketing beats science into a cocked hat. This drug, one of the worst psychotropic drugs ever invented, became a blockbuster, partly because of fraud, harassments via lawsuits against doctors, lawyers, journalists and activists who wanted to tell the truth about the drug, and organised crime that included illegal marketing.[6:31] I have estimated, based on sales and the published meta-analysis in people with dementia,[162] that up to 2007, olanzapine had killed 200,000 patients.[6:232]

The story of olanzapine is a dire one, which students of psychiatry should know about. In 2001, Lilly's best-selling depression pill Prozac (fluoxetine) was running out of patent and the company was desperate to somehow fool people into using Zyprexa also for mood disorders and Lilly misleadingly called it a mood stabiliser.[196] Olanzapine was an old substance and the patent was running out, but Lilly – a US company - got a new patent by showing that it produced less elevation of cholesterol in dogs than a never-marketed drug![197] Olanzapine raises cholesterol more than most similar 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.[197]

A lawsuit revealed that Lilly's documentation for the effect of olanzapine was so bad that the drug should not have been approved, but FDA covered up for all Lilly's manipulations, just as they did with fluoxetine (see Chapter 8).[198] A highly revealing book, *The Zyprexa Papers*, by lawyer Jim Gottstein, describes illegal, forced drugging that destroyed patients.[199] Psychiatrists, lawyers, and Eli Lilly lied shamelessly, and judges didn't care, which I have experienced first-hand as Gottstein's expert witness. Gottstein needed to go to the Supreme Court in Alaska before he got any justice, and he ran a great personal risk by exposing Lilly documents that were supposed to be secret.

One of the reasons why marketing of medicines is so effective is that the salespeople believe they are selling a very good drug. But they have been lied to by their bosses.[6] Lilly's huge commercial success with both fluoxetine and olanzapine illustrates that in psychiatry it doesn't matter which drugs you have. Corruption, marketing and lies will ensure that doctors don't use better and cheaper drugs. And patient organisations willingly contribute to the corruption. They often receive money from the industry and they only know what the drug firms have told them, or what the psychiatrists have told them, which is 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 psychosis pills such as olanzapine and risperidone.[200]

The crimes were massive. In 2009, Lilly agreed to pay more than $1.4 billion for illegal marketing for numerous off-label uses including depression and dementia, and Zyprexa was pushed particularly hard in children and the elderly.[201] The allegations were raised by six whistle-blowers from Lilly who were fired or forced to resign by the company. According to the complaint, one sales representative had contacted the company hotline regarding unethical sales practices but received no response.

Lilly salespeople were posed as persons in the audience who were interested in Zyprexa's expanded use and asked planted questions during off-label lectures and audio conferences for physicians. Another tactic was that, while knowing the substantial risk for weight gain posed by Zyprexa, the company minimised the connection between Zyprexa and weight gain in a widely disseminated videotape called *The myth of diabetes*.

The fraud was massive. In 2007, Lilly still maintained that "numerous studies … have not found that Zyprexa causes diabetes," even though Zyprexa and similar drugs since 2003 on their label had carried an FDA warning that hyperglycaemia had been reported.[202] Lilly's own studies showed that 16% of the patients gained at least 30 kg in weight after a year on the drug, and both psychiatrists and endocrinologists said Zyprexa caused many more patients to become diabetic than other drugs. But Lilly and corrupt psychiatrists produced papers describing schizophrenia as a risk factor for diabetes![4] As always, the drug wasn't the problem; it was the disease.

Zyprexa seems to be more harmful than many other psychosis pills.[196] But Lilly prepared fictitious patient stories for use by the sales force.[196]

An internal AstraZeneca email said that Lilly runs a large and highly effective investigator-initiated trials programme; they offer significant financial support but want control of the data in return; they are able to spin the same data in many different ways through an effective publications team; and negative data usually remains well hidden.[203]

Organised crime and fraud is also the business model for other companies.[6:22] AstraZeneca silenced a trial that showed that quetiapine (Seroquel) led to high rates of treatment discontinuations and significant weight increases while the company at the same time presented data at European and US meetings that indicated that the drug helped psychotic patients lose weight.[204] Speakers Slide Kit and at least one journal article stated that quetiapine didn't increase body weight while internal data showed that 18% of the patients had a weight gain of at least 7%.[196]

In 2010, AstraZeneca accepted to pay $520 million to settle a fraud case where the company illegally marketed quetiapine to children, the elderly, veterans and inmates for uses not approved by the FDA, including aggression, anger management, anxiety, ADHD, dementia, depression, mood disorder, post-traumatic stress disorder and sleeplessness.[205] The company also paid kickbacks to doctors.

In 2012, Johnson & Johnson was fined more than $1.1 billion after a jury found that the company and its subsidiary Janssen had downplayed and hidden risks caused by risperidone (Risperdal).[206] The judge found nearly 240,000 violations under Arkansas' Medicaid-fraud law. Janssen lied about the serious harms of risperidone which include death, strokes, seizures, weight gain and diabetes, and claimed the drug was effective and safe in the elderly. The crimes hit hard also on children.[207] More than a quarter of Risperdal's use was in children and adolescents, including nonapproved indications, and a panel of federal drug experts concluded that the drug was used

far too much. A world-renowned child psychiatrist, Joseph Biederman from Harvard, pushed the drug heavily to children and also extorted the company. Internal emails revealed that Biederman was furious after Johnson & Johnson rejected a request he had made to receive a $280,000 research grant. A company spokesperson wrote that he had never seen someone so angry and that, since that time, their business became non-existing within Biederman's area of control.

It seemed that Alex Gorsky, Vice President of Marketing, was actively involved and had first-hand knowledge of the fraud and kickbacks.[208] Johnson & Johnson's board of directors rewarded Gorsky by selecting him to be the next CEO. Just like in the mob: the greater the crime, the greater the advancement.

A disproportionate number of the drug companies' criminal activities involved psychiatric drugs and included illegal marketing, Medicare and Medicaid fraud, bribery of doctors, civil servants and politicians right up to the ministerial level, and disposal of evidence.[6] The corruption of doctors is also worse than in any other specialty.[7:267,209]

Our academic institutions have also become corrupt. They grant ownership to the collected data to the sponsor and often accept that the doctors will have little influence on any publications.[210] The competition for research funds means that companies can shop around among the various academic centres and choose those that don't raise uncomfortable questions.

The different psychosis drugs

One textbook[17:656] noted that a 2003 meta-analysis showed a better effect of second-generation drugs than of first-generation drugs.[211] This is unlikely and we should remember that head-to-head comparisons are usually flawed. They are almost always conducted by the companies selling the newer drugs and the design is often flawed, e.g. old drugs like haloperidol have been given in too high doses.[6] Fraud is also an issue. For example, AstraZeneca presented a meta-analysis of four trials showing that quetiapine was more effective than haloperidol, but internal documents released through litigation showed that quetiapine was *less* effective than haloperidol.[196]

It is very rare that the title of a paper tells you everything you need to know, but here is an example:

"Why olanzapine beats risperidone, risperidone beats quetiapine, and quetiapine beats olanzapine: an exploratory analysis of head-to-head comparison studies of second-generation antipsychotics."[212] In a mathematical sense, this is impossible. If A is higher than B, and B is higher than C, then C cannot be higher than A. But in psychiatry, the impossible is possible.

I shall not go into detail with what the textbooks claimed about the advantages of individual drugs or classes of drugs, as the research literature is unreliable. I will just mention a few issues.

The 2003 meta-analysis reported that 4 out of 10 second-generation drugs were more effective than first-generation drugs and that it was not because haloperidol had been dosed wrongly.[211] However, the authors noted that another meta-analysis, from 2000, had concluded that, "There is no clear evidence that atypical antipsychotics are more effective or are better tolerated than conventional antipsychotics,"[213] and that other researchers and guidelines shared this view, e.g. the American Psychiatric Association treatment guidelines, the Schizophrenia Patient Outcomes Research Team funded by the US Agency for Healthcare Research and Quality, and the US National Institute of Mental Health.

The authors did not find a dose-response relationship for the old drugs,[211] in contrast to the 2000 meta-analysis that found that high doses of haloperidol *reduced* the effect and that noted

that almost all studies had been sponsored by the pharmaceutical industry, which could lead to bias.[213] These authors also mentioned non-publication of negative studies and publication of only favourable results, which is why they made considerable efforts in obtaining complete data, e.g. from the FDA website.

Head-to-head trials are a risky business for drug companies, and they either don't do them or ensure that the result will be favourable to them. Given this, it is usually futile to try to find out if some drugs are better than others. We should therefore generally forget about head-to-head meta-analyses, but I shall mention one of them.

It is remarkable that it has been possible to show in a meta-analysis of published trials that new drugs ("second generation") aren't better than old ones ("first generation"). A huge 2009 meta-analysis of 150 trials with 21,533 patients showed just that. This means that the psychiatrists have been duped for decades.[214,215]

A textbook noted that atypicals such as risperidone, aripiprazole and olanzapine were increasingly used in mania and that they had more favourable harms profiles than the old drugs.[18:114] My imaginative powers may be limited but I have difficulty accepting that the harms profile of olanzapine can be better than *any* psychosis drug.

In the book about child and adolescent psychiatry, the authors gave a reference to a network meta-analysis by Danish authors[216] in the chapter on schizophrenia.[19:240] I co-authored the guidelines for performing such analyses that many journals refer to in their instructions to authors.[217] To increase power, a network meta-analysis typically includes both placebo-controlled trials and head-to-head comparisons. If drugs A and B have only been compared in two trials but have both been compared with placebo or drug C in three trials, there will be eight trials providing data for the comparison of A with B instead of just two. This method is very attractive but also presents challenges. As in other meta-analyses, one must be very careful that trials with implausibly large effects or few harms do not make the results unreliable and ensure that the data entered in the meta-analysis can be trusted.

The network meta-analysis included 12 trials comparing 8 psychosis pills with one another or with placebo in youth with schizophrenia and concluded that 6 drugs were effective as measured on PANSS and that 5 drugs were effective for negative symptoms.[216] The authors paid no attention to whether the patients were already in treatment before they were randomised, or to whether the effect measured on PANSS was clinically relevant (they were not reported as scores but as effect sizes, without standard deviations). It is not possible to conclude anything based on their analysis, and it is highly suspicious that an effect was also found on negative symptoms because psychosis pills worsen negative symptoms.

The authors wrote that the drugs did not have the same harms, e.g. "Weight gain was primarily associated with olanzapine." This expression downgrades the harm. The term "associated with" belongs to observational research because we cannot be sure that an association is causal, as it can be caused by confounding. We do randomised trials to remove confounding. Therefore, when olanzapine makes people obese in placebo-controlled trials, the harm is real and is *caused* by olanzapine.

Another textbook also mentioned a network meta-analysis of psychosis pills in patients with schizophrenia.[16:569] The authors compared the drugs for their effect on cognition.[218] They included only nine trials but found several significant results, e.g. that quetiapine, olanzapine and risperidone were better than amisulpride and haloperidol, and that quetiapine was better than other

drugs on attention and processing speed tasks. They concluded that quetiapine and olanzapine had the most positive effects. This is also hard to accept, as these drugs *worsen* cognition. Furthermore, many studies have shown that olanzapine is one of the most sedative drugs, which doesn't exactly improve one's attention and speed.

I have played with the idea of taking a pill of olanzapine just to feel what it is like but I won't do it. If I happened to be one of those people who have a long QT interval on the ECG, olanzapine could kill me, as it prolongs this interval and can cause deadly ventricular arrhythmia.[134]

The cognition meta-analysis was particularly untrustworthy.[218] The authors did not assess the included trials critically; three of the nine trials were not even blinded; and some were of appallingly poor quality, as between 20% and 85% of the patients dropped out. This was garbage in, garbage out. The authors even admitted that the observed superiority of the atypicals might reflect a deleterious effect of high-dose haloperidol. Why publish the garbage then?

The textbook authors noted that the differences in effect reported in this meta-analysis had no clinical relevance, apart from clozapine, which they considered more effective than other drugs.[16:569]

Another textbook offered a sobering observation: Atypicals generally have fewer extrapyramidal harms, [16:561,20:416] but the distinction is not entirely logical as these harms can also occur with several atypicals, especially at higher doses.[16:561]

The fairy tale of clozapine

The textbook authors considered clozapine the most effective drug,[16:569,16:576,18:101,18:235,20:418] and some even claimed that it reduces mortality[17:656] and suicides.[16:576,17:656] We are also told that, because of its considerable anticholinergic effect, clozapine does not cause extrapyramidal symptoms.[16:576]

None of this is correct. In the package insert for clozapine, FDA warns that "Neonates exposed to antipsychotic drugs during the third trimester of pregnancy are at risk for extrapyramidal and/or withdrawal symptoms following delivery,"[219] and in one study, 4 of 104 patients treated with clozapine developed tardive dyskinesia.[220]

Claims of highly implausible effects should be accompanied by references, but there were none. It has never been documented in reliable research that any psychosis pill reduces mortality, but it has been documented in randomised trials that these pills increase mortality substantially.

The alleged superiority of clozapine is also highly questionable. There are mediocre meta-analyses that suggest this, but a Cochrane review of good quality did not.[221] It included 27 comparative trials. Attrition was high, 30%, "leaving the interpretation of results problematic." The authors found a higher attrition rate due to adverse effects with clozapine and a lower attrition rate due to inefficacy, which they suggested pointed at a higher efficacy of clozapine.

This cannot be concluded and the statement is flawed. Since the adverse events come immediately, people will drop out earlier because of adverse events than because of a perceived lack of effect, and there will therefore be fewer patients on clozapine who can drop out because of lack of effect than on the comparator drugs. There were no significant differences between clozapine and olanzapine or risperidone in terms of positive or negative symptoms of schizophrenia while clozapine can cause more serious harms, e.g. deadly agranulocytosis and a higher incidence of ECG changes, which can also be fatal. The authors noted that data on important outcomes such as cognitive functioning, quality of life, death or service use are largely missing.

As for the claim that clozapine reduces suicides, I was unable to find any placebo-controlled trials documenting this. Clozapine is the only drug with an FDA approved indication for reducing the risk of suicidal behaviour, but, most curiously, this is not based on a placebo control but on a trial with olanzapine as comparator.[222] It randomised 980 patients with schizophrenia or schizoaffective disorder considered at high risk for suicide. The differences were barely statistically significant, P = 0.03, both for suicidal behaviour and attempted suicide. This in unconvincing. Furthermore, we cannot exclude the possibility that both drugs increase suicides but that clozapine does this to a slightly lesser degree than olanzapine. Finally, with P = 0.03, it could be a chance finding or a result of data torture.

Guess who supported this trial? Novartis, the manufacturer of clozapine, and 6 of the 13 authors had received grants from or were consultants to Novartis. They thanked a contract research organisation for monitoring the study and taking care of data transfers. Not exactly a set-up that instils any confidence in what went on, and the academics likely did not have much, if anything, to do with data analysis and the writing of the manuscript.

Upping the dose, using several drugs concomitantly, and increasing deaths

A textbook noted that it may be appropriate in some cases to increase the dosage above the approved interval.[17:652] This is very bad advice. It will not lead to a better effect[157] but to more harms,[63] including killing more patients.[223-227]

About combining several psychosis pills, a textbook noted that a better effect has not been documented but it can be needed in patients with treatment resistant schizophrenia.[16:577] Another book claimed that combination treatment cannot always be avoided and that it had not been shown that it increased mortality.[18:101] It even claimed that psychosis drugs *decrease* mortality.

This is as absurd as it gets in psychiatry. In a quote whose origin is uncertain, the definition of insanity is doing the same thing over and over and expecting a different result. When drugs don't work, more of the same won't work either. And when highly toxic drugs increase mortality substantially, more of the same will increase mortality even further.

The psychiatrists don't realise that when a patient is "treatment resistant," which is an insulting term as it suggests that the patient is at fault and not the drug, they should not increase the dose or add another drug but taper off the first drug slowly, which will have the best outcome for the patient.

A report from the Danish National Board of Health showed that half of the patients were in treatment with more than one psychosis pill simultaneously,[228] 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 psychosis drugs used simultaneously.

One textbook admitted that combinations with benzodiazepines increase mortality,[18:101] which the Danish National Board of Health has also warned about stating that combinations increase mortality by 50-65%.[228] Nonetheless, half of Danish patients received combination therapy.[228] The report advised against the massive use of depression pills, which was also out of control. Almost half of the patients were in treatment with both psychosis drugs and depression drugs.[228]

Drug use is out of control everywhere. In the UK, half of the prescriptions by general practitioners of psychosis pills are issued to people for non-psychotic problems including anxiety and sleep problems, and they are particularly often used in people with dementia and in old people.[229] In the United States, the use of psychosis pills doubled in adults and went up eight-fold in children

in just 11 years.[230] In 2005, seven kids per 1,000 were in treatment with the pills,[231] and only 14% of the prescriptions were for psychoses. Most were for behaviour problems and mood disorders.[4]

Observational studies show more than a doubling in mortality when more than one psychosis pill is used.[223-225] This is not a confounding effect. As already noted, psychosis pills can cause QT interval prolongation and life-threatening ventricular arrhythmias, and large US studies have shown that the drugs double the risk of sudden cardiac death in a dose-dependent manner.[226,227] They also cause falls and hip fractures due to orthostatic hypotension, sedation and loss of consciousness, and they increase cerebrovascular adverse events.[162]

The huge weight gains and diabetes many patients experience also increase mortality. A systematic review showed that the mortality for patients with schizophrenia has increased markedly 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.[232] The authors noted that an obvious explanation for this development is the increased use of newer psychosis pills, which are more likely to cause weight gain and metabolic syndrome than the old drugs.

There are of course also studies denying this, but they are flawed.[7:174] There are many tricks one can use to make a mortality increase look like a decrease, e.g. ignoring that untreated patients are generally in much poorer health or using person-years in follow-up studies of safety after the randomised phase ended instead of counting dead bodies, which is not biased. In one such study, the authors claimed that psychosis pills lowered mortality by over 50% and lowered suicides while their data showed that 65% more patients died and three times as many killed themselves.[233] This study was published in one of psychiatry's flagship journals, *JAMA Psychiatry*.

Depot injections, which releases the drug very slowly, are recommended for patients who are not keen to take psychosis pills and will often stop if left to themselves.[18:235] It was claimed that this is caused by the patients' lack of insight into their disease, called non-compliance, and that it is important for the prognosis to motivate such patients to adhere to the treatment.[18:235]

This approach to patients is horrible. I have met many patients who have excellent insight into their disease and understand more about psychosis pills than their psychiatrists do. Some of them have suffered from severe harms when treated forcefully or have seen patients suddenly drop dead and are very scared that they might get killed, too.

In the book, *Dear Luise*, Dorrit Cato Christensen writes about her daughter who was killed this way by psychiatry.[234] The main problem with depot drugs is that they cannot be interrupted even if the patient's life has become endangered, e.g. if it is detected that the patient is a slow metaboliser, which many people are. Luise was a slow metaboliser.

Irreversible brain damage and other serious harms

The textbooks provided contradictory information about irreversible brain damage, and one tried to explain it away in a most confusing fashion.[16:222] It noted that psychosis pills probably prevent loss of brain tissue in many patients, but that a harmful effect in others cannot be excluded.

It has not been documented that psychosis pills can prevent brain damage and evidence-based medicine is not about speculations but about what the effect is on average. It is sobering to be reminded that psychosis pills kill nerve cells so effectively that their possible use against brain tumours has been explored. [4:176,135]

These authors noted that the brain shrinkage is related to the dose of psychosis pills but also that the sickest patients receive the largest doses (confounding by indication), and that it is therefore difficult to make a judgment. They added that more recent data had shown a relationship between relapse and progressive shrinkage, which suggested that the psychosis can be toxic.

There were no literature references, but the relationship between relapse and shrinkage does not suggest that psychosis can be toxic. Using their own argument, that a harmful effect of the drugs cannot be excluded, the shrinkage might as well be caused by the drugs.

Another textbook noted that that animal experiments have shown that psychosis drugs reduce the grey substance,[17:314] with a reference.[235] It added that this has partly been confirmed in human studies, but that the literature is equivocal. One of the authors was Merete Nordentoft, a leading schizophrenia researcher. It is strange that she did not mention Nancy Andreasen's well-known studies.[63,64] Despite the huge potential for bias in brain imaging studies (see Chapter 3), such studies and meta-analyses of them – performed by people who, judging from their papers, clearly didn't like what they found – have convincingly shown that psychosis pills shrink the brain.[63,236] They do this in a dose-dependent manner[1,63] and they also shrink the brain in primates, which do not suffer from psychosis.[235] In contrast, the severity of illness has minimal or no effect.[63]

There is no reliable evidence that a psychosis per se can damage the brain,[237] and although a large study claimed this,[64] 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 psychosis pills could lead to brain shrinkage of the grey matter, again with no relation to the severity of the illness.[238]

Imaging studies can always be discussed but if we turn to the extrapyramidal harms of psychosis pills, there is no doubt that they cause permanent brain damage. These harms consist of various involuntary movements, which include akathisia; dystonia (painful muscle spasms); Parkinsonism (which include tremor, difficulty finishing thoughts or speaking, stiff facial muscles and difficulty walking); and tardive dyskinesia (facial movements including sucking or chewing motions of the mouth, sticking out the tongue, blinking the eyes a lot, and inability to sit or lie still, with constant movements of the extremities).[11] Patients with tardive dyskinesia have higher mortality rates, and this harm is dose related.[1]

One textbook noted that tardive dyskinesia is often reversible.[19:286] This is wrong[7,135] and was contradicted by another book that spoke about irreversible movement disorders.[17:314]

Among the harms of psychosis pills, the textbooks mentioned dystonia, dyskinesia, akathisia, Parkinsonism, malignant neuroleptic syndrome, sexual dysfunction, erectile dysfunction, retrograde ejaculation, decreased libido, cardiometabolic harms, influence on heart rhythm, QT prolongation, torsade de pointes, deadly ventricular tachycardia, orthostatic hypotension, sinus tachycardia, metabolic syndrome, type-2 diabetes, stimulation of the appetite centre with weight increase, prolactin increase, galactorrhoea, gynaecomastia, amenorrhea, osteoporosis, possibly breast cancer, nasal stenosis, influence on memory and cognition, dry mouth, constipation, urine retention, and blurred vision.[16:563,18:235,19:236,19:278] A remarkable omission in most of the lists of harms in the textbooks was tardive dyskinesia.

The risk of developing malignant neuroleptic syndrome was largely ignored for many years, 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.[1:208]

One textbook warned against QTc prolongation but only if the patients receive other drugs with such effects.[17:656] This advice is deadly. Some people have a long QTc interval naturally and they can suddenly die if they are treated with a psychosis drug as the only drug.

We are told that clozapine and olanzapine carry the highest risk of obesity, diabetes and cardiovascular disease,[17:655] which makes it difficult to understand why these drugs are so popular. Another textbook mentioned that meta-analyses have shown that the biggest risk of metabolic harms is seen with clozapine and olanzapine, and that patients on olanzapine gain more in weight than those on other drugs.[16:564]

Only one textbook informed honestly about the serious harms.[16:563] It noted that the extrapyramidal harms are dose dependent, and that tardive dyskinesia is a severe harm, which may be irreversible and has an incidence of about 5% per year with first generation drugs but is also seen with second generation drugs. It mentioned that akathisia, in severe cases, can contribute to increased suicide risk and can be confused with psychomotor agitation as a result of the psychotic condition leading to a dose increase, worsening the situation. Akathisia was said to occur in 25% of the patients on first generation drugs and to a lesser degree with second generation drugs.

Not even this book could refrain from semantically downplaying the problems. Akathisia does not *contribute* to increasing the suicide risk, it *causes* an increased risk. No other factors in the causal chain are needed for suicides to happen.[7]

Two books were dangerously dishonest.[17:654,18:235] They claimed that first generation drugs cause extrapyramidal harms, which can be irreversible in the case of tardive dyskinesia,[17:655] and that these harms can be avoided by using second generation drugs.[17:657] As already noted, this marketing message is false; the newer drugs are not any better in this respect.[239] Furthermore, when the authors stated that some patients with akathisia consider suicide, they did not say that this also applies to second generation drugs. A third book that mentioned akathisia[19:286] failed to note that it is a dangerous harm that increases the risk of suicide and violence.[7]

There are videos of children and adults with akathisia and tardive dyskinesia that show how horrible these brain damages can be.[240] It took psychiatry 20 years to recognize tardive dyskinesia as an iatrogenic illness,[7:163] even though it is one of the worst harms of psychosis pills and also one of the most common ones, affecting about 4-5% of the patients per year.[241] In 1984, Poul Leber from the FDA extrapolated the data and concluded that, over a lifetime, all patients might develop tardive dyskinesia.[11:368] Three years later, the president of the American Psychiatric Association said at an Oprah Winfrey show that tardive dyskinesia was not a serious or frequent problem.[242]

Neurologists are much better at spotting tardive dyskinesia than psychiatrists and the same applies to researchers. Among 58 consecutively admitted patients with acute psychosis, 48 of whom were treated for at least a week with psychosis drugs, the researchers found 10 patients with tardive dyskinesia, but the psychiatrists only made this diagnosis in one of them.[243] The akathisia diagnosis is also often missed or misinterpreted, particularly when the symptoms involve the extremities rather than the face. In the same study, the researchers diagnosed akathisia in 27 patients, the clinicians only in 7.[243] In a community sample of patients with schizophrenia, the prevalence was 19%.[244]

There are several reasons why akathisia might go undetected.[245] Its symptoms resemble and often overlap with those of other psychiatric disorders, such as mania, psychosis, agitated depression, and ADHD. In addition, akathisia often occurs concurrently with, and is masked by, akinesia, a common extrapyramidal harm of psychosis pills. Such patients might have the inner

feeling of restlessness and urge to move but do not exhibit characteristic limb movements but sit still, in a state of inner turmoil. When akathisia is mistaken for worsening anxiety, psychosis, or agitated depression, the clinician usually increases the dosage of the offending agent, leading to further harm.

Even less recognised than akathisia is tardive akathisia,[11:70] which has a delayed onset, usually more than three months since a medication or dose change. It is often associated with tardive dyskinesia. A journalist described his experience this way: "And then, one day about four months after my taper [of a depression pill], I woke up shaking, with a sense of impending doom like nothing I'd ever experienced."[246]

Akinesia is frequently overlooked or misdiagnosed as depression. Akinesia should therefore be considered in the differential diagnosis of any patient taking psychosis pills that becomes amotivational, depressed, lethargic, or slowed down. Severe forms of akinesia tend to be overlooked more often than mild cases, which might be because severely affected patients complain less about their symptoms.

What was totally missing in the textbooks were the harms psychiatrists inflict on their patients that are not drug harms. There was nothing about the lack of hope that occurs when psychiatrists stigmatise their patients by telling them they have schizophrenia and say it is a lifelong disease that sometimes requires lifelong treatment with psychosis pills, and subject them to forced treatment with these drugs.

Understandably, this increases the risk of suicide considerably.[7] A 2014 Danish register study of 2,429 suicides showed that the closer the contact with psychiatric staff – which often involves forced treatment – the worse the outcome.[247] Compared to people who had not received any psychiatric treatment in the preceding year, the adjusted rate ratio for suicide was 6 for people receiving only psychiatric medication, 8 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 are more ill than others (confounding by indication), but the findings were robust and most of the potential biases in the study were 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.[248] The editorialists believed that some people who commit suicide during or after an admission to hospital do so because of conditions inherent in the hospitalisation.

One textbook mentioned 10 risk factors for suicide,[18:131] but admission to a psychiatric ward was not among them even though this seems to be the greatest risk of all.

Lithium and antiepileptics

By and large, the information on lithium in the textbooks was incorrect (see also Chapter 8). One book claimed that lithium has a prophylactic effect in schizoaffective disorders and can dampen aggression,[18:241] with no reference. However, a systematic review of 22 trials of lithium for schizophrenia found no reliable evidence that lithium worked.[249] The trials were generally small, with only 35 patients on average, of short duration, incompletely reported, and insufficiently blinded,

and a positive effect disappeared when non-double blind studies or those with high attrition were excluded. I updated the search in April 2022 by searching on *lithium schizo** in the title field on PubMed and did not find any additional trials.

Psychotic patients are often treated with antiepileptics. As far as I can see, lithium and antiepileptics (see below, under bipolar disorder) should not be used in patients with psychosis.

Benzodiazepines

One textbook mentioned that agitation increases the risk of suicide and aggressive behaviour towards staff and other patients, and that it may be due to intoxication with psychoactive substances, abstinences, or harms of psychiatric drugs.[16:84]

These authors recommended non-pharmacological interventions for acute agitated conditions, e.g. de-escalation techniques,[16:85] and they said that psychosis drugs are better than benzodiazepines if drugs are needed. In the same book, another author said that benzodiazepines are important in an acute agitated phase of psychosis,[16:577] and that the effect is equivalent with that of psychosis pills.[16:560]

The drug industry has of course shied away from comparing their highly expensive psychosis pills with off-patent benzodiazepines that can be acquired almost for free, and psychiatrists 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.[5:200]

In 2012, there were 14 head-to-head trials, summarised in a Cochrane review.[165] The desired sedation occurred significantly faster with a benzodiazepine than with a psychosis pill, but the authors paid tribute to the drug industry by providing a conclusion that did not agree with their results: “There is currently no convincing evidence to confirm or refute the practice of administering benzodiazepines as monotherapy.”

There surely was, and we should use benzodiazepines if sedation is needed in the acute phase. The psychiatrists who did the Cochrane review noted that the trials they reviewed were of poor quality, but it is the best evidence we have.

When I lecture for psychiatric patients, I often ask them which drug they would prefer next time they became admitted acutely and needed something to calm them down. All of them have preferred a benzodiazepine. It is therefore unethical to force them to take a psychosis pill or to give them an involuntary injection with a psychosis drug, but this is standard practice.

Since acute psychosis tend to disappear again if left untreated, psychiatrists should be very reluctant to use drugs, apart from a benzodiazepine for a few days.

An Icelandic psychiatrist told me that when he worked at a psychosis ward in London, he and his colleagues waited on average about two weeks before starting psychosis medication on newly admitted people. Most people chose to take some medication, but often in very small doses, so it is very well possible that it was respect, time and shelter that helped the patients, not the “subtreatment threshold doses.”

Psychiatrist Simon Wilkinson from Akershus University Hospital in Norway told me that they don’t have a regime for rapid tranquillisation and have never needed one.

It is all a question of the prevailing culture. Psychiatrists could do vastly better by meeting the patients where they are while mustering all the respect and empathy they can, without forced drugging.

Psychotherapy and caring

As noted above, psychotherapy was generally not a stand-alone treatment option but a supplement to pills.

This is a serious blunder. The authors of the Cochrane review, which pointed out that we don't have evidence that psychosis drugs in an acute early episode of schizophrenia is effective,[154] included a randomised trial by Loren Mosher in their review.[250] Mosher compared 55 patients in hospital, all of whom received psychosis drugs, with 45 patients treated in a non-hospital milieu where 67% did not receive psychosis drugs, and the results after six weeks were virtually the same.

Mosher wasn't against using psychosis drugs.[7:168] He opened a 12-room Soteria house in 1971, as he wanted to treat acutely psychotic people in a humanistic way with empathy and caring. 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' stories, which often revealed traumas with abuse and extreme social failure.[251] Thus, Mosher paved the way for the Open Dialogue approach (see page 50).

The good results obtained by Mosher, also after the randomised trial, by avoiding using psychosis drugs were too threatening to other psychiatrists.[1] His patients had fewer relapses and functioned better in society in terms of holding a job and attending school than those on drugs. It was offensive to the psychiatrists to suggest that ordinary people could help crazy people more than psychiatrists with their 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 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.[1] This is the standard method used in healthcare by those who hold the power when the results of a project threaten 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 who was a treasure for the patients, and a bitter Mosher said 25 year later: "If we were getting outcomes this good, then I must not be an honest scientist."[1:224] The NIMH made Mosher an outcast and threw him out of the NIMH three years later. Others in America who questioned the merits of psychosis pills learned quickly that this would not advance their career, and NIMH did not allot any more funds to this type of project.[5] Many years later, the first author on the Cochrane review analysed the follow-up data from Mosher's study and discovered that they were even more positive than what Mosher had published.[1:225]

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.[252]

It wasn't until 2014 that the first trial of psychotherapy in people with schizophrenia who were not on drugs was published.[253] All the patients had declined to be treated with drugs. The effect size was 0.46 compared to treatment as usual, about the same as that seen in seriously flawed trials comparing psychosis pills with placebo, which is a median of 0.44.[254]

This means that the effect of psychotherapy is likely better than the effect of pills.

US Psychiatrist Peter Breggin has described what a remarkable effect empathy, caring and understanding can have in patients with severe schizophrenia.[135] As an 18-year old college freshman without 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 to be true 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. They 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 patients returned to the hospital.

Breggin's 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 the NIMH. Ever since, the focus has been on co-operative efforts with the drug industry to promote biochemical explanations and drugs.

Psychiatrists have found out recently that if they talk more with their patients with schizophrenia, there is less need of forced treatment. Merete Nordentoft conveyed this positive experience in a TV debate with me. I wondered why this was something psychiatrists should discover. Shouldn't they have known this all along?

8 Depression and mania (affective disorders)

Depression pills are the most widely used psychiatric drugs, and brain scan studies play a major role when psychiatrists try to convince the world that these drugs are very useful and necessary.

In Chapter 3, I rejected the textbook claims that affective disorders may cause brain atrophy and other neurobiological changes. Only very rarely was there any admission or consideration that these changes might be caused by the pills rather than by the disease.

The textbooks had an array of extraordinary claims about what depression pills can accomplish in the brain. But there were no references, and what was claimed is highly unlikely to be true.

We are told that depression pills have an effect on neuroplasticity; that the pills stimulate the formation of new neurons and dendrites in the hippocampus;[16:558] that brain scans have shown a decrease in atrophic changes with treatment; that animal studies have shown a very clear neuroprotective effect of the pills; that the pills can prevent nerve cell death, atrophy of nerve cells, and decreased neurogenesis, glial cell genesis, and angiogenesis; that there is much to suggest that treatment can lessen the structural pathological changes;[16:267] that treatment can prevent deterioration if there are white matter lesions on an MR scan;[18:121] and that the atrophy of the hippocampus, which can be seen in long-term untreated depression, declines during effective treatment.[18:126]

The claim that the atrophy declines during effective treatment is a tautology. If it does not decline, the treatment was not effective. Evidence-based medicine (EBM) is about what a treatment does, on average. Does the treatment heal the claimed atrophy in the hippocampus compared to a group that was treated with placebo? We don't know because such a trial has never been carried out.

We need not waste our time trying to find out which studies the psychiatrists didn't quote, as we already know that brain imaging studies are grossly unreliable (see Chapter 3). Furthermore, if depression pills have no clinically relevant effects on depression; do not increase the patients' quality of life; have common and disturbing adverse effects; and increase the risk of suicide, it is immaterial what happens in the brain.

This is exactly the case,[6,7] which I shall demonstrate below. But first: why are so many people depressed?

Well, they aren't really. Heavily pushed by the drug industry via psychiatric leaders on drug company payroll,[6] the criteria for a depression diagnosis have been markedly lowered over the years, so that it now takes very little to get a diagnosis. Before we had depression pills, very few citizens ever got a diagnosis of depression.[2] It was what we today call very severe depression, previously called melancholia, where people are unable to work for months. Many people feel sad from time to time, which is natural. This is not a disease, but today it is called a disease named not only depression but *major* depressive disorder to underline that you need professional help. Who would decline help if suffering from major cardiac disorder or from a major bone fracture?

In 2010, the US Centers for Disease Control and Prevention (CDC) published a report stating that 9% of the interviewed adults met the criteria for current depression.[255] Do we believe that one-tenth of the US adult population is depressed at any one time?

We should reject this idea. The criteria the CDC used were those listed in DSM-IV (The Patient Health Questionnaire, PHQ-9) and very little was needed. You were depressed if you had had little interest or pleasure in doing things for more than half of the days over the past two weeks plus

one additional 'symptom', which could be many things, for example, trouble falling asleep, or poor appetite or overeating. Little interest or pleasure in doing things for 8 days out of 14 will happen for most people sometimes. Trouble falling asleep is common, and many people overeat.

There is a substantial risk of circular evidence in all of this. In an interview, *The creation of the Prozac myth*, David Healy explained that 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.[256] The drug companies do not primarily sell drugs, they sell diagnoses, which is far more lucrative, and they sell lies about their drugs.[1-11]

In 2013, I was invited to speak at the Selling Sickness conference in Washington DC organised by Kim Witczak, whose husband Woody was driven into suicide by sertraline that was prescribed for insomnia but caused akathisia.[7-89] Another speaker was science journalist Alan Cassels, co-author of the book, *Selling Sickness: How the world's biggest pharmaceutical companies are turning us all into patients*.[129]

The other author of Alan's book was science journalist Ray Moynihan who played the role of a patient in a video about a new epidemic – motivational deficiency disorder.[257] 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." Moynihan described the new disorder in the *BMJ's* 1 April issue in 2006,[258] and some people believed it was a true disease and asked where they could buy the drug against it, Indolebant.

Another video illustrated 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 avafynetyme HCl (have a fine time plus hydrochloric acid).[259,260] 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 that this drug was also for real and folded it into websites for panic and anxiety disorder or for depression.

I showed the two videos as an introduction to my talk about overdiagnosis and overtreatment when I lectured for over 100 psychiatrists in a hospital in Copenhagen in 2012. They laughed out loud but not when I added that what they had just seen wasn't far from their everyday practice. All psychiatrists and family doctors should see these two videos as an antidote against the pervasive influence from the drug industry and their peers.

Bipolar in children rose 35-fold in 17 years in the United States,[1] which is not only because of looser diagnostic criteria. Both SSRIs[261] and ADHD drugs[34] may cause mania, and their harms may lead to a diagnosis of bipolar disorder in one out of ten young people.[262] However, leading psychiatrists hail this as "better" diagnosis, or they say that the drug unmasked the diagnosis.[5:235] That psychiatrists are able to turn even serious drug harms around and make them look like benefits mirrors how the drug industry operates.

In 1987, just before the SSRIs came on the market, only 16,200 children were disabled mentally ill in the United States; 20 years later, it was 561,569, a 35-fold increase.[5:8]

In Denmark, sales of depression pills are now so high that 8.5% of the entire population can be in treatment with an adult dose every day for their entire life.[263] This means that every Dane could be in treatment for 7 years. If this cannot wake people up, what can?

The drug companies are the drivers of this colossal overtreatment. In the period when the sales of SSRIs increased almost linearly by a factor of 18, the number of products on the market - and therefore the marketing pressure - increased by a factor of 16 (r = 0.97, almost perfect correlation).[264] In the United States, the use of SSRIs and similar drugs almost trebled in primary care between 1989 and 2000, with each new agent adding to the aggregate use without a concomitant decrease in previously introduced newer agents.[265]

Depression pills don't have clinically relevant effects on depression

Recent sharp increases in depression pill use have been accompanied by increased prevalence and duration of depressive episodes and rising levels of sickness absence.[1:8,24]

This is a general phenomenon for psychiatric drugs. In all countries where this relationship has been examined, the increased use of psychiatric drugs has been accompanied by an increase in disability pensions for mental health reasons.[119:24] This is one among many indicators that the way we use psychiatric drugs causes more harm than good.

The placebo-controlled trials of depression pills are not of much use. As explained in Chapter 6, they are flawed for eight major reasons, which include the use of rating scales, lack of effective blinding in trials called double-blind, and withdrawal effects in the placebo group that are misinterpreted for depression symptoms.

One textbook claimed that imipramine, a tricyclic depression pill, removes the symptoms in patients with severe depression.[18:307] This is impossible. No drug has ever been shown to cure patients with severe depression. But many psychiatrists believe that the old tricyclics, which they rarely use because of their harms, are more effective than selective serotonin reuptake inhibitors (SSRIs) and serotonin and norepinephrine reuptake inhibitors (SNRIs).

This belief is not based on reliable evidence. Half a century ago, trials were performed with tricyclics that were adequately blinded, as the placebo contained atropine,[266] which causes dryness in the mouth and other adverse effects similar to those seen with the tricyclics. The trials were therefore much more reliable than those using conventional placebos.

A review of nine trials (751 patients) with atropine in the placebo failed to demonstrate an effect of tricyclics.[266] The measured effect, a standardised mean difference of 0.17, was not only statistically uncertain (the 95% confidence interval went from 0.00 to 0.34), but so small that even if it were true, it would have no clinical relevance. The effect was 0.39 if all studies were included, but there was a strongly positive trial, and the authors obtained the more reliable result of 0.17 after they had excluded it from the analysis. This is the appropriate thing to do. Fraud is the most common reason that one study is an extreme outlier (in this case, the effect size was 1.1).

An effect of 0.17 is tiny. In the clinical study reports of depression pills I obtained from the European Medicines Agency, the median standard deviation on the Hamilton scale after treatment was 7.5. This means that 0.17 corresponds to 1.3 on the Hamilton scale, which ranges from 0 to 52. The smallest effect that can be perceived on this scale is 5-6.[267] 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 mean there is enough light to read a newspaper and your depression doesn't lift just because your psychiatrist has noticed a tiny change.

The placebo-controlled trials of SSRIs and SNRIs are not only flawed because of the lack of adequate blinding but also because virtually all patients were in treatment with a depression pill before randomisation. This creates a huge bias because of withdrawal effects.[7:244] Many of the withdrawal symptoms are the same as the symptoms that define depression, and the researchers therefore make a wrong conclusion when they say their trial showed that the drug worked (see page 115).

Some meta-analyses have found that the effect of depression pills is larger if the patients are severely depressed,[268-270] and all over the world the pills are recommended for severe and usually also for moderate depression even though one textbook noted that the effect of the pills is the same or less than that of cognitive behavioural therapy in moderate depression.[19:293]

It is difficult to believe that an intervention that doesn't work when tested in patients with all disease severities, including many with severe disease, should work for those most affected. The difference to placebo is only about 2 on the Hamilton scale,[268,271] even though the trials are flawed in favour of active drug.

The reported effect is also small and irrelevant for patients with very severe depression, e.g. only 2.7 for patients with a baseline Hamilton score above 23[268] which, according to the American Psychiatric Association's Handbook of Psychiatric Measures, is very severe depression.[270] The effect is 1.3 for milder degrees of depression,[268] but this difference is likely just a mathematical artefact.[272] Since the baseline scores for severe depression are larger than for mild depression, any bias will influence the measured result more in patients with severe depression than in those with mild depression. If we assume the bias caused by insufficient blinding because of the drugs' adverse effects is 10% when estimating the effect in the drug group,[7:51] and, for the simplicity of the example, that there is no bias in the placebo group and no improvement between baseline and the final visit, then a Hamilton baseline score of 25 would still be 25 after treatment. But because of the bias, there would be a 2.5-point difference between drug and placebo. If the baseline is 15, that difference would only be 1.5.

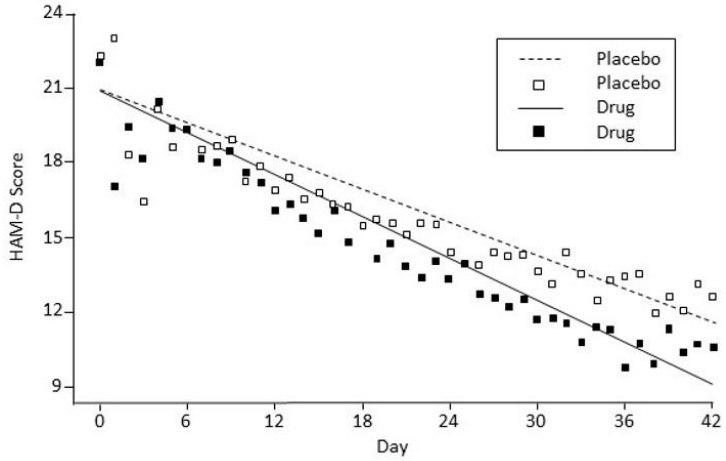
Now that we know that depression pills do not have clinically relevant effects on depression, we may turn to the textbooks. They do not tell us anything of the above.

One textbook claimed that one can notice an improvement on fluoxetine already after a few days.[19:294] This is utter nonsense. Whether the patients are treated with a depression pill or a placebo, it takes about 3 weeks before they become any better, corresponding to the minimal clinically relevant effect of 5-6 on the Hamilton scale (see figure on the next page).[273]

Another book mentioned that most depressions will subside after 2-4 months;[17:357] and a third book noted that 60-80% become healthy after 6-10 weeks.[18:126] None of the books explained that this is not a pill or a placebo effect but the spontaneous remission of the depression.

The latter book was totally dishonest about the benefits of the pills.[18:237] It claimed that psychomotor speed, sleeping pattern, appetite and mood become normalised, and that depressive thoughts about guilt, inferiority and suicide vanish. Nothing becomes normal during pill treatment and the pills double the risk of suicide (see below).

This book also noted that it often takes 2-4 weeks before the effects can be observed, not rarely even longer; and that drugs may often improve cognitive deficits, but that this effect often comes after months.[18:237] This is like selling snake oil. It doesn't work but if you wait long enough, you will be better off.



Depression severity over time in 37 trials of fluoxetine or venlafaxine versus placebo. Redrawn.

It is also misleading to claim that, by testing the patient, one can see an effect earlier than the patient subjectively recognises.[16:273] The doctor's assessment on a rating scale is no less subjective, and what the patient feels about the treatment and its unpleasant adverse effects is more important than what the psychiatrist thinks.

When psychiatrists – rarely – acknowledge that the effect of the pills is small, they often add that this is not important because the patients will benefit from the large placebo effect.

This is a common misconception among doctors and it is due to a logical error. They think the placebo effect is the before-after difference in a group of patients treated with a placebo, which it is not, as the spontaneous improvement is included.

It is difficult to study the placebo effect because we will need an untreated control group to compare with, and such a trial cannot be blinded. One of my PhD students collected all the randomised trials in all diseases that had included both a placebo group and an untreated group and we found that the placebo effect is generally small, if any.[274]

One textbook advised that if a 50% reduction in the Hamilton score has not been obtained after 3-4 weeks, the doctor should try to increase the dose, or switch to a drug with another pharmacodynamic profile, and it claimed that this will result in a satisfactory effect in 60-70% of the patients.[16:273] Yet again, such statements are highly misleading as the spontaneous improvement is included.

This book noted that a dose-response relationship is poorly elucidated for SSRIs, but claimed that escitalopram was a possible exception, and that SNRIs show a clearer dose-response relationship.[16:583] None of this is correct. There are many dose-response studies and they have not shown increased effect with dose (see below).

Another book claimed that a dose increase would lead to full or partial remission in 60-80% of the patients and advised that if a tricyclic had not cured the patient, the patient should be admitted to hospital where the dose could be increased to the upper serum level of what is recommended, or even more.[18:124]

A third book advised to increase to the maximum dose or to switch to a drug from other class.[17:360]

The psychiatric literature is full of advice and claims like this, which are harmful and contrary to the principles of evidence-based medicine.

We can easily see why it is inappropriate to increase the dose using escitalopram as an example because one of textbooks said escitalopram might be an exception.[16:583] The FDA package insert for escitalopram directly contradicts this:[275]

For adults: "Initial: 10 mg once daily. Recommended: 10 mg once daily. Maximum: 20 mg once daily … No additional benefits seen at 20 mg/day dose."

The only thing doctors obtain by increasing the dose is to increase the harms. The package insert noted that in two fixed-dose trials, the overall incidence rates of adverse events was 66% on 10 mg and 86% on 20 mg. The incidence of serious harms, e.g. akathisia and deliberate self-harm, also increase with dose,[276] and self-reports of violence from patients with no apparent background of violent behaviour are also related to dose.[277]

Escitalopram is the S-enantiomer of citalopram, the active half of its two stereoisomers, which are mirror images of each other. The tablets exist in three doses, 5, 10 and 20 mg, which are half the doses of citalopram, 10, 20 and 40 mg, as the inactive moiety is not included.

The initial dose of citalopram is 20 mg once daily, which can be increased to a maximum dose of 40 mg/day.[278] "Doses above 40 mg/day are not recommended due to the risk of QT prolongation. Additionally, the only study pertinent to dose response for effectiveness did not demonstrate an advantage for the 60 mg/day dose over the 40 mg/day."[278]

The package insert for escitalopram mentioned that a cross-over dose-response study in 113 healthy subjects showed that the maximum QTcF change compared to placebo was 4.5 msec on 10 mg and 10.7 msec for 30 mg escitalopram given once daily.[215] Thus, increasing the dose increases the risk of lethal harms for both drugs.

When the fluoxetine trials X065 and HCJE, submitted to obtain approval for using the drug also in children, were being reviewed by FDA, Eli Lilly submitted a license application for R-fluoxetine, an enantiomer of fluoxetine, which was ultimately withdrawn in part because of QTc interval problems.[279] Such problems are an issue with all SSRIs. However, in response to FDA concerns about study HCJE, Lilly argued that the statistically significant increase in mean QTc found with the initial analysis was the product of random variability.[280] FDA's reviewer responded dryly that, with a P-value of 0.009, the result was, by definition, unlikely to be produced by random variability.

When no additional benefit is seen with the 20 mg/day dose of escitalopram then the FDA should also warn against using 40 mg/day of citalopram, the corresponding dose of the parent compound, but there is no such warning.[278]

That no benefit is gained by increasing the citalopram dose from 20 to 40 mg also follows from the shape of the drug's binding curve to brain receptors. As for other drugs, the relationship between receptor occupancy and dose is hyperbolic (see page 163). At 10 mg, 72% of the serotonin receptors are occupied, which increases to 81% with 20 mg and 86% with 40 mg, not much different from 10 mg.[281]

There are many dose-response studies of citalopram and other depression pills and they show that increasing the dose does not increase the effect.[282-287]

In his 2009 book, *The Emperor's New Drugs: Exploding the antidepressant myth*, psychologist Irvin Kirsch explains the fallacy of increasing the dose and why doctors usually do this when their patients do not improve.[127:35] The UK Summary of Product Characteristics for citalopram notes that, "In the fixed dose studies there is a flat dose response curve, providing no suggestion of

advantage in terms of efficacy for using higher than the recommended doses. However, it is clinical experience that up-titrating the dose might be beneficial for some patients."[288]

The summary also has this advice, which comes already on the first page: "The recommended dose is 20 mg daily. In general, improvement in patients starts after one week, but may only become evident from the second week of therapy. As with all antidepressant medicinal products, dosage should be reviewed and adjusted, if necessary, within 3 to 4 weeks of initiation of therapy and thereafter as judged clinically appropriate. Although there may be an increased potential for undesirable effects at higher doses, if after some weeks on the recommended dose insufficient response is seen, some patients may benefit from having their dose increased up to a maximum of 40 mg a day in 20 mg steps according to the patient's response."

The summaries for fluoxetine and paroxetine are much the same, with a recommended dose of 20 mg, but the dose can be increased up to 60 mg and 50 mg, respectively.[289,290]

When doctors increase the dose of depression pills, they are following the manufacturer's misleading advice, echoed by our spineless and much too industry-friendly drug regulators.[2,6,7]

It is pure nonsense when the UK drug regulator states that the improvement in patients starts after one week but may only become evident from the second week of therapy. Absolutely nothing becomes evident at any point in time because the improvement, whether the patient receives a drug or not, is gradual (see the graph above). It is therefore also impossible to provide any meaningful assessment after 3-4 weeks to decide if the dosage should be adjusted, but drug regulators abound in such empty advice. When I was young, a common advice was that drugs should only be used in pregnancy with caution. Either you use a drug, or you don't. You cannot use a drug in pregnancy "with caution."

It is horrendous that a drug regulator, which is supposed to issue instructions based on solid science, says that, "it is clinical experience that up-titrating the dose might be beneficial for some patients." Psychiatrists value their clinical experience a lot without realising how misleading it is, but drug regulators should not support them in this illusion. For an individual patient, the clinician has no idea if the patient improved because of an increase in dose, as they have nothing to compare with, but we know from the randomised trials that this is not the case. The text in package inserts comes from the drug companies selling the drugs, which might be the background for the UK drug regulator's foolish advice.

Kirsch[87] mentions a study conducted by German psychiatrists that illustrates these issues.[291] Depressed patients who failed to respond to paroxetine or maprotiline were given an increased dose of the drug, following which 72% (65/90) of them improved significantly by showing at least a 50% reduction in the Hamilton score. The catch was that this was a randomised trial, and the dose had only been increased for half of the subjects. Yet the response rate was also 72% (60/83) in the group where the dose was not increased.[291]

Receptor occupancy for fluoxetine is very similar for 20 mg, 40 mg and 60 mg.[292] Nonetheless, the UK drug regulator advises doctors to double or triple the dose if the response is insufficient.[289] The only thing they will get out of this is to increase the harms for no increase in benefit while enriching the industry and its friends, some of whom work in drug agencies.[6,7]

Harms are very poorly reported in randomised trials, but it is a basic and logical concept in clinical pharmacology that increased doses cause more harm. Harms are often much better reported in cohort studies, at least if the authors or sponsors had no interests in hiding them.

One such study found that the rate of deliberate self-harm among children and adults up to 24 years of age who were new users and initiated high-dose therapy with citalopram, fluoxetine or

sertraline was twice as high, hazard ratio 2.2 (1.6 to 3.0) as among matched patients initiating therapy with usual doses (20, 20 and 50 mg, respectively).[276] This is a convincing study because depression severity and suicidal ideation at baseline was similar across the dose categories and because any confounding would need to be implausibly large to nullify the findings.

One textbook noted that, for treatment resistant depression, two depression pills could be combined but warned that there is no evidence that "for sure" supports such treatment.[16:275] But we do know for sure that using two drugs instead of one increases the total dose and therefore the harms, for no additional benefit.

The textbooks recommended switching to a drug with another pharmacodynamic profile if the effect is insufficient.[16:273,18:123,18:237]

One book claimed that treatment resistant depression is seen in 30-40% of the patients within 6-8 weeks;[16:275] another book had a lower bet of only 10-20% of the patients.[17:364]

The first book noted that fewer than 20% of the patients would be treatment resistant if a tricyclic was also tried.[16:275] However, this would likely also happen without treatment, as this is the natural course of a depression. If you wait long enough, "treatment resistant" depression disappears in most patients without treatment.

When drugs do not provide meaningful effects, they won't do so by switching between them. A comprehensive 403-page report prepared by McMaster University Evidence-based Practice Center in Canada concluded that, "There is low strength of evidence evaluating relative differences for any monotherapy or combination therapy approach. All but 2 of 44 studies showed no relative differences in response and remission rates."[285]

It was Gordon Guyatt from McMaster University who invented the term evidence-based medicine, in 1992.[293] He advocated for a new paradigm for medical practice, which "de-emphasizes intuition, unsystematic clinical experience, and pathophysiologic rationale as sufficient grounds for clinical decision making and stresses the examination of evidence from clinical research. Evidence-based medicine requires new skills of the physician, including efficient literature searching and the application of formal rules of evidence evaluating the clinical literature."

Rigorous assessment of clinical research is a prerequisite for practicing evidence-based medicine, but critical comments about the research that was quoted were almost totally absent in the textbooks. When the authors used literature references, the research was accepted at face value. My studies of psychiatry have taught me two lessons:

1) Very few psychiatrists have sufficient understanding of the basics in clinical research and can assess what they read critically. They therefore cannot practice evidence-based medicine.

2) Very few psychiatrists read anything at all. They do what their leaders tell them to do who usually do what the industry tells them to do. It is no surprise that psychiatry is a disaster area.

The latest fad in psychiatry is esketamine. It is the S-enantiomer or mirror image of ketamine, a dissociative hallucinogen used as a general anaesthetic for over 50 years.

Esketamine induces dissociative anaesthesia, a trance-like state providing pain relief, sedation, and amnesia. Ketamine is commonly used as a street drug,[294] often called a recreational drug even though it is not particularly recreational to be a drug addict.

In 2019, two psychiatrists praised esketamine for treatment resistant depression in *BMJ*.[295] Together with colleagues, I responded that common sense tells us that a drug cannot possibly

have a dramatic effect on depression within the first day of treatment unless something is terribly wrong.[296]

Esketamine was approved by FDA in March and by EMA in December 2019. It was not mentioned in any of the textbooks, not even in the one from 2021.[20] But the 2018 book mentioned ketamine under hallucinogens in a section about drug abuse.[18:77]

Ketamine seems to work mainly through stimulation of opioid receptors. In a cross-over trial of 12 very severely depressed patients (Hamilton score of 27.4), a colossal "effect" was observed the first day post-infusion of ketamine, a reduction of 22.3, which corresponds to an effect size of 7.0.[294] An effect size of this magnitude is totally unheard of in psychopharmacology, and in the rest of medicine as well. For placebo-controlled trials of depression pills, the effect size is only 0.2 to 0.3, and it takes weeks before this can be measured.[268,271,273]

When ketamine was supplemented with naltrexone (an opioid antagonist), the reduction was smaller, 5.6. As prolonged opioid use can cause depression,[298] long-term use of esketamine might increase the risk of chronic depression.

The two unduly enthusiastic psychiatrists wrote that the effects of ketamine and esketamine fit with "modern theory that depression emerges from an impoverished neural network rather than serotonin deficiency."[295] It is not clear what they meant by this, and newness does not make a theory any more reliable than the discarded hypothesis about a chemical imbalance causing depression (see Chapter 4).

We wrote that we are convinced it could be demonstrated that alcohol, morphine, cocaine and Ecstasy also exert an "effect" on depression within the first day, but that does not make these substances acceptable. They may have acute euphoric effects, but frequent and long-term use often results in dysphoric mood states.

The dream of a quick fix for depression never stops. It has become popular to discuss another hallucinogen for depression, psilocybin, produced by fungi, and even LSD (lysergic acid diethylamide) is being dusted off. In 2020, the authors of a systematic review reported positive results and concluded that LSD is "a potential therapeutic agent in psychiatry."[299]

This is unbelievable. Will psychiatrists ever learn from their mistakes? Psychedelic drugs are not the answer to psychiatric disorders. They make things worse.

Number needed to treat is highly misleading

When psychiatrists want to praise their drugs, they often refer to the number needed to treat (NNT) to benefit one patient, but it is so misleading for psychiatric drugs that any such information should be ignored.[8:85]

Technically, NNT is calculated as the inverse of the benefit difference. If 60% have improved on drug and 50% on placebo, NNT = 1/(0.6-0.5) = 10. Here are the main problems:

1) NNT is derived from flawed trials, with cold turkey in the placebo group, insufficient blinding, and industry sponsorship with data torture and selective publication.[6-8]

2) NNT only takes those patients into account that have improved by a certain amount. If a similar number of patients have deteriorated, there is no NNT, as it would be infinite (1 divided by zero is infinite). If a drug is useless and only makes the condition after treatment more variable, so that more patients improve and more patients deteriorate than in the placebo group, the drug would seem effective based on NNT.

3) NNT opens the door to additional bias. If the chosen cut-off for improvement does not yield the desired result, other cut-offs can be tried till the data confess. Such manipulations with the data during the statistical analysis, where the prespecified outcomes are changed after company employees have seen the data, are very common, also in psychiatry.[6,7,137,279,300]

4) NNT is only about a benefit and completely ignores that drugs have harms, which are much more certain to be experienced than their possible benefits. Thus, in a mathematic sense, the NNT should be negative, but I have never seen a negative NNT in the literature. The NNT is -2 for sexual harms of depression pills (see below), which means that for every two people we do *not* treat, we will spare one from getting sexually harmed.

5) If benefits and harms are combined in a preference measure, it is not likely that an NNT can be calculated because psychiatric drugs do more harm than good. We can only calculate the number needed to harm (NNH). Dropouts during trials of depression pills illustrate this. Since 12% more patients drop out on drug than on placebo,[301] there is a net harm (with a NNH of about 25).

When the top among UK psychiatrists in 2014 tried to convince the world that depression pills are highly effective, they did not take any of these flaws into account.[302] They claimed that depression pills have an impressive effect on recurrence, with an NNT of around three to prevent one recurrence.[302] It was not recurrence but withdrawal symptoms in the placebo group. As only two patients are needed to get one with withdrawal symptoms,[136] there cannot exist an NNT to prevent recurrence, only an NNH, which is two.

There cannot exist an NNT in other depression trials either, as the difference between drug and placebo in flawed trials is about 10%,[303] or an NNT of 10, which is far less than the NNH.

These issues apply to all psychiatric drugs. Thus, NNTs in psychiatry are bogus. They don't exist.

Depression pills lead to dependence

After the authorities at long last in the 1980s, more than 20 years after it had been documented that benzodiazepines cause dependence, admitted that the huge consumption of benzodiazepines was a public health disaster and had started to warn against them, usage went down.[264] At the same time, the American Psychiatric Association tightened the criteria for substance dependence, very conveniently just before the SSRIs appeared on the market.[304]

I have often wondered how much corruption was involved, as this change in the criteria must have been worth many billions of dollars for the companies.

The changes were 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.[304] From being very simple, it became highly complicated, arbitrary and judgmental, and no one can remember all these criteria or apply them consistently from case to case.[7:239]

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[305] – does "often" for paroxetine mean taking three paroxetine pills a day? Hardly).

The new criteria took the power away from the patients to decide for themselves if they had become dependent on depression pills. The time criterion is also foolish. Symptoms should have

persisted for at least one month or should have occurred repeatedly over a longer time. Very many patients are dependent on psychiatric drugs without fulfilling 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 abstinence symptoms they experienced. According to the time criterion, such patients are not dependent, 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 and SNRIs 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.[304] When Lundbeck, which sells several depression pills, was interviewed about our findings, the company called it "nonsense" that people could become dependent on SSRIs.[306]

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, smokers are not dependent on nicotine because they don't increase their consumption of cigarettes, and every smoker could stop smoking overnight, with no ill effects.

To describe similar problems as dependence for benzodiazepines and as withdrawal reactions or the even milder term discontinuation symptoms, invented by Eli Lilly,[307:65] 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. A survey showed that 57% of 493 Danish patients agreed to the sentence: "When you have taken antidepressants over a long period of time it is difficult to stop taking them,"[89] and in another survey, 55% of 1,829 patients in New Zealand taking depression pills mentioned withdrawal effects, which 25% described as severe.[308]

A systematic review showed that half of the patients experience withdrawal symptoms; half of those with symptoms experience the most extreme severity rating on offer; and some people experience withdrawal for months or even years.[136] A survey of 580 people reported that in 16% of the patients, the withdrawal symptoms lasted for over 3 years.[136]

According to Lundbeck, patients who say they have difficulty coming off the drugs talk non-sense,[306] and according to psychiatry professor Lars Kessing, such patients are ignorant.[89] Not much respect for patients in psychiatry.

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."[5:161] But the fact is that the disease has not changed. The psychiatrists and other doctors have failed to understand that they themselves have created an iatrogenic disaster because of their use of depression pills.[7:256] The apparent "chronicity" in mental disorders is an artefact of the medications used.

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.[309] Of those who continued to take drugs, which they were supposed to do according to the guidelines, 60% relapsed in two years. The relapse rate was similar for intermittent users (64%) whereas it was 46% for 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.

Another paper showed that people with uncomplicated episodes of depression (lasting no longer than two months and not including suicidal ideation, psychotic ideation, psychomotor retardation, or feelings of worthlessness) were hardly more likely to have a further episode within

12 months than people with no history of depression, and the relapse rates are very low (3.7% versus 3.0%).[103] Other data show the same.[7:256] In the article, *Medicalising unhappiness*, Allen Frances wrote: "Watchful waiting over multiple visits can enable doctors to see if the problems will resolve without intervention."[103] This is true for all major psychiatric disorders.

We have known for over 50 years that depression pills cause dependence, and the patients have known it, too, but even 50 years after we knew it, the dependence problem was still being trivialised by the UK Royal College of Psychiatrists and the National Institute for Health and Care Excellence (NICE).[8:76] The Royal College of Psychiatrists prioritised the interests of the College and the profession it represents over the wellbeing of patients when they took down an incriminating survey that totally contradicted what they postulated as soon as we had sent a complaint to them. What they falsely claimed was that, "We know that in the vast majority of patients, any unpleasant symptoms experienced on discontinuing antidepressants have resolved within two weeks of stopping treatment."

When the College refused to correct the error, we made our complaint public, and the BBC's Radio 4 program, *Today*, covered it on 3 October 2018. The College refused to participate in the programme.

Later, the Royal Society of Medicine launched a podcast series where the opening topic was about depression pills and withdrawal. One of the two interviewees was psychiatrist Sir Simon Wessely, president of the Royal Society of Medicine (and recent president of the College). Wessely rejected any link between depression pills and suicide and stated, categorically, that depression pills are "not addictive."

Despite the psychiatrists' steadfast denial of the facts, things changed. In September 2019, Public Health England published a 152-page evidence review making important recommendations, including for services to assist people coming off depression pills and other psychiatric drugs, and about better research and more accurate national guidelines.[310] NICE updated its guidelines in line with the evidence the following month.[136]

Drug companies don't care about patient safety if it could harm sales.[6,7] Psychiatric leaders don't care about patient safety if it could threaten their own reputation, the guild they represent, or the flow of money they receive from drug companies. This corruption of a medical specialty also permeates our authorities, which rely heavily on specialists when issuing guidelines and only make changes if critics make a lot of public noise about the wrongdoing.

When the profession cannot avoid addressing public criticism, the replies are often revealing. I have described[7:16,311] how I was met with ad hominem attacks and false and highly misleading scientific arguments[302] from the upper echelons of the UK psychiatric establishment 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: *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 books critical of mainstream psychiatry.[3,4,312,313]

Depression pills don't work for children and double their risk of suicide

One of the textbooks mentioned a meta-analysis of 34 randomised trials of depression pills given to children and adolescents and claimed that fluoxetine was the only drug with a significant effect and also had the highest tolerability.[19:215]

Such claims belong to the section for science fiction. It is fairly impossible for a drug to be both more effective and less harmful than similar drugs from the same class. Even though this textbook had references, there were none to this implausible claim, but I believe the source can only be the 2016 network meta-analysis by Andrea Cipriani and colleagues.[297]

To increase the power of the analyses, the authors included both placebo-controlled trials and head-to-head comparisons, but they were not sufficiently careful. They mostly used published trial reports (only 7 of the included 34 trials were unpublished), which are substantially biased.[2,7,8]

As an example, statistician Hans Melander and colleagues, working for the Swedish drug agency, showed that placebo-controlled trials of SSRIs were more often published when the results were statistically significant, and that many publications ignored the results of intention to treat analyses and reported the more favourable per protocol analyses where only patients who do not drop out of the study are retained in the analysis.[314] This created a misconception about how effective the drugs are.[6:137] Moreover, cross-references to multiple publications of the same trial were missing, and sometimes they had no author names in common and therefore looked like separate trials.

As another example, psychiatrist Erick Turner who worked for the FDA and colleagues noted that 31% of the trials done as part of a licensing application for SSRIs and related drugs viewed by FDA as negative or questionable were published as positive, and the effect size in the published articles was 32% higher than in FDA's reviews of all the trials.[315]

Fluoxetine is unsafe and ineffective and the trials are manipulated

Fluoxetine was approved in the United States in 2002 for depression in children and adolescents based on two placebo-controlled trials, X065 and HCJE, with 96 and 219 participants, respectively, although FDA's statistical reviewer had noted that there wasn't a statistically significant benefit for the drug on the primary outcome in either trial.[316] As both trials appeared to have been misreported in the literature, David Healy and I decided to restore them according to the RIAT initiative (Restoring Invisible and Abandoned Trials).[317]

We reviewed the 3557 pages of clinical study reports Eli Lilly had submitted to the regulators and found serious manipulations with the data, both in the reports and in the publications.[279] Essential information was missing; there was contradictory information, even related to suicide attempts; there were unexplained numerical inconsistencies, which included a mathematical impossibility; there were unexplained exclusions of patients and data, and analyses were called intention-to-treat even though some patients with data were excluded; new outcomes appeared that were not prespecified in the trial protocol (the Texas sharpshooter fraud, see page 40); rating scales and analyses were changed; the trial protocols were violated in other ways; and results that were inconsistent with the conclusion that fluoxetine is safe and effective were side-lined or explained away in a disturbing manner.

The efficacy outcomes were heavily biased in favour of fluoxetine by differential dropouts and missing data at trial endpoint. In trial X065, 6 patients had discontinued on fluoxetine and 12 on placebo after four weeks; in trial HCJE, none had dropped out on fluoxetine versus 10 on placebo after two weeks. Most analyses used the last observation carried forward (LOCF) method, but Lilly did not alert its readers to the large bias this caused: More patients on placebo than on fluoxetine had high depression scores carried forward.

FDA's medical reviewer noted that considerably more patients on placebo than on fluoxetine dropped out and that the pattern was "rather unusual" in trial HCJE because there were more dropouts on placebo than on fluoxetine for adverse events (9 vs 5), patient decision (11 vs 3) and lost to follow up (7 vs 1).[280] In contrast, trial X065 had no dropouts for adverse events on placebo versus 5 on fluoxetine, and there were no losses to follow-up.

Since no statistical adjustments can substitute missing data reliably, we focused on patients with minimal symptoms and those who had recovered after 8 weeks in trial X065 according to Lilly's own criteria. We did not find any differences to placebo.

Lilly's analysis in trial HCJE of the Clinical Global Impressions Efficacy Index, which compares the benefits and harms for each patient, was also misleading. The psychiatrists used an index with 8 categories to "rate overall therapeutic effect in conjunction with side effects for each patient." They assessed for each patient if the improvement in the depression outweighed any drug harms in terms of their interference with daily activities. This subjective process was not defined. Lilly claimed the results indicated that the therapeutic effects outweighed any harms because 58% vs 40% had a favourable score. However, when we combined the data from the 8 categories by subtracting bad outcomes from good outcomes, which was more appropriate, we found that 59% versus 55% had a good outcome (P = 0.58).

Despite all the biases and manipulations we identified, the effects Lilly reported were not clinically relevant. The effect on the Children's Depression Rating Scale-Revised, which is assessed by the psychiatrists or their research assistants, relative to the baseline values was only 4% in both trials for observed cases, and 16% and 9%, respectively, if LOCF is used. By comparison, the least recognisable effect on the equivalent adult scale, the Hamilton depression scale,[267] corresponds to 28% of a median baseline of 25.4 in 35 placebo-controlled trials.[269]

It is more important what the patients think about the effect than what the psychiatrists and Eli Lilly think, and patient ratings did not find fluoxetine effective. Children's Depression Inventory (CDI) was used for those below 13 years of age and Beck Depression Inventory (BDI) for those aged 13 and above. In trial X065, the data were combined in the publication and P = 0.58. In trial HCJE, the children even tended to prefer placebo: "Placebo-treated patients exhibited greater numerical reductions in the change from baseline for CDI and BDI total scores compared with fluoxetine-treated patients."

Suicidal events were missing, both in the study reports and the publications. The published report for trial X065 did not mention that two patients on fluoxetine had attempted suicide, and the adverse events in four additional patients who discontinued fluoxetine were called "minimal," even though three of them developed symptoms of mania and the fourth had a severe rash. Lilly's internal report showed that 32 fluoxetine vs 18 placebo patients experienced at least one adverse event (P = 0.008), 19 vs 6 experienced restlessness (P = 0.005), 9 vs 1 had nightmares (P = 0.02), and 7 vs 4 felt tense inside. These are serious harms. Restlessness, including feeling tense inside, and nightmares increase the risk of suicide and violence.[2,7]

A subsequent publication by Lilly staff was also untrustworthy.[318] It addressed safety in trial HCJE and had other numbers of suicidal events than those in the study report submitted to drug regulators.[279,318]

For trial HCJE, only the 9-week results were fully published, whereas the less positive 19-week results were not. Lilly falsely concluded that "fluoxetine 20 to 60 mg/day is safe" and also that "dose escalation may benefit some patients" even though they only reported on four outcomes for which there were no significant differences.

A 2007 Lilly meta-analysis of violent events, which included all placebo-controlled studies of fluoxetine undertaken in children and adolescents, was also untrustworthy.[319] It is totally implausible that aggression or hostility-related events were experienced by *fewer* children and adolescents treated with fluoxetine, 2.1%, than by those treated with placebo, 3.1%.

Lilly's results contradicted our findings and also FDA's assessment of Lilly's application. FDA created a table of discontinuations because of adverse events in X065, HCJE and HCJW (a trial of obsessive-compulsive disorder comparing fluoxetine 10-60 mg daily with placebo for 13 weeks in 71 vs 32 patients).[280] There were 14 vs 3 discontinuations (P = 0.02, our calculation) among the 228 vs 190 patients for reasons related to suicide and violence (suicide attempt, euphoria, manic reaction, agitation, hyperkinesia, nervousness, personality disorder, hostility, and depression). In these trials, there were three suicide attempts on fluoxetine and one on placebo, and another fluoxetine patient was hospitalised because of suicidality. Six patients (2.6%) on fluoxetine developed mania or hypomania versus none on placebo (P = 0.03).[280]

In our restoration of trials X065 and HCJE, we found that precursors to suicidality or violence occurred more often on fluoxetine than on placebo. For trial HCJE, the number needed to harm was only 6 for nervous system events, 7 for moderate or severe harm, and 10 for severe harm. Fluoxetine reduced height and weight over 19 weeks by 1.0 cm and 1.1 kg, respectively, and prolonged the QT interval.

Lilly claimed in its study report for trial HCJE that "depression is an organic disease that readily responds to treatment" and that "Introduction of effective antidepressant treatments earlier in the progression of the disease state has the potential to effectively treat and control the disease as well as improve daily functioning and overall quality of life."[279] There is no evidence that either of this is true,[7,8] and depression pills seems to worsen quality of life[8] (see below).

If extrapolated from the trial data, the harm fluoxetine causes on growth in children corresponds to an annual loss in height of 2.7 cm and a loss in weight increase of 3.0 kg.[279] FDA requested that Lilly conducted a one-year study of the effect of fluoxetine on growth, which the company declined to do.[280] We do not know if fluoxetine also has deleterious effects on the developing brain but given what we know about psychoactive substances, including alcohol, this is likely.

Based on our reanalysis of the two pivotal trials, we concluded that fluoxetine is unsafe and ineffective.[279] It is a horrible drug.

A textbook described the following treatment priorities for children with affective disorders: 1) psychoeducation and support; 2) cognitive behavioural therapy; 3) drugs.[19:214] However, it also said that first-line therapy in severe depression is a combination of fluoxetine and cognitive behavioural therapy and that, by pronounced suicidal thoughts, hospital admission should be considered due to the risk of worsening suicidal thoughts and plans and a decrease in psychomotor inhibition caused by increasing the drug dose.

It defies logic why an increase in dose is recommended in children who are suicidal when the authors acknowledge that this increases the risk of suicide and when we know that the pills do not even have a beneficial effect on the depression.

This is bad medicine but the lack of logic is ubiquitous. In New Zealand,[8] the drug regulator did not approve the use of fluoxetine for people less than 18 years of age. However, this was no hindrance for the usage of depression pills, which increased by 78% between 2008 and 2016,[320] and a UNICEF report from 2017 showed that New Zealand had the highest suicide rate in the

world among teenagers between 15 and 19, twice higher than in Sweden and four times higher than in Denmark.[321]

I visited John Crawshaw, Director of Mental Health, Chief Psychiatrist and Chief Advisor to the Minister of Health in New Zealand, in February 2018, and I asked him to make it illegal to use these drugs in children to prevent some of the many suicides.[8] He responded that some children were so severely depressed that depression pills should be tried. When I asked what the argument was for driving some of the most depressed children into suicide with pills that didn't work for their depression, Crawshaw became uncomfortable, and the meeting ended soon after.

"Pushing children into suicide with happy pills" is the title for one of the chapters in my 2013 book about organised crime in the drug industry.[6] Doctors cannot do worse than this. Telling children and their parents that happy pills are helpful when they don't work and drive some children into suicide.

The large TADS study of fluoxetine funded by NIH was seriously misreported

There has been one independent trial of fluoxetine, the US National Institutes of Health's Treatment of Adolescent Depression Study (TADS), published in 2004.[322] This trial was very large and has been highly influential.

Adolescents with depression (n = 439) were randomised to four treatment groups: fluoxetine alone (n = 109), cognitive behavioural therapy (CBT) alone (n = 111), fluoxetine with CBT (n = 107) or a pill placebo (n = 112) during TADS' acute 12-weeks phase.

The investigators reported that combination treatment with fluoxetine and CBT "offered the most favourable trade-off between benefit and risk for adolescents with major depressive disorder." However, the reporting has been widely criticised. There are issues with study design, statistical reporting and interpretation, discrepancies between article abstracts and their content in the over 30 publications by the TADS team, and misreporting of harms.[323]

A 2020 systematic review criticised 19 international clinical practice guidelines for their reliance on TADS findings without considering the failure of the TADS authors to report adequately on drug harms.[324]

The TADS authors claimed efficacy and safety for fluoxetine, which is the standard mantra for the drug industry whatever the results are, but both claims are wrong. The effect was not clinically relevant, and there were double as many suicidal events in patients randomised to fluoxetine than in patients randomised to placebo.[322,325]

To this day, the reporting on harms remains highly deficient.[323] Two researchers who wanted to redress this got access to summary data via the National Institutes of Health.[323] These data indicated that, of the 30 serious adverse events recorded during the study's acute phase, 12 were suicide attempts among children taking SSRIs, compared with only two attempts among children not taking SSRIs.

Next, the researchers tried to get access to the case record forms and narratives, which are essential for a rigorous reanalysis, which the MedDRA (Medical Dictionary for Regulatory Activities) coded terms and severity ratings do not allow. The researchers' previous experience with restoring GlaxoSmithKline (GSK) study 329 of paroxetine in children and adolescents had shown that this additional step is very important in order to correct the errors made by the original investigators, which changed the harms significantly in disfavour of paroxetine.[300]

However, Duke University, where the trial data were lodged, refused to hand over serious adverse event forms from the trial even though they had signed an agreement about delivering the data.[323] Their arguments for refusal were invalid.

The researchers then tried to get the missing data from Eli Lilly, which provided the drug for the trial and had received all the reports of serious adverse events from the investigators, but Lilly refused to release the data and also to have any of the correspondence published.

The researchers also tried to get the data from the FDA but were told it would take at least two years before they came up in the queue.

Other depression pills are also unsafe and the paediatric trials are manipulated

The increase in the risk of suicide and violence is not limited to fluoxetine. It is a class effect. My research group used the clinical study reports of the placebo-controlled trials of SSRIs and SNRIs, and we found that these drugs increase suicidality and aggression 2-3 times among children and adolescents, odds ratios 2.39, (1.31 to 4.33) and 2.79 (1.62 to 4.81), respectively.[326]

Prior to the development of the SSRIs, there had been 15 randomised trials of tricyclics and related compounds in children and adolescents, all negative.[327] There was a clinical consensus that children did not get endogenous depression.[279] They might be miserable and unhappy, but this was situational and would respond to psychosocial interventions. Linked to this, there were almost no child psychiatrists with expertise in psychopharmacology.

The SSRIs at that time had an anxiolytic action but were marketed as depression pills in part to skirt around clinical concerns that any new anxiolytic would necessarily produce dependence as the benzodiazepines had.[328] A 2017 meta-analysis of published trials in children and adolescents confirmed that SSRIs are essentially anxiolytic drugs, as the effect sizes were significantly larger for anxiety (0.56) and obsessive-compulsive disorder (0.39) than for depression (0.20).[329]

Our reanalysis of the two pivotal fluoxetine trials[279] made it clear that this drug should never have been approved for use in children and adolescents. But once approved, it paved the way for approval of other ineffective and dangerous SSRIs.

After licensing fluoxetine for children, based on studies negative on their primary endpoint, FDA issued an approvable letter in October 2002 for paroxetine, which came to light because of a court case:[330] "We agree [with GSK] that … the results from Studies 329, 377, and 701 failed to demonstrate the efficacy of Paxil in pediatric patients with MDD [major depressive disorder]. Given the fact that negative trials are frequently seen, even for antidepressant drugs that we know are effective, we agree that it would not be useful to describe these negative trials in labeling."

This is one of the most horrible statements I have ever seen a drug regulator make. "The drug didn't work, but we know it works." If so, then why bother doing randomised trials? This is how practitioners of homeopathy or Chinese medicine and other quacksters argue.

In the initial 2001 publication of study 329, which was a trial of paroxetine in depressed minors, GSK claimed paroxetine was safe and effective.[331] But an internal document from 1998 reveals that GSK knew the study demonstrated its drug to be ineffective, which GSK considered would be commercially unacceptable to publish.[332,333] The document states that the "good bits of the study would be published."

The study was negative for efficacy on all eight protocol-specified outcomes and positive for harm. But GSK tortured the data until they confessed.[332,334] The paper didn't leave any trace of the

torture; in fact, it falsely stated that the new outcomes were declared a priori – a classical Texas sharpshooter fraud.

Based on this information, New York State's Attorney General lodged a fraud action against GSK in 2004.[122] The settlement of this action made it possible to access data on study 329 and restore it in a manner that demonstrated paroxetine's lack of efficacy and increase in suicidal events in contrast to the original publication,[331] which was fraudulent. Seven children on paroxetine versus one on placebo demonstrated suicidal or self-injurious behaviour.[300] In the published paper, five cases of suicidal thoughts and behaviour were listed as "emotional lability" and three additional cases of suicidal ideation or self-harm were called "hospitalisation."[122] 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.

The first author on the fraud, Martin Keller, double-billed his travel expenses; was offered $25,000 for each vulnerable teenager; received hundreds of thousands of dollars to fund research that wasn't being conducted; received hundreds of thousands of dollars from drug companies every year that he didn't disclose; lectured for patients and their relatives on drug company money, which he didn't reveal; and his honoraria were whitewashed.[122]

Keller's misdeeds didn't harm his career likely because his department had received $50 million in research funding. A spokesperson from Brown University School of Medicine said that "Dr Keller's research regarding Paxil complied with Brown's research standards." I see.

The journal that published Keller's paper, *Journal of the American Academy of Child and Adolescent Psychiatry*, was complicit in the fraud. Although the journal's editors were shown evidence that the article misrepresented the science, they refused to convey this information to the medical community and to retract the article.[332] An explanation for this passivity can likely be found by following the money that goes to the journal's owner.

GSK illegally pushed paroxetine for use in children, although it wasn't approved for children, and withheld trial results showing paroxetine was ineffective.[335] The ruthless marketing worked. I have described many heart-breaking stories about children and young adults who were not mentally ill in any way, but who killed themselves by hanging or other violent means because of the harms of the depression pills they took.[6:219,7:79] These people were prescribed depression pills because of insomnia, break-up with a girlfriend, stress at work or at school and other every-day problems.

The approval of fluoxetine for depression in children and adolescents and the publication of many articles since, often ghost written, claiming efficacy for a number of SSRIs swept away the idea of relying on psychotherapy and other forms of support.[279]

The FDA was deceived also by other drug companies. In 2002, when GSK applied to get paroxetine approved for children, FDA wrote to the company:[330]

"You did not provide any analysis of ECG interval data for the controlled studies. The results provided for studies 701 and 704 consisted of a count of the numbers of patients with ECG abnormalities. In study 329, ECG abnormalities were considered adverse events but were not otherwise analyzed. In order to complete our review of this application, we are requesting that you submit the typical kind of analyses conducted for these type of data; i.e., an analysis of mean change from baseline for measured ECG intervals."

The FDA furthermore criticised a table that did not show any data from the placebo groups and listed paroxetine treated children whose adverse events had been coded as hostility, emotional lability or agitation but did not include psychiatric adverse events that were coded under other

terms. FDA requested the narrative case summaries for those events that were either serious or resulted in premature discontinuation.

It is unbelievable that such information was not provided in the application. GSK was also asked to provide its "rationale for coding suicide attempts and other forms of self-injurious behavior under the WHOART term 'emotional lability.'"

Paroxetine seemed to stunt growth, just as we found for fluoxetine. FDA requested GSK to test statistically their data on height and weight and to conduct juvenile animal studies to evaluate paroxetine's effects on growth and neurological, behavioural, and reproductive development. However, as soon as a drug is approved, the drug company tend to "forget" everything the drug regulator has requested. This seemed also to be the case this time. I reviewed FDA's package insert for paroxetine in 2022,[336] and there was nothing that suggested that GSK had done the requested animal studies even though they were very important.

The FDA package insert for fluoxetine shows how dangerous these drugs are.[33] It describes a meta-analysis of short-term placebo-controlled trials. For every 1000 children or adolescents treated with drug instead of placebo for a median duration of only two months, there were 14 additional cases of suicidality. Number needed to harm one kid was therefore only 71.

In 2004, the FDA issued a black-box warning on depression pills based on a meta-analysis that showed that the rate of suicidal thinking or suicidal behaviour was 4% among young patients on a depression pill and only 2% among those on placebo, which was a statistically significant difference.[303,337] However, when FDA published the doubling of the suicide risk in children in a medical journal, they called it a "modestly increased risk."[338]

While the FDA was reviewing the data, the academics at the medical schools who had published positive results of these drugs were worried and issued a report in January 2004 defending the effectiveness of the drugs and disputing evidence that their use increased suicidal behaviour.[339] The academic researchers had contacted the companies to get access to the data they had themselves generated, but some drug companies refused to turn over the data. This decision could not be disputed because the medical schools, in agreeing to run the trials, had signed agreements with the drug makers that kept the data confidential.

Academic medical centres in the United States have set up clinical trials offices and openly court the industry, offering the services of their clinical faculties and easy access to patients.[340] Instead of fighting the corruption of academic integrity, the academics participate in a race to the ethical bottom, making it less and less likely that any outsiders will ever get to see the data. Science has shaded into marketing and the professors end up as promoters while some industry scientists are sickened by the process, they have become involved in,[341] but there is nothing they can do.

The textbook that has only psychiatrists as authors noted that some people experience agitation or anxiety at the beginning of treatment, especially at young ages, with a possible worsening of suicidal thoughts.[18:238] It is not especially in young people; it is not limited to the beginning of treatment but can happen at any time; and it is far worse than just thoughts. Some children kill themselves because of the pills' harms.[7]

It is cruel that most psychiatric leaders say - even on Danish national TV, which Lars Kessing did[342] - that depression pills can be given safely to children because there wasn't a statistically significant increase in suicides in the trials, only in suicidal thoughts and behaviour, as if there is no relation between the two.

The psychiatrists reward the companies for their fraud while they sacrifice the children. We all know that a suicide starts with suicidal thinking followed by preparations and suicide attempts.

Concealing suicide and homicide: fraud, organised crime and FDA's complicity

Fluoxetine (Prozac or Fontex) was the first SSRI that came into widespread use. The story behind it is a grim one which students of psychiatry should know about, also because this drug's approval paved the way for a host of similar drugs. It illustrates that marketing trumps science totally in influencing if doctors use drugs, and if so, which drugs they use.[2,6:202]

Fluoxetine is such a terrible drug that senior management in Eli Lilly wanted to shelve it after having considered to market it for eating disorders.[2] But Lilly was in serious financial trouble, and if fluoxetine failed, Lilly could "go down the tubes."[197,343,344]

The FDA noted serious flaws in Lilly's trials.[2] Patients who didn't do well after two weeks had their code broken, and if they were on placebo, they were switched to fluoxetine.[345] In this way, six weeks of fluoxetine was compared to two weeks on placebo. It also turned out that 25% of the patients had taken an additional drug, and when the FDA in 1985 removed patients on benzodiazepines and other drugs from Lilly's trials, there was no significant effect of fluoxetine.

The FDA went to extremes to make it look like fluoxetine worked.[345] 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.[21]

The German drug regulator considered 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.[2,5,346] This is also the case for other depression pills, and also for children. When the patients evaluate the effect themselves, it is non-existent (effect size 0.05 or 0.06).[347,348,349] Only the psychiatrists think they work (effect sizes 0.25 to 0.29) but they are not the ones to be treated.

When Lilly showed some of its data to Swedish psychiatrists, they laughed and didn't think Lilly was serious.[350] But it was crucial to get fluoxetine approved in Sweden, as it would then be easier to get it approved by the FDA. Lilly's Swedish director, John Virapen, invited doctors to the Caribbean for a week, with plenty of relaxation, including "diving, surfing, sailing, pretty girls and hot nights."[350] He came to Copenhagen to visit me to tell me more about this than he published in his book,[350] and official documents confirm his story.[7:59]

By planting indirect questions to the secretaries of prominent psychiatrists, Virapen identified the independent expert, psychiatrist Anders Forsman, who was going to examine the clinical documentation for the Swedish drug agency. Forsman was one of those who had laughed about the idea of ever getting fluoxetine approved, but already at their second meeting, he suggested $20,000 as a reasonable sum for a speedy approval, which, moreover, shouldn't become known to the taxman but was to be handled by Lilly's office in Genève. He furthermore demanded a good deal of research money. The money was split and the second half was to be paid when the drug was approved. This is how the mob operates when it orders a murder.

Forsman even suggested to falsify the registration application, e.g. suicide attempts were called "miscellaneous effects," and he placed his own personal letter of recommendation.

As the criteria for the depression diagnosis were much more stringent and relevant than today, there weren't many depressed people at the time, and fluoxetine was therefore marketed as a mood lifter, like street pushers sell cocaine.

The approval in Germany also followed "unorthodox lobbying methods exercised on independent members of the regulatory authorities," as Virapen called it.[350] After having been so enormously helpful to Lilly, Virapen was fired. This is also like in the mob. When a lower-ranked person has been asked to murder a well-known political figure, it is safest to kill the assassin afterwards. The official explanation was that Lilly had certain ethical principles. When journalists ask me what I think of the ethical principles of the drug industry, I say I have no answer as I cannot describe what doesn't exist. The only industry principle is money, and the worse the crime, the more money will be earned.[6]

Forsman's name became known in the press, but he just went on and came to work for the court, as a psychiatric assessor for Sweden. Virapen tried to persecute him, but that wasn't possible because he wasn't an employee of the health authority. After this affair, the Swedish anti-corruption law was amended.

Lilly turned their awful drug, which they didn't even like themselves, into a blockbuster, which contributed to making the company one of the world's ten biggest.

Lilly promoted fluoxetine illegally for several non-approved ailments, e.g. shyness, eating disorders and low self-esteem, and concealed that the drug causes suicide and violence.[2,122,351]

In 1990, only two years after fluoxetine came on the market, Martin Teicher et al. described six patients who had become suicidal and reacted in bizarre ways with intense, violent suicidal preoccupation while receiving the drug, which was something completely new to them.[352] Teicher's observations were very convincing. Later, however, internal Lilly documents that came to light during a litigation case[353] revealed that the FDA worked with Lilly on the suicide issue. The psychiatrists Lilly had corrupted came in handy while Lilly's own scientist left out information at the subsequent 1991 FDA hearings that demonstrated that fluoxetine increases the risk of suicide.[122] Earlier, Lilly had submitted data to the German drug agency showing that suicide attempts almost doubled on fluoxetine compared to placebo.

The chair of the FDA committee, psychiatrist Daniel Casey, brutally interrupted Teicher so that he couldn't present his findings and reasons. He was only allowed to present a few slides while Lilly staff presented many. A few years later, Teicher's wife was offered a job at Lilly as their top scientist in oncology without having applied, which she accepted.

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.[353] They 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.[344] 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, which might be the reason why Lilly marketed fluoxetine as a mood lifter.

Already in 1985, an in-house analysis of placebo-controlled trials found 12 suicide attempts on fluoxetine versus one on placebo, but after the code was broken, Lilly's hired consultants threw out six of the attempts on fluoxetine.[111:258]

Lilly was keen to root out the word "suicide" altogether from its database of adverse events experienced by patients and suggested that, when doctors reported a suicide attempt on fluoxetine, Lilly staff should code it as "overdose."

Lilly's fraud was second to none. It is hardly possible to kill yourself by overdosing fluoxetine, and the suicides occur on normal doses. Furthermore, Lilly excluded 76 of 97 cases of suicidality on fluoxetine in a postmarketing surveillance study it submitted to the FDA.[354,355] Lilly instructed its staff to code "suicidal ideation" as "depression,"[197] which is the usual script for drug companies, drug regulators and psychiatrists: Blame the disease, not the drug.[7:208]

Lilly also kept completed suicides from public view. In 2004, the body of a 19-year-old college student was found hanging by a scarf from a shower rod in an Indianapolis laboratory run by Lilly.[354] She had entered a clinical study as a healthy volunteer in order to help pay her college tuition after having undergone thorough medical testing to screen out depression or suicidal tendencies. She had taken duloxetine, another Lilly drug. When a *BMJ* journalist, Jeanne Lenzer, filed Freedom of Information Act requests for all safety data related to duloxetine she received a database that included 41 deaths and 13 suicides. Missing from the database was any record of the college student and at least four other volunteers known to have committed suicide while taking duloxetine for depression.[354]

One of the leaked documents noted that in clinical trials, 38% of fluoxetine-treated patients reported new activation compared to only 19% of placebo-treated patients. Activation may lead to agitation or akathisia, and Lilly recommended early on that, in their trials of fluoxetine, such patients should also take benzodiazepines,[2] which reduce the symptoms. We therefore don't know what the true extent of akathisia is. Other companies adopted the same strategy, and minor tranquillisers were permitted in 84% of placebo-controlled trials of depression pills.[356]

Lilly's widespread criminal activities and corruption of doctors worked. In 1997, Prozac was the fifth most prescribed drug in the United States.[357] It also became the most complained-about drug.[1:287]

In relation to lawsuits, Healy found early drafts of Prozac's package insert that stated that psychosis might be precipitated in susceptible patients by depression pills.[357] The warning about psychosis wasn't included in the final package insert for the United States, and is not even included today,[33] whereas the German drug agency required it. By 1999, the FDA had received reports of over 2000 Prozac-associated suicides and a quarter of the reports specifically referred to agitation and akathisia.[2:171] As always, the FDA protected the drug and not the patients, as it 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.[357]

Other companies also indulged in fraud and organised crime.[6:208] SmithKline Beecham, later merged into GSK, started marketing paroxetine (Paxil or Seroxat) in 1992 and falsely claimed for the next 10 years that it wasn't habit forming[358] even though the licence application showed that paroxetine leads to withdrawal reactions in 30% of the patients.[359] The UK drug regulator also denied there was a problem whereas the BBC reported in 2001 that WHO had found Paxil to have the hardest withdrawal problems of any depression pill. Until 2003, the UK drug regulator propagated the falsehood that SSRIs are not addictive, but the same year, WHO published a report that noted that three SSRIs (fluoxetine, paroxetine and sertraline) were among the top 30 highest-ranking drugs for which drug dependence had ever been reported.[307]

The UK drug regulator also misrepresented the data when it described withdrawal reactions as generally being rare and mild. Independent researchers showed that the reactions had been classified as moderate in 60% of the cases and as severe in 20% by the same UK regulator that told the public that they were mild.[360]

In 2003, 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%,[307] a 100 times increase.

From 2002 onwards, the BBC presented four documentaries about SSRIs in its Panorama series, the first one called *Secrets of Seroxat*. The GSK spokesperson, doctor Alastair Benbow, lied in front of a running camera. He denied 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 the drug in children.

The drug regulator claimed that this information was completely new to GSK, which, however, had known about it for ten years. In addition, the head of the drug agency echoed the drug companies' false assertion that it was the disease, not the drug, that caused the suicidal events.

US senator Charles Grassley asked GSK for how long the company had known that paroxetine carried a suicide risk.[361] GSK lied when it wrote back that they "detected no signal of any possible association between Paxil and suicidality in adult patients until late February 2006." Government investigators found that the company had the data in 1998 and Healy found evidence in internal company documents that 25% of healthy volunteers experienced agitation and other symptoms of akathisia while taking paroxetine.[357]

Healy performed a study of sertraline in 20 healthy volunteers, and to his big surprise two of them became suicidal.[2:179] One 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 in healthy volunteers showed similar deleterious effects, but they hid most of the data in company files.

Drug regulators also hid the lethal harms. When FDA reviewers and independent researchers had found that the drug companies had concealed cases of suicidal thoughts and acts by labelling them "emotional lability," the FDA bosses suppressed this information.[2,362] When FDA's own safety officer Andrew Mosholder concluded that SSRIs increase the suicide risk 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.[355,363]

There were other types of fraud. In data submitted by GSK to the FDA in the late 1980s and early 1990s, the company had added suicide attempts from the washout period before the patients were randomised to the results for the placebo group, but not to those for the paroxetine group. At least three companies, GSK, Lilly and Pfizer, added cases of suicide and suicide attempts in patients to the placebo arm of their trials, although they didn't occur while the patients were randomised to placebo.[2,141,353,364,365]

Healy wrote in 2002[364] that, based on data he had obtained from the FDA, three of five suicide attempts on placebo in a sertraline trial[366] had occurred during washout rather than on placebo and that two suicides and three of six attempts on placebo in a paroxetine trial[366] had also occurred in the washout period. Healy's observations weren't denied by Pfizer and GSK,[367,368] but GSK provided another glaring example that their lies are not of this world:[368]

The "drug" v. "true placebo" analysis Dr Healy describes is not only scientifically invalid, but also misleading. 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.

The systematic fraud can be important for the companies in court cases. In 2001, when a man on paroxetine had murdered his wife, daughter and granddaughter and committed suicide, GSK said in its defence that its trials didn't show an increased risk of suicide on paroxetine.[369] This seemed to be incorrect. In 2004, a researcher published a meta-analysis based on the full reports of GSK's trials that were made available on the Internet as a result of litigation. He found that paroxetine increased significantly suicidal tendencies, odds ratio 2.77 (1.03 to 7.41).[370]

The clinical study reports we analysed also included trials in adults.[326] We could not address the harms fully because some of them appeared only in patient listings in appendices, which we had for only 32 of our 70 included trials. Furthermore, we didn't have case report forms. But we found many alarming events, which you will never see in medical journals and here are some:

Four deaths were misreported by the company, in all cases favouring the active drug.

A patient receiving venlafaxine attempted suicide by strangulation without forewarning and died five days later in hospital. Although the suicide attempt occurred on day 21 out of the 56 days of randomised treatment, the death was called a post-study event as it occurred in hospital and the drug had been discontinued because of the suicide attempt.

Although patient narratives or individual patient listings showed they were suicide attempts, 27 of 62 such attempts were coded as emotional lability or worsening depression, which is what you see in the publications, not the suicide attempts.

A suicide attempt (intentional overdose with paracetamol in a patient on fluoxetine) was described in the adverse events tables as "elevated liver enzymes," which you can get if you drink alcohol.

For Eli Lilly's drugs, fluoxetine and duloxetine, we compared our findings with the summary trial reports on the company's website. Lilly's reporting was seriously misleading.[8,326] In most cases, adverse events were only shown if they occurred in, for example, at least 5% of the patients. In this way, the companies may avoid reporting many serious harms. Only 2 of 20 suicide attempts (17 on drug, 3 on placebo) were documented. None of 14 suicidal ideation events (11 vs 3) were mentioned, and only 3 akathisia events (15 vs 2) were mentioned.

In three sertraline trials where we had access to both the verbatim and the coded preferred terms, akathisia was coded as "hyperkinesia," and miscoding seemed to have been prevalent also in paroxetine trials since we didn't find a single case of akathisia.

As explained earlier, akathisia increases the risk of suicide, violence and homicide. We could only identify akathisia if we had access to the verbatim terms, but we nonetheless found that, like aggression, akathisia was seen double as often on the pills than on placebo.[326]

It is of particular relevance for the many school shootings that the following events for 11 patients on a depression pill were listed under aggression in patient narratives for serious adverse events: homicidal threat, homicidal ideation, assault, sexual molestation, a threat to take a gun to

school, damage to property, punching household items, aggressive assault, verbally abusive and aggressive threats, and belligerence.

Many of the killers were on depression pills. The authorities routinely hide such information in order not to raise concerns about the safety of the pills, and it therefore took quite a while before we learned that the German Wing pilot that took a whole plane with him when he committed suicide in the Alps, and that the Belgian bus driver who killed many children by driving his bus into a wall, also in the Alps, were both on a depression pill.

In 2014, ten years after the FDA had issued a black-box warning on depression pills because the rate of suicidal thinking or behaviour was double as high among young patients on a depression pill as on placebo,[303,337] a psychiatrist argued in *New England Journal of Medicine* that the FDA should consider removing the warning entirely.[337]

His arguments were untenable. He found it disturbing that the warning had decreased the use of depression pills also in adults, "for whom there is solid evidence of a positive effect of antidepressant medication on suicide risk." As we shall see, the truth is the opposite.

He opined that "the risk posed by untreated depression — in terms of morbidity and mortality — has always been far greater than the very small risk associated with antidepressant treatment. We need to better educate physicians, to help them understand that although they cannot ignore that small risk, they can safely manage it by carefully monitoring their patients, particularly children and adolescents, during pharmacotherapy."

It is typical for the journal, which is so beholden to drug companies that it is nicknamed the *New England Journal of Medicalisation,* to publish such nonsense. The harms are far greater than the benefits, which are invisible, and the suicide risk cannot be safely managed. Many children and young people have committed suicide by violent means, e.g. hanging, while their parents or peers had no idea that they were endangered.[2,7:79]

But this is how psychiatrists and drug regulators think. In 2007, the FDA humbly "proposed" to the drug makers that they update their black box warning:[7,371]

"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 - after 20 years of foot dragging - 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.

The FDA also admitted, at least indirectly, that depression pills increase the suicide risk in adults, too.

Three years earlier, in 2004, FDA issued a warning that depression pills can cause a cluster of activating or stimulating symptoms such as agitation, panic attacks, insomnia and aggressiveness.[353] Such effects were expected, as fluoxetine is similar to cocaine in its effects on serotonin.

However, when EMA in 2000 continued to deny that the use of SSRIs leads to dependence, it nonetheless stated that SSRIs "have been shown to reduce intake of addictive substances like cocaine and ethanol. The interpretation of this aspect is difficult."[372] It is only difficult for those who are so blind that they *will not* see.

It has been difficult to demonstrate the danger of depression pills because many suicidal events are missing in the trials.[2,6,7] This has been demonstrated by the FDA itself. When FDA in 2006 published its meta-analysis of 100,000 patients who had received depression pills or placebo in randomised trials, after having asked the companies how many suicides they had, the suicide rate on the pills was 1 per 10,000 patients.[7,303]

Five years earlier, Thomas Laughren, who chaired the large FDA meta-analysis, published his own meta-analysis of the drugs, based on data in FDA's possession, and this time the suicide rate on pills was 10 per 10,000 patients, or 10 times as many.[373] Laughren interpreted his findings in a dishonest way: "There is obviously no suggestion of an excess suicide risk in placebo-treated patients." Surely not, but there were four times as many *suicides* – not just suicidal thoughts - on depression pills than on placebo, which was statistically significant (P = 0.03, my calculation).[373] Laughren left the FDA and established Laughren Psychopharm Consulting to help pharmaceutical companies "meet the high standards of FDA and other regulatory agencies."[7:74] He certainly knows how to speak and behave like a drug company.

What is abundantly clear - and which has been demonstrated by many researchers - is that the companies have deliberately concealed many cases of suicide and suicide attempts in their trials and in their reports to the drug regulators.

It is difficult to comprehend discrepancies of this magnitude, but it can be explained. When the FDA asked the companies to adjudicate possibly suicide-related adverse events, the agency didn't verify if they were correct or if some had been left out. Why would the companies, which had cheated shamelessly earlier about suicidal events caused by their drugs not continue cheating when they knew that FDA didn't check what they reported? If they didn't cheat this time, it would be too obvious how much they had cheated earlier.

Another issue is that the collection of adverse events was limited to within one day of stopping randomised treatment, although stopping an SSRI increases the risk of suicide for several weeks. As I have documented in detail, the huge FDA meta-analysis[303] is grossly underestimates the risk of suicide.[6,7] In trials on some drugs included in FDA's analysis, there were *more* suicides than in the *whole* FDA analysis of *all* the drugs. For example, a memo from Lilly Germany listed nine suicides in 6993 patients on fluoxetine in the trials.[374] This is a suicide rate 14 times bigger than the five suicides *in total* in FDA's analysis of 52,960 patients on SSRI drugs.[303]

Many suicides disappeared and the data I found were remarkably consistent. There were likely 15 times more suicides on depression pills than reported by the FDA in its large meta-analysis.[7:70] This is an error of 1,400%. The fraud has been so massive that it is difficult to comprehend and it has killed many patients all over the world. I consider it a crime against humanity.

Even missing by far most of the suicides and other suicidal events, FDA found that paroxetine increased suicide attempts significantly in *adults* with psychiatric disorders, odds ratio 2.76 (1.16 to 6.60).[303] GSK limited its analysis to *adults with depression*, but it also found that paroxetine increases suicide attempts, odds ratio 6.7 (1.1 to 149.4).[375] GSK USA sent a "Dear Doctor" letter that pointed out that the risk of suicidal behaviour was increased also *above* age 24.[376]

Does anyone think that paroxetine is an exception and that all other depression pills do not increase the suicide risk in adults? Apparently, many psychiatrists think so, but this is irrational.

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 the trials had a follow-up phase where all patients could receive the active drug. As those who continue with the drug are those who tolerate it, patient-years are added "for free" to the drug group in terms of suicidality.[7:78]

In 2016, my research group found that, compared to placebo, depression pills double the occurrence of FDA defined precursor events for suicide and violence in healthy adult volunteers.[377] In 2017, we demonstrated with similar methods, based on unpublished clinical study reports submitted to drug regulators, that duloxetine increased the risk of suicide and violence by 4-5 times in middle-aged women with stress urinary incontinence, and that twice as many women experienced a core or potential psychotic event than those who got placebo.[378] Later, the FDA announced that, in the open label extension phase of the randomised trials in urinary incontinence, the suicide attempt rate was 2.6 times higher on duloxetine than for other women of similar age.[379]

Leading psychiatrists did not like our results and criticised our use of precursor events, but this is a red herring. Precursor events are used throughout medicine, e.g. prognostic factors for heart disease. As smoking and inactivity increase the risk of heart attacks, we recommend people to stop smoking and to start exercising.

Suicide attempts and suicides are not only concealed during the trial. Most often, they are also omitted when they occur just after the randomised phase is over.[8:52] When Pfizer in 2009 did a meta-analysis of its trials of sertraline used in adults, they reported a halving of the suicidal events (risk ratio 0.52).[380] But when they included events occurring up to 30 days after the trial phase ended, there was an increase in suicidality events of about 50% (risk ratio 1.47).

A 2005 meta-analysis conducted by independent researchers using UK drug regulator data found a doubling in suicide or self-harm when subsequent events were included.[381] These researchers noted that the companies had underreported the suicide risk in their trials, and they also found that nonfatal self-harm and suicidality were seriously underreported compared to reported suicides.

Another 2005 meta-analysis was also carried out by independent researchers but this time of the published trials.[382] It found double as many suicide attempts on drug than on placebo, odds ratio (which is the same as risk ratio when events are rare) 2.28 (1.14 to 4.55). The investigators reported that many suicide attempts must have been missing. Some of the trial investigators told them that there were suicide attempts they had not reported, while others didn't even look for them. Further, events occurring shortly after active treatment was stopped were not counted. These researchers found that, for every 1,000 patients treated for one year, there were 5.6 additional suicide attempts on active drug compared to placebo (all ages). Thus, by treating 179 patients for a year with an SSRI, one additional patient will attempt suicide.

The reason why it is so important to include suicidal events that occur after the randomised phase is over is that it reflects what happens in real life rather than in a tightly controlled trial where the investigators motivate the patients to take every single dose of the trial drug. In real life, patients miss doses because they forget to take the pills to work, school or a weekend stay, or they introduce a drug holiday because the pills have prevented them from having sex.[383]

It differs from trial to trial what happens when it is over. Sometimes, the patients are offered active treatment, sometimes only the treated patients continue with active treatment, and sometimes there is no treatment.

In 2019, two researchers reanalysed the FDA data and included harms occurring during follow-up.[384] Leading psychiatrists disliked the results and criticised the researchers who then published additional analyses.[385] Like other researchers, they found that suicide events had been manipulated, e.g. they removed two suicides that had erroneously been assigned to the placebo group in the paroxetine data.[385] They reported double as many suicides in the active groups than in the placebo groups, odds ratio 2.48 (1.13 to 5.44).

The suicide issue in relation to depression pills has been one of the most hotly debated issues in psychiatry. But the debate should stop now. Researchers have again and again demonstrated that depression pills double suicides both in children and adults and are even supported by foot-dragging drug regulators in this.[7]

It is very threatening to the psychiatric guild that the most used drugs in psychiatry increase suicides and violence, and the textbooks reflect that, unfortunately, the organised denial continues. They were highly untrustworthy about the suicide risk, which they consistently downplayed or denied to such an extent that the advice was outright dangerous.

One textbook noted that there is an increased risk of suicidal thoughts and behaviours up to 25 years of age,[16:584] which is what the FDA stated in 2004, but many reviews have been published later showing there is no age limit. Two textbooks that referred to this young age group failed to warn that any dose change, also a decrease, increases the suicide risk.[16:538,19:215]

A third textbook mentioned under harms gastrointestinal symptoms, sweating, headache, insomnia, sedation, weight gain, sexual dysfunction, serotonin syndrome, and inner unrest.[17:659] It noted that, in some cases, particularly when treating children and youngsters, akathisia can be seen at the start of treatment, which can be extremely uncomfortable, and that, possibly, the akathisia may even give rise to suicidal thoughts or actions, and it is therefore very important to follow the patients closely at the start of treatment.

There are several errors in this advice. Akathisia is not “particularly” seen in children; it is not “possible” that akathisia can cause suicidality, it is certain; and the patients should not only be followed closely at the start of treatment, but also later, particularly at times of dose changes. In fact, every minute they are on the drug, as suicide can come out of the blue. It is a fake fix.

The level of ignorance and denial about one of the most important issues in psychiatry is astonishing and deadly. One textbook mentioned that there is considerable debate about the suicide risk, and that suicide awareness programmes in Sweden and Germany have educated doctors, increased the use of depression pills, and decreased suicides.[16:538]

This is the UFO trick at its worst. The best evidence we have shows that the pills double suicides, but the psychiatrists used flawed evidence based on before-after studies with no control group that tells them what they want to hear.

One textbook noted that randomised trials have shown that depression pills tend to increase the suicide risk, especially in young age groups, in connection with the start of treatment.[18:132] Yet again: It is not a tendency, it is a fact, and it is not only at the start of treatment.

Later, this book claimed that it is highly disputed if SSRIs can increase suicidal thoughts in the beginning of the treatment even though it acknowledged that large meta-analyses of randomised trials “suggest” that suicidal thoughts and acts can occur.[18:238] All the authors of this book are psychiatrists. They dispute unequivocal facts to protect their guild interests, and to say “suggest” is dishonest. When placebo-controlled trials have proved something, against all odds as no one is

interested in finding out that the pills increase suicides, it is not a suggestion, it is a fact. Moreover, it is not only at the start of treatment; it can occur at any time (see FDA's warning above).[7,371]

This textbook explained that the psychomotor inhibition often subsides before the mood rises, which gives the necessary energy to carry out any suicidal ideation.[18:132] This was also stated in another book, which described an increased suicide risk only at the start of treatment.[19:294] It has never been documented that the pills increase the suicide risk because they remove any psychomotor inhibition. This is part of the psychiatric folklore and a smart way of turning a drug harm into something that looks positive: You see, it is because the drugs are so good, isn't it?

A third textbook was also dangerously wrong. It mentioned that untreated depression can be harmful and cause suicidality and recommended SSRIs.[17:668] In a 20-page chapter about preventing suicides, a psychiatrist and a psychologist claimed that SSRIs seem to reduce the extent of suicidal thoughts.[17:811] They did not provide any references to this blatantly false statement, and in the next sentence, they contradicted themselves by adding that it has not been shown that depression pills or "mood stabilising" medication have an effect on the extent of suicidal behaviour or suicide.

It is a false dichotomy to distinguish between suicidal thoughts or behaviour and suicide. But the nonsense abounds in the literature because the drug industry and the psychiatrists have an interest in ignoring the suicides the pills cause.

Lundbeck's research director, Anders Gersel Pedersen, once argued, in reply to my criticism of Lundbeck,[386] that it has never been shown that there is a clear relationship between suicidal behaviour, suicide attempts and suicide.[7:95,387] But a suicide starts with a thought about suicide, which leads to preparations for suicide, a suicide attempt and suicide. Evidently, the risk factors for serious suicide attempts are very similar to those for suicide,[388,389] and the placebo-controlled trials have shown an increase in suicidal thoughts, suicidal behaviour and suicides.[7,381,382,384,385] That not all meta-analyses have shown a *significant* increase in suicides is only because the drug industry has hidden them. We should not reward the industry for committing fraud that is lethal for our patients, but this is what mainstream psychiatry has done for decades.

It is wrong when the "suicide experts" claimed in this textbook that an effect has not been demonstrated of depression pills or mood stabilising drugs on suicidal behaviour or suicide.[17:811] It is surely an effect, albeit a harmful one, that both depression pills[7, 381,382,384,385] and antiepileptics[390] double the risk of suicide.

One textbook noted that the serotonin metabolite 5-hydroxyindoleacetic acid is decreased in people who have had several suicide attempts or who died by violent methods.[16:537] If this were correct, we would expect SSRIs to decrease the suicide risk, as they increase serotonin, but they do the opposite. The biochemical pseudoexplanations for psychiatric phenomena do not add up.

Leading psychiatrists don't abandon their wrong and dangerous ideas. Leading professors of psychiatry and spokespersons for general practitioners still claim that depression pills *protect* even children and adolescents against suicide,[7,159] and websites are also misleading. Our 2018 review showed that 25 (64%) of 39 popular websites from 10 countries stated that depression pills may cause suicidal ideation, but 23 (92%) of them contained incorrect and sometimes dangerous information.[90] Only two (5%) websites noted that the suicide risk is increased in people of all ages.

A textbook noted that, in most Western countries, the suicide rate dropped markedly while the consumption of depression pills increased.[18:131] This is one of psychiatry's most horrible UFO tricks. There is a wealth of such studies; they are all of poor quality; and some are fraudulent. I discuss these studies over six pages in another book,[7:96] which I shall briefly summarise here.

In a 2011 radio programme, Ulf Wiinberg, the CEO of Lundbeck, which sells several depression pills, claimed 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 they 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 depression pills.

Already back then, only four years into my explorations in psychiatry, I had seen and heard an overwhelming amount of nonsense about psychiatric drugs but this was so much over the top that I published an open letter to Lundbeck about the radio programme on a science website.[386] The next day, Anders Gersel Pedersen, responded,[387] citing several studies that were so deeply flawed that I failed to understand how a research director could misinform to this degree.

An example was a 2007 paper by Robert Gibbons who reported an increase in suicide rates after FDA and EMA in 2003 and 2004 had warned against using depression pills in young people.[391] Critics quickly pointed out the dishonest science Gibbons had employed to make his case.[392] He didn't use the same calendar years for SSRI prescriptions as for suicides, and the fact was that the number of suicides for people below 24 years of age *declined* when the prescribing of SSRIs to youth *decreased*.

This is not the sort of error a scientist accidentally makes. It seems to be a deliberate attempt to tell a story that fits a preconceived end.[392] In the Netherlands, which Pedersen also referred to, the academics were incensed with Gibbons and his statistical antics (Gibbons is 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 found Gibbons' conclusions astonishing and misleading and stated that he and his co-authors had been reckless to publish such claims.[392]

Gibbons has published at least ten papers telling stories that are false.[7:96] Sweden has its own version of Gibbons, Göran Isacsson, who has also published study after study that are entirely misleading.[7:97] Like Gibbons, he has concluded the opposite of what his data show.

So-called experts in suicide prevention aren't any better than Gibbons and Isacsson. They are highly biased towards drug use and cherry-pick the studies they quote despite calling their reviews systematic.[393] Suicide prevention strategies always seem to incorporate the use of depression pills,[393] even though they increase suicides, which also happened in a suicide prevention programme for US war veterans.[394]

One textbook listed 10 risk factors for suicide and commented on suicides during and after hospitalisation,[18:131] but it did not mention the specialty's own contribution to the suicide risk, which is increased 44 times for patients admitted to a psychiatric ward.[247]

Another book was contradictory and lacked important information.[16:538] It claimed that "only a few" randomised trials had been performed of psychosocial and psychotherapeutic interventions to prevent suicide and suicide attempts in risk groups. But on the next page, it stated that "several" trials had been performed in patients with a previous suicide attempt to find treatments that reduce the risk, and that several of these studies had shown an effect of outreach treatment, possibly with home visits, and of cognitive behavioural therapy and dialectical behaviour therapy, specifically for borderline patients.

The authors referred to only one study in their literature list,[395] which was not a randomised trial, but an observational study. Perhaps it played a role for the citation that 10 of the 12 authors

of this study were Danish. It showed that patients who, after deliberate self-harm, received a psychosocial intervention at suicide prevention clinics in Denmark had a significantly lower risk of self-harm, suicide and death by any cause than patients who did not receive such an intervention. The researchers had used a propensity score and 31 matching factors, but no amount of statistical adjustment can correct for the fact that patients who decline to get the intervention will have a poorer prognosis than other patients (confounding by indication).

It is unscientific to write that "several" studies have shown this and that and to quote a flawed study instead of randomised trials. We do systematic reviews of randomised trials to find out what we may conclude when we include *all* relevant studies in our assessments.

Self-harm does not always imply a suicidal intent. My research group therefore did a review of suicidality where we focused on cognitive behavioural therapy because most trials had used this method. We found that psychotherapy halves the risk of a new suicide attempt in people acutely admitted after a suicide attempt.[272]

This is a very important result, and it is not limited to cognitive behavioral therapy. Emotion regulation psychotherapy and dialectical behaviour therapy are also effective for people who harm themselves.[396]

We have the unfortunate situation that mainstream psychiatry recommends depression pills, even for children, to prevent suicide even though they double the suicide risk whereas we do not hear much about using psychotherapy to prevent suicide, even though it halves the suicide risk.

This is a sign of a specialty in ruins. It is also bizarre that when a textbook mentioned that the suicide risk is increased at the start of treatment with depression pills, it added that this is also seen at the start of psychotherapy.[18:132] It looks like an excuse for using harmful pills to postulate that other interventions also increase the suicide risk. There was no reference, but the fact is clear: Psychotherapy *decreases* the risk of suicide.[272]

Since 10% of patients with affective disorders commit suicide, and their life length is reduced by about 10 years,[17:373] it is very important that all psychiatrists become thoroughly educated in psychotherapy. This is currently not the case. Many psychiatrists don't even know how to practice psychotherapy and others have had a short course. I have been taught obstetrics at medical school – a short course - but have never felt qualified to deliver a baby.

In 2015, I arranged an international meeting about psychiatry in Copenhagen in relation to the launch of my first book about psychiatry. Five women who had lost a son, a daughter or a husband, to drug induced suicide, when there was no good reason to prescribe a depression pill, decided to come on their own account and tell their story.[7:79] My program was full, but I made room for them. This was the most moving part of the whole day. There was stunning silence while they recounted their stories, which can be seen on YouTube.[397]

Something can be done. The usage of depression pills in children and adolescents increased by 59% in Denmark from 2006 to 2010, but in the following six years, I constantly made clinicians and the general public in Denmark aware of the suicide risk of depression pills. During this period, the usage dropped by 41% while it increased by 40% in Norway and 82% in Sweden.[8:84,398]

In 2018-19, I alerted the Boards of Health in the Nordic countries, New Zealand, Australia and the UK to the fact that two simple interventions, a reminder from the Danish Board of Health to family doctors and my constant warnings on radio and TV, and in articles, books and lectures, had caused usage of depression pills to children to be almost halved in Denmark, from 2010 to 2016, whereas it increased in other Nordic countries.[399]

I noted that this was a serious matter and explained that "The consequence of the collective, professional denial is that both children and adults commit suicide because of the pills they take in the false belief that they will help them."[7:149]

I urged the boards to act but got no replies, late replies, or meaningless replies that looked like bullshit to me, which philosopher Harry Frankfurt considers short of lying.[400] I received a report from the Swedish Drug Agency that contradicted the package insert for fluoxetine in Sweden, and some of the so-called experts the agency had used had financial ties to manufacturers of depression pills, which they had not declared.

In 2020, I wrote to the boards again, this time attaching a paper I had published about their inaction.[399] The Icelandic Directorate of Health replied that they had asked the psychiatrists in charge of child and adolescent psychiatry to give their opinion nine months earlier, but that they had not responded despite a reminder. Their excuse was that they did not have time. I replied: "They should be ashamed of themselves. Children kill themselves because of the pills and they don't have the time to bother about it. What kind of people are they? Why did they ever become psychiatrists? What a tragedy for the children they are supposed to help."

I informed Whitaker about this. He replied that the inaction by the medical profession regarding the prescribing of psychiatric drugs to children and adolescents is a form of child abuse and neglect, and institutional betrayal.

More about SSRIs and SNRIs causing homicide

Some critical psychiatrists believe that the suicide risk has been better documented than the homicide risk. Perhaps so, but the main reason is that SSRIs and SNRIs cause suicide much more commonly than they cause homicide, which is therefore more difficult to prove.

The evidence, which I have described in detail in another book,[7:103] is nonetheless overwhelming.[2,6,7,21,401,402]

The main mechanisms of action are that depression pills can cause akathisia, emotional blunting and psychosis. Many people who have committed homicide were, by all objective and subjective measures, completely normal before the act, with no precipitating factors; they had akathisia; and they returned to their normal personality when they came off the offending drug.[135,402]

There are numerous reports in the literature and on websites that people of all ages have killed other people or came close to it after having experienced akathisia. Many of these people were healthy and had been prescribed the drug for non-disease-related reasons, e.g. for fun, stress, distress, insomnia, worry, harassment at work, family problems, or economic problems.[2,6,277,402]

In many cases, the treatment provided by the psychiatrists constituted medical malpractice and contributed directly to the violent actions. I was an expert witness in a double homicide case in Holland in 2016[46:114] and emphasized in my written statement that professional malpractice played a crucial role. A mother had killed her two children while she had indisputable symptoms of akathisia on paroxetine but her pleas for help were ignored. After three months on the drug, the mother became suicidal but instead of withdrawing it, her psychiatrist advised continued use.

The mother told two people about nightmares where she slit her children's throats (which she ultimately did, and also tried to commit suicide). Two days prior to the homicides, she reported to her "supervisor" that she was ill and told several people that she was not feeling well. She also went to her family doctor (who had prescribed paroxetine) with her complaints and visited her

company doctor who dismissed her. Finally, she contacted her psychologist who did not have time for her.

It was a gruesome story. She was not herself, which a forensic psychiatrist confirmed three days after the homicides. And her doctors continued to harm her. They stopped paroxetine abruptly when she was in the psychiatric penitentiary six months after the homicides, causing serious harm that persisted for five months. She got a long jail sentence but questions were raised in parliament if the judicial system in Holland was not too harsh. Indeed. She should have been freed for reason of drug induced insanity.

The expert for the prosecution, Anton Loonen, did not have any good arguments against my testimony, which included a criticism of his own report to the court. In the middle of the proceedings, he suddenly handed over a document to the court where he had written in Dutch that he suspected I suffered from a mental disorder that made me seriously disinhibited and advised that I should be examined by a doctor in order to protect myself from myself. This was the third time I had been "diagnosed" by someone with a psychiatric background who did not know me and had not examined me but had some grudge against me.

Another example of medical malpractice is a 26-year old woman who tried to kill her two children on two occasions.[7:105,402] She was prescribed paroxetine for stress but experienced an episode of rage and attempted suicide and then stopped taking the drug. Despite this, she was prescribed paroxetine again two years later and was reassured about its safety. This time she experienced intense restlessness, surges of rage and anger, panic attacks, impulsive spending sprees, and constant suicidal ideation. She overdosed and was admitted to hospital where the paroxetine dose was increased.

She tried to kill herself again and was diagnosed with an "adjustment disorder." She was switched to venlafaxine, and after each dose increase, she was unable to get out of bed (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 product information for venlafaxine fit well with her experiences, e.g. intentional injury, malaise, suicide attempt, depersonalisation, 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 drug harm), akinesia, increased energy, homicidal ideation, and impulse control difficulties.[402]

In 2001, for the first time, a jury found a drug firm liable for deaths caused by a depression pill, paroxetine.[7:106] Donald Schell, aged 60, had been taking it for just 48 hours when he shot and killed his wife, his daughter, his granddaughter and himself.[403] 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 but did not warn about it. Company documents stamped "confidential" showed that some volunteers experienced anxiety, nightmares, hallucinations and other harms – definitely caused by the drug – within two days of taking it, and two of the volunteers attempted suicide after 11 and 18 days, respectively.

However, GSK, which took over SmithKline Beecham, lied blatantly. Even in 2011, ten years after the verdict, GSK denied that paroxetine can cause people to commit homicide or suicide and that there are withdrawal problems.[404]

On the Internet, there is a collection of media stories of massacres, homicides, suicides, and school and college shootings that involve depression pills and ADHD drugs.[405]

Does the disease or the pills increase the risk of dementia?

Three textbooks warned that depression doubles the risk of dementia,[17:358,18:126,20:429] and another book noted that some patients with recurrent depression develop dementia.[16:260] We are also told that if the depression is not treated, the risk increases for new depressions and permanent reduction in the ability to concentrate.[17:358,18:126,18:237]

Only one book had any references to the claim that depression doubles the risk of dementia.[20:429] There were two. The first was to a Danish register study that compared patients admitted to a psychiatric ward with mania or depression with patients who had osteoarthritis or diabetes.[406] The authors argued that treatment of the two latter conditions was not known to increase the risk of cognitive dysfunction, but they said nothing about the risk with psychiatric drugs. They adjusted their analyses for various confounders and noted that drug abuse and alcohol increased the risk of dementia.

In the Discussion, they quoted another researcher who suggested that treatment for depression might increase the risk of dementia. But the Danish researchers had no data on treatment for their own study. They tried to circumvent this essential problem in a most remarkable way:

"If treatment explained the findings in our studies of an increased risk of developing dementia in affective disorder (hypothesis 1), then this treatment should be given for long periods of time to patients with unipolar or bipolar disorders. Antidepressants are usually only given for short periods in patients with bipolar disorder (Frances et al., 1998), however anxiolytics may often be given to both patient groups for a longer time. As indicated by Jorm, the literature is inconsistent as benzodiazepine use has been associated with cognitive decline (Prince et al., 1998) as well as a lower incidence of Alzheimer's disease (Fastbom et al., 1998)."

This explanation was misleading, for at least five reasons:

1) There is no evidence that psychiatric drugs need to be given for a long time before they cause dementia.

2) It is misleading to say that depression pills are usually given short-term to patients with bipolar disorder, as 84% of the included patients in their study were not bipolar but had depression.

3) Depression pills are not given for short periods. In 2006, only 20% of the patients in Denmark who got a prescription for a depression pill were first-time users.[113] Ten years later, 33% of all patients who were prescribed a pill in 2006 had received a new prescription every single year and were still on treatment. And many of them were on treatment also before 2006. I also studied psychosis pills and found the same: 20% first-time users in 2006 and 35% of all users were still on them in 2016. This is iatrogenic harm of epic proportions.

4) The authors wrote that their patients were the most severely affected ones because they had all been hospitalised. Drug usage would therefore be expected to be much more pronounced and long-term in their patients than what I found.

5) Whatever benzodiazepines do to the brain, it is of minor importance in this context because the standard treatments for unipolar and bipolar depression do not include these drugs. They include depression and psychosis pills.

The other study the textbook authors referred to wasn't any better.[407] It was a meta-analysis of case-control studies and cohort studies, which didn't say anything about previous treatments. There wasn't the slightest hint that the increased risk of dementia could be due to the medication rather than to the depression, although this is far more likely. In contrast to the first study, this possibility was not even considered in the paper.

Poul Videbech, an influential depression researcher who edited one of the textbooks,[18] uncritically quoted this meta-analysis as evidence that depression doubles the risk of dementia.[408] He added that depression pills can help the brain regenerate. The wishful thinking in psychiatry has no limits, it seems.

Other harms of depression pills

Other harms of depression pills were also consistently downplayed. One textbook claimed that children may experience mild, often temporary, harms at the start of treatment.[19:294] It is far more important to know about the harms that are *not* temporary, but there was no information about them. A fact box showed harms that occur in over 10% of the children: fatigue, diarrhoea, nausea, dry mouth, drowsiness, headache, dizziness and insomnia.

One book noted that sexual harms are seen in "some" children.[19:294] Some? The pills disrupt the sex lives in about half of those treated.[383] In a carefully conducted study, 59% of 1022 people who had a normal sex life before they came on a depression pill developed sexual disturbances: 57% experienced decreased libido; 57% had delayed orgasm or ejaculation; 46% no orgasm or ejaculation; and 31% had erectile dysfunction or decreased vaginal lubrication.[383] About 40% of the patients considered their sexual dysfunction unacceptable.

The sexual dysfunction can persist long after the patients came off the offending drug and can likely become permanent.[409-411] David Healy has described that, in some unpublished phase 1 trials, over half of the healthy volunteers had severe sexual dysfunction that in some cases lasted after treatment stopped.[410] Rats can become permanently sexually impaired after having been exposed to SSRIs early in life,[412] which we have confirmed in our systematic review of animal studies.[413]

In the upside-down world of psychiatry, the pills that destroy your sex life – which, in contrast to their claimed effect on depression, people can surely feel - are called happy pills.

When the patients find out that they will never again be able to have intercourse, e.g. because of impotence, some kill themselves.[8:170,409,410,414] When I lectured for Australian child psychiatrists in 2015, one of them said he knew three teenagers taking depression pills who had attempted suicide because they couldn't get an erection the first time, they tried to have sex. This is cruel.

About harms, another textbook also mentioned sedation, orthostatic hypotension, cardiac conduction disorders, anticholinergic harms, gastrointestinal harms and serotonin syndrome (which is very dangerous and can be deadly).[16:582]

A third textbook, where all the authors are psychiatrists, was different to the two others. It claimed that SSRIs have few harms, which are rarely severe;[18:124] and that they are first and foremost sexual ones: delayed ejaculation, decreased libido, and difficulty in obtaining orgasm.[18:238]

This is not true. In drug trials, a severe side effect is one that is incapacitating with inability to work or do usual activity. By this definition, is it a severe harm to be unable to have sex, which is a usual activity for most people. And this incapacity is certainly not rare either.

The companies were also untruthful about this predominant problem. 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."[307] The companies claimed that very few patients become sexually disturbed, e.g. only 1.9% in the registration application for fluoxetine,[172] whereas the true occurrence is 30 times higher.

One textbook noted that depression pills can cause mania;[18:113] which in another book was downplayed to short-term hypomanic episodes that may occasionally be seen in association with depression pills.[16:252]

About the prolongation of the QTc interval, we are told that tricyclics can be fatal and that an ECG is therefore needed before starting them (to see if the patient has a genetically determined prolongation of the interval).[18:124] Later, the same book noted that other drugs than tricyclics can cause QTc prolongation in rare cases and that an ECG is recommended if the patient has heart disease, electrolyte disturbances, some other diseases, or is treated with methadone.[18:238] Another textbook only mentioned QT prolongation under tricyclics.[17:660]

This is confusing, and it is not true that SSRIs rarely cause QT prolongation. This is what these drugs do, and it has been known for decades.[279] I therefore believe that if doctors want to prescribe a depression pill – which they shouldn't – they should have an ECG taken before, and not only if there are other problems.

Given these drugs' common and severe harms, we would expect them to decrease the quality of life. However, this was well hidden from public view. We showed in our large systematic review that there is an extreme degree of selective reporting of quality of life not only in the published literature,[326] but even *within* the clinical study reports of the placebo-controlled trials of depression pills.[415]

These drugs likely decrease quality of life. We found that 12% more patients dropped out on drugs than on placebo ($P < 0.000{,}01$).[301] The patients weigh any perceived benefit from the pills against their harms when they decide if they want to continue in a study till the planned end and drop-out for any reason it is therefore a highly relevant outcome. The patients prefer to be treated with a placebo!

Bipolar disorder

The hospital based psychiatry in one of the five regions in Denmark mentions on its homepage that "Drugs for bipolar disorder – mood stabilising drugs - can prevent and cure depression, mania and mixed conditions in most people."[416]

This is very misleading. Psychiatric drugs only have symptomatic effects. They are not disease-modifying and they cannot cure people; they can only lessen some of the symptoms of the emotional pain. Similarly, aspirin cannot cure a broken leg, only lessen the physical pain. Psychiatric drugs cannot prevent psychiatric disorders either.

About bipolar in children, a textbook said that the risk is increased if the children have had hypomanic or manic symptoms after treatment with a depression pill, and that there is a "family relation" between ADHD and bipolar.[19:216] It is not clear what the authors meant by this, e.g. if it is a genetic or environmental issue they describe. They did not mention that the harms of ADHD drugs are much the same as the diagnostic criteria for bipolar, and that many children will there-

fore get a false diagnosis of bipolar that will harm them, as it is treated with lithium, psychosis pills, and antiepileptics.

These are serious omissions. In USA, particularly Joseph Biederman has pushed the diagnosis bipolar in children, which was virtually unknown half a century ago. He and his co-workers made a diagnosis of bipolar in 23% of 128 children with ADHD and reported this in a paper with the telling title, *Attention-deficit hyperactivity disorder and juvenile mania: an overlooked comorbidity?*[417] There is no overlooked comorbidity, only overlooked harms.

One textbook mentioned that beta-blockers, alpha-blockers, prednisolone and cytostatics can elicit and maintain both mania and depression.[17:370] In another book, the same first author, Lars Kessing, noted that beta-blockers, alpha-blockers, adrenal cortex hormones and cytostatics can trigger and maintain mania; that, according to clinical experience, depression pills can trigger mania during treatment of bipolar depression; and that intoxication with central stimulants gives rise to a clinical picture that confusingly resembles mania.[16:292]

This information is seriously misleading. Kessing protected psychiatry's guild interests. He should have said that depression pills and ADHD drugs *in usual dosage* can cause mania or hypomania *when given to anyone* (even healthy volunteers).

The dramatic rise in numbers of patients with a diagnosis of bipolar disorder, previously called manic depression, is a man-made catastrophe. As noted above, this epidemic has hit children particularly hard in the United States where the prevalence rose 35-fold in just 17 years.[1] The fact that doctors in America make this diagnosis in children 100 times more often than in the United Kingdom[418] also illustrates that it is a fake diagnosis in most cases.

A US study of nearly 90,000 patients aged 5 to 29 years showed that treatment with depression pills caused a conversion rate to bipolar of about 5% a year.[262] A systematic review of trials in children and adolescents showed that 8% of people treated with depression pills developed mania or hypomania on drug and only 0.2% on placebo.[419] A systematic review including all ages also found an 8% rate.[420] As already noted, ADHD drugs cause symptoms that are misdiagnosed as bipolar and they can also induce bipolar disorder, as they are stimulants.

Lithium: no reliable evidence that is prevents suicide or dementia

The textbooks advised that patients with bipolar disorder should always be treated with mood preventing [sic] drugs (e.g. lithium),[17:371] or first be treated with lithium,[16:297] also in children from 12 years of age.[19:220] For treatment resistant depression, one book noted that augmentation with another type of drug was best documented for lithium.[16:275]

I tried to find out if these recommendations are based on good evidence, but that was difficult because most trials and meta-analysis are of poor quality.

One of the better meta-analyses was about relapse prevention in bipolar disorder.[421] The authors excluded studies that randomised patients to suddenly discontinuing lithium in the placebo group, which was prudent because the cold turkey symptoms after lithium can be severe and pronounced.[422-427] The authors reported a substantial effect of continued lithium on relapse, risk ratio 0.65 (0.50 to 0.84). However, they did not report which tapering regimes that were used in the trials for those randomised to placebo, and it is therefore impossible to know if there was a true effect on relapse, or the studies just measured what happens when patients on placebo are exposed to a cold turkey. Furthermore, the criteria for relapse were very subjective and such studies are not adequately blinded. I therefore agree with the authors when they noted that "A

wholly unbiased measure of average preventive efficacy would require recruitment of patients without pre-trial exposure to lithium."

One book claimed that lithium prevents suicidal behaviour in children,[19:220] but there is no reliable evidence that this is correct.[428] Another book claimed, without referring to age groups, that Danish and foreign studies suggest that lithium prevents suicide,[16:306] which was called a unique antisuicidal effect 280 pages further ahead.[16:586]

In a chapter about affective disorders, another textbook also claimed that lithium reduces the risk of suicide, according to a foreign and a Danish study.[17:376] The foreign study was not referenced but was likely a meta-analysis of the randomised trials by Cipriani and colleagues,[429] which was not convincing (see below). Instead of referencing this study, one of the two authors quoted his own study, even though it was observational.[430]

It reported that purchasing lithium at least twice was associated with a halving of the suicide rate compared to purchasing lithium only once, a 0.44-fold reduced rate (0.28 to 0.70). This result is unreliable. The authors noted themselves that "Undefined individual factors associated with acceptance and adherence to long-term treatment might tend to select for lower suicide risk during treatment" and that "nonadherence may be associated, for example, with alcoholism, drug addiction, and personality disorders that, in themselves, are associated with an increased risk of suicide."

Furthermore, the relation between number of prescriptions and suicide was not straightforward. The authors noted that "for men, the rate of suicide was greatest for patients purchasing lithium 2 to 5 times whereas patients who purchased lithium 6 to 10 times or 11 or more times had reduced rates as compared with patients who purchased lithium only once." They did not present any data for this curiosity or explain how one jumps from one purchase to at least 6 purchases without passing the dangerous territory of 2 to 5 purchases on the way to safety.

Most importantly, although they showed data for total mortality except suicide, they did not say anything about them. I added the suicides (23 vs 79) to deaths from other causes to get total mortality rates, which were 14.7% (198/1348) among those with only one purchase and 10.5% (1239/11838) among those with two or more purchases (P = 0.000,006). Thus, the group with only one purchase had an extremely poor prognosis. This study is so misleading that it should never have been published.

The claim that lithium prevents suicides has a long and convoluted history. One of the books mentioned that Danish psychiatrist Mogens Schou in 1954 had included placebo in his studies. The book noted that 80% of the patients recovered,[17:910] which is misleading, as it does not take the recovery in the placebo group into account. The authors noted that Schou published his results in 1967,[431] which were later criticised for the methodology. They did not reveal what the problems were.

The lithium story started a little earlier than Schou's studies. Australian physician John Cade fed lithium to guinea pigs and observed that it made them docile.[5:183] In 1949, he reported that he had successfully treated ten manic patients with lithium. But he forgot to mention in his published article that he killed one patient and made two others severely ill.

In 2019, I published a systematic review with a Swedish psychiatrist about lithium's effects on suicide and mortality.[428] We were uncertain about whether lithium worked whereas there was no uncertainty among psychiatric leaders. According to the 2003 practice guidelines from the American Psychiatric Association, "there is strong and consistent evidence in patients with recurring

bipolar disorder and major depressive disorder that long-term maintenance treatment with lithium salts is associated with major reductions in risk of both suicide and suicide attempts."[432]

A systematic review that included 37 observational studies and 8 randomised trials found that the annual suicide risk was 0.4% with lithium treatment and 2.6% without lithium.[433] However, confounding by indication was very likely, e.g. failure to comply with lithium treatment could be associated with a more serious condition with a worse prognosis.

In a 2013 systematic review, Andrea Cipriani and colleagues included 48 trials, 24 of which were placebo controlled, and they found that lithium reduces suicide in people with mood disorders, odds ratio 0.13 (0.03 to 0.66).[429]

However, the trials were small and there were only six suicides in total, all in the placebo group. The authors pointed out that only one or two moderately sized trials with neutral or negative results could materially affect their results. The estimate for total mortality was also uncertain, odds ratio 0.38 (0.15 to 0.95) and was based on only 5 deaths in the lithium groups and 14 in the placebo groups.

There are other reasons for caution. As noted earlier, about half of the deaths and half of the suicides occurring in trials of psychiatric drugs have been left out in published trial reports.[125] In order to address this problem, Cipriani et al. contacted all study authors and manufacturers. They reported that unpublished information was obtained for "most of the studies," which was important for the outcome deliberate self-harm, for which no statistically significant benefit was found. It is not clear if the few suicides were included in this outcome and only one trial provided data for both.[434] Cipriani et al. did not report if their contacts with authors and companies had resulted in additional information about deaths and suicides; if all authors and manufacturers replied; or if they considered that the replies were reliable and comprehensive.

They did not explain either that they had included trials where the patients were already on lithium before they were randomised. Lithium withdrawal may trigger depression and mania,[422-427] which might explain the increased risk of suicide after lithium withdrawal.[426] An observational study found that the median time to disease recurrence was 4 months after abrupt lithium discontinuation (over 1-14 days) and 20 months after more gradual discontinuation (15-30 days),[425] which was still far too quickly.

The withdrawal effects can come quickly. In a study of 18 euthymic patients (17 with a diagnosis of bipolar disorder and one with unipolar disorder) who had received lithium for 3 to 58 months, one developed mania and two developed depression within the first four days after lithium discontinuation.[422] Another study found that the number of suicidal acts per year before lithium was instituted was lower than during the first year after lithium discontinuation (2.3% versus 7.1%).[426]

There were additional problems with the trials Cipriani included. It is not clear if the patients were followed up after the trial ended and if events were included from such follow-up. If people come off lithium abruptly, it will increase the risk of suicide in the lithium group. Furthermore, the review included "enriched studies," which is a euphemism for flawed trials where only patients who respond to lithium and tolerate it are randomised.

We included 45 trials in our review where none of the patients were on lithium before they were randomised to lithium (1978 patients) or placebo (2083 patients). They covered a wide array of diagnoses and phases of the disorders, and some were therapeutic, some about preventing relapses. They were of very poor quality. Only four of the 45 eligible trials reported data on total

mortality or suicides in a total of only 449 participants; the causes of deaths were not clear; and the risk of bias was high or unclear in all four trials.

In one of the trials,[434] there were pronounced differences at baseline between the lithium and the placebo group with respect to previous suicide attempts and personality disorders. With a most remarkable statistical stunt and incorporating "available person-years" in the analysis although it was a randomised trial, the authors managed to turn three suicides versus none into a statistically significant difference (P = 0.049). We used Fisher's Exact test on the same data, which is the appropriate analysis, and got P = 0.12.

Total mortality was significantly lower in the lithium group than in the placebo group (two versus nine deaths, odds ratio 0.28, but the 95% confidence interval was very wide, 0.08 to 0.93. If we included an additional four deaths on lithium in one of the trials that we had excluded according to our protocol because they did not have depressive comorbidity, the odds ratio was 0.70 (0.27 to 1.85). Only one study reported any suicides (none versus three); odds ratio 0.13 (0.01 to 1.27).

That lithium reduced total mortality but not suicides is the opposite of what would be expected if lithium alleviated bipolar symptoms, acute mania in particular, but with somatic harms. Our results could be related to the fact that we based our review on published trial reports. Clinical study reports likely no longer exist because lithium is a very old drug. We asked EMA that replied ten months later that they did not have them.

The investigators might think it is not important to report one or two deaths on lithium, particularly if they believe that the deaths are not related to lithium and also because psychiatrists for many years have believed that lithium saves lives. We cannot know how many deaths that were missing in the 41 lithium trials where there was no information about deaths.

The answer to the question if lithium decreases the risk of suicide and total mortality is: We don't know. New placebo-controlled trials are needed with treatment naïve patients and without any run-in period where all patients receive lithium and become stabilised on the drug. The dose titration should take place after randomisation.

To maintain the blinding, plasma values for lithium should remain blinded for the treating physician. If there is no blinding, or inadequate blinding due to lithium's harms, the use of other treatments, e.g. psychosis pills and electroshock, might differ in the two groups.

Trials should be very large, as suicide is a rare event, and they should last several years, as the outcome might be influenced by study length. If, for example, lithium reduces manic symptoms, it could lead to fewer accidents with a fatal outcome, but also to a higher mortality in the long run because of lithium's toxicity. Furthermore, to obtain information about long-term harms and clinical effects of lithium, trials should end with a long tapering period, and patients should be followed up for several years after they have come off the drug or placebo. Finally, the analysis of the data and the writing of the manuscript should be performed under blind conditions to reduce the risk of reporting bias;[435] details about causes of deaths should be published; and all the raw anonymised patient data should be made freely available so that other researchers may check for themselves whether they agree with the authors.

Does lithium do more good than harm? We cannot use the four trials we found to answer that question. They had highly subjective outcomes, such as if the patients had relapsed or had improved by a certain amount, and the trials must have been poorly blinded because the harms of lithium are pronounced. If we want to know what lithium does to people, we need large trials with

something in the placebo that gives adverse effects so that it is more difficult to break the blinding.

One book noted that bipolar disorder causes dementia, which can likely be prevented with medication, including lithium, which has neuroprotective properties.[18:118] Another book repeated this[16:294,16:586] and claimed that lithium seemed to reduce or totally remove the risk of dementia.[16:294] A third book claimed that newer studies indicate that lithium has a protective effect on brain cells in bipolar patients.[17:662]

There was no documentation for this wishful thinking.

One book claimed that the effect of lithium on acute mania was certain.[18:115] But what does it mean to have an effect on acute mania? There is a Cochrane review of this, which included 36 trials.[436] It is 300 pages, the size of a book, and there are 390 analyses. This is Cochrane cook-book science at its worst. Considering how unreliable psychiatric drug trials are, and how common selective reporting is, this is way over the top.

Lithium was more effective than placebo at inducing a response, odds ratio 2.13 (1.73 to 2.63), but it was less effective than olanzapine, odds ratio 0.44 (0.20 to 0.94) and risperidone, mean difference 7.28 (5.22 to 9.34). Response is a very subjective and biased outcome in trials that are not adequately blinded, and being less effective than major tranquillisers, which do not have clinically relevant effects on psychosis, is not a convincing finding.

The Cochrane authors protected the psychiatric guild by propagating the nonsense I have debunked earlier in this book. They wrote that lithium is a neuroprotective agent in the brain that reduces cell death and enhances new neuronal growth; that functional imaging studies have shown that people treated with lithium have a global increase in grey matter, especially concentrated in the prefrontal cortex, amygdala and hippocampus, which is important because bipolar disorder may well be a neurodegenerative condition. They also wrote that lithium reduces the risk of suicide.

Harms of lithium

Lithium is a highly toxic drug that requires tight monitoring of the serum level. The FDA warns that "lithium toxicity … can occur at doses close to therapeutic levels."[437]

This fact was ignored in a textbook which claimed that lithium is generally well tolerated, and that its harms are few and well known.[18:115] If that were true, it is surprising that 40% of the patients interrupt the treatment prematurely, which the book mentioned on the same page.[18:115]

Another textbook respected the evidence. It mentioned that the most common adverse effects are polydipsia, polyuria, weight increase, hand tremor, gastrointestinal symptoms such as nausea, dyspepsia and diarrhoea, minor oedema, and skin reactions, and that bothering mental harms are difficulty concentrating, affected memory, and decreased vitality and creativity.[17:662] The book noted that long-term harms are more serious: up to 10% of the patients have morphological changes in their kidneys, 1% have irreversible kidney damage, and hypothyroidism and teratogenicity occur in rare cases.[17:662] A third textbook confirmed the risk of malformations.[16:301]

In package inserts, patients and their families are warned that the patient must discontinue lithium therapy and contact the doctor if they experience diarrhoea, vomiting, tremor, mild ataxia (not explained even though few patients know that it means loss of control over bodily movements), drowsiness, or muscular weakness. The risk of lithium toxicity is increased in patients with

renal or cardiovascular disease, severe debilitation or dehydration, or sodium depletion, and for patients receiving medications that may affect kidney function, e.g. some antihypertensives, diuretics and pain-relieving arthritis drugs. Very many drugs can change serum levels of lithium, which is therefore very difficult to use safely.[437]

There are other serious harms, e.g. lithium may cause cardiac conduction disturbances.[16:299]

One book claimed that cessation of lithium therapy increases the risk of a new manic episode beyond the risk associated with the natural course of the disease before lithium therapy.[16:589] There was no reference to this statement, and – as for other psychiatric drugs - it is likely that what is seen when stopping lithium are withdrawal effects rather than relapse. The only relevant reference in this section was not about lithium but a network meta-analysis of psychosis pills in patients with schizophrenia.[218]

Lithium is similar to psychosis pills in its effects, which include emotional blunting, apathy, a decline in cognitive functioning and impoverished lives with little social contact.[5,135] Patients who come off lithium may end up worse than ever before,[3] and the time to a recurrence following lithium withdrawal is several times shorter than it is naturally.[427]

Just like depression and schizophrenia, bipolar disorder appears to have taken a more chronic course because of the drugs being used. Earlier, about one-third of manic patients suffered three or more episodes in their lives, but now it is two-thirds, and depression pills and ADHD drugs may cause rapid cycling between ups and downs.[5]

The list of serious harms lithium can cause is very long and frightening,[437] and we don't know if the brain damage is reversible.[11:204] This is not a drug I would recommend to anyone.

Psychosis pills, antiepileptics and ECT

The textbooks recommended that, instead of lithium, one might use atypical psychosis pills or antiepileptics.[16:297,18:241,19:220] One textbook did not recommend lithium as first choice for mania but psychosis pills, which could be combined with benzodiazepines to avoid high doses.[18:114] I doubt there is any good reason not to use benzodiazepines alone as the idea of treating mania is to calm the patient down, which is a question of dose.

This book noted that patients with mania and depression can usually be treated effectively with "modern" psychotropic drugs, which were claimed to prevent relapse in most patients, but there was no reference to this statement,[18:110] which is false.[438] Further on, it was specified that modern drugs mean psychosis pills.[18:116]

As noted earlier, "modern" is an inappropriate term to use, as it suggests that newer drugs are better than old ones, which is rarely the case, and psychosis pills do not prevent anything apart from letting the patients live more normal and productive lives. This book also claimed that, on medication, most manic episodes were over in 6-8 weeks whereas an untreated manic episode lasted from a few months (most often) to several years.[18:115] Obviously, this claim was not derived from placebo-controlled trials.

One book noted that there is no evidence for using antiepileptics for treatment resistant depression.[16:275] The same book stated that valproate has a well-documented antimanic effect and that lamotrigine is approved for prophylaxis.[16:302] It is not surprising that doctors think antiepileptics work for mania, as everything that knocks people down "works" for mania. The main effect of antiepileptics is that they suppress emotional responsiveness by numbing and sedating people.[135]

Like most other psychiatric drugs, antiepileptics are used for virtually everything. I have seen many patients entering the door of psychiatry with a variety of starting diagnoses – very often depression or nothing at all that qualifies for drug treatment - all ending up being prescribed a gruesome cocktail of drugs that include antiepileptics. Antiepileptics not only sedate people, they can also make them manic[390,439] and thereby give the patients a false diagnosis of bipolar.

The trial literature has been distorted to an extreme degree. For gabapentin (Neurontin), for example, there was selective reporting of trials and of statistical analyses and outcomes that happened to be positive; patients were inappropriately excluded or included in the analyses; and spin made negative results appear positive.[440,441]

Bias was already introduced at the design stage, e.g. by using high doses 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 and company bosses: "We would need to have 'editorial' control;" "The results, if positive, will … be published;" "'We are using a medical agency to put the paper together which we will show to Dr. Reckless. We are not allowing him to write it up himself."

Gabapentin was only approved for people 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.[6:151] Almost 90% of influential thought leaders were willing to tout gabapentin at meetings after having been updated on the company's promotional strategies. A company executive told a salesperson about "Neurontin for everything ... I don't want to hear that safety crap."[442] The company insisted on pressing doctors to use much higher doses of Neurontin than those approved, which means more deaths.

In 2010, a jury found Pfizer guilty of organised crime and a racketeering conspiracy.[443] Six years earlier, Pfizer had paid $430 million to settle charges that it fraudulently promoted Neurontin for unapproved uses.[444]

We have seen similar problems with other drugs. For lamotrigine, seven large, negative trials remained unpublished and invisible for the public, whereas two positive trials were published.[7:193]

Drugs for epilepsy have many harmful effects, e.g. 1 in 14 patients on gabapentin (Neurontin) develops ataxia.[439]

One textbook claimed that some antiepileptics can be used for prophylaxis of bipolar.[18:242] There were no references, but systematic reviews do not seem to provide support to this claim.[445,446] I did not find it worthwhile to go any further, as the trials in this area are of such poor quality that it is a major undertaking to do a systematic review of each agent, and there are many antiepileptic drugs. Furthermore, antiepileptics are so toxic that I doubt their usage can be justified.

One textbook described several harms with antiepileptics,[17:663] but not the most important one, which is that these drugs double the suicide risk. FDA's package insert for pregabalin (marketed with great success by Pfizer under the seducing name Lyrica) mentions a meta-analysis of 199 placebo-controlled clinical trials of 11 antiepileptics that showed an adjusted risk ratio of 1.8 (1.2 to 2.7) for suicidal thinking or behaviour.[390]

Mood stabiliser is a euphemism the psychiatrists never defined. They usually mean antiepileptic drugs and lithium. Eli Lilly also calls olanzapine a mood stabiliser,[7] which is Orwellian newspeak. Psychosis pills don't stabilise anything but sedate people, render them passive, and make it more difficult for them to live normal lives. This term should be abandoned, as it is intensely misleading.

This textbook admitted 345 pages later that there is sparce evidence for an effect of anti-epileptics, but that they are nonetheless used to some extent.[16:577]

I would not recommend antiepileptic drugs for any mental disorder.

A book claimed that ECT is the only monotherapy that is effective in over 60% of the patients.[16:302] Another book went even further and said that 80% of patients with treatment resistant depression responded to ECT,[17:360] which is a meaningless statement, as there is no control group.

One book claimed that there is great potential in preventing more depressions and manias by offering a combination of drugs and psychoeducation as soon as the diagnosis bipolar has been made.[16:307] There is no reliable evidence that drugs can prevent relapse.

Depression pills increase total mortality substantially

In 2015, I tried to find out how many people are killed by the three major drug groups, depression pills, benzodiazepines and similar drugs, and psychosis pills.[7:307] I used the most reliable research I could find and restricted my analyses to patients at least 65 years of age. The estimated number of drug deaths in Denmark (population 5.8 million) based on current usage was 2831 for depression pills, 721 for minor tranquillisers, and 141 for major tranquillisers. I estimated that fluoxetine alone had killed 311,000 people worldwide in the age group 65 and above up to 2004.

The high number of deaths on depression pills may be surprising. It is partly due to the fact that so many elderly people take them (12% in the age group 65 to 79 and 19% in those at least 80 years old).[7:310] A UK cohort study of 60,746 patients older than 65 showed that SSRIs lead to falls more often than if the depression isn't treated, and that the drugs kill 3.6% of patients treated for one year.[447] The study was very carefully done, e.g. the patients were their own control in one of the analyses, which is a good way to remove the effect of confounders.

A textbook advised that in the elderly, we should try a depression pill even on a vague suspicion of depression because it can be difficult to distinguish between dementia and depression, and because the consequences for the patients are very serious if we overlook "this treatable condition."[18:121] This advice is deadly. Even if the death risk in the UK study for some reason was exaggerated, it is the best evidence we have, and we have an obligation to follow the evidence.

The psychiatrists are not keen to hear about how deadly their drugs are, and they did not communicate any data about this in their textbooks. Absolutely nothing.

In October 2017, I gave two invited talks at the World Psychiatric Association's 17th World Congress of Psychiatry in Berlin.[8:27] This was arranged by Peter Lehmann, a German reformer who wants to make psychiatry more human, with self-determination and less use of toxic drugs. He contacted the international advisory committee and urged them to invite "users/survivors of psychiatry" as speakers, which they did, and they also invited me.

I spoke at two symposia. One was an *International withdrawal symposium* that highlighted the increasing gap between knowledge about withdrawal problems and the lack of support for withdrawal. The other was *Responding to the frightening reduction of psychiatric patients' life expectancy*.

When I spoke about withdrawal from psychiatric drugs, there were around 150 psychiatrists in the audience. The atmosphere was hostile, and several people asked irrelevant questions such as if I did not believe that lithium worked?

Fifteen minutes later, I spoke at the other symposium and my title was *Why are psychiatric drugs the third leading cause of death after heart disease and cancer?*[7] Three psychiatrists out of the over 10,000 at the congress attended. They refused to give interviews and carefully avoided being filmed by a documentary film team that followed me to Berlin, as if they were on their way to see a porn movie. This was a no go zone. A taboo.

If you read the package inserts or look up relevant published papers, you will realise that psychiatric drugs have harms that can lead to falls and traffic accidents.[448-453] These harms include sedation, dizziness, orthostatic hypotension, confusion and impaired coordination and balance. Depression drugs double the risk of falls and hip fractures in a dose-dependent manner,[452,453] and within a year after a hip fracture, about one-fifth of the patients will be dead.

These harms won't be noticed by the doctors, as many people fall and break their hip anyway. The drugs are therefore silent killers, and doctors don't learn anything from their much overvalued clinical experience, which, in psychiatry, lead them astray more often than not.

Depression pills do not prevent relapse

It's tricky that withdrawal symptoms and disease symptoms are often the same. If a drug is stopped abruptly or over a short period of time, and the patient becomes depressed, it doesn't mean that the disease has come back.

However, when the patients have tried to stop taking their drug because of its harms or because they feel it doesn't work, psychiatrists, other doctors, social workers and relatives will usually tell them that the symptoms demonstrate that they still need the drug.

It's an uphill battle to try to stop taking a depression pill,[8] but usually, what we are seeing is what I call an abstinence depression. This term is a precise description of what happens, but I might be the only person to use this term. A search on PubMed with *"abstinence depression"* in the Title field yielded no records, and not even a Google search found any. I shall explain below why it is correct to say that patients become dependent on depression pills, even though mainstream psychiatry continues to deny this.[7,8,90]

My new concept should become part of the language psychiatrists use and should be included in the disease manuals. I define abstinence depression as a depression that occurs in a patient who is not currently depressed but whose depression pill is stopped abruptly or over a few weeks. Its hallmark is that the depression-like symptoms come quickly (depending on the half-life of the drug or its active metabolites) and disappear within hours when the full dose is resumed. Re-introducing the drug can therefore be regarded as a diagnostic test separating an abstinence depression from a true depression, as true depressions do not respond quickly to depression pills.

A 1998 trial of 242 patients with remitted depression illustrates the difference between an abstinence depression and a true depression.[45] After they had become well, the patients received open maintenance therapy with fluoxetine, sertraline, or paroxetine for 4-24 months. They then suddenly had their therapy changed to a double-blind placebo for 5-8 days, but the timing of the treatment interruption was unknown to them and their 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.

The three most common withdrawal symptoms were worsened mood, irritability and agitation, which are not signs of a relapse of the depression. As expected, relatively few people had symptoms on fluoxetine:

	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%

Withdrawal symptoms in patients with remitted depression during a 5-8-day placebo period.

On sertraline or paroxetine, 40 of 122 patients had an increase in their Hamilton score of at least 8, which is a clinically relevant increase.

There would have been many more withdrawal symptoms if the drugs had been withdrawn for 2-3 weeks, particularly on fluoxetine, but even with an interruption of only 5-8 days, 25 of the 122 patients on sertraline or paroxetine fulfilled the authors' criteria for depression.

This study shows why doctors get it wrong when they think the disease has come back. We might ask how many patients are likely to get a true depression in a random week in a group of 122 patients whose depressions have remitted. I worked this out based on a study of 362 high school students who had experienced one or more episodes of depression.[454] Of the patients who recovered, 5% relapsed within 6 months and 12% within a year, which suggests a rather constant relapse rate over time. Using these data, I calculated what the expected number of patients relapsing is. This is 122 x 12% x 6.5/365 = 0.03, which suggests that not a single patient of the 25 that "relapsed" in Lilly's study would have relapsed if they had not been exposed to a cold turkey.

Two years later, Eli Lilly conducted another unethical trial with a similar design that harmed the patients.[305] The abstinence symptoms after paroxetine withdrawal were severe. The patients experienced "statistically significantly worsened severity in nausea, unusual dreams, tiredness or fatigue, irritability, unstable or rapidly changing mood, difficulty concentrating, muscle aches, feeling tense, chills, trouble sleeping, agitation and diarrhoea during placebo substitution."

The various harms the patients suffered because of Lilly's cruel trial design increase the risk of suicide, violence and homicide.[7] This was known long before the trials were carried out.[2,7,21]

Unsurprisingly, the patients that had been harmed after withdrawal of paroxetine reported "statistically significant deterioration in functioning at work, relationships, social activities and overall functioning."[21]

It is only in cold turkey trials I have seen such outcomes. According to the American Psychiatric Association's disease manual, DSM-5, major depression is present when the patient exhibits 5 or more of 9 symptoms that "cause clinically significant distress or impairment in social, occupa-

tional, or other important areas of functioning."[8] Given how the disorder is defined, it makes no sense that drug trials avoid using these outcomes, which are far more important and relevant than a score on a rating scale. The reason is of course that the drug industry knows that their pills do not have positive effects on these essential outcomes.

Since psychiatrists usually confuse withdrawal symptoms with relapse, it is not surprising that two textbooks claimed that if the drug is stopped too early,[16:276,17:661] it increases the risk of relapse, and one noted that at least 50% will relapse.[16:276]

The misconception leads to harmful advice about long-term treatment. A continuation phase of 6-12 months after remission is advised,[16:276,18:126] and the longer, the better,[16:276] e.g. by severe depression with imminent suicide risk.[18:126] This advice is deadly. The drugs might push a patient in imminent danger of suicide over the edge. The same textbook claimed, with no references, that the preventative effect of psychotherapy is not so pronounced as that of drugs.[18:126] This false information is also lethal[7,272, 381,382,384,385] because psychotherapy halves the risk of suicide.[272]

If a patient has had two depressions within 5 years, the doctor should consider continuing with the drug for an extra year; if three depressions, for 5-10 years or lifelong.[18:127] If onset after 50-60 years of age, the treatment should be lifelong because the risk of recurrence is almost 100%. It was claimed that an excellent preventative antidepressant effect is achieved. This advice is also lethal because of the high death rate in elderly people given depression drugs.[7:310,447]

The absurdities were endless. A third book recommended continuing with the drug for the same number of years as the number of depressive episodes.[17:360] Even if we imagine there was a drug that worked for depression and prevented new episodes, it would be bizarre. The advice means that the poorer the effect, including no effect, the longer the patient should take the drug. If seven depressions, the patient would be "sentenced" to an additional seven years on the pill and be called treatment resistant as well. This gives connotations to criminal law. The more treatment resistant a criminal is, i.e. the more offences and jail sentences, the longer the last jail sentence will be.

Some pages later, the same book claimed that the risk of relapse in bipolar is about 85% but only 35% when medically treated.[17:377] This is also wrong. All maintenance studies are seriously flawed as they measure withdrawal effects in the placebo group, not relapse.

It was claimed that quetiapine significantly reduces relapse of mania – which is unlikely to be true - and that such an effect has not been shown for other psychosis pills.[16:305] It is even more unlikely that one psychosis pill, and not all the others, should work. This is pain wrong.[436]

This textbook recommended maintenance treatment already after a single manic episode, for 2-10 years or lifelong, unless caused by psychoactive drugs.[16:305] It was not explained if this only means street drugs or could also be prescription drugs, but many psychiatric drugs can cause mania, including depression pills and ADHD drugs.[7]

The book explained that abrupt discontinuation always increases the risk of relapse because it is expected that the disease will last a long time.[16:306] This statement is ludicrous. It is not because the disease will last a long time but because the patients get withdrawal symptoms. This has been described for all psychiatric drug classes.[135]

Gradual cessation over at least four weeks[16:584,19:295] or a couple of months[18:239] was advised, but only one book advised particularly small dose changes by the end.[19:295] One book offered dangerously misleading advice, as it postulated that withdrawal symptoms could be avoided if the

pills were discontinued over two weeks.[17:360] Elsewhere in this book,[17:660] the authors recommended what they called slow withdrawal over 1-2 months, which is not slow.[8,136]

According to the textbooks, about 20-30%[18:239] or one-third[16:584] will get withdrawal symptoms by abrupt cessation. This is not correct either. Half of the patients will suffer from such symptoms after depression pills, and in half of them, they will be very severe.[136]

The information about withdrawal symptoms varied but included dizziness, headache, tiredness, gastrointestinal symptoms, influenza-like symptoms, insomnia, anxiety, irritability, agitation, sweating, sadness, increased dreaming, muscle contractions, and electric zap feelings in the extremities.[16:584,17:360,17:660,18:239,19:295] Absent were the most serious harms, akathisia, increased risk of suicide and violence, and abstinence depression.

Two books claimed that the patients do not become dependent on depression pills,[17:661,18:239] and one of them noted that, because of this, relapse should not be misinterpreted as withdrawal symptoms, and it added that recurrence will typically occur several weeks after stopping treatment.[18:239] A third book noted that withdrawal symptoms usually occur within a few days, varying from one day to two weeks, and that the duration varies from a few days to several weeks.[19:295]

These statements are also wrong. Depression pills lead to dependence (see page 79), and withdrawal symptoms can occur much later, after months, e.g. if the patient becomes stressed, and they may last for years.[8,136] Another book noted that one-third would get withdrawal symptoms if the drug was suddenly stopped and advised tapering over at least four weeks but did not explain how.[16:584]

In one book, the authors warned that about 40% of patients with bipolar stop treatment and that this carries a great risk of new episodes.[16:296] Obviously, the patients don't like the drugs but the psychiatrists don't care.

The widespread professional denial of the drug harms patients experience was displayed when I mentioned on the TV news in 2011 that depression pills can change the personality. In a commentary to this, the president of the Danish Psychiatric Association wrote that it is misleading to focus on a side effect that is so scary for patients, and which is extremely rare.[455]

It isn't. Six years earlier, Danish psychiatrists had published a study in which 43% of 493 patients agreed that the treatment could alter their personality and 42% that they had less control over their thoughts and feelings.[89] 82% agreed that as long as they took the pills, they didn't really know if they were necessary. The patients' replies correspond closely with what other researchers have found,[308] but the Danish psychiatrists refused flatly to believe what the patients had told them. They called the patients ignorant and wrote that the patients needed "psycho-education." However, the relatives had the same opinion as the patients about the pills. Perhaps they should also be taught they were wrong?

The different treatments and combinations

In case of insufficient response in patients with depression or anxiety, one textbook suggested adding another drug (so-called augmentation), e.g. mirtazapine in the evening if the patient cannot sleep.[17:661] It noted that augmentation with lithium, thyroxine or lamotrigine is reserved for doctors with particular experience, and it suggested that a depression pill can be combined with a psychosis pill and that it is often a problem that the patients are underdosed. This advice increases drug harms for no benefit.

The literature is full of studies and meta-analyses claiming that some depression pills are better than others. Almost all of them are financed by the drug industry, either directly or indirectly. Many of the academic authors are on industry payroll as advisors, consultants or lecturers and have little to do with writing the manuscript but just lend their well-known names to it.[2,6,7] This is called guest authorship and those who write the manuscript are often ghost authors, as their names are not in the byline.[140] When a person is acknowledged for her help without specifying for what, or is thanked for "editorial assistance," this person is usually the real author of the paper.

I shall mention a recent network meta-analysis by Cipriani and colleagues, as it got enormous attention in the media. It was published in 2018 in *The Lancet*,[271] which many consider a highly prestigious journal. The authors included 522 trials but by far most of the data came from published trial reports. They reported an effect size of 0.30 for drugs compared to placebo, very similar to earlier meta-analyses.[268,269]

However, even though they found an effect that is far below what is clinically relevant (see page 72), they ranked the drugs according to their effect (response rate) and acceptability (drop-out for any reason), which were the two primary outcomes.

This is futile, and when I first saw this network meta-analysis, my immediate thought was that the authors had rewarded those companies that had cheated the most with their trials, as I indicated in the title when I published my observations.[456] My suspicion was strengthened when I looked at the results in the abstract. The authors claimed that in head-to-head trials, agomelatine, escitalopram, and vortioxetine are more effective than other drugs and that these drugs are also better tolerated than other drugs. One doesn't need to be a clinical pharmacologist to know that this is extremely unlikely to be true. I therefore took a closer look at the three drugs.

Agomelatine was touted in *Lancet* as being an outstanding drug by two authors,[457] one of which was the Australian psychiatrist Ian Hickie who had numerous financial conflicts of interest. They claimed that fewer patients relapsed on agomelatine (24%) than on placebo (50%), but a systematic review by other psychiatrists found no effect on relapse prevention; no effect as evaluated on the Hamilton scale; and also that none of the negative trials had been published.[458] Three pages of letters - which is extraordinary – in *Lancet* pointed out the many flaws in Hickie's review.

Escitalopram and vortioxetine are sold by Lundbeck. It is far-fetched to believe that escitalopram can be better than citalopram because the active substance is the same. As already noted, citalopram is a stereoisomer consisting of an active part and an inactive mirror molecule, and escitalopram only contains the active substance.

When studied by Lundbeck in its own head-to-head trials, and meta-analysed under Lundbeck's control, with Jack M Gorman as first author, the active molecule was better than itself.[459] All three authors of the meta-analysis worked for Forest, Lundbeck's US partner, one as a consultant and the other two in the company.[7:224] What are we supposed to make out of a paper published in a bought supplement to a journal which, on top of this, was edited by a person – the first author of the paper[6] - who was also bought by the company?[459] Absolutely nothing.

Even if Lundbeck's meta-analysis of its own trials is believed, there were no relevant differences between the parent drug and the "me-again" drug.[7:225,460] Four independent reviews of the evidence -- by the FDA, the American advisory group Micromedex, the Stockholm Medical Council and the Danish Institute for Rational Drug Therapy – all concluded that escitalopram offers no significant benefit over its mother molecule.[461]

The Cochrane review of escitalopram is disgraceful. It is from 2009 and has not been updated even though what it says is totally misleading: "Escitalopram was shown to be significantly more effective

than citalopram in achieving acute response (OR 0.67, 95% CI 0.50 to 0.87). Escitalopram was also more effective than citalopram in terms of remission (OR 0.53, 95% CI 0.30 to 0.93)."[462] It's first author is Andrea Cipriani who was also behind the untrustworthy network meta-analyses of depression pills published in *The Lancet* (see pages 82 and 118).

The official task of the government-funded Institute for Rational Drug Therapy is to inform Danish doctors about drugs in an evidence-based fashion. In 2002, the institute noted that escitalopram didn't have clear advantages over the old drug.[463] Lundbeck complained loudly about this in the press and said it was beyond the institute's competence to give statements that could affect the international competition and damage Danish drug exports.[464] It wasn't beyond the institute's competence, but the institute was reprimanded by the Minister of Health, Lars Løkke Rasmussen, who later became Prime Minister. Our highly praised governmental institute was only allowed to tell the truth about imported drugs, not about drugs we export. Principles are only valid if they don't cost too much.

Two years later, the institute announced that escitalopram was better than citalopram and might be tried if the effect of citalopram hadn't been satisfactory.[465] The institute must have stepped on its toes to find a politically correct way to express themselves.[466]

I had a big laugh when I saw the four references in support of the positive statements.[7:226] I laughed again when an employee from the institute was interviewed in the TV news. She was pressured by the journalist who asked her if she couldn't imagine any situation where it might be an advantage that the drug worked faster. Desperate for finding an appropriate answer, she said: "Yes, if a patient is about to throw herself out the window!" This was doubly ironic, as SSRIs increase the risk of suicide.

In 2003, Lundbeck breached the UK industry code of practice in its advertising on five counts, notably by claiming that "Cipralex [escitalopram] is significantly more effective than Cipramil [citalopram] in treating depression."[461] Lundbeck also attributed harms to citalopram in its literature on escitalopram that weren't mentioned in promotional material for citalopram. This confirmed the adage that it's surprising how quickly a good drug becomes a bad drug when the patent expires.

In 2013, the European Commission imposed a fine of €94 million on Lundbeck and fines totalling €52 million on several producers of generic citalopram, which, in return for cash, had agreed with Lundbeck in 2002 to delay market entry of the drug in violation of EU antitrust rules.[467] Lundbeck had also purchased generics' stock for the sole purpose of destroying it.

When independent researchers made a meta-analysis based on indirect comparisons, escitalopram vs placebo, and citalopram vs placebo, there was no difference.[468] Their results are telling. For the adjusted indirect comparison of 10 citalopram and 12 escitalopram placebo-controlled trials (2,984 and 3,777 patients, respectively), escitalopram wasn't any better than citalopram, indirect OR 1.03 (0.82 to 1.30). The researchers also did a meta-analysis of seven head-to-head trials (2,174 patients), and the efficacy was now significantly better for escitalopram than for citalopram, odds ratio 1.60 (1.05 to 2.46). A similar discrepancy was found for treatment acceptability.

Such results tell us we should distrust network meta-analyses of depression pills. The cheating is pervasive and obvious. A drug containing the same active substance as the molecule that it out of patent, is claimed to be more effective and better tolerated. How dumb does Lundbeck think doctors are? Very dumb, indeed. Lundbeck made the rejuvenated drug a commercial success via a huge fraud scheme where the outcomes of the trials were already established before the trials were begun.[6:229]

When Lundbeck's American partner Forest had performed a trial of citalopram for compulsive shopping disorder, Gorman appeared as an expert in Good Morning America and said that 80% of the compulsive shoppers had slowed their purchases on the drug.[131] The viewers were told that this new disorder could affect as many as 20 million Americans of which 90% were women.

In 2010, Forest pleaded guilty for obstruction of justice and illegal promotion of citalopram and escitalopram for use in treating children and adolescents with depression.[469] Forest agreed to pay over $300 million to resolve criminal and civil liability arising from these matters and faced numerous court cases from parents to children who had either committed suicide or had tried.[470]

Forest lied to Congress and kept negative trials out of public view.[469,471,472] Forest had 19,000 so-called advisory board members,[472] and the periodical *Pharmaceutical Marketing* has provided the answer as to why so many advisors are needed:[473]

The advisory process is one of the most powerful means of getting close to people and of influencing them. Not only does it help shape medical education overall, it can help in the process of evaluating how individuals can best be used, motivate them to want to work with you – and with subliminal selling of key messages ongoing all the while.

The most important of these corrupt doctors are paid obscene amounts of money for doing very little or nothing.[6:78] A professor of psychiatry stated:

"'It is very dismaying to find academic psychiatrists that one has hitherto respected supporting one drug on a Monday and another on Tuesday ... I can think of a well-known British psychiatrist I met and I said, 'How are you?' He said, 'What day is it? I'm just working out what drug I'm supporting today.'"[341,369]

The generous honoraria for lectures attract a large army of physician "educators." A 2002 survey found that American psychiatrists were paid about $3,000 for a symposium lecture and some earned as much as $10,000.[369] Doctors working for multiple companies are called drug whores by drug reps,[343] and their work as lecturers or advisors are sometimes used as "payback" for participation in trials, which allows the doctors to say that they had no financial conflicts of interest while doing the trial.[474]

A psychiatrist reported how generous Wyeth was when he sold its drug, venlafaxine (Effexor), to colleagues:[475]

We were all handed envelopes as we left the conference room. Inside were checks for $750. It was time to enjoy ourselves in the city … Receiving $750 checks for chatting with some doctors during a lunch break was such easy money that it left me giddy. Like an addiction, it was very hard to give up.

When he said at a lecture that other drugs might be equally effective as Effexor, he was immediately visited by Wyeth's district manager who asked him if he had been sick. The doctor salesman then abandoned his lucrative career as an academic prostitute on top of his private practice.

Forest used illegal kickbacks to induce physicians and others to prescribe Celexa and Lexapro, which allegedly included cash payments disguised as grants or consulting fees, expensive meals and lavish entertainment. Lundbeck's reaction to the crimes was: "We know Forest is a decent and ethically responsible firm and we are therefore certain that this is an isolated error."[470] Of course. In the eyes of those who collect the cash,[471] organised crime is "an isolated error."

The corruption was total. Forest recruited about 2000 drug pushers (psychiatrists and primary care physicians) whom the company trained to "serve as faculty for the Lexapro Speakers' Bureau Program."[476] It was obligatory that speakers used the slide kit prepared by Forest. Forest provided "unrestricted grants" to professional societies including the American Psychiatric Association, so that they could develop "reasonable practice" guidelines, and Forest became a corporate sponsor of the American College of Physicians "which provides additional marketing opportunities," and this organisation was also involved with developing the "reasonable practice" guidelines.

Vortioxetine seems to be an exceptionally poor drug. Every author in all of the published short-term trials had significant commercial ties to Lundbeck, which is a sure way for a company to control that

what gets published supports its marketing ambitions. But when independent researchers compared vortioxetine with duloxetine and venlafaxine in meta-analyses, they found that these drugs were significantly more effective than vortioxetine at three of the four dose levels tested.[477]

It has been documented that network meta-analyses (NMA) of published trial data are unreliable. In an elegant study of this, the authors used data from 74 FDA-registered placebo-controlled trials of 12 depression pills and their 51 matching publications.[478] For each dataset, NMA was used to estimate the effect sizes for 66 possible pair-wise comparisons of these 12 drugs. To assess how reporting bias affecting only one drug may affect the ranking of all drugs, the researchers performed 12 different NMAs where they used published data for one drug and FDA data for the 11 other drugs. They found that the effect sizes for drugs derived from the NMA of published data versus those from the FDA data differed in absolute value by at least 100% in 30 of 66 pair-wise comparisons. This is a huge bias.

The *Lancet* NMA by Cipriani et al. contained nothing new and what was claimed to be new was unreliable. However, Cipriani hyped the paper to the extreme, e.g. in BBC News:[479]

"This study is the final answer to a long-standing controversy about whether anti-depressants work for depression … Scientists say they have settled one of medicine's biggest debates after a huge study found that anti-depressants work. The study … showed big differences in how effective each drug is ... The authors of the report … said it showed many more people could benefit from the drugs … The Royal College of Psychiatrists said the study 'finally puts to bed the controversy on anti-depressants' ... Researchers added … at least one million more people in the UK would benefit from treatments, including anti-depressants."

The mean baseline severity score on the Hamilton Depression Rating Scale was 25.7, which is considered very severe depression.[270] Thus, it was confirmed once again that the oft-heard claim that these drugs work for very severe depression is wrong, as the average effect was far below the minimal clinically relevant difference.

The NMA did not report the average drop-out rate for the drugs, but it was very close to 1, which is also a misleading result. As noted above, when my research group used the clinical study reports from the European drug regulators, we found 12% more drop-outs on drug than on placebo ($P < 0.000,01$), so if one million more people in the UK should benefit, the treatment should be placebo.[301]

Later, my research group showed that the outcome data reported in *Lancet* differed from the clinical study reports in 12 of the 19 trials they examined.[480]

The *Lancet* meta-analysis was garbage in, garbage out, to an unusual degree, which is surprising considering who the authors were. Among Cipriani's 17 co-authors were two with whom I have published the guidelines for reporting network meta-analyses,[217] and a third author was Cochrane statistician Julian Higgins, editor of the Cochrane Handbook of Systematic Reviews of Interventions that describes over 636 pages how do to Cochrane reviews.[481] This suggests that some of the most experienced co-authors did not contribute much to the paper. None of these three people were among the 12 authors who selected the articles and extracted the data, four of whom declared they had received money from drug companies.

In Denmark, the NMA was hyped to the extreme on the homepage of one of the five regions, which referred to a newspaper article:[482] "The conclusion is clear: Antidepressants work. And those who did the study assert that some antidepressants work better than others. One of those 'happy pills' which can best alleviate the depression is, for example, Cipralex."

Of course. Cipralex contains escitalopram, marketed by Lundbeck, and the Danish drug export is the largest source of national income. It beats agriculture although we have four pigs for every citizen, and also have pig farms abroad, e.g. in Spain.

Not only the Cochrane authors but also Poul Videbech functioned as Lundbeck's useful idiots. Videbech praised the NMA in the most bizarre way.[482] He noted that the "very large study" was "far more credible" than a Danish study published a year earlier,[268] which he claimed concluded that the drugs have no significant effect. He also claimed that Cipriani had taken into account the sources of error that the Danish researchers had not been aware of.

As so often before, which is also clear in the textbook he edited,[18] Videbech was highly manipulative. First, there were no errors in the Danish meta-analysis by Jakobsen et al., which was exemplary and highly rigorous, and Cipriani et al. did not point to any errors. They did not even cite the Danish meta-analysis although it was published 12 months before their own.

Second, the NMA was far *less* credible than the Danish meta-analysis that only included comparisons with placebo, which is the reason for the smaller number of patients. As already noted, head-to-head comparisons are notoriously unreliable and they are not research but marketing disguised as research.[6,7]

Third, it is false that the Danish meta-analysis did not find a significant effect of the pills. The effect was highly significant ($P < 0.000,01$), which the researchers reported.

Fourth, the effect in the Danish meta-analysis was about the same as that found in the NMA; the effect sizes were 0.26 and 0.30, respectively.

The main difference between the two meta-analyses was how the researchers interpreted their results. In their abstract, Jakobsen et al. concluded: "SSRIs might have statistically significant effects on depressive symptoms, but all trials were at high risk of bias and the clinical significance seems questionable. SSRIs significantly increase the risk of both serious and non-serious adverse events. The potential small beneficial effects seem to be outweighed by harmful effects."

In contrast, Cipriani et al. concluded: "All antidepressants were more efficacious than placebo in adults with major depressive disorder," with no caveats about the risk of bias and absolutely nothing about the drugs' harms. The only thing they reported was the proportion of patients who dropped out early because of adverse events, which was higher for all active drugs than for placebo.

This whole affair was hugely embarrassing for Cipriani et al., for Cochrane, and for *Lancet*.

Cipriani's two NMAs in depression, one for adults[271] and one for children and adolescents[297] (see pages 82 and 118) and numerous other meta-analyses, e.g. virtually all Cochrane reviews, are seriously biased and should be distrusted. This is the indisputable conclusion when we compare with the results obtained in studies based on clinical study reports submitted to drug regulators.[279,300,314,315,326,378]

This important message was not in the textbooks. Leading psychiatrists do not *want* to hear the truth about psychiatric drugs. They prefer to be fooled by the drug industry and corrupt colleagues and to propagate false statements about the drugs in their textbooks and elsewhere.

Pregnancy

Depression pills are among the most commonly used drugs by females in their reproductive age. In Denmark, 8% in the age group 18-44 years take them.[263] This is worrying, as the drugs seem to increase miscarriages, voluntary terminations, birth defects, and behavioural abnormalities in newborns,[483,484] and they cause many other serious harms in the offspring.[336]

The advice about pregnancy was inconsistent and confusing. The textbooks generally put the blame on the disease, not on the pills. For example, one book warned that depression doubles the risk of developing cardiovascular disease[16:259] and potentially increases heart malformations and neonatal complications.[16:584]

Another textbook was confusing, contradictory and misleading. It warned that depression increases the risk of abnormal bleeding during pregnancy, spontaneous abortion, premature birth, foetal death, eclampsia, other birth complications, poor quality of life for the child, and lack of breastfeeding.[17:364] However, on the same page, the authors noted that depression pills are possibly associated with a slightly increased risk of premature birth and perinatal complications, and 13 pages later, that the drugs likely increase the risk of malformations.[17:377]

After another 291 pages, this book contradicted itself again and tried to have it both ways in a most confusing fashion:[17:668] Untreated depression can cause premature birth and perhaps also malformations. Depression pills can perhaps increase spontaneous abortions, but the newest studies speak against malformations. Nonetheless, the authors noted that paroxetine is possibly associated with heart malformations and neonatal complications and that there is an increased risk of pulmonary hypertension in newborns, which can be deadly.

If you don't know what to say, it is prudent to say nothing instead of confusing your readers totally. I cannot make any sense out of the above, and it got worse. This book noted that the Danish National Board of Health recommend always to consider psychotherapy for pregnant women who are depressed.[17:365] Indeed; none of them should get pills. But lo and behold, just one page earlier, the book advised that pregnant women who have been depressed earlier should be treated *prophylactically* with depression pills to reduce the risk of relapse from about 70% to about 25%.[17:364] It is impossible to justify this horrific recommendation.

The Board of Health also contradicted itself. It recommended routine screening of pregnant women for depression and subsequent treatment with depression pills, although the available data do not support these recommendations.[485] It acknowledged that SSRIs increase the occurrence of spontaneous abortions, decrease birth weight, likely increase the occurrence of birth defects, 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, and increase neonatal complications such as irritability, tremor, hypertonia and difficulty sleeping or breast feeding.[485] An article about this appropriately called it neonatal abstinence syndrome.[486]

Excuse me, but have they gone mad at the Board of Health? A large Danish cohort study of 500,000 children showed that the risk of heart septum defect is doubled.[487] This is not trivial, as 1% of the treated foetuses 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 deadly pulmonary hypertension in adults who took diet pills that increase serotonin levels, and these drugs have been withdrawn from the market.[6:144]

The Board's recommendation of screening was so absurdly harmful that I wrote a little sketch about it,[488] which a psychologist and I spontaneously performed as the introduction to my lecture about psychiatry by reading it aloud from my computer. It is on YouTube with English subtitles.[489]

Among its many weird postulates in relation to pregnancy, this book also claimed that the risks of depression and behavioural disorders are increased in 18-yr-old children of mothers who were not treated during pregnancy for their depression.[17:365]

As I didn't believe this could be true for drugs that don't work, I looked up the evidence the authors referred to, which was a 2014 clinical guideline for the use of psychiatric drugs during

pregnancy produced by the Danish Psychiatric Association, Danish Society for Obstetrics and Gynaecology, Danish Paediatric Society and Danish Society for Clinical Pharmacology.[490] With so many knowledgeable people involved, one would expect the guideline to be reliable, but it can best be described as being blatantly dishonest.

The guideline stated that there is "an increased incidence of depression in 18-year-old children of mothers who were not treated during pregnancy for their depression (Pearson et al., 2013)" and that "untreated depression during pregnancy seems to increase the risk of developing behavioural disorders in the child (Pedersen et al., 2013)."

None of this was true. The article by Pearson et al. didn't say anything about whether the women were treated or not during their depression. What the paper showed was that if a mother was depressed, the risk of her offspring becoming depressed was increased, but only for mothers with low education.[491] This has nothing to do with treating or not treating a depression, but with poor living conditions, which is often also the case for the offspring. When living conditions are depressing, people become depressed. No great wonder here.

The article by Pedersen et al. did not document at all that untreated depression increases the risk of behavioural disorders in the child.[492] This was clear already in the abstract: "Prenatal antidepressant exposure was not associated with abnormal SDQ scores compared with prenatal exposure to untreated prenatal depression or to no exposure." But the abstract also reported the results of what we call a fishing expedition. When a result is negative, it is very bad research practice to report on subgroups of patients or on selected items on a scale, but this is what the authors did: "Untreated prenatal depression was associated with abnormal SDQ scores in the subscales of conduct [adjusted odds ratio (aOR) 2.3 (95% CI, 1.2-4.5)] and prosocial problems [aOR 3.0 (95% CI, 1.2-7.8)] compared with unexposed children." They not only selected items on a scale, they also did not compare untreated depression with treated depression but with people who were not depressed at all but were healthy!

In the tables, there wasn't a single significant difference between the total score or any of the subitems in the score when treated women with depression were compared with untreated women with depression. But the authors had been fishing again to find what they reported in the abstract for conduct: "Including only women with normal MDI [depression] score at time of follow-up". For prosocial problems, I was unable to find the adjusted odds ratio of 3.0, which was claimed to be statistically significant. It was nowhere in the paper, but there was this information: "The prosocial association was no longer statistically significant, OR 2.2 (95% CI, 0.8-6.5)" (when including only women with a normal depression score)."

It is amazing that such rubbish with tortured data analyses can get published but the research literature is full of this. A systematic review found that subgroup analyses in trials were more common in high-impact journals; and in trials without statistically significant results for the primary outcome, industry-funded trials were twice as likely to report subgroup analyses as non-industry-funded trials and twice as likely not to have prespecified the subgroup hypotheses.[493]

This textbook noted that valproate and carbamazepine are contraindicated due to a high risk of neural tube defects.[17:669] I wonder why the authors did not warn against all antiepileptic drugs.

Psychotherapy and psychoeducation

Psychotherapy is not a magic bullet against psychiatric disorders. It doesn't always work, but it is the best intervention we have.

The textbooks were sometimes contradictory and misleading. One noted that 50% of patients with depression are not treated; that many of them likely have mild depression; and that psychotherapy will shorten the disease phase, prevent chronicity and provide obvious relief for the patients.[16:257] Unfortunately, the book advised that SSRIs or tricyclics could be used instead of psychotherapy for moderate depression or in combination with it. For severe depression, psychotherapy was not advised, but hospital admission, tricyclics, tricyclics plus psychosis pills, and electroshock were.[16:272]

This is a familiar theme. The worse the disease, the more the patients shall be harmed by treatments that don't help them. This is not evidence-based medicine.

The book where all the authors are psychiatrists denigrated psychotherapy stating that pills can be combined with talk therapy with advantage, which also increases compliance.[18:238] So, pills first, even though they don't work, and psychotherapy is only aimed at keeping the patients on pills that harm them and which many patients would rather avoid. When asked what they prefer, six times as many people prefer psychotherapy for pills,[494] but they get the exact opposite. A 2002 survey of US child and adolescent psychiatrists showed that 91% of their patients were treated with psychiatric drugs.[495] Only in the remaining 9%, was psychotherapy used without drugs.

In Sweden, the National Board of Health recommends that all adults with mild to moderately severe depression are offered psychotherapy, but only 1% get it.[496]

This illustrates that psychiatry is a perverse trade. It doesn't help the patients as they want to be helped but helps itself.

This textbook recommended watchful waiting or supportive conversations for mild depression, psychotherapy for moderate depression, and depression pills for more severe depression.[18:123] The authors claimed that the preventative effect of drugs was more pronounced than that of psychotherapy,[18:126] which is false and was contradicted by another book, which noted that the effect of psychotherapy lasts longer than that for drugs.[16:277] As expected, studies with long-term follow-up show that psychotherapy has an enduring effect that outperforms pharmacotherapy,[180,497-501] and when psychiatrists believe pills prevent relapse, they mistake withdrawal effects for relapse (see Chapters 7 and 8).

A third textbook advised psychotherapy for moderate and severe depression,[17:359,17:363] but not for severe depression requiring hospital admission.[17:359] It noted that psychotherapy should most often be considered when the patient is in remission[17:363] and claimed that a halving of the depression score was obtained in 60% of the patients treated with drugs and psychotherapy.[17:359]

This statement is meaningless. It cannot be interpreted without knowing what happened to the other 40% of the patients. If their score increased markedly, the overall effect might be zero. Evidence-based medicine is not about what happened in some selected subgroup of patients, but about what happened on average. These authors considered psychotherapy a secondary option, which contradicted a chapter about psychotherapy in the same book where other authors noted that the effect size in a meta-analysis was quite high, and that in many cases, psychotherapy was cost-effective in comparison with drugs.[17:675] It seems to be correct that psychotherapy is more cost-effective than other forms of therapy.[502]

A fourth book also put the pills first even though it noted that the effect of psychotherapy and pills was about the same for mild and moderate depression.[20:435] This is misleading because the effect is also about the same in severe depression.[503] The book noted that the National Board of Health had found a better effect of combining psychotherapy with pills than of pills alone, but it did not mention that the Board's guideline strongly recommended to offer psychotherapy, in

combination with pills, to patients with moderate or severe depression.[504] This was contradicted by another book, which noted that for mild or moderate depression, there was no evidence of a greater effect of the combination than of drug or psychotherapy alone, while it claimed that this was the case for chronic depression.[16:278]

It was totally confusing. And why would a combination work for chronic depression when it did not work for moderate depression, and what is chronic depression? The guideline from the Board of Health had an important reservation: "Combination therapy has demonstrated an increased effect over monotherapy, but patients have often not been followed beyond the end of the intervention. The working group wants to clarify the long-term effects of combination therapy consisting of antidepressant pharmacotherapy and psychotherapy."

It is remarkable that three textbooks did not recommended psychotherapy for severe depression,[16,18,20] and that a fourth book did not recommend it for depression requiring hospital admission.[17] The only book that advised psychotherapy for severe depression was the one about child and adolescent psychiatry,[19:214] but, unfortunately, this book advised that psychotherapy should be combined with fluoxetine, which is unsafe and ineffective (see page 82).

One book stated that treatment of bipolar in children involves drugs, in addition to psychoeducation, but did not say that drugs should only be used if psychoeducation did not work.[19:220]

Two books stated that psychoeducation may halve the risk of new depressions or manias in bipolar patients and reduce hospital admissions but added that this was probably because of better treatment compliance (with drugs).[16:306,17:376] One of the books gave a reference to this statement,[17:376] which was a randomised trial of psychoeducation.[505]

It turned out that the textbook claims about better compliance with drug treatment were false.[505] The researchers randomised 120 bipolar patients to 21 weekly group psychoeducation sessions or nonstructured group meetings and the effect was assessed blindly. During treatment, 23 vs 36 patients had a recurrence ($P < 0.05$); at the end of the follow-up, these numbers were 40 vs 55 ($P < 0.001$); and there were markedly fewer hospital days, 4.8 vs 14.8 ($P < 0.05$).

At the 2-year follow-up, a tiny difference was found in lithium levels, 0.76 vs 0.68 mEq/L ($P = 0.03$), whereas there were no differences in the levels of valproate or carbamazepine, and no differences regarding drug treatment.

The authors wrote in the discussion that, "compared with control patients, psychoeducated patients had higher lithium levels at the 2-year follow-up, which may suggest an effect of psychoeducation on pharmacotherapy adherence."

So, the trial authors *did not* suggest that the tiny difference in lithium levels could explain the pronounced effects they found of psychoeducation. It is bending the data to the extreme when the textbook authors wrote that this was likely, instead of just accepting that psychoeducation is highly effective.

A textbook noted that, although PET studies are preliminary, there is much to suggest that symptom reduction during psychotherapy may normalise metabolism in certain cerebral areas found to be affected during depression.[16:269] Brain scan studies are highly unreliable (see Chapter 3), but this was a rare occasion where they were not used to promote drugs but psychotherapy.

9 ADHD

The sections on ADHD in the textbooks can best be described as being seriously dishonest even though there was useful advice interspersed here and there.

An epidemic of ADHD diagnoses

As mentioned earlier, Allen Frances, chairman for the DSM-IV task force, noted that DSM-IV created a false epidemic of ADHD because the diagnostic criteria were too wide.[116] The criteria for the ADHD diagnosis have changed with each iteration of the DSM, with each updated volume making it easier to make the diagnosis.[57] Prevalence studies reflect this. The percentage of youth said to have ADHD increased from 3% with DSM-III, to 5% with DSM-IV and to 10% with DSM-5.

The scientific literature is also dishonest, and it starts with the diagnosis. The American Psychiatric Association invented ADHD for DSM-III in 1980, and in 1998, the US National Institutes of Health (NIH) held a 3-day consensus conference about its diagnosis and treatment.[7:137,506]

At the meeting, the chairman asked a leading ADHD expert, Mark Vonnegut, what ADHD is, but although he talked for 2-3 minutes, he couldn't explain it (see the YouTube video; starts after 4 minutes):[507]

"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 ravings included that a teacher might say that a kid was two standard deviations different from the other kids in the classroom. I don't think teachers argue this way.

Furthermore, 5% of us are by definition beyond two standard deviations from the average of everything that follows a normal distribution, but this doesn't mean we are sick. If we measure people's 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.

The consensus document stated that "The diagnosis of ADHD can be made reliably using well-tested diagnostic interview methods."[506] This was a huge lie, which Vonnegut contradicted: "The diagnosis is a mess."

The document is embarrassing in many other ways. It uses 15 pages to tell us what ADHD is.[506] It says that ADHD is one of the most common childhood "brain disorders" and that imaging studies have shown abnormalities in the brain.

ADHD is not a brain disorder and the brains of these children are not different from the brains of other children (see Chapter 2).

The first page mentioned that "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 gets and cannot justify calling ADHD a brain disorder. There is a hilariously funny video that mocks this pseudoscience, which I strongly recommend.[508]

Many children qualify for the diagnosis because they are talented and therefore bored and cannot sit still in poorly disciplined classrooms, or because they have emotional problems generated by their parents.

I have lectured a lot for various audiences, both professionals and lay people, and I often expose people to the recommended test for adult ADHD (see table).[7,509] Between one-third and one-half test positive. When I lectured for 27 therapists in 2022, 21 tested positive and 10 of these had a full house, which is six out of six criteria (only four positive replies to the questionnaire are needed for the diagnosis). I told them they were a great audience because some of the most interesting people I have ever met qualify for the ADHD diagnosis. They are dynamic and creative and have difficulty sitting still on their chairs pretending they are listening if the lecturer is dull.

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

When I tested my wife, she also scored a full house. Once, when we discussed the silliness of psychiatric diagnoses, I subjected one of my daughters and her boyfriend to the test. My daughter scored five, like I did, and her laid-back boyfriend whom I would never suspect would be positive, scored four. So, we all got a bogus diagnosis that should land us on narcotics on prescription (amphetamine or amphetamine-like compounds).

My little exercise makes people realise how foolish and unscientific psychiatric diagnoses are. If you don't test positive for adult ADHD, then try a few other diagnostic questionnaires for other disorders, and you are likely to get one.

I do not deny that there are people with symptoms labelled ADHD that we can help to get a better life, but the big mistake is that psychiatrists tend to equate help with drugs.

Psychoeducation and psychotherapy or drugs?

The advice in the textbooks was highly inconsistent, sometimes even within the same book. Some texts praised psychosocial interventions while others praised drugs, with loads of false claims.

The focus was predominantly on drugs. One textbook chapter demonstrated a total subjugation to the biological model.[17:620] Its authors, both psychiatrists, noted that there is no evidence that cognitive behavioural therapy works on the fundamental "neurologically conditioned core symptoms" in adults with an ADHD diagnosis but that therapy should be offered in the form of general psychoeducation and training in practical and social skills as a supplement to medical treatment. They claimed that there are only a few large studies of psychotherapy and that they all have methodological problems. The reference for these remarks about psychotherapy was a whole book written by one of the two authors, psychiatrist Marianne Geoffroy.

But there is actually a review of 14 trials of psychotherapy, and it showed an effect on core symptoms, in contrast to the textbook statement.[510]

When I published my first psychiatry book in 2015, *Deadly psychiatry and organised denial*,[7] there was enormous media interest all over the world because I had documented in detail why psychiatry is a disaster area. It was very threatening to mainstream psychiatry, and two weeks after it came out, Geoffroy wrote in an industry supported throwaway magazine that I used public funds to publish private, non-scientific books, which she compared to Scientology books.[8:23]

She claimed that I scared citizens suffering from psychiatric disorders away from getting relevant treatment (which means drugs). I complained about her libellous misinformation, and a tribunal concluded that she had violated both the ethical guidelines and the collegiate guidelines from the Danish Medical Association and had used a language that was totally beyond the borders of a decent debate about healthcare issues.

I mention this because Geoffroy's under the belt reaction is typical of the way psychiatrists react in the public debate when they have no counterarguments and perceive their opponent as being dangerous for their interests.[1,5,7,8]

The textbooks direly warned of the consequences if ADHD is not treated with drugs. It was claimed that untreated ADHD can increase the risk of poor educational course, risky behaviour, crime and drug abuse; that there are effective treatments, primarily in the form of drugs;[19:107] and that several follow-up studies suggest that stimulants protect against drug abuse.[19:291]

Another book claimed that drugs reduce the risk of developing drug abuse, traffic accidents and committing crime.[16:475]

A third book, the one with no references at all, also mentioned the risk of drug abuse and crime, and that children might not get the education they could otherwise get, and it said that studies suggest that treatment can counteract this.[18:224] This book mentioned psychoeducation, support, training and rules for adults diagnosed with ADHD and claimed that the vast majority experience a good effect of the medicine and that venlafaxine may also be effective.[18:229] None of these claims can be substantiated (see below).

A fourth textbook wasn't any better. It said that medication is central and that many benefit from it.[17:618] It noted that a Cochrane review raised doubt about the effect of methylphenidate because there was substantial bias in the trials,[511] but added that many clinicians and patients say that they have a positive experience that methylphenidate works, which is indisputable.

So, clinical experience is all we need, right? If that is the case, then why bother to do trials? Did it ever occur to the psychiatrists that some patients say they like the drugs, not because they work, but because they are speed on prescription that makes them high?

It was also claimed that placebo-controlled studies have shown an effect of central stimulants in 70-80% of the children,[19:289] and that 50% of adults have significant positive effects on the core

symptoms, compared to 75% in children and adolescents.[16:475] These claims are meaningless, as the percentages are before-after observations from a treated group, with no placebo control.

It was claimed that methylphenidate reduces hyperactivity and impulsivity in adults with an effect size of 0.50-0.56 and increases clinician-assessed global functioning with an effect size of 0.87.[16:475] These large effects are also wrong (see below).

Trials of ADHD drugs are biased to an exceptional degree, even by psychiatric standards. A review of 43 studies in children, of which 34 were randomised, showed that very few of the reported adverse drug reactions were called serious, although many children dropped out of the studies precisely because of serious adverse drug reactions.[512]

Many of the 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 the industry rich.

Most systematic reviews of the trials are therefore also biased. Two Cochrane reviews performed by my former employees, who paid attention to the flaws, found that every single trial ever performed was at high risk of bias, both for trials in children,[511] and in adults.[513]

An earlier Cochrane review from 2014, of immediate-release methylphenidate for adults, showed positive effects for hyperactivity, impulsivity and inattention, but the trials were short-term and biased.[514] The results varied so hugely that I would not have performed meta-analyses on these data. Most worryingly, the authors could not even determine if adverse effects were not discussed because none occurred, or because the data had not been collected. The review was so bad that the criticism we and others raised led to its withdrawal from the *Cochrane Library*.[515]

My research group found that also the drug agencies' reporting of harms can be highly unreliable.[513] In the British drug agency's review, "psychosis/mania" was reported to occur in 3% of patients treated with methylphenidate and in 1% of those on placebo. The 3% estimate is *30 times higher* than the 0.1% risk of "new psychotic or manic symptoms" that the FDA's Prescribing Information warns about. We even encountered discrepancies *within* the regulatory documents.

We observed huge differences across trials that could not be explained by trial design or patient populations, e.g. decreased libido on methylphenidate was experienced by 11% in one trial versus only 1% in a pooled analysis of three other trials. As quality of life was measured in 11 trials but only reported in 5, where a tiny effect was found,[513] it is reasonable to assume that quality of life worsens on ADHD drugs, which is also what the kids experience. They don't like the drugs if asked while their parents are not in the room.

In 2022, my research group published a systematic review of extended release methylphenidate in adults.[513] We found that every single trial had a flawed design and there were many other flaws. A medical student involved with our research was shocked when he saw this; he had never imagined that clinical trials could be of such poor quality, with many missing patient-relevant outcomes. He wondered, for example, why blood pressure measurements were missing when we know that stimulants increase blood pressure and that many people die from high blood pressure.

We used proper methods and could not confirm the large effects described in the textbooks. We included 24 placebo-controlled trials of extended-release methylphenidate for ADHD in 5066 adults. We also included documents from six drug regulatory agencies covering eight of the trials. We rated 20 trials at high risk of bias, primarily due to unclear blinding of participants and investigators, attrition bias, and selective outcome reporting.

All trials were impaired in at least one of three design characteristics related to generalisability, e.g. by excluding patients with psychiatric comorbidity or by including only participants with a previous positive response to stimulants. We rated the certainty of the evidence as very low for all outcomes.

For the primary outcomes, we found that methylphenidate had no effect on days missed at work and a minor effect on self-rated ADHD symptoms, effect size -0.37 (-0.43 to -0.30). Methylphenidate improved self-rated quality of life slightly, -0.15 (-0.25 to -0.05) investigator-rated ADHD symptoms, -0.42 (-0.49 to -0.36), and ADHD symptoms rated by peers, -0.31 (-0.48 to -0.14). These confidence intervals did not include the large effects of 0.50 to 0.87 claimed in a textbook, see just above.[16:475] Methylphenidate increased the risk of several adverse effects.

We concluded that the benefits and harms of extended-release methylphenidate are uncertain.

The large MTA trial and institutional corruption

A textbook recommended psychoeducation for adult AHDH,[17:617] and the book about child and adolescent psychiatry mentioned non-pharmacological treatment first and pointed out that there can be a vicious circle where the parents scold the child a lot.[19:114]

This little glimpse of non-pharmacological hope vanished already on the next two pages, which said that drugs should be used in children above 6 years of age with moderately severe and severe ADHD, and that they often have quick and pretty dramatic effects. The authors noted that the effect was well documented up to 12-18 months; that the long-term effect was insufficiently elucidated; and that newer register studies suggested a reduction in number of accidents and emergency visits and a positive long-term effect on learning, marks, and schooling.[19:116]

It is misleading to mention highly positive effects up to 12-18 months; to pretend we do not know what happens after this; and then say that register studies, which are less reliable than randomised trials, suggest the drugs have important long-term effects. The authors presented 19 references but none of them was to the short-term results of a large study, which child and adolescent psychiatrists otherwise cite a lot.

This is the famous MTA trial sponsored by the US National Institute of Mental Health,[7:148] in which 579 children were randomised to methylphenidate, behavioural therapy, both, or routine community care. The NIMH published the 14-month results in 1999.[516]

Many scales and outcomes were used, with no less than 19 primary outcomes, which is extraordinarily many, but the only differences between drug and behavioural therapy were that the children were less hyperactive or impulsive and paid more attention when on the drug. Combined treatment was no better than drug alone for core ADHD symptoms.

The authors considered ADHD a chronic disorder and advocated ongoing treatment, which agreed poorly with the improvement in symptoms, which, in all four groups was sometimes much larger than the differences between the treatments, e.g. for inattention and social skills.

More importantly, did the reported differences in scores translate into anything useful for the children? They 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.[517]

This time, the investigators revealed their financial conflicts of interest, which were extreme. Sixteen authors listed a total of 214 drug companies, 13 per author. These relationships were mostly described as research funding, "unrestricted grants" (a euphemism for corruption[6]), con-

sulting and being on speakers' bureaus and advisory boards. Not exactly a group of people that would be likely to give us an unbiased view of the value of methylphenidate.

After three years, the four treatment groups didn't differ significantly for any of the numerous efficacy outcomes. The investigators partly ascribed this to the fact that many children in the two non-drug groups took drugs, diluting the treatment contrast. But they also mentioned that the results were possibly due to an age-related decline in ADHD symptoms, thereby contradicting their claim that ADHD is a chronic disorder.

This was cognitive dissonance. Most people experience this from time to time, but for many leading psychiatrists, it seems to be a chronic disorder.

A companion paper was close to impossible to interpret, as the findings were drowned in advanced statistics, but the limited relevant data the authors presented showed a lower rate of substance abuse in the behaviour therapy group than in the drug group.[518] Methylphenidate didn't protect against delinquency and substance abuse; if anything, it caused them.

A priori, one would expect stimulants to increase these risks. But a very large and long-term study about this was never published. The main investigator, Nadine Lambert, died in a car accident in 2006[519] and her colleagues did not ensure that her unique research got published. Perhaps because they disliked the results.

There is an account of her study in a news release from the University of California, Berkeley.[520] She presented her report to the NIH for the 1998 consensus meeting where Vonnegut couldn't explain what ADHD is, which was the basis for the University's news release.

Lambert conducted a 26-year study of 492 children, half of whom were diagnosed with ADHD. While nearly half of the youngsters treated with methylphenidate had become regular smokers by age 17, only 30% of those who had never been treated smoked daily. Only 2% of those who had never smoked or taken methylphenidate were dependent on cocaine as adults, compared to 40% of those who both smoked and were treated with stimulants. We cannot know to which degree confounding might explain her results, but I mention them because it is one of the biggest prospective studies ever made of this important issue. After she had presented her results in 1998, the National Institute on Drug Abuse stopped funding her work.[5:306]

The results in the MTA study after six and eight years were also discouraging.[521] The treatment groups didn't differ significantly for grades earned in school, arrests, psychiatric hospitalisations, 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.

The follow-up papers are also difficult to grasp, as they confuse readers with unnecessarily complicated statistics, which looks like deliberate obfuscation, as it would have been much easier to simply describe the disappointing results. One of the investigators was honest, not in any of the over 100 scientific papers that the MTA study generated, but in a newspaper interview:[522]

"I think that we exaggerated the beneficial impact of medication ... 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. It became clear in another newspaper article that the public was duped, seduced and lied to.[523] A news release issued by NIMH presented the negative results in a favourable light with the title, *Improvement following ADHD treatment sustained in most children*. Free fantasy.

It was not possible to see any difference to drug company propaganda. 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 deceives people in the same way. 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, which nonsense the *New England Journal of Medicine* allowed Merck, a US company, to publish.[6:156]

The stunting of growth methylphenidate caused was huge. After 16 years, those who consistently took their pills were 5 cm shorter than those who took very little, and there were many other harms.[524] We can only speculate which permanent effects these drugs might have on the children's developing brains but it seems likely – based on what we know about other brain active substances[11] - that they harm the brain.

The short-term effect is that the children sit still in class, but that effect disappears quite quickly. Short-term harms include tics, twitches, and behaviours consistent with obsessive compulsive symptoms,[506] all of which can become common.[4,525] Stimulants reduce overall spontaneous mental and behavioural activity, including social interest, which leads to apathy or indifference, and many children - more than half in some studies - develop depression and compulsive, meaningless behaviors.[135,526] Mental activity and interaction with other people are important for brain development, so this also suggests that the drugs may harm the brain permanently.

Animal studies have confirmed these findings,[526] and my research group has documented other harms, e.g. that the drugs impair reproduction even after the animals were taken off them.[527]

At school, the compulsive behaviour is often misinterpreted as an improvement even though the child may just obsessively copy everything shown on the board without learning anything. The drugs used for ADHD have hallucinogenic properties,[506] and some children develop mania or other psychoses.[135,528] The harms of the drugs are often mistaken for a worsening of the social construct called a "neurodevelopmental disease," which leads to additional diagnoses, e.g. depression, obsessive compulsive disorder or bipolar – and additional drugs, leading to chronicity.[526]

Patients and their relatives commonly refer to depression pills as "Psychiatry's Starter Kit." This is because many people start their psychiatric "careers" by consulting their family doctor with some problem many of us have from time to time and leave the doctor's office with a prescription for a depression pill, which starts a chronic course with multiple diagnoses and multiple drugs. ADHD drugs are also one of Psychiatry's Starter Kits.

Misleading textbook information and advice

One textbook advised to continue with drugs for years, namely for as long as there is clinical effect and harms are tolerated.[19:118] However, it is impossible to judge if there is any benefit in the individual case. We can only say what the randomised trials have shown and they do not lend support to treating people for years, particularly not when the harms are also taken into consideration. This section had 11 references, none of which were elucidating, and not a single one was to the MTA trial, although this was a textbook in child and adolescent psychiatry from 2019, and the 16-year results from the MTA trial were published in 2017.[524]

A section in another book, written by two psychologists, advised that the treatment should be broad, flexible and long-term and should start with a series of non-pharmacological methods.[20:472] But they also bought into the misleading psychiatric narrative. They claimed on the next page that

drugs effectively reduce symptoms of impulsivity, hyperactivity and inattention; improve social interaction with children of the same age and with parents; may alleviate aggression; have a moderate to large effect in children aged 6-18 years; and appear to reduce the risk of subsequent drug abuse.

The authors also claimed that drugs will improve symptoms significantly in adults. They cited the MTA study but only for use in a figure about co-morbidity with overlapping Venn circles. There wasn't a single word about the results of this trial in the whole chapter even though the book was published in 2021. The MTA paper in their literature list was 20 years old and only reported the misleading 14-month results.[529]

The authors provided three other references.

One was a meta-analysis of 28 placebo-controlled trials of stimulants in children with ADHD, all of which were "published in a peer-reviewed scientific journal."[530] This provides no comfort, as the peer-reviewed literature is full of unreliable research. The authors used a home-made quality score for assessing the quality of the trials, which is a method firmly recommended against.[481] They gave two points to double-blind studies and one point to single-blind studies, but single-blind studies should have been excluded, and nowhere in the paper did they say which studies were single-blind. The trials were very small, with an average of only 28 patients. The authors reported a huge effect on aggression, effect size 0.84.

This looks impressive, but: How is it possible to find such a result when it is widely known that stimulants *cause* aggression; when the FDA warns that "anxiety, psychosis, hostility, aggression, and suicidal or homicidal ideation have also been observed;"[34] when 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;[261] and when these drugs – including atomoxetine – cause hallucinations and violence?[261,401]

The answer is simple: Meta-analyses of published placebo-controlled trials of psychiatric drugs are highly unreliable. As already noted, it seems that all trials in ADHD are at high risk of bias and the degree of underreporting harms like aggression is pronounced in psychiatric drug trials.

The second reference was to a 2017 editorial commenting on a study of health care claims from 3 million adolescent and adult patients diagnosed with ADHD.[531] The editorialist noted that the use of stimulants could sensitise people to the rewarding effects of drugs, increasing the risk of substance use disorders, and that the sensitising effects of amphetamine are well established in animal studies. On the other hand, he also noted that, by reducing the symptoms and impairments of ADHD, stimulant medications may decrease the risk for substance use disorder.

The study found a reduction of events related to substance use disorders, such as emergency department visits, during periods of treatment with ADHD medications whereas the use of SSRIs increased such events. The patients were their own controls, but they could be more motivated to reduce substance use during periods in which they were engaged with medical treatment.

The editorialist noted that a 2003 meta-analysis found a 1.9-fold reduction in risk for substance use disorders in the treated group. He didn't provide a reference to this meta-analysis, and I was unable to find it even though I tried many search strategies on PubMed and browsed hundreds of records. However, I doubt this meta-analysis can be important because the MTA trial of 579 children and adolescents found the opposite, a *higher* rate of substance abuse in the drug group than in the behaviour therapy group.[518] Furthermore, the MTA trial did not find that the drug reduced arrests, psychiatric hospitalisations, or other clinically relevant outcomes.[521]

It is therefore difficult to believe the results of research on registry data from Sweden and Denmark that reported 30-50% reductions in criminal convictions, which the editorialist mentioned.

When searching on PubMed, I found a systematic review from 2021 that appeared to have been carefully conducted.[532] The authors concluded that, based on the limited evidence available, strong clinical recommendations are not justified, but that provisionally, stimulant treatment in children with ADHD "may prevent" the development of substance use disorders. "More studies are needed." I am not so sure about this. It seems likely that ADHD drugs increase substance abuse, and, at any rate, these drugs should not be used because they are harmful.

The editorialist had numerous financial conflicts of interest and had been a speaker at drug company sponsored events. An editor wrote in the paper that he had "found no evidence of influence from these relationships." It is funny how people always try to get away from the indisputable fact: Financial conflicts of interest corrupt, which is why the drug industry buys doctors.[6,7,533]

The third reference was a meta-analysis of adults from 2010.[534] The authors acknowledged that all meta-analyses are limited by the quality of the studies they included, but they did not provide any assessment of the risk of bias in the individual studies. They included 7 placebo-controlled trials of short-acting stimulants (459 patients) and 5 trials of extended release preparations (637 patients) and reported huge effect sizes, 0.96 and 0.73, respectively. They translated these effects, measured on scales, to binary data and reported that the number needed to treat to benefit one patient was only about 2-3.

As already explained, these miraculous results are fake and there cannot exist any number needed to treat for psychiatric drugs, only number needed to harm (see page 79). Furthermore, one of the world's finest biostatisticians, Douglas Altman, who was statistical advisor for the *BMJ* for many years, has advised against dichotomising continuous variables.[535]

The first author of this meta-analysis had received consulting fees or research support from or had been on advisory boards or a speaker for companies selling ADHD drugs. These were called "Potential conflicts of interest," which is a misnomer often used to downplay the problems. Conflicts of interest cannot be potential; they are real.

About ADHD in adults, child and adolescent psychiatrist, professor Søren Dalsgaard, advised in a textbook that psychoeducation should be one of the first things offered; that there is good evidence for the effect of cognitive behavioural therapy, especially when combined with drugs; and that the combination is clearly better than drugs alone.[16:473]

This information is strange. It starts with psychoeducation, goes on with psychotherapy, and ends by saying that the combination is better than *drugs* alone. Since Dalsgaard prefers psychological interventions, he should have told us if the combination is better than *psychotherapy* alone. Perhaps drugs are not needed?

This text is an example that psychiatrists are totally absorbed in the drug focused paradigm. In 2015, I participated in a panel at a conference with hundreds of patients in the audience. After I had advocated for psychotherapy instead of drugs, also for patients with schizophrenia, the psychiatrist on the panel, Merete Nordentoft, remarked that drugs could not always stand alone. I turned the argument around and said that everyone should get psychotherapy and that this could not always stand alone. The audience applauded my remark. Few patients get the psychotherapy they so much want and need and many hate psychosis pills but are forced to take them.

About side effects – which was the term always used in the textbooks that never spoke of harms - Dalsgaard mentioned that, in high doses, the drugs may trigger or aggravate depressive and psychotic symptoms if the patient is predisposed toward a psychotic disorder.[16:475]

This is wrong. Dalsgaard provided a thinly veiled attempt at blaming the victim and not the drug, which permeates the whole of psychiatry. Depressive and psychotic symptoms may occur on usual doses and without any predisposition.

Dalsgaard's claims were not referenced, but there was a list with 19 references of which only two were research papers related to his claims. Both were his own publications; they were observational studies; and they were not illuminating.

One article noted that 47% of children with ADHD had criminal convictions in adulthood, five times more than in the general population.[536] What should we make out of that? We all know that children with this diagnosis have more problems in life than other people, but we cannot help them avoid crimes with drugs.

The other article included 208 youths with ADHD.[537] The risk of substance use disorder in adulthood was 8 times higher than in the background population, and for every year older at start of treatment, the risk increased by a factor of 1.5. This suggests that kids should be medicated from birth if only doctors could identify symptoms of ADHD that early. It also means that the risk of drug abuse is 130 times higher (1.5^{12}) if a child starts treatment at age 18 rather than at age 6.

It can be calculated from the article that the background rate in the population is 0.69%. This means that 0.69% x 130 = 90% of all children with an ADHD diagnosis from age 6 will become substance abusers if they are not treated before age 18. The article did not specify the age span that provided the data for the 1.5 times annual risk increase, and I might have extrapolated too liberally by using a span of 12 years, but the study is absurd. There must have been huge confounding because children starting drug treatment late are very different to other children.

Dalsgaard did not mention the MTA trial in his book chapter, which is the best evidence we have about crime and drug abuse when children with the ADHD diagnosis grow up.

Harms of ADHD drugs

The information on harms of stimulants was inconsistent and the most important harms received little or no attention in the textbooks.

Among the listed harms were headache, dry mouth, nausea, stomach pain, tics, irritability, sadness/depression, nervousness, worsening of anxiety symptoms, sedation, increased blood pressure in 5% of the patients, insomnia, anorexia, and possibly weight loss.[16:475,18:229,18:244,19:117] One book regarded anorexia as the likely cause of a reduction in hight of 2 cm,[19:117] which is speculative.

The harms were said to be frequent but often reversible. The readers were not told which harms that are *not* reversible. [18:229,19:117]

One book mentioned the potential for drug abuse.[18:229] Indeed, and ADHD drugs are easily available on the black market. This book noted 15 pages further ahead that, in rare cases, the drugs can cause arrhythmias, palpitations, mania or psychosis.[18:244] It did not mention reduced height and weight, even though they are irreversible harms, or misdiagnosis of bipolar due to adverse effects of the drug, which is also pretty irreversible, or violence.[401] The book did not mention the MTA study, but in a section called *Abuse and dependence on illegal drugs*, the book noted that stimulants include cocaine, "amphetamine (speed)" and methylphenidate.[18:76]

Thus, this book did not hide that ADHD drugs can be abused. It also noted that abstinence reactions could include depression, and that some people become depressed after a few doses of stimulants, which the authors believed, with no references, was likely because of the drugs' effect on the brain's serotonin system.

This is one of the extremely few instances where a textbook admitted that a psychiatric drug can harm the brain. But as always, the authors were so absorbed in the drug focused paradigm that even though they noted that the depressions could come suddenly, be long-lasting and cause a great risk of suicide, they did not advice tapering of the offending drug but that the depression can be treated with depression pills.[18:76] This advice is deadly, as it will increase suicides.

A third book listed only anorexia, insomnia, and cardiovascular harms under adult ADHD,[17:618] but 48 pages further ahead it listed some more harms and stated that the most common ones were insomnia, anorexia, headache, weight loss, dry mouth, mood swings, and an increase in blood pressure and pulse.[17:666] The book noted that the drugs can cause mania, worsen tic diseases and destabilise bipolar disease, but not that bipolar is often misdiagnosed because of the drugs' harms.

The only mentioning of withdrawal symptoms I found was in a book that noted that the symptoms can lead to decreased ability to drive, use machines and work.[19:118]

Death, the most severe of all harms, was not mentioned in any of the textbooks even though sudden death, stroke, and myocardial infarction are listed on the first page in the package insert for methylphenidate.[34]

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.[538] This drug is now being sold in USA to children with an ADHD diagnosis. It was withdrawn in Canada in 2005 after 14 children suddenly died and two had strokes.[539] The FDA did nothing, apart from trying to convince their Canadian colleagues not to withdraw the drug.

Children have killed themselves or suddenly dropped dead while playing with friends.[261,401] Psychiatrists writing textbooks do not think this is important information.

These addictive drugs are stimulants and work like amphetamine; in fact, some of them *are* amphetamine. WHO describes amphetamine-type stimulants, including methylphenidate and MDMA (Ecstasy) this way:[540]

"Over the past decade, abuse of amphetamine-type stimulants (ATS) has infiltrated its way into the mainstream culture in certain countries. Younger people in particular seem to possess a skewed sense of safety about the substances believing rather erroneously that the substances are safe and benign ... the present situation warrants immediate attention."

It is hardly surprising that young people think these substances are safe, as so many of their friends get them on prescription.

Crystal meth is the common name for crystal methamphetamine, a strong and highly addictive drug. In 2017, about 0.6% of the US population reported having used methamphetamine in the past year.[541] The usage of stimulants on prescription was 0.8% of the Danish population, also in 2017.

The WHO did not mention with one word that the increasing use of stimulants on prescription is also a huge problem. This is taboo.

There were 10,333 drug overdose deaths in USA in 2017 involving stimulants,[541] compared to only 1,378 in 2007. Meth is regarded as particularly dangerous. We don't know how many people are killed by stimulants on prescription.

But we do know that stimulants increase the risk of violence,[34,401] including suicidal and homicidal ideation,[34] which is not surprising, given their pharmacological effects.

We should not change children's brains but their environment

Doing the right thing in psychiatry is often not allowed by the psychiatric guild. An Irish child psychiatrist told me he was suspended because he didn't put his children on psychiatric drugs, including ADHD drugs.

Instead of changing our children's brains, likely permanently, we should change their environment. ADHD medications are prescribed much more to children if the parents have low-skilled jobs.[542] The drugs are used as a form of social control, just as psychosis pills are, and the soma pill was in Aldous Huxley's novel, *Brave new world*.

Sexual abuse of children is frighteningly common. You can easily find references on the Internet to the fact that about one in ten children have been sexually abused before their 18th birthday. If a child behaves badly, is provocative and defiant, this can easily lead to a diagnosis of ADHD or borderline personality disorder, although it might be a reaction to a horrible situation of ongoing sexual abuse that the child doesn't dare talk about to anyone.

Not even when the patients talk about it, it is always taken seriously. A young woman told me that when she mentioned to her psychiatrist that she had been sexually abused as a child, he responded: "This is beside the point." Of course. He used the foolish questionnaires for making diagnoses, which was all that mattered to him. Many patients have told me it took many years before they met a psychiatrist who took an interest in the serious trauma they had experienced.

One of my critical colleagues, child psychiatrist Sami Timimi, often asks parents who want him to drug their child for ADHD:[46] "Imagine this drug working perfectly; what changes are you hoping will result from this?" That question may surprise parents, but if you say no more, one of them will break the silence and start talking about what changes they are hoping for. That helps Timimi understand the parents' specific areas of concern. Is it, for example, behaviour at home, peer relationships, academic performance at school, a lack of a sense of danger? Timimi might then respond that no drug in the world can alter these things in their child. Drugs don't make decisions, have dreams and ambitions, or perform actions.

By discovering the specifics of what the parents want to see change, Timimi can divert their interest from drugs to more targeted measures such as developing parental management skills for children who are more "intense" than most. He helps them understand the anxieties and stress their children may be feeling, or he supports them getting more structured interventions in schools. He also reminds parents that one thing is certain: Children change as they grow older and often the problems labelled as ADHD (particularly the hyperactivity and impulsivity) tend to go away as the child matures during adolescence.

As noted earlier, this agrees with observations in school classes: 50% more of those born in December were in drug treatment than those born in January.[51]

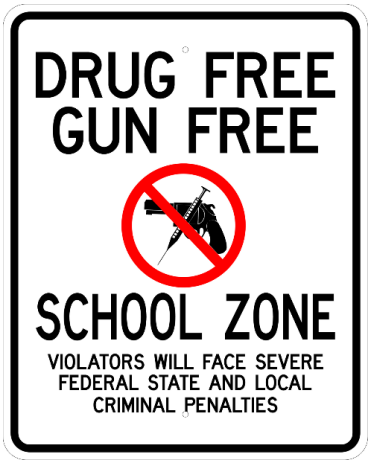
One textbook claimed that most neurobiological studies of patients with ADHD suggest that the abnormal findings in the brain are gradually normalised by late maturation of the brain as well

as by treatment.[18:224] This is not correct. The brains are not abnormal (see page 16 and Chapter 3), and drugs cannot normalise what is already normal.

The ADHD diagnosis should not be a prerequisite for getting extra help or money for schools, which it is now. This requirement drives the prevalence of the diagnosis upwards all the time, and the use of ADHD drugs, too, which increased by 240% in Denmark from 2007 to 2017.[263]

Some countries have experienced an increase in the use of psychiatric drugs in children that is directly attributable to school partnerships with hospitals. A colleague informed me that, in one Canadian province, the hospitals aggressively lobbied special services personnel and high school guidance counsellors, who in turn referred any child under stress to the psychiatric department. The school board hired a school psychiatrist who consulted with staff on school refusal situations and behavioural issues and recommended depression pills or ADHD drugs.

Schools and hospitals have become dangerous places for children and adolescents. How sad this is. Schools should stimulate children, not pacify them with speed on prescription. In USA, you can be met with this warning:



But inside the gate, about 10% of the children have an ADHD diagnosis[543] and are on speed.

It is a paradox that teachers act as more effective drug pushers than those in the streets. 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.[544]

I have this advice to people:[8:67]

1) Don't ever accept that your child be treated with speed on prescription.

2) Don't ever accept this yourself but resist becoming a faceless number in the new legal market for speed for adults.

3) Approach children with patience and empathy that allow them to grow up and mature, without drugs.

4) Work on changing the mechanisms that label more and more children with a psychiatric dis-order; they must be able to get the help they need without getting a diagnosis first.

ADHD is a disaster area, both in terms of the diagnosis, clinical research, and the harms inflicted on hundreds of millions of healthy people, including 10% of our children. All ADHD drugs should be removed from the market and the diagnosis should be banned.

Danish child and adolescent psychiatrist Lisbeth Kortegaard and US psychiatrist Peter Breggin[135] have gradually withdrawn ADHD drugs from every child that came their way and have both experienced that it improves the child's condition given the parents agree and work on improving their parental skills.

A British documentary was very revealing about what is needed.[8:65] It showed highly disturbing children, which were so difficult to deal with that even critical psychiatrists might conclude that ADHD drugs are necessary. "We cannot have children hanging around in the curtains," as a child psychiatrist told me at a hearing in Parliament about the drugging of children. The families in the documentary got help from psychologists and it turned out that the children were disturbed, which was why they were disturbing. One mother who always reprimanded her "impossible" daughter was taught to praise her instead, and somewhat later, she had developed into a very nice child that was no longer difficult or hostile towards her mother.

10 Anxiety disorders

Although the textbooks advised psychotherapy for anxiety disorders, their focus was on drugs.

One book noted that cognitive behavioural therapy is the best documented intervention for anxiety disorders in children, possibly supplemented with an SSRI, and it recommended this also for obsessive-compulsive disorder (OCD).[19:143,19:167] Drugs were only indicated if there was a lack of effect of psychosocial support and cognitive behavioural therapy and where the anxiety was severely invalidating.[19:157]

This is the standard script for psychiatry. Even when drugs are generally not recommended, they must always be used if the condition is severe enough.

Another book noted that psychotherapy is the preferred treatment of agoraphobia,[18:136] while a third book was more positive towards drugs. It noted that agoraphobia can be treated with cognitive behavioural therapy, depression pills, or both in combination.[16:349] For social phobia and generalised anxiety,[16:351,16:357] cognitive behavioural therapy was considered first choice, but it was mentioned that SSRIs also have well documented effects.

There is no doubt that shyness – now called social phobia, as it is better for industry's marketing[7:214] - should be treated with psychotherapy. A 24-week trial that randomised 375 patients with social phobia to sertraline or to gradual exposure to the feared symptoms found a similar effect of exposure and sertraline, but during an additional six-month follow-up, the exposure group continued to improve, which the sertraline group did not.[545] This was expected. People on drugs don't learn anything about how to cope with their anxiety. It is like alleviating the tension with alcohol. In contrast to drugs, psychotherapy usually has enduring effects on psychiatric disorders.[180,497-501]

A Cochrane review of 41 trials in children and adolescents with anxiety showed very large effects from cognitive behavioural therapy.[546] The outcome was assessed blindly in 32 of the 41 trials. The odds ratio for remission, compared with waiting-list controls, was 7.85 (5.31 to 11.60), and the reduction in anxiety symptoms had an effect size of -0.98 (-1.21 to -0.74). Other psychological therapies were similarly effective.

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 (-0.23 to 0.40).[547] These results agree with those from numerous other studies.[14,547] Patients can also help themselves. 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 (0.55 to 0.80).[548]

For OCD, a book recommended psychoeducation, self-help material and cognitive behavioural therapy, and SSRIs in severe cases.[16:360] The evidence for psychotherapy is strong. A Cochrane review of trials in adults found that psychotherapy resulted in far fewer symptoms than if the patients had received treatment as usual, effect size -1.24 (-1.61 to -0.87).[549] The effect of SSRIs was substantially smaller, effect size -0.46 (-0.55 to -0.37) (calculated by me).[550] There were few direct comparisons, but a review found that psychotherapy was better than depression pills, effect size -0.36 (-0.72 to 0.00) (calculated by me, three trials with 118 patients).[551]

The book that recommended SSRIs in severe cases also stated that psychosis pills could possibly be used as augmentation.[16:360] There were no references to original research, [16:369] only to a national guideline from 2007,[552] which was very brief: "There are no studies of monotherapy with

antipsychotics that have shown an effect on OCD. Various open and a few double-blind placebo-controlled studies of the effect of combination therapy with a serotonergic antidepressant and an antipsychotic (p. 162) have been carried out. On this background, it is concluded that there is some evidence that risperidone and quetiapine may have an effect in augmentation treatment of OCD."

The guideline referred to page 162 in a NICE report, which was not illuminating.[553] It describes a few small trials and there was no systematic review of these trials.

It is difficult to understand the thinking behind the weak conclusion that risperidone and quetiapine "may" have an effect based on "some evidence." Severe OCD can ruin the lives not only for the patients but also for their relatives, but it is not a deadly disease. In contrast, psychosis pills are some of the deadliest drugs ever invented (see page 46), apart from cancer chemotherapy, and they should be avoided, also for patients with OCD.

As SSRIs double the risk of suicide and have many other important harms, these pills should also be avoided. The book noted that SSRIs and SNRIs may increase anxiety, and that it takes longer than for depression before they work, but that there is continued improvement after several months.[16:368] To say that it takes longer than for depression before they work means they don't work, but the psychiatric mindset doesn't allow such admissions.

On the same page, this book offered horrible advice also about benzodiazepines.[16:368] It mentioned that a study had found an effect after years of treatment, especially with alprazolam and clonazepam, but that generally only a few weeks of treatment is recommended while treatment with a depression pill is started. Alprazolam is a very harmful drug. After a few weeks, many people have become dependent on it, and the rebound effect when it is stopped is so pronounced that the patients become worse than they were when they started therapy.[5:295]

This book also claimed that pregabalin, an antiepileptic, works and is approved for anxiety disorders. It is bad medicine to use antiepileptics for anxiety given their many serious harms, including a doubling of the suicide risk.[390,439]

The literature list did not provide support to the harmful recommendations launched by a psychiatrist as sole author.

Further ahead, another author wrote more soberly about benzodiazepines, contradicting the first author:[16:585] The information on the anxiolytic effect is conflicting and there is a lack of long-term studies. Furthermore, there is development of tolerance (the effect vanishes over time), and cessation causes physical abstinence symptoms, including anxiety, restlessness, irritability, difficulty sleeping, tremor, photo- and phonophobia, flu-like symptoms and rebound phenomena.

Even though the abstinence symptoms are very much the same for SSRIs and SNRIs as for benzodiazepines (see page 80),[554]they were not called abstinence symptoms in any of the textbooks.

The psychiatrist author wrote that when stopping, 10-20% of the starting dose should be removed every other week, but in the last part of withdrawal it may be necessary to reduce with even smaller doses and extend the intervals.[16:586] Another book had similar advice, a dose reduction of 10-20% with 1-2 weeks intervals and possibly even slower in the final phase.[18:71]

It is of utmost importance that the dose reductions are much smaller by the end of a tapering.[281] But none of the books explained that the binding curves for psychiatric drugs are hyperbolic (see page 163), and that the tapering therefore needs to be exponential. This is unfortunate, as very few doctors know about correct withdrawal and cause terrible harms by withdrawing the drugs much too quickly.

A third book recommended either cognitive behavioural therapy or SSRIs for social phobia, for 6-12 months, if there is effect.[18:136] It noted that benzodiazepines should not be used long-term due to dependence, and because abstinence symptoms can be difficult to distinguish from the primary anxiety symptoms. It is true that rebound anxiety is a very common abstinence symptom. But why was the same not said about SSRIs and SNRIs in any of the textbooks? The same problems occur,[554] but the authorities also failed badly, as they ignored the dependence problems with depression pills for two decades.[304]

In the same book, other authors contradicted this, as they said that benzodiazepines are used long term for anxiety such as panic attacks when cognitive behavioural therapy or depression pills have not had sufficient effect.[18:240] This is horrible advice.

The two remaining books escalated the confusion. Two psychologists claimed that SSRIs and cognitive behavioural therapy should often be combined to get the best result in OCD and that most studies had shown remission in 60% of the patients,[20:485] a meaningless statement, as there is no control group. There were 47 references but none of them were about the effect of SSRIs.

The fifth book contradicted this, noting that, according to the National Board of Health,[555] the effect is not increased by adding depression pills to psychotherapy,[17:420] which, also considering the harms, was the reason the Board does not recommend pills.

The authors noted that, on SSRIs, 60-70% will experience a 50% reduction in panic symptoms and 50-60% will have an effect on social phobia, but they added that 60% will experience an effect of placebo on panic attacks.[17:404] What is the reader supposed to conclude based on this?

The authors also claimed that, according to a meta-analysis, about half of the patients with OCD will come in remission but none of their 13 references were to a meta-analysis.[17:420] There was a reference to an article about escitalopram, but it was irrelevant and there was no mention of it in the text.[556]

From then on, it became worse in this book.[17:423] The authors spoke about extensive evidence for the effect of SSRIs and that we should try another one or increase the dose beyond the maximum (in rare cases) if the effect is insufficient. We could also add a small dose of a psychosis pill, which is effective according to clinical experience. But they added that the National Board of Health says that no clinically relevant effect has been shown and that there is risk of harms and that, in some cases, psychosis pills can cause or worsen OCD.

This is confusing and contradictory, and the authors felt that clinical experience is more important that advice from the Board of Heath. Furthermore, it seems that the 2007 national guideline for anxiety disorders[552] is in conflict with the one specifically for OCD.[555] The guideline for OCD was updated in 2019. During these 12 years, the apparent effect of psychosis pills in 2007 disappeared:

"As there is insufficient evidence from the paediatric literature on augmentation therapy with antipsychotics, the question is solely addressed in adults … Use only after careful consideration an atypical antipsychotic as augmentation therapy for adults with severe OCD who have had no effect of treatment with cognitive behavioural therapy and antidepressants (SSRIs), as no clinically relevant effect has been demonstrated and as there is a risk of side effects."

This textbook opined that the primary drug treatment for anxiety is depression pills.[17:664] Benzodiazepines should not be used for more than four weeks but can be used for longer, e.g. if the patient has panic attacks. We are even told that pregabalin can be used because the harms are relatively mild. Antiepileptics have many harms, one of which is to double the risk of suicide.[390,439]

This horrible and harmful advice suggests that patients with anxiety disorders should avoid seeing a psychiatrist. It is too dangerous.

11 Dementia

As noted earlier, an editor of one of the textbooks,[18] Poul Videbech, wrote in 2014 that depression doubles the risk of dementia,[408] but the meta-analysis he cited did not mention with one word which treatments the patients had received.[407] Other studies suggest that it is depression pills and other psychiatric drugs that make people demented.[557,558]

The information about treatment of dementia was highly misleading. In one book, the chapter about Alzheimer's disease was written by two psychologists[20:341] who went into detail about the drugs, even though this is none of their business, as psychologists are not allowed to prescribe drugs in Denmark. They claimed that acetylcholinesterase inhibitors can dampen the development of symptoms[20:351] but none of their 19 references were to research documenting this.

Two other psychologists wrote that the drugs have a better effect on Lewis body dementia and dementia in Parkinson's disease than in Alzheimer's disease on cognitive functions, apathy, visual hallucinations, delusions and other neuropsychiatric symptoms.[20:375] These finger-tip sensations are far-fetched for drugs that don't work (see below), and none of their 38 references were about drug effects whereas several were about psychotherapy and other therapies, e.g. a meta-analysis of the effect of dancing in patients with Parkinson's disease. Very strange, indeed.

Another book claimed that drugs can inhibit the progression of Alzheimer 's disease for months to a few years, and that donepezil, galantamine and rivastigmine have equal effect.[18:48]

A third book mentioned that acetylcholinesterase inhibitors may delay the decline in functional level and behaviour.[17:243] This became more concrete 424 pages later: Drugs, primarily acetylcholinesterase inhibitors, may to some extent re-establish lost cognitive skills as well as postpone further deterioration. The progression in Alzheimer's can be delayed for 6-12 months.[17:667]

A fourth book did not pull any punches either.[16:127] It claimed that, in a minority, a clear improvement of cognitive functions is experienced, with resumption of earlier activities and possible disappearance of hallucinations or other neuropsychiatric symptoms. The authors also claimed that acetylcholinesterase inhibitors may have a beneficial effect on behavioural and psychological symptoms of dementia and may delay their onset.

Dementia was of course not an issue in the textbook about child and adolescent psychiatry.[19]

All these statements are totally wrong. There wasn't a single reference to placebo-controlled trials or meta-analyses, which would have told a story of drugs that don't work and are harmful.[7:197]

The small subjective effects registered in drug trials are likely spurious, as they can easily have been caused by unblinding bias because of the drugs' conspicuous adverse effects.

A 2006 Cochrane review of donepezil, 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."[559] Even without considering the unblinding problem, this conclusion was unwarranted. 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.[560] We may also compare with the smallest effect that can be perceived on the Hamilton scale for depression, which is 5-6, although the maximum is only 52.[267]

The author of the Cochrane review wrote that "donepezil appears to have no serious or common side effects." This is so egregiously false that I don't think Pfizer would have dared claim this in one of their advertisements for Aricept (donepezil).

The harms are both common and serious, which the author of the Cochrane review actually demonstrated herself, as 29% of the patients dropped out of the drug groups, as compared to only 18% in the placebo groups, partly because of more adverse events.[559] The most common harms of donepezil are nausea, diarrhoea, insomnia, vomiting, muscle cramps, fatigue, and anorexia.[561] This is not what we would want for an old person who might already have problems with bad sleep, feeling tired, and eating too little.

The list of frequent adverse effects in Pfizer's product information for Aricept is very long.[561] The drug causes syncope in 1% of the patients and when old people fall, there is a considerable risk that they break their hip and die. A large Canadian cohort study showed that if people with dementia took dementia drugs, they had almost a doubled risk of hospitalisation for syncope, and they had more pacemakers inserted and more hip fractures.[562] More than half of the patients who were admitted to hospital for bradycardia were retreated with the same type of drug after discharge.[562] This is yet another proof that doctors cannot handle psychotropic drugs safely.

A 2014 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, another dementia drug.[563] The title of the paper was appropriate: *Use of medications of questionable benefit in advanced dementia*.

It is interesting that no benefits for society have been found,[564] as we so often hear about the economic burden of dementia and how important it is to intervene with drugs.

The 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, and the authors concluded that donepezil isn't cost-effective, with benefits below minimally relevant thresholds.[565]

In contrast to other trials, this trial was publicly funded. It was excluded from the Cochrane review,[559] and the author used 511 words on explaining why. The main reasons appeared in a table: "Results for the 5 and 10 mg/day groups were not reported separately. Complex design and high numbers of dropouts made analysis and interpretation difficult."

It is not acceptable to exclude a study because it combines two dose groups in the results. And that the design was complex is not a valid reason either for its exclusion. Furthermore, as it was a long-term trial, where more people drop out than in short-term trials, the high drop-out rate was also an invalid reason for exclusion.

The outcome after three years was similar on drug and placebo for institutionalisation, progression of disability, and behavioural and psychological symptoms.[565]

Extremely few trials in psychiatry run for three years but such trials are exactly those we need instead of the thousands of short-term trials we have, which are useless for an assessment of drug effects, as very few patients are treated for only a few weeks.

Six years after the trial was published, 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; "Don't wait. Talk to your doctor about Aricept."[566] The FDA told the company that - with these huge lies - it had broken the law.

You should not talk to your doctor about dementia drugs because, as the textbooks so clearly showed, your doctor is highly likely to mislead and harm you. These drugs should not be used by anyone to prevent or treat dementia.

Three critical comments have been published on the 2006 Cochrane review, including mine.[559] Unfortunately, contrary to good scientific practice, they are undated. The author apologised for an

error, which she said would be corrected in the next version, and she replied to me that another error had "also now been corrected." It has not been corrected. In 2015, I was told that "An update of the review … is in preparation."[559] The review has not been updated. It stands as a gravestone over a once magnificent organisation, which is currently facing big financial trouble because it has not lived up to the expectations of its major funder, the UK National Health Service.[146]

There are other Cochrane reviews of these drugs, e.g. one in vascular dementia, which is not encouraging either.[567] The authors concluded that donepezil and galantamine have a small effect on cognition but that it is unlikely to be clinically important.

One of the textbooks noted that psychosis pills cause considerable harms, e.g. an increased risk of thrombosis in the heart and brain and an increased risk of death.[16:127] It claimed that risperidone and olanzapine have a documented minor effect in dementia.[16:127] This was in a chapter about dementia written by two doctors who work with these patients. In another chapter, about psychopharmacology, the author contradicted this, as she noted that psychosis pills should be avoided in elderly people with dementia and behavioural disorders due to the lack of evidence for an effect, increased sensitivity to harms, and an increased risk of stroke.[16:561] She did not mention the most important reason to avoid these drugs: To avoid killing patients in large numbers (see Chapter 7).

This demonstrated a general issue. People who treat patients become carried away by their "clinical experience" and other biases and are much too positive towards the effects of psychiatric drugs. They are therefore not the most trustworthy textbook authors. They have many vested interests, too, very often financial ones related to the drug industry.

Even people who should know better can be disappointing. A clinical pharmacologist acknowledged at a public meeting that the drugs don't work but he recommended that they should be tried, as they work better in some people than in others. I asked him if he had never heard about statistical variation. With his argument, we could use whatever we pleased that doesn't work.

The perspective is chilling. Doctors are like children. They cannot keep their fingers away from dangerous toys, which is why we should take all the ineffective and dangerous psychiatric drugs off the market. I suggested this in a newspaper article in 2014.[189]

As I doubted it could be true that risperidone and olanzapine work for dementia,[16:127] which no drugs do, I browsed the Internet and found a trial of olanzapine.[568] I had been duped again. It was not about having an effect on dementia but about calming down disturbing Alzheimer patients with a major tranquilliser, and the patients became somnolent and developed gait disturbances. I also found a Cochrane review, but this was also not about treating dementia but about treating aggression and psychosis in people with dementia. Everything I found was about this.

This book noted that the effect of depression pills is very limited and added that a minority without depression develop depression after discontinuation.[16:131] This is interesting because it is an iatrogenic harm, an abstinence depression (see page 114).

Yet again, this was not about treating dementia, it was about treating depression in people with dementia. I found a Cochrane review, which was also discouraging.[569] It noted that the data were of variable quality and unsupportive: "On the only measure of efficacy for which we had high-quality evidence (depression rating scale scores), antidepressants showed little or no effect."

As noted earlier, it is likely that all psychotropic drugs can cause chronic brain damage,[5,135] which may be permanent. A hallmark of this is impaired cognitive function. 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, the patients should have become worse when the drugging was reduced.[135] A 17-year follow up of the Framingham Heart Study found that use of depression pills increased the risk of developing dementia by about 50%,[570] and benzodiazepines seem to double the risk of dementia.[571]

We should avoid drugging demented people. We should care for them. A systematic review of 33 trials of agitated demented people showed pretty large effects of care, e.g. communication skills training, activities, music, touch, massage and talking to people.[572]

12 Electroshock

Electroshock, also called electroconvulsive therapy (ECT), was highly praised in the textbooks. One book recommended ECT or pulsating electromagnetic fields (PEMF) for treatment resistant depression,[16:275] and another book noted that ECT must always be considered for this condition.[17:364]

It was claimed that ECT stimulates the formation of new neurons and the maintenance of the dendrite tree,[16:558] and the development of new neurons in hippocampus.[17:746] A third book noted that no acute or permanent brain damage had been demonstrated in the many scanning studies, and that a few studies suggest that the neurogenesis in the hippocampus increases.[18:245]

The truth is that the brain reacts to harm by producing new neurons.[11] A harmful effect was therefore praised as being beneficial, which is common in psychiatry. There were no references.

One book claimed that it has not been possible to detect brain damage; that retrograde amnesia is difficult to interpret and difficult to distinguish from problems triggered by the disease; that some studies suggest a slight memory loss a year after ECT whereas other studies do not find it; and that long-term symptoms experienced by the patients after ECT are extremely rare and not with certainty related to it.[17:745] By using the word "experienced," the author downgraded what the patients tell their psychiatrists about the harms of ECT.

In another book, the same author claimed that brain damage has never been diagnosed after ECT while noting that almost all patients get amnestic symptoms in a treatment series.[16-556] This is full-blown cognitive dissonance. If amnesia after ECT is not a sign of brain damage, what is it then? How can anyone argue this way? People who become amnestic after a concussion are told it is because they had a brain damage.

The author explained that the anterograde amnesia recovers two weeks later while retrograde amnesia is more uncertain. He noted that some studies suggest a slight memory loss 6-12 months later whereas prolonged experiences of inconveniences are extremely rare. This author, a professor of psychiatry, ignored the facts when asking if the problems were due to ECT or the disorder.

Other authors also denied the facts. They noted that, rarely, a few patients experience "subjective inconveniences" in the form of lacunas in retrograde memory and claimed that it is difficult to judge if they are harms of ECT because patients with severe depression also often have such lacunas.[18:244]

The memory problems are not just subjective (which is the standard script: Blame the victim, not the treatments); they have been verified in numerous studies.

Elsewhere in this book, the authors wrote about a short-term memory dysfunction, and that thorough studies with imaging methods had not shown damage to the nerve tissue.[18:231]

This is just incredible. ECT causes memory loss in most patients[573-575] and permanent memory loss in some patients, which means irreversible brain damage.[96,121] ECT furthermore kills some patients,[573] which means that every single brain cell is dead.

The organised denial of the harms caused by ECT was astounding. My translation of the above is: We psychiatrists do not worry about the memory problems we cause; the patients already had memory problems before we electroshocked them; the memory problems patients tell us about are not real (only "subjective);" and we need not pay attention to what the patients tell us anyway because they are mentally ill. In my view, psychiatrists are too dangerous to have around.

The descriptions of what ECT does to people are among the most dishonest I encountered when reading the five textbooks, and this also applies to the postulated benefit. We are told that

ECT is extremely effective against severe depression;[18:231] and that it can be lifesaving.[18:244] This agrees poorly with the information in the same book that, usually, 8-16 shocks are given.[18:244] ECT is also used in patients with mania to prevent delirium acutum.[18:114]

It was claimed that 80% of patients with affective disorders respond to ECT,[17:360] but there was no control group and no reference.

Here is an account of the facts.[7:207] In the Cochrane review of ECT for patients with schizophrenia, which is from 2005,[576] more people improved on ECT than on placebo or sham ECT, risk ratio 0.76 (0.59 to 0.98), but this finding is unreliable. It was barely statistically significant; the trials were small (only 392 patients in 10 trials); the larger the trial, the smaller the effect, which suggests that negative trials exist that haven't been published; and the authors only excluded trials from their review if more than 50% of the patients were lost to follow-up, which is far too generous. Other researchers have concluded that all the sham ECT trials are grossly flawed.[577]

The Cochrane authors reported that, using the Brief Psychiatric Rating Scale, ECT was better than sham ECT, but there were only 52 patients in the analysis, and we have no idea how many patients or data that were missing or why. Further, the difference was only 6 on a scale that goes to 126, which is not a clinically relevant effect (see page 45 about a similar lack of a relevant effect of psychosis pills).

Even more worrying, ECT was considerably *less* effective than psychosis pills, e.g. twice as many patients weren't improved in the ECT group, risk ratio 2.18 (1.31 to 3.63).

The authors didn't draw firm conclusions about any short-term benefit, and there was no evidence for a long-term benefit.

A 2003 review found that ECT was more effective than simulated ECT for depression (6 trials, 256 patients, effect size –0·91 (–1·27 to –0·54), corresponding to a Hamilton score difference of 10, and ECT was also better than drugs (18 trials, 1,144 patients, effect size –0·80 (–1·29 to –0·29).[578] This looks impressive, but these are short-term effects; 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 outcomes relevant for clinical practice; and the data suggested that ECT caused cortical atrophy in the brain. 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.

Psychiatric researchers often avoid saying in plain language what they found and what it means, as it would be threatening to the psychiatric guild. They should have said that it is uncertain if ECT for depression does more good than harm, particularly as it caused brain damage and as only short-term studies were evaluated. Systematic reviews have failed to find benefits beyond the treatment period, both for schizophrenia and for depression.[573,578]

Many psychiatrists believe ECT can be life-saving, but there are no reliable data in support of this belief,[573,578] whereas we know for sure that ECT can be deadly. A systematic review found a death rate of about 1 per 1000,[573] which is 10 times higher than what the American Psychiatric Association says. When I lectured in Brisbane in 2015, a mother told me that the psychiatrists killed her son with ECT but they resuscitated him. When he woke up, he had severe burns and 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.

In 2003, the UK Royal College of Psychiatrists' fact sheet stated that more than 80% of depressed patients respond well to ECT and that memory loss is not clinically important.[575] We do not ask

a hairdresser if we need a haircut. The patients disagreed and the lowest satisfaction levels were obtained in studies led by patients rather than by psychiatrists.

If we want to know the truth about psychiatric drugs and electroshock, we need to listen to the patients and not to the psychiatrists.[121] One Danish patient couldn't remember even the commonest things, like the name of the Danish capital, after she was electroshocked.[121] She was permanently brain damaged by electroshocks she should never have received because her problem was that she had been sexually abused as a child. She didn't have any psychiatric disorder. Her book is a frightening account of what is wrong with psychiatry.[121]

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 damning.[575] With a strict definition of memory loss, between 29% and 55% of the patients are affected. With looser criteria, the range goes from 51% to 79%.

Other studies also show that ECT may cause permanent brain damage.[573] 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.

It is blatantly dishonest to say, as the psychiatrists who authored a Cochrane review of depressed elderly did,[579] 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".

The 2010 official guidance for general practitioners in Denmark on depression was even worse. It stated 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."[580]

ECT "works" by making people confused and by destroying their memories, which are what define us as humans, but doctors describe this as positive. They also described lobotomy and the many other harmful treatments they used in the past as positive.[1]

As illustrated by the case in Brisbane, what happens in practice is far from what should happen. This has been studied systematically. Repeated audits by the Royal College of Psychiatrists showed that many hospital trusts failed to adhere to the college's standards.[575] One audit found that only a third of ECT clinics met the standards.[578] There are also huge variations in clinical practice and in rates of usage.[573,575,578]

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.[581]

There is a very moving documentary about Mette Askov, a Danish nurse who had heard voices since she was eight years old and was a psychiatric patient for 15 years.[582] She was diagnosed with paranoid schizophrenia and received vast amounts of medicine, 150 electroshocks and a disability living allowance. She was stigmatised and surrounded by prejudice but after she reclaimed her own life and left psychiatry, she achieved some of her greatest goals. Her story illustrates so well what the psychiatrists' abuse of forced treatments lead to. Even when they so clearly don't work, the psychiatrists continue to use them.

I have heard many stories where psychiatrists describe miraculous improvements and grateful patients. I was once asked at a meeting after my lecture about drugs what my view was about a

woman who was so depressed that she could hardly be contacted but asked for a glass of water after an electroshock.[8:87] I said that since this was an anecdote, I would reply with another anecdote. I examined a newly admitted man, an unconscious alcoholic, and as I needed to rule out meningitis, I tried to insert a needle in his back to tap cerebrospinal fluid for microscopy and culture. It was very difficult to get in and I hit his bone several times. All of a sudden, the drunkard exclaimed loudly: “Bloody hell, stop stinging me in the back!” Had I caused a miracle with my needle and cured the guy? No. Odd things happen all the time in healthcare. Could I have woken up the deeply depressed woman with my needle? Who knows, but maybe?

Some psychiatrists I have met have never used electroshock. This barbaric treatment should be made illegal, just as lobotomies were. In particular, no one should be forced to get electroshocks against their will.

13 Forced treatment

The textbooks were rather silent about this important issue, which is remarkable, as forced treatment is highly controversial.[7:314]

As power corrupts, there needs to be a power balance in human relations. However, involuntarily admitted patients are powerless. This extreme power imbalance is a recipe for disaster, and there is nothing psychiatric patients fear more than forced treatment. Some psychiatrists have administered electroshocks to the patients they disliked the most, and doctors have regularly prescribed shocks for those patients who were fighting, restless, noisy, quarrelsome, stubborn and obstinate.[1:106]

There is a high risk that forced treatment is being used to benefit staff rather than patients to make their work less stressful, which is the major reason for the popularity of psychosis pills when they appeared in the 1950s.[1] In Europe, the oversight of forced treatment comes under the convention prohibiting torture, and a committee has observed that deliberate ill-treatment of patients in psychiatric establishments still occurs.[583]

I have provided a long account of the abuses in another book[7:314] and shall only comment on a few issues here.

The European Committee for the Prevention of Torture has noted that, on inspection, it all too often finds that fundamental components of effective psychosocial rehabilitative treatment are underdeveloped or totally lacking, and that the treatment consists essentially of drugs.

The laws about forced treatment are 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.

Are there any treatments that can cure insane patients or 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, also because its premise is false.

The other lawful reason for forcing drugs on people is if they present an obvious and substantial danger to themselves or others. This is also an invalid argument. Psychiatric drugs *cause* suicide and violence[7,8] and they cannot *protect* against violence unless the patients are drugged to such an extent that they have become zombies. According to the National Italian Mental Health Law, a reason for involuntary treatment cannot be that the patient is dangerous. This is a matter for the police.

Rare cases like forced feeding for life-threatening anorexia are already covered by other laws than those that apply specifically to psychiatry. And severe mania where the patient may be busily spending his entire wealth can also be handled without forced hospitalisation and treatment. For example, an emergency clause could be introduced that removes the patients' financial decision-making rights at short notice. Furthermore, a few difficult cases cannot justify that massive harm is inflicted on the patients in general,[7] which also makes it difficult to recruit good people to psychiatry. No one likes coercion, and it destroys the patient's trust in the staff, which is so important for healing and for the working environment in the department.

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 do if she is 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 about 1000 emails I have received from patients and relatives describe exactly this.

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 the widespread abuse if we keep it. There are no randomised trials that have compared the use of force with no use of force but we know enough already. Mechanical restraint and ECT can be fatal; and, as explained earlier, psychosis drugs, other psychiatric drugs, and contact with a psychiatric ward kill an enormous amount of people.

One of psychiatry's 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 didn't find any differences in service use, social functioning or quality of life compared with voluntary care or brief supervised discharge.[584] In clinical practice, this initiative has also failed. After the UK had introduced these treatment orders, hospital admissions increased.[585] 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. Some psychiatrists find treatment orders unethical and many patients find them stigmatising.

In 2007, the UK mental health charity, Mind, expressed many concerns.[586] 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 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. Many people consulted by Mind felt their relationships with professionals would be harmed by the increased threat of compulsion, with those professionals being turned into "Mental Health Act police officers."

The therapeutic relationship is what matters the most, and if you have been a cop and have used force, it becomes nearly impossible to change that role into the role of the physician as a healer and advocate for the patient.[7:327] This is why psychiatrists should stay out of the job of being police. Another reason is that violence breeds violence. Loren Mosher testified in a Supreme Court case in Alaska and 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.

Lawyer Jim Gottstein convinced the court to rule 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. Gottstein used scientific data to prove that it was not in the patients' best interest to treat them forcefully.[7:328]

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.[587,588]

Psychiatrists should consider 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 and reminds us of the living conditions in concentration camps where it is important to never provoke the guards, which will lead to harsh punishment.

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 tells us the opposite; forced treatment kills patients.[7] A patient told me that she likened forced treatment to rape and that there cannot be good rapes. This patient was raped by a man in her family when she was only nine years old and became terrified when the staff subjected her to forced treatment.

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 insightless because he had alerted the community to the brain-damaging effect of psychosis drugs! Another person I met was a psychiatrist who was considered insane by her colleagues, also because she spoke out about psychiatric drug harms. They tried to have her involuntarily confined to hospital but failed.

In 2014, the Danish Ministry of Health issued a licence to kill. It allowed psychiatrists to use extraordinarily large doses of psychosis drugs 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.[589] It's insane. These patients should have their drug withdrawn. Giving more of what was already not working doesn't help, it kills.

Since forced treatment is not evidence-based but culture-based, it is no surprise that practices vary enormously between countries. Involuntary hospital admissions in Europe range from 12 per 100,000 inhabitants in Italy to 233 in Finland.[587] Once admitted, rates of coercion also vary enormously. In Austria, mechanical restraint is used 45 times more often than in the Netherlands, where forced drugging is also used very little.[590]

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.[184]

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. 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."

The Convention makes it clear that "unsoundedness 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.[7:335]

Everyone who argues for forced treatment and involuntary detention should read a heart-breaking book, *Dear Luise*, [234] which I have summarised[7:337] and briefly mentioned in Chapter 7.

In his foreword, *You need to be strong in order to be vulnerable*, former Danish Prime Minister Poul Nyrup Rasmussen describes the book as heart-breaking. It truly is. It could be used as a screening test for doctors who contemplate to become psychiatrists. If they get through it without crying, they should find themselves another job.

Luise's best friend at the care home, who 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," which she became six months later. She and her mother protested against her treatment. The psychiatrist didn't care and killed her with a depot injection.[7:337]

The level of ignorance and the lack of respect for Luise and her mother who knew a lot about the drugs was astounding. Luise's mother did everything she could to avoid that Luise got over-dosed and begged the staff not to overdose, but Luise died from an overdose.

When Luise's mother complained to the authorities after the death, the system replied that Luise had received the highest standard of specialist treatment while it congratulated itself with its first-class homicide which they called a "natural death." Many relatives have experienced that psychiatrists killed their loved ones, and in Denmark they have united in the association *Death in psychiatry*, which demonstrates in front of the hospital every year on Luise's death day.

The book, which has been translated into English,[234] describes virtually everything that is wrong with psychiatry including making incorrect diagnoses. Whenever I open it again, I get overwhelmed with sadness because I know the author and also that many psychiatric patients are abused and die under similar circumstances as Luise and her best friend. Luise was a slow metaboliser, and her mother had begged the psychiatrists never to use a depot injection, which was what killed her daughter.

Being treated humanely is difficult in today's psychiatry. If you panic and go to a psychiatric emergency ward, you will probably be told you need a drug, and if you decline and say you just need rest to collect yourself, you might be told that the ward is not a hotel.[591]

This is bad medicine. Impending psychoses can sometimes be fended off before they develop if we provide patients with the shelter and rest they need. There should be 24-hour support facilities without any compulsion, so that the hospital is no longer the only place patients in acute crisis can go to.[592] There could be refuges with the possibility of accommodation and the money should follow the patient and not the treatment.

Psychiatry seems to be the only area in society where the law is systematically being violated all over the world - even Supreme Court and Ombudsman decisions are being ignored.[7:328,593,594]

We studied 30 consecutive cases from the Psychiatric Appeals Board in Denmark and found that the law had been violated in every single case.[594,595] All 30 patients were forced to take psychosis pills they didn't want, even though less dangerous alternatives could be used, e.g. benzodiazepines.[165] The psychiatrists had no respect for the patients' views and experiences. In all 21 cases where there was information about the effect of previous drugs, the psychiatrists stated that psychosis pills had had a good effect whereas none of the patients shared this view.[595]

The harms of prior medication played no role either in the psychiatrist's decision making, not even when they were serious, e.g. we suspected or found akathisia or tardive dyskinesia in seven patients, and five patients expressed fear of dying because of the forced treatment. An expert confirmed our suspicion that a patient had developed akathisia on aripiprazole (Abilify) but on the

same page, the expert - a high-ranking member of the board of the Danish Psychiatric Association - recommended forced treatment with this drug even though it was stopped because of the akathisia.[595]

The power imbalance was extreme. We had reservations about the psychiatrists' diagnoses of delusions in nine cases, and there is an element of catch-22 when a psychiatrist decides on a diagnosis and the patient disagrees. According to the psychiatrist, the disagreement shows that the patient has a lack of insight into the disease, which is a proof of mental illness. The abuse involved psychiatrists using diagnoses or derogatory terms for things they didn't like or didn't understand; the patients felt misunderstood and overlooked; their legal protection was a sham; and the harm done was immense.[595]

The patients or their disease were blamed for virtually everything untoward that happened. The psychiatrists didn't seem to have any interest in traumas, neither previous ones nor those caused by themselves or their staff. Withdrawal reactions were not taken seriously - we didn't even see this term, or a similar one, being used although many patients suffered from them.

It is a very serious transgression of the law and of professional ethics when psychiatrists exaggerate the patients' symptoms and trivialise the harms of the drugs to maintain coercion, but this often happens, and the patient files can be very misleading or outright wrong.[7,121,234,595] In this way, the psychiatrists can be said to operate a kangaroo court, where they are both investigators and judges and they routinely lie about the evidence,[7:329] where after they sentence the patients to a treatment that is deadly for some of them and harmful for everyone.

In Denmark, when the patients complain about this unfair treatment, which isn't allowed in any other sector of society, it is the same judges (or their friends that won't disagree with them) whose evidence and judgments provide the basis for the verdicts at the two appeal boards, first the Psychiatric Patients' Complaints Board, and next, the Psychiatric Appeals Board. It doesn't matter the slightest bit what the patients say. As they have been declared insane, no one finds it necessary to listen to them. This is a system so abominable that it looks surreal, but this is the reality all over the world.

In one of the textbooks, under the section, *The violent and aggressive patient*, the authors mentioned some drugs that, in rare cases, can cause motor restlessness and increase restlessness and aggression. These drugs are benzodiazepines, amphetamine, anabolic steroids, and testosterone.[17:821]

It is inexcusable that the authors did not mention that depression pills, methylphenidate and psychosis pills can also cause such symptoms and did not mention akathisia either, one of the most dangerous drug harms. This is yet another example that psychiatrists protect their guild.[596]

Further ahead, the authors noted that studies suggest that the patients' aggression can be seen as a reaction to conflicts among the staff, and they said that a newer study pointed out that increased patient autonomy can reduce violent behaviour and the use of coercion.[17:828] We all know it can reduce aggression to respect other people. This is what international diplomacy is about, and no scientific studies are needed to confirm this.

However, the respect for the patients lasted only one page. On the next page, we are told that not using psychotropic drugs for patients that are agitated, aggressive or violent and where belt fixation might be needed should only occur exceptionally and then accompanied by a clear argumentation for this in the patient file. A table with suggested interventions included lorazepam, olanzapine, ziprasidone or haloperidol in the acute phase, and clozapine, antiepileptic drugs,

depression pills, or ECT in the follow-up phase. This is a prescription for death and for creating zombies.

The book mentioned the rule about using the least intrusive treatment, but then argued that some patients are permanently incompetent, i.e. permanently lack the ability to consent, and that these include mentally ill people with mental disabilities, chronic mentally ill people, and mentally ill people with long-term illnesses, and that the issue is whether the patients can give a reasonably meaningful informed consent.[17:927] As noted above, these arguments have been rejected by the United Nations Convention on the Rights of Persons with Disabilities.[184]

The same book had a section about forensic psychiatry where it was argued that randomised trials studying the effects of using force cannot be carried out for ethical reasons.[17:926] This is wrong. There are good intentions behind using force in psychiatry, but the harms are massive, and it is not at all clear if force, on average, benefits or harms the patients. Most likely, it is harmful. Therefore, it is ethically acceptable to do randomised trials. During a trial, half of the patients will avoid coercion, and when the trial is over, perhaps all future patients will avoid coercion. What is unethical is to continue subjecting patients to force against their will.[184]

The book argued that, during forced treatment, one should only use medication in usual doses and with the fewest possible harms.[17:929] This contradicts what other authors wrote in the same book 277 pages earlier, that it is appropriate in some cases to increase the dose of psychosis drugs above the approved interval.[17:652]

14 Psychotherapy and the role of psychologists

There wasn't much mention in the textbooks of an independent role of psychologists in mental health. Psychotherapy was often listed as an option, but almost always in a context that also involved drugs. It was implicitly understood that even psychotherapy was the responsibility of psychiatrists. When reading the books, I did not doubt that the psychiatrists had won the decades old battle with the psychologists and had absolute power over everything in mental health.

It was almost as if the psychological profession did not exist. When anything was specifically mentioned in relation to psychologists, they were reduced to being servants of the psychiatrists.

This was particularly clear in the textbook about child and adolescent psychiatry.[19] All the editors were psychiatrists and they protected their guild. The book started out by saying that children and young people with mental disorders must be referred to a child and adolescent psychiatrist if there is psychopathology and the problem is too complicated for general practitioners or social workers.[19:13] There was nothing about which help psychologists can offer and the advice contained a pleonasm: If a person has a mental disorder, there is psychopathology, which is just another name for the same thing.

Psychologists were mentioned only as testers.[19:15,19:25] They test the cognitive level and attention and do projective tests like the Rorschach test where the patients are shown a series of irregular, symmetrical inkblots and explain what they see.

It was noted that the first clinical assessment could be made by a general practitioner, in healthcare, at a paediatric ward or in an emergency room.[19:14] Psychologists were not mentioned but referrals could also come from school psychologists. And older children and young people could take the initiative themselves, for example by contacting a psychologist.[19:14] However, many parents take their children, also young ones, to a psychologist and would never contact a psychiatrist as the first step. In one of my books, I write:[8:4]

"If you have a mental health issue, don't see a psychiatrist. It is too dangerous and might turn out to be the biggest error you made in your entire life."[597] The quote is from Peter Breggin, a psychiatrist who avoids using drugs. As noted on the first page in this book, the public knows very well that there is a great risk that they or their children will be harmed if they contact psychiatry.[12]

In 1992, the UK Royal College of Psychiatrists, in association with the Royal College of General Practitioners, launched a five-year *Defeat depression campaign*.[8:1,494] Its aim was to provide public education about depression and its treatment in order to encourage earlier treatment-seeking and reduce stigma. Campaign activities included newspaper and magazine articles, television and radio interviews, press conferences, production of leaflets, factsheets in ethnic minority languages, audio cassettes, a self-help video and two books.[598] The colleges had accepted donations from all the major manufacturers of depression pills for the campaign, and the president of the Royal College of Psychiatrists, Robert Kendall, acknowledged that their motive was to sell more pills.[8:2]

When 2,003 lay people were surveyed before the launch of the campaign, 91% thought that people with depression should be offered counselling; only 16% thought they should be offered depression pills; only 46% said they were effective; and 78% regarded them as addictive.[494]

The psychiatrists replied pompously: "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 that prescribe psychiatric drugs, but they are so much out of touch with reality that no amount of training will get them close to where the patients and the general public want them to be.

There is also institutional corruption.[599] Just before fluoxetine (Prozac) reached the market in 1988, NIMH surveyed the public about its views on depression, and only 12% wanted to take a pill to treat it.[5:290] However, the NIMH was determined to change this attitude and launched a public awareness campaign claiming that 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. The postulated 45% difference in effect is fraudulent; even the FDA found only 10% in flawed trials,[303] and the patients do not get better on drugs. They get worse, which is why 12% more patients leave the trials when they are on drug than when they are on placebo.[301] The campaign was immensely successful, and the media praised Prozac as the new wonder drug.

A chapter on psychotherapy written by a psychologist, professor Nicole Rosenberg, was unusually well documented. She wrote that cognitive behavioural therapy has a small effect in schizophrenia; is effective against depression, also in preventing relapse and in getting people back to work; and works for anxiety, with large effects for generalised anxiety, social phobia and post-traumatic stress disorder (PTSD).[16:597]

This is important information, particularly that psychotherapy can get depressed people back to work. It has never been documented that depression pills have such an effect, and they seem to have the opposite effect. The rate of disability pensions follows the usage rates for psychiatric drugs,[5:8,119:24] and most of these drugs are depression pills.[7]

Rosenberg mentioned many names in the text, e.g. a Cochrane review by Niewenhuijsen, a 2006 meta-analysis by Butler, and a 2007 meta-analysis by Norton and Price of 108 studies, but many of the papers didn't appear in the literature list, which only had 16 references.

Textbook authors should not play hide and seek with the readers about important statements. It is often difficult, and sometimes impossible, to find the papers.

I found three Cochrane reviews with Niewenhuijsen as author. One was about interventions to improve return to work in depressed people, published in 2012 and updated in 2020.[600] It found moderate quality evidence based on three studies that telephone or online cognitive behavioural therapy was more effective in reducing sick leave than usual primary or occupational care, effect size -0.23 (-0.45 to -0.01). In the 2020 update, there were more studies of psychotherapy, and the effect was now -0.15 (-0.28 to -0.03).[601]

When I searched on Butler in the author field, 2006 in the publication year field, and meta-analysis in the title field, there were no records on PubMed. People named Butler had published 663 articles in 2006, but only 161 had Butler as first author. Sorting these by best match yielded a review of meta-analyses as the top record.[602]

The authors had reviewed 16 methodologically rigorous meta-analyses and reported that the effect sizes for cognitive behavioural therapy were large for unipolar depression, generalised anxiety disorder, panic disorder with or without agoraphobia, social phobia, posttraumatic stress disorder, and childhood depressive and anxiety disorders, and that the effect of cognitive behavioural therapy was somewhat superior to depression pills in the treatment of adult depression.

When I searched on Norton as I had done for Butler, there were no records, but after having tried various strategies, I found "a meta-analytic review."[603] It included 108 trials of cognitive behavioural therapy and reported that this therapy and exposure therapy - alone, in combination,

or combined with relaxation training - were efficacious for anxiety disorders, which included generalised anxiety disorder, posttraumatic stress disorder and social phobia.

The aim of psychological treatments is to change a brain that is not functioning well back towards a more normal state.[8:89] Psychiatric drugs also change the brain, but by creating an artificial third state – an unknown territory - that is neither normal nor the malfunctioning state the patient came from.[604]

This is problematic because you cannot go from the chemically induced third state back to normal unless you taper off the drugs, and even then, it will not always be possible, as you might have developed irreversible brain damage.

A humane approach to emotional pain is very important, and treatment outcomes depend more on therapeutic alliances than on whether psychotherapy or pharmacotherapy is used.[605] Furthermore, the more in agreement physicians and patients are about what is important when being cured from depression, the better the outcomes for positive affect, anxiety and social relationships.[606]

Most of the problems patients face are caused by maladaptive emotion regulation. Psychiatric drugs make matters worse, as their effects constitute exactly this, maladaptive emotion regulation.[607] In contrast, psychotherapy aims at teaching patients to handle their feelings, thoughts and behaviour in better ways, which constitutes adaptive emotion regulation. It may permanently change patients for the better and make them stronger when facing life's challenges.

In accordance with this, meta-analyses have found that the effectiveness of psychotherapy compared with depression pills depend on the length of the trial, and psychotherapy has an enduring effect that clearly outperforms pharmacotherapy in the long run.[497-501,503] In one meta-analysis, the effect size was 0.26 (P = 0.003).[498] In another meta-analysis, there was a trend toward better long-term effect of acute psychotherapy compared with ongoing pharmacotherapy, odds ratio 1.62 (0.97 to 2.72).[499] As in other meta-analyses, there were also more dropouts in the acute phase on drug than on psychotherapy, odds ratio 0.59 (0.34 to 0.99). The patients are better helped by psychotherapy, which is also what they prefer but rarely get (see page 125).[494-496]

Short-term results are misleading. We should only take results into consideration if they have been obtained after at least a year. We also need to consider that trials that have compared psychotherapy with drugs are not effectively blinded, neither for psychotherapy nor for drugs. The prevailing belief in the biomedical model would be expected to influence the psychiatrists' behaviour during the trial and to bias their outcome assessments in favour of drugs over psychotherapy.

Trials that show that the effects of a drug and psychotherapy combined are better than either treatment alone should also be interpreted cautiously, and I will not advocate the combination. Providing effective psychotherapy can be difficult when the patients' brains are numbed by psychoactive substances, which may render them unable to think clearly or to evaluate themselves. As noted earlier, the lack of insight into feelings, thoughts and behaviours is called medication spell-binding.[135,159] The main biasing effect of medication spellbinding is that the patients underestimate the harms of psychiatric drugs, which they have gotten used to.

In June 2022, I witnessed a PhD defence in Copenhagen.[607] One of the examiners, a psychologist, made a lot out of saying that psychotherapy wasn't any better than drugs for depression. It provoked me so much that – when I was allowed to comment after the defence was over – I noted that it was not appropriate to refer to short-term results obtained with the Hamilton rating scale when comparing the two treatments because this ignores that psychotherapy does not cause

withdrawal symptoms or destroy people's sex lives; that pills cannot teach patients anything which psychotherapy can; and that pills double the risk of suicide whereas psychotherapy halves this risk.[272]

The examiner did not reply, but the other examiner, a psychiatrist, noted that psychotherapy does not always work and when the patients come to him, they have already tried it in vain. This reply is typical for psychiatrists. But pills that do not have clinically relevant effects and double the risk of suicide, the most feared outcome of a depression, cannot be legitimised this way.

I shall not go into detail about psychotherapy. There are many methods and schools, and it is not so important which method you use. It is far more important that you are a good listener and meet your fellow human being where he is, as Danish philosopher Søren Kierkegaard advised us to do two centuries ago. As there are many trials with cognitive behavioural therapy, this tends to be the preferred method, but if used too indiscriminately, it can be a sort of cook-book approach that pays too little attention to the concrete patient's special circumstances, wishes and history.

Psychotherapy seems to be useful for the whole range of psychiatric disorders including psychoses[7,253] (see also earlier chapters). It does not work for everyone. But this should not make us use inefficacious and harmful drugs. Some people cannot be helped no matter what we do, also in other areas of healthcare. We cannot help most patients with cancer and use chemotherapy far too much out of desperation,[46] ruining people's lives, rushing them in and out of hospital, instead of giving them a peaceful time with their loved ones without drugs.

Physical and emotional pain have similarities. Just like we need physical pain to avoid dangers, we need emotional pain to guide us in life.[591] According to a Swedish psychiatrist who does not use drugs, we learn something important through the process of healing that can be useful if we get in trouble again, which can boost our self-confidence. In contrast, doctors may think they need not engage themselves as much when a patient is taking drugs.[591]

15 Withdrawal of psychiatric drugs

Psychiatrists and other doctors know very little about abstinence symptoms, which they mainly reject, and about how to taper off psychiatric drugs safely.[135] One should never start psychiatric drug treatment without having a tapering plan, but no one taught doctors how to stop the drugs, whereas they have learned from their professors and the pharmaceutical industry when to start them and always to blame the disease for untoward symptoms, ignoring the troubles they have caused.

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 in a day.

Patients who want to stop drugs are mostly left to fend for themselves and they share their experiences on the Internet and on social media. This is the reason why I thought it might be valuable to write a book about why and how to withdraw psychiatric drugs.[8] Volunteers found the book so important that they translated it into Spanish, French and Portuguese. It is available in these languages on my website, scientificfreedom.dk, and has also appeared in print in English,[8] Danish, Swedish, Dutch and Italian.

Few people can taper off the drugs themselves, and psychiatrists may feel disrespected when patients ask to come off the drugs they have instituted. A common 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.

Most patients are unable to judge themselves because the drugs have changed their brains. When 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. 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."[21,135]

This is one of the reasons it is so essential that patients are not alone, but that close relatives or friends can observe them carefully. It can be dangerous if the patient's false explanations are accepted. The patient should therefore allow friends and family to contact the therapist if they are concerned.[135] When patients have left suicide notes, only very rarely is there any indication that the drug was the problem; patients don't know this and think they have gone mad.[7:79]

It often requires strong determination, a lot of time, patience, and a long tapering period to come off the drugs while making the abstinence symptoms bearable. It can usually be done within a few months but can take more than a year. 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,[135] 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.

What we need more than anything else in psychiatry are withdrawal clinics, with easy and quick access free of charge, and education about the harmful effects of psychiatric drugs, how to stop them, and above all: How to avoid starting them. Public investment in such clinics would be highly profitable and beneficial in terms of fewer disability pensions, fewer suicides and other drug deaths, much healthier citizens, and fewer serious crimes.

Nurses, psychologists, social workers, teachers and other non-prescribing people have often been taught that their task is to push people to get a diagnosis and to comply with the prescribed medication. They should be taught the opposite, that psychiatric diagnoses should be avoided and that drugs should be used for as short a time as possible and preferably not at all.

Patients don't care about the academic wordplays whose only purpose is to allow the drug companies to continue intoxicating whole populations with mind-altering drugs. The patients know when they are dependent; they don't need a psychiatrist's approval that their experience is real, and some say the withdrawal from a depression pill was worse than their depression.[608]

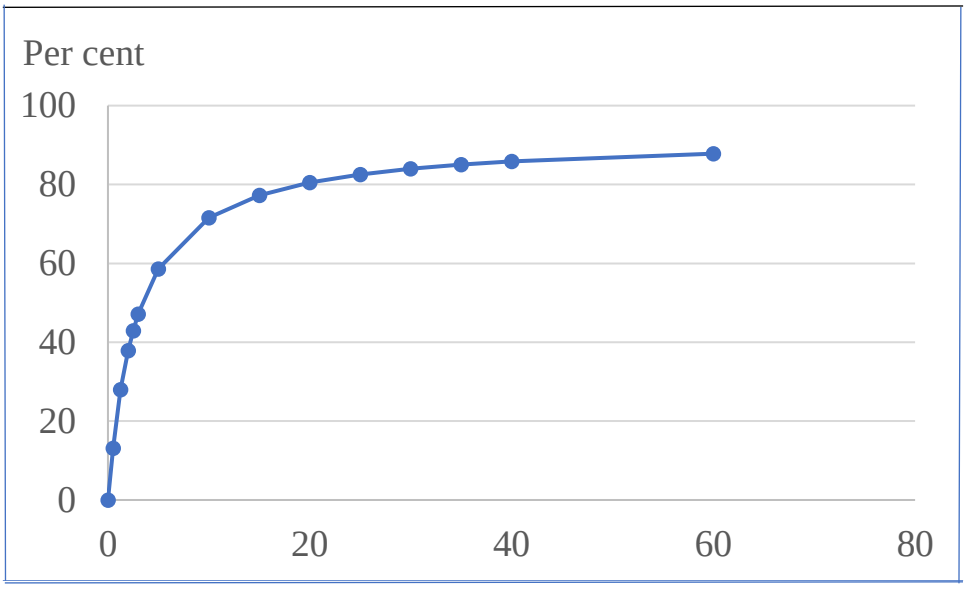
The patients have been fooled by their doctors who were fooled by their leaders who were fooled by the drug industry. A recent survey of 1829 New Zealanders who were on depression pills showed that only 1% had been told anything about withdrawal effects or addiction.[609]

Progress is very slow. In 2020, the UK mental health charity Mind said it signposted people to street drug charities to help them withdraw from depression pills because of the lack of available alternatives. A voiceover said on BBC about this initiative: "Although they are not addictive, they can lead to dependency issues." What's the difference?

In November 2019, the Danish National Board of Health issued a guideline about depression pills to family doctors that was dangerous. As I knew from experience that it doesn't lead anywhere to complain to the authorities, I published my criticism in a newspaper article.[195] The Board of Health was given the opportunity to respond but declined – a sign of the arrogance at the top of our institutions related to important public health issues. They won't admit they got it wrong.

Although the author group for the guideline included a psychiatrist and a clinical pharmacologist, they didn't seem to know what a binding curve for depression pills to receptors looks like (see graph).

Hyperbolic relationship between receptor occupancy and dose of citalopram in mg



(Courtesy of Mark Horowitz[281])

As with other medicines, the binding curve is hyperbolic. It is very steep in the beginning when the dose is low, and flattens out and becomes almost horizontal at higher doses.[281,610]

It is important to be familiar with these issues. With my Danish colleagues, who have withdrawn many patients, I have written repeatedly about the principles in Danish newspapers and elsewhere since 2017. It is therefore strange that the board recommends halving the dose of depression pills every two weeks, which is far too risky.

At usual dosages, most receptors are occupied because we are at the top of the binding curve where it is flat. Since virtually all patients are overdosed, they might remain on the flat part of the binding curve after the first dose reduction and not experience any withdrawal symptoms. It could therefore be okay to halve the dose the first time. But even this might cause problems because psychiatric drugs are nonspecific and influence more than one type of receptor.[281] We don't know the binding curves for all these receptors. The patient could be on the steep part of the curve for one of the receptors at the start, or on the steep part in particular regions of the brain.

Already the next time, when going from 50% of the starting dose to 25%, things can go wrong. Should the withdrawal symptoms not occur this time either, they will almost certainly come when you take the next step and come down to 12.5%.

It is also too fast for many patients to change the dose every two weeks. The physical dependence can be so pronounced that it takes many months or years to fully withdraw from the pills.

As noted earlier, fast withdrawal can cause akathisia, which predisposes to suicide, violence, and homicide.

A withdrawal process must respect the shape of the binding curve, and become slower and slower, the lower the dose. These principles have been known for many years and were explained in an instructive paper in *Lancet Psychiatry* in March 2019,[281] eight months before the Danish National Board of Health published its dangerous guideline.

After decades of inaction and denial,[7] some progress is now being made. I co-founded Council for Evidence-based Psychiatry in 2014 and, as noted earlier, I was immediately attacked by the top of British psychiatry.[302] I requested and was granted an opportunity to publish a rebuttal of their nonsense.[311] The Council was established at a meeting in the House of Lords by filmmaker and entrepreneur Luke Montagu who had suffered horribly from withdrawal symptoms for many years after he came off his psychiatric drugs,[8:97] and he wanted to highlight their harms.

After setting up the Council, Luke founded the All-Party Parliamentary Group on Prescribed Drug Dependence (APPG), which successfully lobbied the British Government to recognise the issue. He also succeeded to get support from the British Medical Association and the Royal College of Psychiatrists. That led to a ground-breaking review by Public Health England with several key recommendations, including a national 24-hour helpline and withdrawal support services.[310] These recommendations do not only focus on the traditional culprits, opiates and benzodiazepines, but also on depression pills.

In December 2019, the APPG and the Council published the 112-page *Guidance for Psychological therapists: Enabling conversations with clients taking or withdrawing from prescribed psychiatric drugs*.[611] This guide is very detailed and useful, both in relation to the individual drugs and in terms of the guidance offered to therapists.

In 2016, I co-founded the International Institute for Psychiatric Drug Withdrawal (iipdw.org), originally based in Sweden, now in the UK.

I have not had success with Danish parliamentarians. Although they were always positive when I explained why major changes are needed in psychiatry, they are afraid of going against the psychiatrists who are quick to tell them that psychiatry is outside their area of expertise.

Mind, the most influential organisation for psychiatric patients in Denmark, wasn't forthcoming either. When I tried to get an advertisement in their member journal in 2017 for a withdrawal course I planned for psychiatrists, patients and others, they refused to accept my ad.[8:99] But after I went to their headquarters with a documentary film crew, they felt pressured to give in to my reasonable request, which was in their members' interest.

When I informed *Psychiatry in the Capital Region* about our course, Poul Videbech complained about to the Patient Safety Authority, which did not react to his complaint until five months later when we had already held the course. They noted they did not intend to take any action.

By the end of 2017, psychiatrist Jan Vestergaard tried to get a two-hour symposium about drug withdrawal on the programme for the annual meeting of the Danish Psychiatric Association in 2018. Even though the meeting lasted four days, with parallel sessions, the board declared there wasn't room for the symposium. Vestergaard had asked me to speak at his meeting and I did not accept this censorship. I booked a room at the conference hotel and held a two-hour symposium for the psychiatrists in the morning, which we repeated in the afternoon. I mentioned in the ad in the *Journal of the Danish Medical Association* that several psychiatrists had urged us to hold a course on withdrawal of psychiatric drugs at the same time as their annual meeting.

My PhD student on the subject, Anders Sørensen,[607] also lectured. Later, when we strolled around in the corridors, we learned that young psychiatrists had been scared away from attending because their bosses would see them as heretics and might retaliate, but the room was pretty full, nonetheless.

On other occasions, psychologists, social workers, and nurses who wished to attend my lectures or courses have told me similar stories about receiving dire warnings from their superiors that if they showed up, it would not be well received at their department. This is diagnostic for a sick specialty. It tells a story of a guild that behaves more like a religious sect than a scientific discipline because in science, we are always keen to listen to new research results and other points of view, which make us all wiser.

The Cochrane Collaboration, which I co-founded in 1993, was also uncollaborative.[8:106] Anders and I had submitted a protocol for a Cochrane review of studies of withdrawal of depression pills, but the editors sabotaged it. The Cochrane depression group sent us on a two-year mission that was impossible to accomplish, raising their demands to our protocol to absurd levels with many irrelevant requirements, including demands of inserting marketing messages about the wonders that depression pills can accomplish, according to psychiatric dogma. Cochrane did its utmost to defend the psychiatric guild, its many false beliefs, and the drug industry, forgetting that its mission is to help patients.

It was bizarre. In the midst of all our troubles, Anders wrote to me that our review was quite simple, as we just wanted to help people wishing to come off their drugs but weren't allowed to do so: "What kind of world is this?"

The 8th and final reviewer functioned as hangman. He denied a long array of scientific facts and used strawman arguments accusing us of things we had never claimed. We were accused of "painting a picture" about avoiding depression pills, which did not represent the scientific consensus.

The reviewer wanted us to "Start with a statement as to why antidepressants are considered by the scientific community to be beneficial … in treating a broad range of highly disabling and debilitating mental health problems" and accused us of being unscientific because we had not mentioned the beneficial effects. We responded that our review was not an advertisement for the

drugs and that it was not relevant to discuss their effect in a review about stopping using them. Furthermore, a Cochrane review should not be a consensus report.

The editors also asked us to write about the benefits and to mention that "some antidepressants may be more effective than others", with reference to the fatally flawed 2018 network meta-analysis in *Lancet* by Andrea Cipriani and colleagues (see page 118).[271]

A Cochrane editor asked us to describe how depression pills work and what the differences are between them, and a reviewer wanted us to explain when it was appropriate and inappropriate to use depression pills. But we were not writing a textbook in clinical pharmacology, we were just trying to help the patients come off their drugs.

We wrote in our protocol that "Some patients refer to the discredited hypothesis about a chemical imbalance in their brain being the cause of their disorder and therefore also the reason for not daring to stop." The hangman, who believed in the chemical imbalance nonsense, opined that we dismissed many decades of evidence of neurochemical changes observed in depression and accused us of having suggested with no evidence that prescribers perpetuate untruths to justify drug prescription. He also wanted us to mention ongoing prophylactic depression pill treatment, "a well-accepted clinical strategy," which was outside the scope of our review. Moreover, all the maintenance studies are flawed. We were wrongly accused of having conflated relapse with withdrawal symptoms, and the hangman argued that most people who had taken depression pills for extended periods could stop safely without problems, which is blatantly false.

He also wanted us to remove this sentence: "the patients' condition is best described as drug dependence" referring to the DSM-IV drug dependence criteria. We replied that, according to these criteria, no one who smokes 20 cigarettes every day is dependent on cigarettes.

The level of denial, obfuscation, confusion and censorship was so high that I saw this as one of several signs of the impending death of Cochrane as an organisation.[146]

We will publish our review in a journal whose editors are not morally and scientifically corrupt and who have the patients' interests at heart. We uploaded all 8 peer reviews, our comments to them, and our final protocol, as part of an article we published about the affair in 2020.[612]

The psychiatrists and other doctors have made hundreds of millions of people dependent on psychiatric drugs and yet have done virtually nothing to find out how to help them come off them again. They have carried out tens of thousands of drug trials but only a handful of studies about safe withdrawal.

Many psychiatrists continue to turn a blind eye to the disaster they have created and argue that we need more evidence from randomised trials, but such evidence is unlikely to be helpful, as withdrawal is a highly individual and varying process. Furthermore, isn't over 150 years of waiting enough? There has been no good evidence base either about how to come off opium, morphine, bromides, and barbiturates.

I shall not repeat the extensive advice I gave in another book about drug withdrawal,[8:93] only repeat a few things and add some more.

The patient needs a support person during withdrawal. It is rare that such a person can be a doctor, as most doctors expose their patients to a cold turkey and then conclude that the patients still need the drugs. But it is a good idea to inform the usual doctor that a withdrawal is about to start and hopefully get the doctor interested in helping out. Automatic renewal of prescriptions over the phone should not occur, as the risk is that drug treatment will continue for many years.

The patient should try to find a person who has succeeded with withdrawal, a recovery mentor, and involve that person in the withdrawal.

Psychologists can be very helpful. It can be overwhelming when the emotions, which have been suppressed for so long, come back, and in this phase, it can be crucial to get psychological support to handle the transition from living emotionally numbed to living a full life.

A health professional or recovery mentor will rarely be able to support a patient on a daily basis. Other support people are needed, which can be relatives or friends.

It is often huge work to help a patient get through withdrawal, and it doesn't end there. The support person should wrap it all up together with the patient and summarise the withdrawal process, including the most important symptoms experienced along the way. The patient should be offered continued support, as there is a risk that the patient would want to come back on the drug if a situation is stressful, which can cause some of the withdrawal symptoms to return, even long after a successful withdrawal. It can take many years before the brain becomes normal again.

The patient needs to know that the support person will always be available, and the feeling of security and that someone cares can have a strong healing effect.

One should not try to taper off a patient who doesn't have a genuine wish of becoming drug-free. It is unlikely to work. But this should not be used as an excuse for doing nothing. We need to explain to the patients that long-term treatment is very harmful and we should try to persuade the patients to start a withdrawal process.

With three experienced colleagues, I have written a short guide to psychiatric drug withdrawal, with tips about how to divide tablets and capsules. We also made an abstinence chart that allows the patient to follow the symptoms over time, and I have provided a list of people willing to help with withdrawal and links to videos of our lectures on withdrawal.[613] There are many websites set up by psychiatric survivors[8:198] that offer good guidance, e.g. theinnercompass.org, created by Laura Delano who lost 14 years to psychiatry[7:298] but reclaimed her life after she had read Whitaker's famous book, *Anatomy of an epidemic*.[5]

In Holland, former patient Peter Groot and psychiatrist professor Jim van Os have taken a remarkable initiative. A Dutch pharmacy produces tapering strips, with smaller and smaller doses of the drug, making it easier to withdraw. Their results are also remarkable. In a group of 895 patients on depression pills, 62% had previously tried to withdraw without success, and 49% of them had experienced severe withdrawal symptoms (7 on a scale 1 to 7).[614] After a median of only 56 days, 71% of the 895 patients had come off their drug.

Each strip covers 28 days and patients can use one or more strips to regulate the dose reduction. There is a website dedicated to this, taperingstrip.org. People in other countries currently try to convince pharmacies to produce tapering strips.

It is important to get a successful start. It is often best to remove the most recently started drug,[135] as withdrawal gets harder the longer the patient has been on a drug.[135,614] It is also important to withdraw psychosis pills and lithium early on, as they cause many harms.[135] Withdrawal can cause sleeping problems, which is a good reason to remove sleep aids last.

It is not advisable to withdraw more than one drug at a time, as it makes it difficult to find out which drug causes the withdrawal symptoms.

It is rarely a good idea to substitute one drug for another, even if the new drug has a longer half-life in the body and would be expected to be easier to work with. Some doctors do this, but a switch can lead to additional withdrawal problems because the two drugs may not target the

same receptors, or to overdosing, as it is hard to know which doses should be used for the two drugs in the transition phase. But it may be necessary, e.g. if the tablet or capsule cannot be split.

It is generally not advisable to introduce a new drug, e.g. a sleeping pill if the withdrawal symptoms make sleep difficult. It is better to increase the dose a little.

The dose reduction must follow a hyperbolic curve. This means that you reduce the dose every time you taper by removing the same percentage of your previous dose. If you reduce the dose by 20% each time, and you have come down to 50%, you should remove 20% again next time, which means that you now come down to 40% of the starting dose.

One layperson withdrawal community found that the least disruptive taper is when you reduce the dose by 5-10% per month,[615] but I would not recommend this approach. If you reduce by 10% per month, it will take two years before you come down to 8% of your starting dose, so if you are on four drugs, it may take you eight years to become medicine-free. And the longer you take a drug, the greater the risk of permanent brain damage, and the harder it is to come off it.

The last small step can be the worst, not only because of physical issues but for psychological reasons. The patient may ask himself: "I have taken this pill for so long; dare I take the last small step? Who am I when I don't take the pill?" The doctor may laugh and tell the patient that it's impossible to have withdrawal symptoms when the dose is so low.[616] If that doctor is involved in the withdrawal and behaves like a "know-it-all" guy, the patient should find another doctor.

Citalopram is recommended to be used at dosages of 20 or 40 mg daily, and it will surprise any doctor to know that even at a dose as low as 0.4 mg, 10% of the serotonin receptors are still being occupied.[281] This means that the patient might experience withdrawal symptoms when going from that small dose to nothing. Psychiatrist Mark Horowitz admitted that if the patients had come to him before he had experienced the withdrawal symptoms himself, he would probably not have believed them when they said how difficult it was coming off a depression pill.[616]

16 Is there any future for psychiatry?

> The industry has bought doctors, academics, journals, professional and patient organisations, university departments, journalists, regulators, and politicians. These are the methods of the mob.
>
> Richard Smith, previous editor of *BMJ*[6:viii]

> What makes this book new and worth your attention? The answer is simple: the unique scientific abilities, research, integrity, truthfulness, and courage of the author. Gøtzsche's experience is unequaled.
>
> Drummond Rennie, editor of JAMA[6:x]

These are extracts from the forewords to my 2013 book about organised crime in the drug industry.[6] I have shown in this book that you cannot trust the randomised trials, the drug industry, or the psychiatric leaders. The editors say in their forewords to my 2013 book that I can be trusted but, more importantly, I have tried to document what I say so you can make up your own mind.

You cannot even trust the drug regulators. As David Healy has pointed out, in contrast to drug agencies, airline pilots are critically concerned with our safety because if we go down, they do too.[617] There is widespread corruption in the FDA at the highest levels, including several commissioners,[6] and in 2009, nine FDA scientists wrote to President Obama about this.[618,619] In 2012, it was revealed that FDA management had installed spyware on the computers of five scientists who had alerted the FDA to safety problems to no avail and therefore had informed the politicians.[620]

It must be very tempting for drug companies to bribe officials at drug agencies. There is an enormous amount of money at stake and the approval of a new drug can be the difference between life and death for a company. In 2012, Danish Lundbeck and its Japanese partner Takeda submitted vortioxetine, an SSRI, for regulatory approval in the United States.[621] Lundbeck's blockbuster, escitalopram, was running out of patent, and the company would receive a $43 million milestone payment from Takeda if FDA accepted the drug.

It is paradoxical that, while drug firms don't trust each other, drug agencies are supposed to trust the entire industry because they cannot review more than a tiny fraction of the mountains of documents they receive.[622] The regulators don't even check that everything is included. I have found numerous examples that whole appendices or many pages in the middle of a report were missing,[279,326] and also of missing cases of suicidality,[279] in clinical study reports of placebo-controlled trials submitted to European drug regulators for marketing approval.

Psychiatry's narrative is that drugs are very often needed, both in the acute phase and long-term to prevent relapse; that specific drug treatments have been known for about 65 years;[18:232] that the drugs are generally effective and safe; and that the new psychiatric drugs are highly beneficial.[18:307]

The truth is that none of the many psychiatric drugs have specific effects; the drugs rarely have clinically relevant effects and are therefore rarely needed, not even in the acute phase; an effect on relapse has not been demonstrated; and the drugs are far from being safe. There is an epidemic of overdiagnosis and overtreatment with psychiatric drugs to such an extent that, based on the most reliable research I could find, I estimated that psychiatric drugs the third leading cause of death, after heart disease and cancer.[7:12,7:307]

The denial of the facts in the psychiatric profession is massive. In 2011, a group of prominent psychiatrists wrote:[623]

"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 … 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 the suicide rate tends to go down as the incidence of antidepressant treatment goes up."

I fail to understand how Stefan Leucht, who has published much good research and is an editor in the Cochrane Schizophrenia Group, could co-author this harmful nonsense. It shows that the collective delusions and denial in psychiatry hit even the best psychiatrists. It is very tragic for the patients, their relatives, and psychiatry itself.

A 2012 newspaper article written by four leading Danish psychiatrists called *Behind the myths about antipsychotics* was similarly tragic.[624] 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 psychosis drugs 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 psychosis drugs; that they found no indications that polypharmacy with psychosis drugs increases mortality in their large study; and that large register-based studies in Denmark and Finland show that concomitant treatment with several psychosis drugs is not associated with increased mortality.

Leading psychiatrists constantly tell the public such nonsense, which is dangerous for their patients. They claim that psychosis pills reduce mortality when the truth is the opposite, and they happily continue their Titanic course towards the iceberg, which they refuse to see.

Here is a patient story from one of the psychiatrists' university hospital in Copenhagen.[7:277] A patient was admitted with mania, and although he asked not to be treated with drugs, he received forced treatment with olanzapine. In his own words: At discharge, when I had been declared cured after my first-episode mania, I tried to behave well, fearing that I might not be released. 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 should not be afraid of the harms.

The reason why the patient didn't take the drug was that he had read the newspaper article I published in January 2014 about ten harmful myths in psychiatry, which also exists in English,[189] and he has been well ever since without drugs.

The same day my article about the ten myths appeared, Thomas Middelboe, chairman of the Danish Psychiatric Association declared in the same newspaper, on its website:[625] "Antidepressant drugs protect against suicide." A month later, 16 Danish professors in psychiatry responded to my article[626] without mentioning my name, just like one was not supposed to mention the evil

Voldemort's name in Harry Potter. They wrote that a number of studies show that treatment with psychosis drugs increase longevity, compared with no treatment.

I have given many examples in this book that leading psychiatrists have no problem with claiming the exact opposite of the truth. In 2005, Steven Sharfstein, then president of the American Psychiatric Association, wrote that "Pharmaceutical companies have developed and brought to market medications that have transformed the lives of millions of psychiatric patients."[627] Sure, but not for the better. He added that "Big Pharma has helped reduce stigma associated with psychiatric treatment and with psychiatrists."

Is there any hope for a specialty like this? I have heard critical psychiatrists say that their leaders suffer from cognitive dissonance, as what they see and hear doesn't influence them. Many books have documented that the psychiatric leaders have given up rational thinking for the benefits they acquire themselves from supporting a totally sick system. Even psychiatrists who have used monstrous overdoses of psychosis pills are allowed to practice.[8:143] Why don't our politicians care that incompetent psychiatrists kill hundreds of thousands of their patients every year (see Chapters 7 and 8)? Or that the lives of many millions of children get destroyed?

Psychiatric drugging of children is a form of child abuse that should be prohibited, with rare exceptions. We are not allowed to beat our children but are allowed to destroy their brains with drugs. We medicalise the conflicts that arise between parents and children, and methylphenidate has become the modern version of the cane. This is a flagrant abuse of a faulty disease model.

Little has changed in recent years. If you google *what causes ADHD*, you can find this misinformation from the UK National Health Service, directed toward the public and last reviewed in December 2021:[10:39,628]

"ADHD tends to run in families and, in most cases, it's thought the genes you inherit from your parents are a significant factor in developing the condition … Research has identified a number of possible differences in the brains of people with ADHD from those without the condition … Other studies have suggested that people with ADHD may have an imbalance in the level of neurotransmitters in the brain."

The drugged child's brain cannot develop in its intended manner but develops in response to a toxic internal environment. The stigmatisation and loss of self-esteem, which often follows psychiatric diagnosis and treatment, 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.[526] This cruelty must be stopped.

Imagine if a virus suddenly appears that makes people sleep 12-14 hours a day and move around slowly and become emotionally disengaged.[5:207] Some gain 30 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. Scientists find out that the virus blocks a multitude of receptors in the brain – dopaminergic, serotoninergic, 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 hit millions of children and adults. It is not a virus. It is Eli Lilly's bestselling psychosis drug, olanzapine (Zyprexa). But since it is a drug, we do nothing. Drugs are taboo.

The only hope we have is if people protest so vigorously that it becomes an unstoppable revolution.

In 2017, a young Swedish psychiatrist, Joakim Börjesson, came to Copenhagen to do research with me.[428] He became very impressed during his medical studies when a psychiatrist told the students that they knew so much about the brain and the drugs that they could use drugs that were specifically targeted to work on a disorder's biological origin, the so-called chemical imbalance idea. He found it so fascinating that he decided to become a psychiatrist.

Joakim is cleverer than most of his colleagues. After having read books by Robert Whitaker and me, he realised that he had been totally fooled and considered leaving psychiatry.

In January 2018, he arranged a session in Göteborg during the annual conference for 150 Swedish psychiatrists in training where I debated with clinical pharmacologist and professor Elias Eriksson about SSRIs.[8:147]

During the session, I mentioned that Eriksson had entered a secret agreement with Lundbeck against his university's rules, which meant that Lundbeck could prevent publication of his research if they didn't like the results. I said this because Eriksson routinely "forgets" to declare his conflicts of interest, but I was immediately stopped by the chair. Later, the Ombudsman criticised the university for covering up the affair.[629]

What is typical for debates with people who try to defend a sick system also happened this time. Eriksson broke the rules for the debate, he lied, and he used dirty tricks in his attempts at convincing the audience that I could not be trusted. Joakim informed me that Eriksson had said before the session that he had the intention to "'reveal that Peter Gøtzsche is a charlatan' during his lecture. We then discussed this for about an hour and I fruitlessly tried to convince him to adhere to the rules for the debate with no success."

Eriksson claimed that none of the harms of the pills were irreversible; that they were not addictive; that criticism of the pills was "ideologically founded;" and that their use according to the critics was the result of a worldwide conspiracy that included psychiatrists, researchers, authorities and drug companies. Five months earlier, when I debated with Eriksson on Swedish radio, he said the pills helped dramatically and prevented suicide.

After the meeting, I was told that many psychiatrists had not understood my explanations about depression pills causing suicide. When I present the same slides for a lay audience, they *always* understand them. The psychiatrists *don't want* to understand what is too painful for them.

In 2013, when Robert Whitaker was invited to speak at a meeting in Malmö that child psychiatrists had arranged, other psychiatrists intervened and got control of the meeting. They requested that he should only speak about the dopamine supersensitivity theory and not present any data on long-term outcomes.

When he arrived, Bob was told that Eriksson would be his opponent, and he spent his time denouncing Bob in an unbelievably dishonest fashion. In Bob's own words: "The whole thing was a disgusting setup that stands out for its complete dishonesty, from start to finish." Eriksson declared that he considered Bob to be a "charlatan who tortures patients."

I had planned on coming, but Eriksson declared that he would not participate if I showed up.

It is strange how psychiatry's apologists constantly call their opponents charlatans or worse and use strawman arguments. None of us have ever postulated anything about a "conspiracy."

Censorship in medical journals and the media

It is very difficult to get anything published in a psychiatric journal that the psychiatric guild perceives as threatening for their carefully pruned self-image and wrong ideas.[8:151]

Editors of specialty journals are often on drug industry payroll and journal owners also often have too close relations to the drug industry.[6-8,27,630]

At the inaugural symposium for my Institute for Scientific Freedom in 2019, Robert Whitaker spoke about scientific censorship in psychiatry. He focused on two topics of great importance for public health: *Do antidepressants worsen long-term outcomes?* and *What do we know about post-SSRI sexual dysfunction?*[631] None of 13 and 14 pivotal studies, respectively, about these subjects had been published in the top five psychiatric journals, which did not even appear to have discussed the issues.

The censorship in mainstream media is also pronounced. When my first psychiatry book had been translated into Swedish, I was interviewed by journalists from two major newspapers in Stockholm.[8:152] They were very interested, but as nothing was published, I asked why. One journalist didn't reply. The other said that her editor thought it would be too dangerous to explain to Swedish citizens that depression pills are dangerous, as they can cause suicide. Both newspapers were right-wing. In contrast, a third newspaper, *Aftonbladet*, popular with Social Democrats, allowed me to publish an article that filled the whole back page, with no censorship.

It is also very difficult to get critical documentaries on national TV, and if you succeed, you can be dead sure that the best parts have been removed, "so we don't upset anyone or get too many complaints from the psychiatrists, the drug industry or the Minister." And there is an untruthful voiceover telling the audience that "many people are being helped by psychiatric drugs."[8]

It is difficult to publish relevant books, too.[8] In one case, a former patient and a filmmaker came to film me for a documentary.[632] The patient had an agreement with a book publisher about what she thought was a psychiatric success story. But psychiatry had stolen 10 years of her life and when I explained that she had been horribly harmed by her psychiatrists, which were very close to driving her into suicide with fluoxetine, she accepted my explanations. When her psychiatric "career" was no longer a success story but a scandal, the publisher backed out. Her drug list is one of the worst I have ever seen.[8:154] It is a miracle she survived all this.

Another Norwegian filmmaker wanted to have me on the panel when her documentary, *Cause of death: unknown*,"[633] had world premiere in 2017 at the Copenhagen documentary film festival. The cause of death was not unknown. The filmmaker's sister was killed by her psychiatrist who overdosed her with olanzapine, which turned her into a zombie. The psychiatrist was so ignorant that he didn't even know that olanzapine can cause sudden death. Such iatrogenic deaths are called natural deaths by the authorities.

I appeared in the film and my name was the only one in the announcement: *Medicine or manipulation? Film and debate about the psychiatric drug industry with Peter Gøtzsche*. Seven days before the film was to be screened, I was kicked off the panel under the pretence that the organisers couldn't find a psychiatrist willing to debate with me. This was not the real reason. It turned out that the Lundbeck Foundation, whose objective is to support Lundbeck's business activities, had provided a major grant to the festival. CPH:DOC never contacted me about it, even though I could easily have named several psychiatrists willing to debate with me.

I have described this scandal elsewhere.[8:155] The panel discussion was a farce that protected the status quo and people in the audience became angry. It was deeply insulting to them to show

a film about a young woman killed by Zyprexa without allowing any of those who had lost a family member in the same way to say anything. It was a brutal dismissal and a total prostration for Lundbeck.

Another recent instance of censorship involved Danish public TV. Independent documentary filmmaker Janus Bang and his team had followed me around the world for several years because they wanted me to play a central role in their documentaries about how awful and deadly psychiatry is. Janus ran into a huge roadblock and needed to compromise extensively to get anything out on TV. He broadcast three interesting programmes in 2019, *The dilemma of psychiatry*, but the public debate he so much had wanted to have major reforms introduced was absent. Drug exports are Denmark's biggest source of income, and there were embarrassing, false voiceovers paying lip service to Lundbeck and the psychiatrists. And me? I wasn't allowed to appear at all.

Journalists have told me that the reason Danish public TV doesn't dare challenge psychiatry or Lundbeck is due to two programmes sent in April 2013.

I was interviewed for *Denmark on pills*, which featured three patients. One was prescribed "happy pills" when she was 15 and suffered from massive harms. Another had lost his sex drive and shouldn't have had the pills at all, as he was not depressed but suffered from stress. The third was a boy diagnosed with ADHD by a psychiatrist who had never met him.

Already the next day, the psychiatric empire stroke back. In a magazine for journalists, Poul Videbech said:[634] "It's a scare campaign that can cost lives. I know several examples of suicide after friends and family advised the patient to drop antidepressant medication." Videbech compared this with journalists making programmes advising patients with diabetes to drop their insulin even though he, at the same time, fiercely denied that he believed in the myth about the chemical imbalance (see page 27).

There were many commentaries to the article about Videbech in the magazine. One noted that it was interesting to see that there were virtually no tapering programmes in psychiatry and that people often ended up on lifelong medication.

One mentioned that she was a member of a large and diverse group of people who had warned for years against the uncritical use of drugs and had spent time on helping the victims, but every time they opened a debate on this topic, they were accused of not thinking about those who benefit from the medicines.

One wondered why we heard nothing from psychiatry about the suicides and suicide attempts the drugs cause: " … dismissed as non-occurring. Nevertheless, it was on the list of side effects in the package insert of the medication I received. And I felt the impulse on my own body. But I was told that it was my depression that was the trigger for suicidal thoughts and plans. The strange thing about that was that the impulse came shortly after I started on the drug ... But the doctor and others involved concluded that my dose should be increased, which I luckily declined and I decided to taper off the drug on my own. That people change their personality totally - become aggressive and hot-headed, paranoid, etc. - is also dismissed."

One noted that I was right that the media had been uncritical in their coverage of psychiatric drugs. He pointed out that many people had tried to warn against them for many years but had been silenced or fired from their positions from where they could reach the population.

This also happened to me which I wrote a book about.[635] I updated it[146] (freely available) because Janus Bang and I are currently making a documentary film about the affair, which we base on crowd funding (see scientificfreedom.dk/donate/).

Only four days later, journalist Poul Erik Heilbuth showed a brilliant 70 minute documentary, *The dark shadow of the pill*. He documented in detail how Eli Lilly, GSK and Pfizer had concealed that their depression pills cause some people to kill themselves or commit murder or cause completely normal and peaceful people to suddenly start a spree of violent robberies in shops and gas stations they were unable to explain afterwards and were mystified about. The pills changed their personality totally.

Heilbuth had whistleblower Blair Hamrick in his film, a US GSK salesman who said that their catchphrase for paroxetine (Paxil or Seroxat) was that it is the happy, horny and skinny drug. They told doctors that it will make you happier; you will lose weight; it will make you stop smoking; it will make you increase your libido; everybody should be on this drug. Hamrick secretly copied documents, and GSK was fined $3 billion in 2011 for paying kickbacks to doctors and for illegal marketing of several drugs, also to children.[6:27]

An editorial in one of Denmark's national newspapers, Politiken, condemned the documentary in an unusually hostile fashion and called it "immensely manipulative," "sensationalism," "merely seeking to confirm or verify the thesis that the programme had devised as its premise," and they called one of the well-argued experts a "muddled thinker."

Two days after Heilbuth's documentary, I debated with Lars Kessing on live TV about suicides caused by depression pills. Bits of this debate appears in the documentary, *Diagnosing psychiatry*.[636] Kessing totally denied the science and the drug agencies' warnings, saying that we know with great certainty that SSRIs protect against suicide. He added that the risk of suicide is large when people stop SSRIs but failed to mention that this is a drug harm, as the patients get a cold turkey.

Three days later, I was in a TV debate again with Kessing, this time about how we could reduce the consumption of depression pills. Kessing claimed that they are not dangerous. Lundbeck's director of research, Anders Gersel Pedersen, said that the most dangerous thing is not to treat the patients, and he claimed that the patients don't become addicted but get a relapse when they stop taking the pills. Kessing claimed that perhaps only 10% of those who visit their family doctor are not helped by the medicine, quite a remark about drugs that don't work (see Chapter 8).

When Kessing was asked by the interviewer how the consumption of pills could be reduced - no matter what he might think about its size – he didn't answer the question. He said we knew for sure that there had been a rising incidence of moderate to severe depression over the past 50 years. I replied that we could not tell because the criteria for diagnosing depression had been lowered all the time during this period.

Kessing was wrong. Psychiatrists constantly tell me that the prevalence of severe depression has not increased.[103] Most patients who get a diagnosis of depression live depressing lives, e.g. are married to the wrong person, have a bullying boss, a tedious job, or a chronic disease. It is not the job of doctors to try to get them out of this predicament and a pill won't help.

I have experienced that when journalists react violently and go directly against the scientific evidence and the authorities' warnings, it is almost always because they think the pills have helped them or someone close to them, or because a relative works for Lundbeck or is a psychiatrist. I have been exposed to many vitriolic attacks. It is sad that journalists throw everything overboard they learned at journalism school and explode in a cascade of rage and ad hominem attacks, but that can happen if you tell the truth about depression pills. You are attacking a religion and violating one of the most sacred taboos in healthcare.

In a radio debate, Mind's National Chairman, Knud Kristensen, argued that some of their patients had said that depression pills had saved their life. I responded dryly that it was an unfair argument because all those the pills had killed couldn't raise from their graves and say the pills killed them.

Robert Whitaker has provided a long list of important and large studies whose results were threatening to the psychiatric narrative and which were not mentioned in any US newspapers.[5:307] When the WHO study came out (see page 50), the *New York Times* reported that "schizophrenics generally responded better to treatment in less developed countries."[5:311] This is highly misleading because any reader would think they were treated with psychosis pills, which they rarely were.

A few mainstream psychiatric journals have started to wake up to the disaster. A 2007 paper in the *British Journal of Psychiatry* 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 future,[596] and a 2012 paper in this journal predicted that the current biology-based model will be ruinous to the profession due to its consistent failure to deliver.[638]

More issues with unreliable diagnoses and poor drugs

A textbook called it a psychopharmacological revolution that we can alleviate or cure 80-90% of people with severe depression, and it claimed that patients with schizophrenia can get their symptoms so much under control or even become cured that they do not need to be hospitalised.[18:232] These claims go directly against the evidence. Drugs cannot cure depression or schizophrenia, and if we wait long enough, most patients, also those with severe depression or schizophrenia, will improve, which is not a drug effect.

This textbook claimed, with no references, that studies from the London School of Economics show that it is a really good business for society to offer treatment of psychiatric disorders.[18:288] Since treatment always means drug treatment – when psychiatrists don't say otherwise – the claim is false. It is the other way around. The less we use psychiatric drugs, the greater the savings for society, and the more people will be able to work and contribute to society.[5:8,119:24]

Apart from this, the textbooks did not mention economic aspects of their recommended treatments. Prices of drugs change, but there wasn't a single remark that off-patent drugs should be preferred because they are vastly cheaper than patented drugs and not any worse than these. The psychiatric narrative was the opposite of what it should have been. We are told about new drugs that are "modern" or second generation or third generation drugs. Some of the drugs that have been most widely used are also some of the worst ones in terms of the harms they cause, e.g. olanzapine, paroxetine, and alprazolam.

This has nothing to do with EBM but everything to do with corruption of the science and of the psychiatric leaders.[7,8,533] Psychiatry has sold out to the drug industry. Psychiatrists collect more money from drug makers than doctors in any other specialty,[209,639] and those who take the most tend to prescribe psychosis drugs to children most often.[639] Psychiatrists are also "educated" with industry's hospitality more often than any other specialty.[209,640]

Lundbeck patented the active half of citalopram (Celexa or Cipramil) before the patent ran out and called the rejuvenated drug escitalopram (Cipralex or Lexapro), which it launched in 2002. When I checked the Danish prices in 2009, the rejuvenated drug cost 19 times as much for a daily dose as the original drug.[6:224] This enormous price difference should have deterred the doctors from using escitalopram, but it didn't. Its sales were six times higher in monetary terms than the

sales of citalopram. I calculated that if all patients had received the cheapest citalopram instead of escitalopram or other SSRIs, Danish taxpayers could have saved around €30 million a year, or 87% of the total amount spent on SSRIs.

Corruption, both of the science (see page 118) and of the doctors, was behind this disregard for the public purse. A psychiatrist described vividly that when Lundbeck launched escitalopram in 2002, most Danish psychiatrists (there are more than a thousand psychiatrists in Denmark) were invited to an enjoyable meeting in Paris: "With expensive lecturers - of course from Lundbeck's own 'stable' – luxurious hotel and gourmet food. A so-called whore trip. Under influence? No, of course not, a doctor doesn't get influenced, right?"[641]

The textbooks claimed, without any reliable evidence, that early detection and intervention with drugs are very important for the prognosis, e.g. for psychosis, depression and ADHD. This is not correct.

A chapter on psychopharmacology written by three professors of psychiatry, Anders Fink-Jensen, Poul Videbech and Erik Simonsen, glorified the drugs.[17:645] They claimed that knowledge of brain functions has increased dramatically over the last half century; that our understanding of the mechanisms of the drugs' effects has been strengthened; that new drugs with fewer harms and better effects have been developed; and that there is no doubt that this has decisively contributed to better psychiatric treatment for the benefit of the patients and their relatives.

All of this was wrong. Psychiatrists turn the evidence on its head to suit their own interests, which align with those of the drug industry.

A 2007 paper surveying US department chairs of medicine and psychiatry reported that 67% of them had received "discretionary funds" from industry within the last year.[7,642] This is likely an underestimate, as the survey was not anonymous. The donations to department chairs and other decision-makers are sometimes called unrestricted educational grants, which is a euphemism for corruption, as the industry doesn't just give its money away. They are restricted uneducational grants, as their purpose is to buy doctors.[643]

The three professors' praise of the drugs continued.[17:650] They wrote that the lack of compliance is worst for psychoses, which leads to lack of recovery, relapse and readmissions, and that the patients must understand that the diseases will have health and social consequences if the treatment is not being followed.

It is the other way around. It is very rational when some patients refuse to take toxic drugs that have no meaningful beneficial effects; that will likely harm them irreversibly; and that might even kill them. But in the psychiatrists' delusional world, these patients are the problem, not the drugs they use.

One book was different to the others in terms of what it admitted. Right from the start, in the first chapter of the 1065-page textbook, a psychologist and a psychiatrist noted that it is important to counteract the one-sided reductionism, neuropsychiatry has led to.[17:58] They said that diagnoses do not have much validity and have no direct consequence for the treatment and for the patients; that there is an epidemic of diagnoses, which have a life of their own; and that psychiatry has not been sufficiently cautious about the consequences of the many false positive diagnoses.

They quoted an interesting paper by Jerome Wakefield.[644] His major point is that the shift to symptom-based, operationalised diagnostic criteria in DSM-III and subsequent editions of the manual missed the context in which the symptoms appear, which has led to colossal overdiag-

nosis - false positive diagnoses - of psychiatric disorders because the symptoms are often a normal reaction to a stressful situation.

Wakefield noted, with examples, that physicians used context for about 2500 years to distinguish conditions like depression from normal sadness, but that this was now gone. He mentioned that the DSM-IV criteria for primary insomnia do not consider one of the commonest non-medical reasons for difficulty sleeping, a noisy environment.

Wakefield considered that this problem had urgency because the DSM's symptom-based criteria are often applied in studies and screening instruments outside the clinical context and by nonprofessionals.

He noted, with examples, that flaws in the diagnostic criteria, which lay people can recognise immediately, remain unaddressed, and that the use of symptom checklists gives a diagnosis to many people who do not self-identify as disordered and are often not disordered. Wakefield mentioned a colleague who was seeing a depressed unemployed person and suggested medication, at which point the patient said indignantly, "I don't need medication; I need a job."

Wakefield noted that symptomatic criteria cannot diagnose an underlying dysfunction. For example, adjustment disorder is evaluated in part by whether there is "marked distress that is in excess of what would be expected from exposure to the stressor," but if "what would be expected" is construed in a statistical sense, then this criterion potentially pathologises the upper range of normal variation.

Wakefield wondered why the psychiatric experts behind the DSM revisions had not looked systematically for counterexamples to the proposed criteria that could lead to false positive diagnoses.

I did exactly that in my two books about psychiatry.[7,8] I mentioned above that one of my colleagues, Danish filmmaker Anahi Testa Pedersen, got the erroneous diagnosis schizotypy when she became stressed over a difficult divorce.[8] She should never have had a psychiatric diagnosis or been treated with drugs.

Since I suspected it was a dubious concept, I looked it up on the Internet and found a test for schizotypal personality disorder.[8:145,645] It is defined in various ways in different sources but the test reflects quite well the criteria on the Mayo Clinic website that notes that the symptoms are those in the DSM.[646] You should reply true or false, or yes or no, to nine questions.

1. "Incorrect interpretations of events, such as a feeling that something which is actually harmless or inoffensive has a direct personal meaning." This is a vague question, and many people interpret events incorrectly, particularly psychiatrists, or take them personally.
2. "Odd beliefs or magical thinking that's inconsistent with cultural norms." When a psychiatrist disagrees with the "cultural norms" about preventative treatment of schizotypy, as recommended in a textbook,[18:106] is he then abnormal? And what about monstrous overdoses, which is also a "cultural norm" in some places? It seems that those in the staff who protest are normal but would be considered abnormal according to this question.
3. "Unusual perceptions, including illusions." I have provided evidence in my books including this one that most psychiatrists would need to say yes to this question. Just think about the illusion called the chemical imbalance.
4. "Odd thinking and speech patterns." Most psychiatrists display odd thinking, about the chemical imbalance and many other issues, and they deny totally what other people see clearly, including their own patients, e.g. that psychiatric drugs do more harm than good.

5. “Suspicious or paranoid thoughts, such as the belief that someone’s out to get you.” If you are detained in a psychiatric department, such a reaction is normal and understandable. The staff is surely out to “get you,” namely to treat you forcefully with psychosis pills against your will. When psychiatric leaders use terms about their critics such as “anti-psychiatry” and “conspiracy,” is it then a “yes” to this question?
6. “Flat emotions, appearing aloof and isolated.” This is what psychiatric drugs do to people. If they were normal to begin with, the psychiatrists will ensure that this won’t last.
7. “Odd, eccentric or peculiar behaviour or appearance.” One definition of madness is doing the same thing again and again expecting a different result, which is what psychiatrists do all the time with their drugs. I would call that an odd, eccentric, and peculiar behaviour.
8. “Lack of close friends or confidants other than relatives.” This is what psychiatric drugs do to people, particularly psychosis pills; isolate people and make zombies out of them.
9. “Excessive social anxiety that doesn’t diminish with familiarity.” If you are detained in a psychiatric department, such a reaction is normal and understandable.

Many, perhaps even most, psychiatrists would test positive. What is less amusing is that the test provides circular evidence because patients who are normal might test positive after they have been treated inhumanely by psychiatrists.

When I discuss the state of psychiatry with critical psychiatrists, psychologists and pharmacists I collaborate with, they sometimes ask: “Who are most mad, the psychiatrists or their patients?” An Oxford dictionary defines delusion as “An idiosyncratic belief or impression maintained despite being contradicted by reality or rational argument, typically as a symptom of mental disorder.” According to this, the most vocal leading psychiatrists suffer from delusions.

I was once invited to follow the chief psychiatrist’s round at a closed ward.[8:68] We talked with several patients, and one of them appeared normal and reasonable to me, but to my big surprise, the psychiatrist asked me if I could see that he was delusional. As I couldn’t, he explained that the patient was delusional because he had been on the Internet and had found out that psychosis pills are dangerous. I replied that they are indeed dangerous and that there is nothing delusional in believing this. I was so stunned that I said no more. This psychiatrist was not just anybody. He had a high position at the Danish Psychiatric Association.

On another occasion, I phoned a psychiatric department that has a bad reputation because of the patients the psychiatrists have killed there with their drugs, including Luise.[234] A desperate patient in great distress had rung me, but I couldn’t get through to a psychiatrist, even though I was a colleague and it was within normal working hours. I was transferred to a head nurse who told me not to become involved because the patient was delusional. When I asked her in what way, she said he had found out that psychosis pills were dangerous. I asked her if she knew whom she was talking to. Oh yes, she knew about me.

Psychiatry is characterized by such insanity. The psychiatrists’ delusions are not shared by people considered sane, e.g. the general public, but they forcefully maintain them, even when the most reliable science has clearly shown that their beliefs are wrong. When I point this out to them, they have no shame or regrets.

If psychiatry had been a business, with competition, it would have gone bankrupt long ago.

The disappointing CATIE and STAR*D studies

The two sane authors of the first chapter of the 1065-page textbook noted that naturalistic studies - which they did not reference but mentioned by name, CATIE, STAR*D, and Storebø 2016 - have shown smaller effects than those the drug companies have advertised.[17:57] They also said that psychiatry is plagued by a bad reputation after cases of overmedication and that more caution is needed when using psychiatric drugs.[17:58]

For CATIE, there were 191 records on PubMed. It was an NIMH financed trial, which randomised 1493 "real world" patients with schizophrenia to olanzapine, quetiapine, risperidone or ziprasidone, or to a very old drug, perphenazine, marketed in 1957.

The results must have agonised the key opinion leaders in psychiatry. The primary outcome was very reasonable, time to discontinuation for any reason, which reflects both the benefits and the harms of the drugs. After 18 months, only 26% of the patients were still on the randomised drug, and perphenazine was not worse than the "atypicals" and did not produce more extrapyramidal harms than these agents.[239]

So much for the highly praised "modern" psychosis pills, which are far more expensive than a 65-year old drug off patent. But psychiatry's narrative was not affected. The study authors talked about the comparable levels of effectiveness of the five drugs,[239] but they should have talked about comparable levels of *ineffectiveness*, as all the drugs failed according to the primary outcome. Psychiatrists are masterminds in this type of semantic deception.

STAR*D was also financed by the NIMH. It is a remarkable story of fraud.[7:118] Like CATIE, it was a highly relevant study of real world patients. With 4041 included patients,[647] it is the largest effectiveness study ever conducted of depression pills. The investigators announced that the study would produce results with "substantial public health and scientific significance,"[647] which it did, but not in the way they had imagined.

There was no placebo group, and all patients started on citalopram, manufactured by Lundbeck, which was motivated by horrendously erroneous claims of citalopram's "absence of discontinuation symptoms" and its "safety" in elderly patients. In their disclosure statements, ten of STAR*D's authors reported receiving money from Forest, Lundbeck's partner in the United States.

When the study was over, NIMH announced falsely that "about 70% of those who did not withdraw from the study became symptom-free." 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." 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 honest professional would describe such patients as having become symptom-free.

The researchers noted in their abstract that, "The overall cumulative remission rate was 67%." In the main text, however, they said 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.

The investigators cherry-picked the data they reported. This involved the Texas sharpshooter trick (see page 41) by changing the measurement scale. They also included patients that, according

to the protocol, should have been excluded. This, the French call "sauve qui peut" (save those you can), which characterises a state of panic or disorder.

The data were presented in such a confusing manner that it is extremely difficult to correct for all the errors and find out what really happened, even for a seasoned research detective like me. Ed Pigott et al. did the hard detective work for us.[647] It turned out that only 3% of the patients who entered the trial remitted, stayed well, and stayed in the trial during the one-year follow-up!

This publicly funded study bombarded doctors and the public with the totally mendacious message that depression pills enable about 70% of depressed outpatients to recover. The medications were said to be "far more effective" than placebo, which was also mendacious, as there was no placebo group in the trial.

A journalist interviewed one of the STAR*D investigators, Maurizio Fava, a prominent psychiatrist, who acknowledged that the 3% success rate was accurate and that the investigators knew this all along.[648]

The many STAR*D papers display highly selective reporting of outcomes, numerous false claims, contradictory statements, and even pure fiction. As of mid-2011, despite over 100 papers having been published, 11 prespecified outcomes had still not been reported.[147] 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 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.

It is remarkable that suicidality can differ by a factor of 10 or more in different publications of the same trials, but this was also the case when the FDA investigated this issue (see page 95).

The STAR*D study is so fraudulent that all its 100+ papers should be retracted. Ed Pigott says about this:[649]

"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 these errors – without exception – had the effect of making the effectiveness of the antidepressant drugs look better than they 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."

I could not find any naturalistic study published by Storebø in 2016. The textbook authors might have referred to his 2015 Cochrane review, which found that every single trial ever performed of stimulants in children with an ADHD diagnosis was at high risk of bias.[511]

Thomas Insel and the NIMH: A total betrayal of public trust

Thomas Insel, called "America's psychiatrist," was director of the US National Institute for Mental Health for 13 years, till 2015.[650] In 2022, he published the book, *Healing: our path from mental illness to mental health*.[651]

The book makes an unintended case for abolishing psychiatry even though Insel tries to support it.[650] He takes on the role of a drug salesman, and already the title is misleading. There has been no path from mental illness to mental health, only one to even more mental illness.

Insel is aware of this and promises to investigate why mental health outcomes in the United States are so poor. The publisher presents the book as a roadmap for change, but this is not what it is about; in fact, Insel shies away from suggesting what is so obviously needed.

Coming from the most prestigious institution in the world in mental health, it is worth looking more closely at this book, as it reflects the thinking of psychiatric leaders all over the world. This is what Robert Whitaker did in his book review.[650] The book encapsulates how psychiatry has consistently betrayed public trust and misinformed the public. It underlines that psychiatry will never tell the public the truth about psychiatric drugs, and Whitaker concludes that the real source of the poor mental health outcomes in the United States is the psychiatric establishment, including the NIMH, which – although being a governmental agency – cannot be trusted.

Being a former NIMH director, Insel should have told his readers about the poor long-term outcomes of treatment with psychiatric drugs, as documented in expensive and prestigious research funded by the NIMH, e.g. CATIE and STAR*D. He didn't, even though he had an obvious ethical obligation to do so.[650] Whereas drug companies have funded the short-term studies of drugs, it was the NIMH, dating back to the 1970s, that funded studies of their long-term effects.

This made it even more deplorable that Insel avoided commenting on them. The public expects that a medical specialty will be an honest purveyor of scientific findings about the benefits and harms of its interventions, and if its research tells of treatments that *worsen* long-term outcomes, then the medical specialty will inform the public of those outcomes and rethink its practices.

For 65 years, psychiatry has failed to do this. Insel could have remedied this betrayal of public trust with this book and put psychiatry on a new path, but he sacrificed the patients and protected the psychiatric guild by keeping the long-term studies hidden.

When Whitaker wrote his book, *Anatomy of an epidemic: magic bullets, psychiatric drugs, and the astonishing rise of mental illness in America*, first published in 2010,[5] he started out with a medical puzzle.

The conventional history of psychiatry tells of how the introduction of psychosis pills in 1954 kicked off a psychopharmacological revolution, which was said to take another step forward with the SSRIs in 1988. The prescribing of psychiatric drugs soared, but why did the burden of mental illness soar, too? According to Insel, the number of adults in USA receiving a social security payment due to a mental disorder rose from around 1.3 million in 1987 to around 6 million today.

Whitaker dug through the research literature, and with each class of drugs, he tried to find out what the clinical course was before and after the introduction of drugs, and if the medicated or unmedicated patients had better long-term outcomes in clinical studies. Whitaker found that psychosis pills, depression pills and benzodiazepines worsen long-term outcomes, and that bipolar disorder, which is regularly treated with polypharmacy, runs a much more chronic course than manic depressive disorder - the diagnostic precursor to bipolar - once did.[5]

Whitaker is a careful researcher and his book is highly convincing. There was a great deal of pushback from prominent American psychiatrists when it came out but when a filmmaker interviewed Insel five years later and asked him about Whitaker's book, he responded that Whitaker's observations needed to be taken very seriously and noted that, in other areas of medicine, if you increase the use of your medication several times, you will see reductions in morbidity and mortality.

This short glimpse of sanity in psychiatry quickly disappeared. Insel asked the right question in the first chapter of his book:[650]

"When it comes to mental illness, there are more people getting more treatment than ever, yet death and disability continue to rise. How can more treatment be associated with worse outcomes?"

But he didn't give the right answers. In a most appalling fashion, Insel dismissed any worry that psychiatric drugs could be the cause of the poor outcomes. He used the tactic, philosopher Arthur Schopenhauer calls making a diversion (see page 25). Insel suddenly began talking of something else, as though it had a bearing on the matter. He wrote that Whitaker argues that drugs against depression and psychosis create a "supersensitivity" that makes patients dependent and chronically disabled. This is a red herring. Whether supersensitivity occurs or not (which I believe it does; see also below) is immaterial for Whitaker's convincing findings.

Insel claimed that Whitaker writes that the psychiatric establishment, in collaboration with the pharmaceutical industry, has conspired to overmedicate and overtreat children and adults with disastrous results, and that not everyone buys this conspiracy theory.

This is mendacious. The only time Whitaker used the term conspiracy was when he quoted a patient with schizophrenia who spoke about conspiracies.[5:21] Insel used the diversion trick again and another of Schopenhauer's tricks: "Postulate what has to be proven."[83]

Insel turned sand into gold by making yet a third horrific diversion. He claimed that current treatments are necessary but not sufficient to cure complex brain disorders. This has absolutely no bearing on the case. He quoted his predecessor Steven Hyman who said we need to know much more about the biology of mental illness before we "can illuminate a path across very difficult scientific terrain" and develop medications that are as effective as insulin or antibiotics.

The pompous mumbo jumbo covered up for the fact that biological psychiatry is a total failure, which history has so clearly shown. Furthermore, Insel's ill-founded fantasies about a better future do not remove the immense harm his specialty currently inflicts on hundreds of millions of people.

Insel went further into adventure land. He thinks clinicians are more effective today than they were 25 years ago. Indeed. They are harming their patients more than ever!

Insel's diversions multiplied. He noted that most people with mental illness are not treated; that many of those receiving drugs do not take them; and that patients receive little more than drugs. He cleverly put the blame for the poor outcomes on society for not investing in necessary social supports and on patients for failing to take their drugs and stay engaged in treatment.

This is the standard script for psychiatrists. The disaster they have created is not their fault. Others are to blame, including the patients and society. But if more patients took their drugs, the disaster would only be worse.

Nothing in Insel's narrative would harm psychiatry's guild interests or pharmaceutical interests. Insel described himself as taking on the role of a journalist as he explored humanistic supports that are needed to complement drugs to promote lasting recovery.

This is a win-win position to take. Anyone will welcome social support. Critics of psychiatry have advocated for such efforts for decades, and Insel now positioned himself as the advocate for this societal response. This was manipulation at the highest level. With that framework in place, there would be no place in his 300-page book for research that told of drug treatments that worsen long-term outcomes.

Instead of criticising the drugs, Insel praised them. In the chapter, *Treatments work*, he claimed that psychiatric drugs, ECT, and transcranial magnetic stimulation work and that depression pills have an effect size as high and often higher than medications used in other areas of medicine. A remarkable statement about drugs that have no clinically relevant effects. My comment on this

type of argument is that one unlawful parking does not make the next parking lawful. There are many ineffective drugs in medicine that should not be used.

Insel didn't cite a single study that told of psychiatric drugs providing a long-term benefit. This glaring omission leads to the conclusion that the former director of the NIMH is unable to find a single study to cite that told of a drug improving long-term outcomes. Insel's book is a superb example of *The Emperor's new clothes*. The Emperor is totally naked but so well dressed up that few readers will notice it.

In his book review, Whitaker provided a summary of studies Insel did not dare mention.[650] I present below a brief of this summary. The links to the papers can be found in the original, which is open access, and in my reference list.

After psychosis pills were introduced in the mid-1950s, clinicians began speaking about the "revolving door syndrome" that now appeared in asylum medicine. First-episode patients would be discharged and then return in droves, which led the NIMH, during the 1970s, to fund four studies to assess whether psychosis pills were increasing the chronicity of psychotic disorders.

Bockoven[652] reported that the rehospitalisation rate for discharged patients was higher for patients treated after the arrival of psychosis pills and the medicated patients were also more "socially dependent" than those treated before 1955. Carpenter,[653] Mosher,[654] and Rappaport[655] reported superior outcomes for unmedicated patients after 1-3 years, which led Carpenter to "raise the possibility that antipsychotic medication may make some schizophrenic patients more vulnerable to future relapse than would be the case in the natural course of the illness."

By this time, researchers were fleshing out the adaptive brain changes stirred by psychosis pills. Chouinard concluded that drug-induced dopamine supersensitivity "leads to both dyskinetic and psychotic symptoms. An implication is that the tendency toward psychotic relapse in a patient who has developed such a supersensitivity is determined by more than just the normal course of the illness."[656] This understanding of how the brain adapts to psychosis drugs provided a biological explanation for why drug treatment increased the chronicity of psychotic disorders and a causal explanation for the findings reported by Bockoven, Carpenter, Mosher and Rappaport.

Nancy Andreasen, also funded by NIMH, reported in a large MRI study of patients with schizophrenia that psychosis pills shrink brain volumes over time,[63] and that this shrinkage is associated with a worsening of negative symptoms, increased functional impairment, and, after five years, cognitive decline.[657]

In the late 1970s, with funding from the NIMH, Martin Harrow and Thomas Jobe launched a long-term study of 200 patients diagnosed with schizophrenia or other psychotic disorders, most of whom were experiencing a first or second episode of psychosis. They found that the outcomes of those who got off their psychosis pills by year two began to dramatically diverge from those who stayed on the drugs, and that at the end of 15 years, the recovery rate for the off-med patients was eight times higher than for the medication compliant patients (40% versus 5%).[658] They also reported that the medication compliant patients were much more likely to remain psychotic over the long term than those who got off the medication, and it was the off-medication patients who had dropped out of treatment that had the better outcomes.[659] They referred to drug-induced dopamine supersensitivity as a likely reason for this difference in outcomes.

In the past two decades, longer term studies of psychotic patients conducted in the Netherlands (the only long-term randomised trial of drug discontinuation, see page 55),[192] Finland,[660] Australia,[661] Denmark,[662] and Germany[663] all told of higher recovery rates for those off drugs.

Similarly, users of psychosis pills tell of how these drugs compromise functional recovery over the long-term.[664]

The history of depression pills is much the same. Prior to their introduction, depression - and this finding came from studies of hospitalised patients - was understood to be an episodic disorder. Patients could be expected to recover, and around half of the patients who suffered a first episode would never be rehospitalised for depression.

After the introduction of depression pills, some clinicians observed a "chronification" of the depression. In the 1980s, several studies found high relapse rates in patients treated with depression pills, and an expert panel convened by the NIMH concluded that, in contrast to older studies of mood disorders, "new epidemiological studies [have] demonstrated the recurrent and chronic nature of these illnesses."[665] The elephant in the room was ignored.

Two NIMH studies in real-world patients treated in outpatient settings confirmed that this was the long-term course for medicated patients. The STAR*D trial,[647] with its 3% stay-well rate at the end of the one-year follow-up on depression pills stood in sharp contrast to another NIMH funded trial that sought to identify the long-term outcome of untreated depression in recent times. In that study, 85% of the included 84 patients had recovered by the end of one year.[666] The researchers concluded that "If as many as 85% of depressed individuals who go without somatic treatment spontaneously recover within one year, it would be extremely difficult for any intervention to demonstrate a superior result to this."

Many studies over the past 35 years have compared outcomes for medicated and unmedicated patients over longer periods of time.

In an NIMH study that randomised 250 patients to imipramine or to two forms of psychotherapy or to placebo, the stay-well rate was highest for cognitive therapy (30%) and lowest for imipramine (19%) and placebo (20%) after 18 months.[667]

In an NIMH study of 547 patients that compared six-year outcomes for depressed people treated for the disorder and those who eschewed medical treatment, the treated patients were three times more likely than untreated ones to suffer a cessation of their principal social role and nearly seven times more likely to become incapacitated.[668]

A WHO study of 640 depressed patients found that those treated with medication had worse general health and were more likely to still be mentally ill than those who weren't treated at the end of one year.[669]

A Canadian study of 1281 people who went on short-term disability due to a depressive episode found that 19% of those who took a depression pill went on to long-term disability compared to 9% of those who never took such medication.[670]

In a five-year study of 9508 depressed patients in Canada, medicated patients were depressed on average 19 weeks a year, versus 11 weeks for those not taking drugs.[671]

Two reviews of the long-term outcomes of patients diagnosed with depression found that use of a depression pill was associated with worse outcomes at nine years[672] and at 30 years.[673]

As these findings have piled up, researchers - led by Italian psychiatrist Giovanni Fava - have pointed to drug changes induced by depression pills as a likely explanation for the "bleak long-term outcome of depression ... use of antidepressant drugs may propel the illness to a more malignant and treatment unresponsive course."[674-677]

In a 2011 paper, American psychiatrist Rif El-Mallakh observed that 40% of depressed patients initially treated with a depression pill were now ending up in a chronically depressed "treatment resistant" state.[678] He wrote that continued drug treatment may induce processes that may "cause

a worsening of the illness, continue for a period of time after discontinuation of the medication, and may not be reversible."

Given this literature, it is no surprise that depression is now the leading cause of disability in the United States for people ages 15 to 44, and that in country after country that has adopted widespread use of SSRIs, the number of people on government disability due to a mood disorder has increased in lockstep with the increased use of these drugs.[119:24]

Whitaker also mentioned the MTA trial (see page 131). The investigators noted that, at the end of three years, being on a stimulant was a significant marker not of beneficial outcome, but of deterioration.[517] At the end of six to eight years, the results were much the same.[521] Longer term ADHD studies in Australia[679] and Quebec[680] also found worse outcomes for medicated youth than for those treated without stimulants.

As Whitaker noted, the research literature shows that psychosis pills and depression pills increase the chronicity of the disorders, and the same is true for stimulants, benzodiazepines, and drugs used for bipolar disorder. He also mentioned that a longer list of over 100 papers that tell of these outcomes can be found on the Mad in America resource pages for psychosis pills, depression pills, benzodiazepines, polypharmacy for bipolar disorder, and stimulants.[650]

None of this history is found in Insel's book or on NIMH's website.[650] A search for Martin Harrow shows nothing even though he was considered one of NIMH's experts on schizophrenia. A search for STAR*D shows the press release about the short-term results that tells of "particularly good results" with depression pills that "highlight the effectiveness of high-quality care."[681] The one-year stay-well rate for patients treated with depression pills of 3% is missing (that information was hidden in the journal article that reported one-year out-comes[648]). And the NIMH website information about ADHD[682] does not inform parents that in the MTA study, medication use was a marker of deterioration by the end of year three, and that those taking stimulants had worse ADHD symptoms and were more functionally impaired at the end of six years.

The chemical imbalance myth is derided as a hypothesis that fell out of favour[97] decades ago, with Ronald Pies, former editor in chief of *Psychiatric Times*, describing it as an "urban legend" that was never "seriously propounded by well-informed psychiatrists."[97] Allen Frances,[683] and other prominent figures in the field, including Insel[684] and his predecessor Steven Hyman,[685] acknowledge that the disorders in the DSM manual have never been validated as discrete illnesses, and that the diagnostic categories are constructs. In his book, Insel admits that the so-called second-generation psychiatric drugs are no better than the first, the notion that they were "breakthrough medications" having been put to rest some time ago.

In 2015, Whitaker and Lisa Cosgrove published *Psychiatry under the influence,*[599] a book that arose from their time as fellows at the Safra Center for Ethics at Harvard University, in a lab devoted to studying institutional corruption. In a democratic society, the expectation is that institutions that serve a public interest - and this is particularly true for medical disciplines - will adhere to ethical standards. This includes rising above financial influences; being objective in their design of studies and their analysis of the data; reporting the results in an accurate and balanced way; and putting the interests of patients first.

In a 2009 essay,[686] Daniel Wikler, a professor of ethics at the Harvard School of Public Health, wrote that a medical discipline that fails to adhere to this standard doesn't deserve to retain its privileged place in society.

On Whitaker's Mad in America website there are two more reviews of Insel's book.[650] These reviews also describe how the book functions as a work of propaganda for a sick system.

The erosion of medical integrity is complete for psychiatry[1-11,533] and the psychiatric narrative has collapsed. Yet, drug prescribing increases. If the psychiatric profession told the public the truth, psychiatry would have to completely reorganise its care.

As Whitaker wrote,[650] this is the bridge that psychiatry, as a guild, cannot cross. The profession needs to keep the truth out of sight, even to itself, and it is not presented in psychiatric textbooks or in continuing medical education seminars. By keeping the history hidden, the field is breaking its compact with the public and itself - with every prescriber and all those who enter the field.

A 2022 misleading seminar in *The Lancet* about suicide

A recent *Lancet* seminar was yet another proof that psychiatry has degenerated to a point from which there is no return. Honest information about suicide is of utmost importance but the article *Lancet* published, *Suicide and self-harm,*[687] was dishonest.

The seminar was very long, 14 pages, with 142 references. Many people consider *Lancet* a highly prestigious and influential journal, which should therefore be open to criticism and debate. But it isn't. A journal that does not accept letters for publication unless they arrive within two weeks of publication of the original item and unless they are no longer than 250 words does not invite criticism and a sound scientific debate. Many people will not know that an article has been published before it is too late to criticise it.

The *Lancet* seminar is one of the most misleading articles about suicide I have ever seen.[688] The authors wrote that research has identified "associations between suicidal behaviour and dysregulation of the hypothalamic–pituitary–adrenal axis and serotonergic neural transmission."

They tried to resurrect the stone dead myth about a chemical imbalance in the brain being the cause of psychiatric disorders, and the two references they cited are untrustworthy (see Chapter 4). The first alluded to epigenetic modification of genes, alterations in key neurotransmitter systems, inflammatory changes, and glial dysfunction in the brain as causal factors. The second suggested hypo-thalamic-pituitary-adrenal axis dysfunction, which "in turn can be traced back to genetic predisposition" and "early life stress-related epigenetic mechanisms."

Among risk factors for suicide, the authors mentioned "harmful substance use" but not depression pills, antiepileptics, or the psychiatric profession itself. These are taboos for suicide researchers.

The authors wrote that "The use of medication to prevent suicide is controversial" and that there is a "possibility of exacerbating suicidal thoughts, particularly in young people."

As I have explained in my book, it is seriously dishonest to speak about a *possibility* of *exacerbating suicidal thoughts.* These drugs not only exacerbate suicidal thoughts, they cause them, and they also cause suicidal behaviour, suicide attempts, and suicide.

The seminar authors did not quote any of the many meta-analyses of placebo-controlled trials that showed that depression pills increase the suicide risk. Instead, they quoted a book written by the last author of the seminar and by Robert D Goldney who has published a fatally flawed review about depression pills and the risk of suicide.[689]

His paper is a classic example of how one *should not* do a review.[7:100] He cherry-picked those observational studies that supported his idea that depression pills protect against suicide, e.g. studies in the Nordic countries that linked prescribing of depression pills with a reduction of suicide, but these studies are untrustworthy.[7:97] Nordic researchers have shown that there is no statistical association between the increase in sales of SSRIs and the decline in suicide rates in the

Nordic countries.[690] They reported that the decline in suicides in Denmark and Sweden predated the introduction of SSRIs by ten years or more.

The Nordic researchers had no conflicts of interest while Goldney had "received honoraria and research grants from a number of pharmaceutical companies." With his flawed reviews, Goldney must be worth far more than his weight in gold for the drug industry.

The seminar authors wrote that "treatment of underlying psychiatric conditions through medication can reduce suicidal behaviour." They gave no references to this information. Which are the miraculous drugs that can *reduce* suicides? All we know is that psychiatric drugs *increase* suicides.

A little later, they wrote: "Evidence from several studies, most of which were observational, suggests that antidepressants might reduce the risk of suicide." They used the UFO trick. They quoted a 2021 review that reported that meta-analyses had found that "antidepressants prevent suicide attempts, but individual randomized controlled trials appear to be underpowered."[691] These meta-analyses were of observational studies. All meta-analyses of randomised trials have shown the opposite and they are not underpowered.

In the next sentence, they wrote: "However, some research has found an association with increased risk of suicide-related outcomes in young people." This is also blatantly false. When the FDA looked at *all* relevant research, not just *some research*, and indeed the best we have, the randomised placebo-controlled trials, it was a causal relation and not just an "association."

In the ensuing sentence, they wrote: "The evidence base is far from complete, since many randomised trials exclude people at heightened risk of self-harm or suicide." This is utter nonsense. We have all the data we need to conclude that depression pills double suicides. The authors used the familiar trick Schopenhauer calls diversion by suddenly talking of something else that has no bearing on the matter.

The authors claimed that "Lithium has been associated with reduced suicide rates in people with bipolar disorder and depression, which might be a specific effect not seen with other drugs designed to stabilise mood." As noted earlier (see page 106), there is no reliable evidence that lithium reduces suicides.

About the latest fad in psychiatry, the authors wrote that "Ketamine has shown promise." It hasn't (see page 78).

There was a glimpse of light in all the psychiatric darkness. The authors wrote that "cognitive behavioural therapy and related treatments have the strongest evidence base for reducing suicidal ideation and repeat self-harm compared with treatment as usual."

This is correct, but they quoted a review that included self-harm. Self-harm does not always imply a suicidal intent. My research group therefore did a systematic review where we excluded self-harm studies. We found that psychotherapy halves the risk of a new suicide attempt in people acutely admitted after a suicide attempt.[272] Our review was published in 2017 in a well-known journal but was not among the seminar authors' 142 references even though it sends a very strong message: Do not use pills but psychotherapy if you want to prevent suicide.

Lancet is not the source to go to if one wants reliable information about depression pills. It is the extended marketing arm of the pharmaceutical industry,[692] just like the *New England Journal of Medicine* is, also in relation to articles denying that depression pills cause suicide.[337]

Final words about a specialty in ruins and what to do about it

The authors of the five textbooks count some of the most prominent professors of psychiatry in Denmark. There is no reason to believe that the systematic betrayal of public trust would be any different in other countries. We see the same lies, denial and misleading information about psychiatry everywhere,[7] as illustrated so convincingly in Whitaker's review of Insel's book.

Those who shape psychiatry are often deeply corrupt,[7,533] and they often "forget" to declare their conflicts of interest against the rules.[7] These people are highly effective drug pushers. Court documents revealed that, in 1999, two such US psychiatrists, Charles Nemeroff and Alan Schatzberg, published a psychiatry textbook that was ghostwritten by GlaxoSmithKline.[335]

In 2000, they co-authored a report of a depression pill trial in *New England Journal of Medicine* where the authors had so many ties to drug companies that there wasn't room for them in the print journal (they took up 1067 words).[693] This made the journal's editor, Marcia Angell, publish an accompanying editorial: "Is academic medicine for sale?"[694] She explained that it had been difficult to find a psychiatrist to write an editorial who was not conflicted. This showed that the whole specialty has been corrupted by industry money. Nemeroff and Schatzberg declared 17 industry ties each:

Dr. Nemeroff has been a consultant to or received honoraria from Abbott, AstraZeneca, Bristol-Myers Squibb, Forest Laboratories, Janssen, Eli Lilly, Merck, Mitsubishi, Neurocrine Biosciences, Organon, Otsuka, Pfizer, Pharmacia–Upjohn, Sanofi, SmithKline Beecham, Solvay, and Wyeth–Ayerst. He has received research support from Abbott, AstraZeneca, Bristol-Myers Squibb, Forest Laboratories, Janssen, Eli Lilly, Organon, Pfizer, Pharmacia–Upjohn, SmithKline Beecham, Solvay, and Wyeth–Ayerst.

Dr. Schatzberg has served as a consultant to or received honoraria from Abbott, Bristol-Myers Squibb, Corcept Therapeutics, Forest Laboratories, Janssen, Eli Lilly, Merck, Mitsubishi Pharmaceuticals, Organon, ParkeDavis, Pfizer, Pharmacia–Upjohn, Sanofi, Scirex, SmithKline Beecham, Solvay, and Wyeth–Ayerst. He has received research support from BristolMyers Squibb, Pfizer, and SmithKline Beecham. He has equity ownership in Corcept, Merck, Pfizer, and Scirex.

I wonder if such people have time for seeing patients, or for listening to those they see.

The many erroneous and misleading statements I found cannot be explained by the advent of new, important knowledge, as the publication dates for the textbooks were recent, from 2016 to 2021. Furthermore, even though I have sometimes used recent articles to demonstrate that the authors are wrong, the knowledge I convey has existed for many years prior to 2016.

In the protocol for my study, I noted that the textbooks should mention that the causes of psychiatric disorders are mainly environmental, and not genetic or related to a visible brain abnormality. The textbooks conveyed the opposite message, and strongly so, although there is no foundation for a biological model of psychiatric disorders. The psychiatrists have not even been able to explain what exactly they mean by this.[9]

I also noted in my protocol that there are no reliable trials that have shown that drugs are better than placebo for overall functioning, quality of life, return to work, sick leave, and social relationships. The textbooks were remarkably silent on this important issue, even though there is clear evidence, particularly from non-industry funded randomised trials and from good observational studies that long-term drug treatment is harmful.[1,5]

It was disappointing that psychologists mostly said the same as the psychiatrists, and they were sometimes even more radical and uncritical than them, e.g. in their praise of the imaging studies and the drugs. I think there are two reasons for this. In a radicalised group, newcomers tend to be even more radical than their leaders to become accepted as their equals. Therefore fringe groups tend to become more radical with time. The other reason is related to the first one. Some psychologists want to get permission to prescribe drugs and their scientific associations often support this idea. They will not succeed if they are seen as critics of mainstream psychiatry.

One of the textbooks, *Clinical neuropsychology*, which has three psychologists as editors, exemplifies this issue.[20] It has three full pages describing imaging studies in depression, with many references.[20:432] It conveys the impression to the students that we know a lot about the brain based on reliable studies, which is totally false. Students believe what they read in their university books of psychiatry, even though it can best be characterised as brainwash, and they may spread their false ideas even more forcefully when confronted with irrefutable evidence to the contrary.[14]

Many psychologists do not realise that they have a great advantage over psychiatrists, which is that they are educated with the aim of understanding the patients where they are and helping them with psychotherapy and other forms of support. It is very sad when psychologists buy into the false narrative the psychiatrists and the drug industry have created about their drugs instead of criticising it. If we lose the leading psychologists, there is little hope for the patients who would then need to consult therapists with lesser educations. Some of them are very good, but they do not have an academic background for understanding the science.

When I announced in the Critical Psychiatry Network that I was writing a critical textbook of psychiatry that would explain what was wrong with the current textbooks, a general practitioner reported what she experienced when she went to a regional meeting about adult ADHD three years earlier to learn something. Here is what she learned:

The psychiatrist that lectured was in the pay of three drug companies. He presented no peer reviewed research and said he didn't like rules; he just knew what worked. The audience wasn't allowed to ask him direct questions. We were put in groups to discuss how we should implement what we had heard. Members of my group were stunned when I was chastised for asking two questions, one about how conflicts of interests might interfere with good prescribing and the other about the lack of long-term studies. I was told I was a dinosaur and too old to be flexible and innovative and go with modern medicine developments. I've never experienced anything like this before! I confronted the bully face to face when the group work was finished and left him with a stern reminder to keep his mind open.

Whether drugs are legal or illegal, it is unhealthy to perturb brain functions with them. Brain active substances can lead to violence, including murder. An analysis of adverse drug events submitted to the FDA between 2004 and 2009 identified 1937 cases of violence, 387 of which were homicide.[401]

The violence was particularly often reported for psychotropic drugs - depression pills, sedatives/hypnotics like benzodiazepines, ADHD drugs and a smoking cessation drug that also affects brain functions. Depression pills are being suspected of having a causal role in mass shootings, but when one of the teenage shooters in the Columbine High School massacre was found to have taken a depression pill, the American Psychiatric Association denounced the notion that there could be 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.[695]

This is sickening. It is marketing speak and standard industry tactic to blame the disease and not the drug, but this is what psychiatrists do all the time. The other murderer had taken both sertraline and paroxetine.

Drugs and guns are a dangerous cocktail, but America abounds in both, including easy access to opioids on prescription, which makes this country the most backward in the Western world.

There are many other high-profile cases where the mass murderers were on depression pills,[696] but in many cases, information about the shooters' prescription drug use and other medical history has been kept from public records. Drugs causing homicide is taboo.

The hypocrisy is all over the place. As an example, universities are happy to accept enormous gifts from industry at the same time as they implement stringent conflict of interest policies for their faculty and their relationship with commercial sponsors.[697]

One of the chapters in my book about organised crime in the drug industry was *Psychiatry, the drug industry's paradise*.[6] Psychiatry is second to none in exploiting people with harmful drugs and in killing, incapacitating or maiming hundreds of millions of people. In 1990-92, 12% of the US population aged 18–54 years received treatment for emotional problems, which went up to 20% in 2001–2003.[698] Although there are hundreds of diagnoses in DSM-IV, and even more in DSM-5, only half of people who were in treatment met diagnostic criteria for a disorder. In 2012, the US Centers for Disease Control reported that 25% of Americans have a mental illness.[699]

We must put an end to this insanity in a profession that is supposed to take care of the insane. We have a chance of influencing those who study psychiatry before it is too late and they have accepted the false narrative. This was my motivation for writing this book.

As child and adolescent psychiatrist Sami Timimi explains, psychiatry ignores much of the genuine science there is and instead goes on supporting and perpetuating concepts and treatments that have little scientific support.[10:20] He calls this scientism. It means that psychiatry likes to talk in the language of science and treats this as more important than the actual science.

In Timimi's debates with fellow psychiatrists about the evidence, three defences are common. The first is the use of anecdote - such and such a patient got better with such and such a treatment, therefore, this treatment works. The second is an appeal about taking a "balanced" perspective. But each person's idea of what a balanced position is depends on where they are sitting. We get our ideas on what is balanced from what is culturally dominant, not from what the science tells us. The third is that when molecular genetics has consistently failed to produce anything about diagnoses being related to specific genes, we are told that the area is "complex."[10:63] This is bullshit.

When I published my ten myths about psychiatry, which are harmful for people, in a major newspaper in January 2014, I ended my article this way:[189]

"Psychotropic drugs can be useful sometimes for some patients, particularly in short-term use, in 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."

My article caused an outcry that lasted for a couple of months, spearheaded by the drug industry and their paid allies among doctors and journalist friends. I got the whole Danish establishment on my back, and the Minister of Health threatened that I could get fired.[7:278] The only thing I had done was to tell people the truth. But this cannot be tolerated when the subject is psychiatry.

Outside the power circles, my paper was much appreciated.[700] Numerous articles followed, some written by psychiatrists who agreed with me. For more than a month, there wasn't a single day without discussion of these issues on radio, TV or in newspapers, and there were also debates at psychiatric departments. People in Norway and Sweden thanked me for having started a discussion that was impossible to have in their country, and I received hundreds of emails from patients who confirmed with their own stories that what I had written was true.

Nothing changed, however. Perhaps a little here and there, but nothing material. On the other hand, it matters for some people that we protest. Many patients and relatives have told me that my books have saved lives, as they gave the patients the courage to withdraw from their drugs against their doctor's advice.[8:167] These emails documented a high level of ignorance and arrogance among psychiatrists and here is a typical example:

Her psychiatrist told her she had an incurable genetic disease and needed psychosis pills for the rest of her life. When she complained that she could no longer concentrate, slept a lot and believed the drugs affected her memory, making it hard to study, the reply was that the problem wasn't the drugs but that she lost neurons due to the psychosis and that her brain wasn't the same anymore. So, she needed to take psychosis pills indefinitely to protect her brain from losing more neurons; otherwise she would become demented. When she had withdrawn the drugs despite this advice, she was told she would have a new psychotic episode. When she said she didn't want to take the drugs for the rest of her life, her psychiatrist replied that she would then not see her anymore because she only worked with patients who wanted to be treated.

What should we do about this? I have these suggestions:[8:172]

1) Leave mental health issues to psychologists and other caring professions. They are not medical diseases. Consider involving recovery mentors who have lived experience.

2) Psychiatry as a medical specialty should be disbanded. In an evidence-based healthcare, we do not use interventions that do more harm than good, which psychiatry does. Let psychologists who are against using psychiatric drugs be heads of psychiatric departments and give them the responsibility for the patients.

3) Psychiatrists should be re-educated so that they can function as psychologists. Those who are not willing to do this, should find themselves another job.

4) The focus should be on getting patients off psychiatric drugs, and to avoid starting them. Never start a drug without having a tapering plan.

5) Establish a 24-hour national helpline and associated website to provide advice and support for those adversely affected by prescribed drug dependence and withdrawal.

6) Provide tapering strips and other aids at no cost to help patients withdraw from their drugs. This would lead to huge savings for society.

7) Apologize. It means a lot for victims of abuse to get an apology.

8) Change psychiatry's misleading narrative, which starts with the semantics. Speak about depression pills, psychosis pills, speed on prescription, etc. Stop using words such as psychiatry, psychiatrist, psychiatric disorder, psychiatric treatments, and psychiatric drugs, as they are stig-

matising and as patients and the general public associate them with bad outcomes. Talk about mental health instead.

9) Discard the psychiatric diagnosis systems entirely and focus on the patients' problems.

10) Drop the rating scales, both in research and practice, and focus on recovery, i.e. a return to a normal productive life.

11) Make forced treatment unlawful.

12) Make psychiatric drugs available only for use under strictly controlled circumstances:

a) while patients are tapering off them; or

b) in rare cases where it is impossible to taper off them because they have caused permanent brain damage; or

c) in patients with alcoholic delirium, as sedatives under operations and other invasive procedures, e.g. colonoscopy, and in other circumstances to be defined.

13) Make it unlawful to use drugs that are registered for nonpsychiatric uses, e.g. anti-epileptics, for mental health issues.

14) Avoid financial conflicts of interest with manufacturers of psychoactive drugs or other treatments, e.g. equipment for electroshock.

15) Forbid all rules about demanding a psychiatric diagnosis to get social benefits, or extra economic support to schools.

16) Make it illegal for general practitioners to prescribe psychiatric drugs, which they cannot handle. In relation to depression, 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.[701] They therefore hand out depression pills liberally. A US study showed that over half of the physicians wrote prescriptions after discussing depression with patients for three minutes or less.[172]

17) Tell the patients that it is rarely a good idea to see a family doctor or a psychiatrist if they have a mental health issue. There is a huge risk that they will be harmed.

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 with clinical trials and regulatory affairs in the drug industry 1975-1983, and at hospitals in Copenhagen 1984-95. Co-founded the Cochrane Collaboration and established the Nordic Cochrane Centre in 1993. Became professor of Clinical Research Design and Analysis in 2010 at the University of Copenhagen. Co-founded Council for Evidence-based Psychiatry in the UK in 2014 and the International Institute for Psychiatric Drug Withdrawal in Sweden in 2016. Founded the Institute for Scientific Freedom in 2019. Peter is officially retired but currently works as researcher, lecturer, author and independent consultant, e.g. in lawsuits.

Peter's greatest contribution to public health was when he, in 2010, opened the archives in the European Medicines Agency after a 3-year long battle that involved a complaint to the European Ombudsman. The agency was solely concerned with protecting the drug industry's interests while ignoring those of the patients. The Ombudsman ruled there was no commercially confident information in the clinical study reports.

Peter has published more than 75 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 150,000 times. His H-index is 82 according to Web of Science, which means that 82 papers have been cited at least 82 times. Peter is author of several books. The most recent ones are:

- The Chinese virus: Killed millions and scientific freedom (2022).
- Mental health survival kit and withdrawal from psychiatric drugs: a user's guide (2022).
- The decline and fall of the Cochrane empire (2022)
- Vaccines: truth, lies and controversy (2021, with an updated corona chapter).
- Mental health survival kit and withdrawal from psychiatric drugs (2020, in 8 languages).
- Vaccines: truth, lies and controversy (2020, in 7 languages).
- Survival in an overmedicated world: Find the evidence yourself (2019, in 7 languages).
- Death of a whistleblower and Cochrane's moral collapse (2019).
- Deadly psychiatry and organised denial (2015, in 9 languages).
- Deadly medicines and organised crime: How big pharma has corrupted health care (2013, in 16 languages). Winner, British Medical Association's Annual Book Award, Basis of Medicine in 2014.
- Mammography screening: truth, lies and controversy (2012). Winner of the Prescrire Prize in 2012.
- Rational diagnosis and treatment: evidence-based clinical decision-making (2007).

Peter has given numerous interviews, one of which - about organised crime in the drug industry - has been seen 450,000 times on YouTube. He was in The Daily Show in New York on 16 September 2014 where he played the role of Deep Throat revealing secrets about big pharma. A documentary film about Peter's reform work, *Diagnosing Psychiatry*, appeared in 2017, and another documentary film about the downfall of the Cochrane Collaboration is under preparation.

Peter has an interest in statistics and research methodology. He has co-authored guidelines for good reporting: CONSORT for randomised trials, STROBE for observational studies, PRISMA for systematic reviews and meta-analyses, and SPIRIT for trial protocols. Peter was an editor in the Cochrane Methodology Review Group 1997-2014.

Peter is Protector for the Hearing Voices Network in Denmark.

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