DEPRESSION: WHY DRUGS AND ELECTRICITY ARE NOT THE ANSWER

Abstract

The dominant view within mental health services and research suggests that feeling depressed is a kind of medical illness partially caused by various biological deficits which are somehow corrected by physical interventions. This article critically appraises evidence for the effectiveness and value of antidepressant drugs and ECT, the two principle physical treatments recommended for depression. It also describes the negative effects of these procedures and raises some concerns about how they impact on the brain. We propose an alternative understanding that recognises depression as an emotional and meaningful response to unwanted life events and circumstances. This perspective demands that we address the social conditions that make depression likely and suggests that a combination of politics and common sense needs to guide us in providing help for people suffering in this way. This alternative view is increasingly endorsed around the world, including by the United Nations, the World Health Organisation and service users who have suffered negative consequences of physical treatments that modify brain functions in ways that are not well-understood.

Introduction

With the World Health Association and the United Nations calling for a paradigm shift away from the medicalization of human distress, new evidence about millions of people struggling to get off antidepressants, and ongoing debate about the value and safety of ECT, it seems timely to discuss these two longstanding treatments offered to us by biological psychiatry's 'medical model' when we become sad, or depressed.

Firstly, we acknowledge that treatments rest on, and are justified by, assumptions about the nature, and causes, of what is being 'treated'. Advocates of so-called 'antidepressant' medications and electroconvulsive therapy {ECT} argue that the treatments work by correcting underlying biological dysfunctions. These hypothesised dysfunctions are proposed to be among the causes of the problematic cognitions, emotions and behaviour that are referred to as 'symptoms' and that form the criteria for diagnosing 'mental disorders' such as 'Major Depressive Disorder'. We, and others, have provided detailed reviews of evidence demonstrating that no biological dysfunction that can be corrected by current treatments has been found, and explained how alternative mechanisms can account for apparent treatment effects including treatment-induced alterations to normal brain functions as well as placebo effects (Breggin, 2008; Fosse & Read, 2013; Moncrieff & Cohen, 2005; Moncrieff, 2008; Read et al., 2015; Read, Kirsch & McGrath, 2019; Read & Sanders, 2013; Valenstein, 1988).

Although most clinicians subscribe to a biopsychosocial model of mental disorder, the idea that treatments work by rectifying underlying biological dysfunctions relegates the role of social and psychological factors to secondary or indirect considerations, such as triggers of a supposed genetic predisposition. Although holistic care is important in general medicine, the primary focus is treating the physiological processes that give rise to symptoms and health risks (Moncrieff, 2020). Therefore, equating psychiatric conditions and treatments with medical ones implies the pre-eminence of biological factors. This is reinforced with psychiatric language. Depression, for example, is described as somehow *causing* abnormal feelings and behaviours, *as if* it were a physical condition, even though those same feelings and behaviours form the criteria for the diagnosis in the first place. For example, the American Psychiatric Association (2021a) proclaims:

> Depression (major depressive disorder) is a common and serious medical illness that negatively affects how you feel, the way you think and how you act.

Until January 2021, the APA website advised:

> Psychiatric medications can help correct imbalances in brain chemistry that are thought to be involved in some mental disorders. (APA, 2021b)

The idea that biological factors 'cause' depression, even if in conjunction with social circumstances, also presupposes that there is a mechanical and predictable relationship between biology and human feelings and actions that excludes the possibility of meaning and agency (Moncrieff, 2020). Hence viewing depression as a medical disorder that somehow originates in the brain and responds to brain-based interventions is fundamentally inconsistent with understanding it as a 'normal' human emotion, albeit sometimes extreme and disproportionate - that is as a meaningful reaction to depressing circumstances (Moncrieff, 2020)

Although we will focus on the failure to establish that antidepressants and ECT are effective or safe, we do so from the perspective that this approach, focussed as it is on decontextualized, pathologised, individuals or brains, is flawed from the outset. For example, it cannot address the issues underlying women being about twice as likely as men to receive either 'treatment'. We are not alone in calling instead for approaches that acknowledge the meaning of depression, and address the common social origins of misery and sadness (Brown & Harris, 1978; Cromby, Harper, & Reavey, 2013; Johnstone et al., 2018; Puras, 2019).

Antidepressants

Are antidepressants active placebos?

Certain drugs have been referred to as 'antidepressants' since the 1950s. Despite this appellation, it is not clear that they have any specific antidepressant effects. Hundreds of placebo-controlled trials suggest that antidepressants are marginally better than placebo at reducing depressive symptoms as measured by depression rating scales. Combining

published and unpublished studies suggests an effect size of around 0.3 across different meta-analyses, which translates into a difference of around 2.0 points on the commonly used Hamilton Depression rating scale (HAM-D) (Hamilton, 1960), which has a maximum score of 54 points. This has not been shown to be a clinically relevant difference (Leucht et al., 2013; Moncrieff & Kirsch, 2015). Matching HAM-D scores against Clinical Global Impression scale scores (Guy, 1976) suggests a difference of 8 points is required to indicate 'mild clinical improvement' and that a difference of 3 points and below does not register as indicating any change.

Moreover, the small difference between antidepressant drugs and inert placebo tablets does not confirm that the drugs have an antidepressant action. There are other explanations for these small differences. At present, most drugs are assumed to work according to a 'disease-centred' model of drug action, which proposes that they act on the biological processes assumed to underpin symptoms, in the same way as drugs do in most medical conditions, including non-curative, symptomatic treatments (such as salbutamol for wheezing). However, an alternative, 'drug-centred' model suggests that psychiatric drugs change mental states and behaviour through the modification of normal brain functions (Table 1). This model highlights that psychiatric drugs are psychoactive substances that alter normal thoughts, sensations, emotions and behaviours. These alterations, along with physical alterations, may unblind people in placebo-controlled trials. This may lead to amplified placebo effects among those taking active drugs, especially since several antidepressant studies show that people's beliefs about whether they are taking an active drug or a placebo have substantial effects on outcome (Chen et al., 2011; Faria et al., 2017). Consistent with this, it seems that almost any drug with psychoactive properties (one that produces noticeable mental alterations) has equivalent effects to antidepressants in depression, including benzodiazepines, stimulants, opiates, buspirone and antipsychotics (Moncrieff, 2008).

TABLE ONE ABOUT HERE

The mental and behavioural alterations produced by antidepressants (and other psychiatric drugs and ECT) may also reduce or mask depressed feelings or other 'symptoms' of depression. SSRIs and some other antidepressants have emotion-numbing effects, which may lead to a reduction in intensity of both depression and anxiety (Goldsmith & Moncrieff, 2011; Price, Cole, & Goodwin, 2009; Read & Williams, 2018). Antidepressants with sedative properties, for example, such as the tricyclic antidepressants and some newer agents like mirtazapine, may help with insomnia or reduce anxiety or agitation, all of which feature in depression rating scales. The fact that differences from placebo are so small suggests these effects are not particularly useful, however.

Other artefacts may also account for the differences between drugs and placebos in randomised trials, such as selective publication (Turner, Matthews, Linardatos, Tell, & Rosenthal, 2008), the conversion of continuous data into categorical outcomes (Kirsch & Moncrieff, 2007), and the fact that many participants in drug trials are already on medication, and are then withdrawn, leading to withdrawal effects and increased likelihood of unblinding in the placebo group (Hunter et al., 2015).

Antidepressants perform poorly in clinical practice

The majority of placebo-controlled trials have been conducted by the pharmaceutical industry, which has an investment in inflating results, but government-funded research also fails to confirm that antidepressants have beneficial effects. The massive STAR-D study of gold-standard naturalistic antidepressant treatment produced dismal results. Although the original publication suggested reasonable remission rates of 37% at 12 weeks (Rush et al., 2006), the study has been criticised for the use of a secondary outcome measure, exclusion of early dropouts and numerous other protocol deviations (Pigott, 2015; Pigott, Leventhal, Alter,

& Boren, 2010). A subsequent analysis found that only 108 participants out of a total of over 4000 recovered, remained well and completed the study, a figure that is still based on the secondary outcome (Pigott et al., 2010). For 14 years no data was published on the primary outcome measure, the Hamilton rating scale. When independent researchers eventually obtained the data it emerged that people given antidepressant treatment along with high quality care showed a reduction in their scores of 6.6 points after 12 weeks (Kirsch, Huedo-Medina, Pigott, & Johnson 2018). This is at the lower end of the range of change seen in people on placebo in meta-analyses of randomised trials (Gibbons, Hur, Brown, Davis, & Mann, 2012; Kirsch, Moore, Scoboria, & Nicholls, 2002; Sugarman, Loree, Baltes, Grekin, & Kirsch, 2014) and roughly half that in randomised trials comparing different antidepressants (Rutherford, Sneed, & Roose, 2009). This suggests that the conditions of being in a randomised trial inflate drug effects and that, in real life, antidepressants are no better than placebos.

Do antidepressants correct an underlying biological abnormality?

Despite claims by professional organisations and the pharmaceutical industry that depression is due to a chemical imbalance that can be rectified by drugs (e.g. APA, 2021b), there is no evidence that there are any neurochemical abnormalities in people with depression, let alone abnormalities that might cause depression. Where differences between people with and without depression have been found, these are likely to be explained by prior use of antidepressants and other medications; but in most areas no consistent differences have been found in any case (Moncrieff et al., submitted). Although the public, internationally, continue to favour psycho-social explanations of depression (Hagmayer & Engelmann, 2014; Read, Cartwright, Gibson, Shiels, & Haslam, 2015), an increasing proportion have been influenced to believe that depression is caused by a chemical imbalance (Pilkington et al.,

2013) and across the world increasing numbers now take antidepressants (OECD, 2020; Olfson, Wang, Wall, Marcus, & Blanco, 2019). 17% of the population of England were prescribed an antidepressant in 2018 (Taylor et al., 2019), and 14% of US adults by 2015 (Olfson et al., 2019). Yet the number of people requiring services or going onto long-term disability due to depression is increasing (Olfson et al., 2019; Viola & Moncrieff, 2016).

Antidepressants alter normal mental activity and behaviour

Besides the fact that millions of people are taking drugs with little demonstrable benefit, the dominance of the disease-centred model of drug action has inhibited research into the nature of the various antidepressant drugs. We therefore don't know the full implications and long-term consequences of taking these drugs. Like other psychiatric drugs, they are psychoactive substances that cross the blood-brain barrier and alter normal mental processes and behaviour by changing the normal functioning of the brain. Evidence suggests that SSRIs reduce the intensity of emotions, and produce apathy and demotivation, which are associated with impairment of sexual function (Goldsmith & Moncrieff, 2011; Padala et al., 2020; Read & Williams, 2018; Zahodne et al., 2012). A drug-centred model suggests that we need to evaluate whether such changes might have worthwhile effects, when balanced against their likely negative impact and other adverse effects.

Adverse effects of antidepressants

Modern antidepressants are generally less toxic than their predecessors and therefore less likely to be used for self-poisoning or suicide, as older antidepressants frequently were (Henry & Antao, 1992). Many have fewer adverse effects, but they are not innocuous. SSRIs cause sexual dysfunction in a large proportion of users, and more worryingly, some people report that this persists after stopping the drug (Bala, Nguyen, & Hellstrom, 2018). This is

consistent with research with young animals finding that sexual behaviour is negatively impacted by previous use of SSRIs (Simonsen, Danborg, & Gotzsche, 2016). The prevalence of persistent effects is unknown, but even if it is rare, it is a potential catastrophe given the huge numbers now using antidepressants, and the increasing number of younger users. That long-term antidepressant use may lead to persistent brain modifications is also evidenced by the prolonged and severe withdrawal state they can induce (Hengartner, Schulthess, Sorensen, & Framer, 2020; Framer, 2021).

It has been recognised since the 1990s that current antidepressants are associated with withdrawal effects (Haddad, Lejoyeux, & Young, 1998), but this has only recently started to receive serious attention. Recent evidence suggests that around 56% of people experience withdrawal effects after discontinuing antidepressants, and for 46% of those the effects are severe (Davies & Read, 2019). In general, the longer someone takes an antidepressant, the more likely they are to experience a withdrawal reaction, and the more severe it will be (Horowitz & Taylor, 2019). The adverse effects of withdrawal can be so intolerable that some people trying to discontinue treatment have to reduce by tiny amounts over many years, and accumulating evidence suggests that the effects may even persist for months or years after the drugs are finally stopped (Framer, 2021; Hengartner et al., 2020).

The use of antidepressants also has potential negative psychological consequences. Since antidepressants are associated with beliefs that depression is caused by biochemical perturbations, their use may discourage people from addressing the circumstances that caused their depression in the first place, whether they be relationship problems, financial difficulties or something else. If people attribute their improvement to taking antidepressants, rather than recognising how they helped themselves, they will not develop confidence in their own resilience and abilities which is likely to make them more vulnerable to future episodes.

Research confirms that people may come to believe they need antidepressants to stay well, and therefore become fearful of stopping them, leading to ever increasing numbers of long-term users (Eveleigh, Speckens, van Weel, Oude Voshaar, & Lucassen, 2019; Maund et al., 2019). The longer antidepressants are used, the greater their adverse effects, including the likelihood of severe and protracted withdrawal syndromes.

Electroconvulsive Therapy

Correcting a biological deficit?

As is the case for antidepressants, the various biological deficits that are supposedly corrected by ECT have never been demonstrated. The earliest two claims about how ECT works, and what it was supposedly correcting, are interesting.

The first was a supposed 'biological antagonism' between 'schizophrenia' and epilepsy (Fink & Sackeim, 1996). If you had one you couldn't have the other. While some doctors treated epilepsy by injecting the blood of 'schizophrenics' (Kalinowsky, 1986), others were using various techniques, including insulin and eventually electricity, to induce seizures in 'schizophrenics'.

The second claim was that ECT works because it causes brain damage, thereby erasing painful memories and/or simplifying thought processes. In 1941, Walter Freeman, who imported ECT to the United States, wrote, in a paper entitled 'Brain Damaging Therapeutics':

> The greater the damage, the more likely the remission of psychotic symptoms. . . . Maybe it will be shown that a mentally ill patient can think more clearly and more constructively with less brain in actual operation (p. 83).

Another psychiatrist explained:

> There have to be organic changes or organic disturbances in the physiology of the brain for the cure to take place. I think the disturbance in memory is probably an integral part of the recovery process. I think that it may be true that these people have

> for the time being at any rate more intelligence than they can handle and that the reduction in intelligence is an important factor in the curative process. . . . Some of the very best cures that one gets are in those individuals whom one reduces almost to amentia. (Myerson, 1942, p. 39)

These quotations concerned the use of ECT for 'schizophrenia', but the idea that the procedure worked in this way was also applied to depression, at least until the 1960s. The principle UK psychiatry textbook of the period attributed the effects of ECT to the 'disruption by the fit and the subsequent period of amnesia of recently acquired morbid patterns of behaviour and reaction' (Henderson & Gillespie, 1962, p. 335).

A neutral observer would assume that the effects on the brain of repeatedly passing sufficient electricity through it to cause seizures are likely to be negative. ECT advocates, however, tend to interpret abnormal brain changes caused by multiple electrocutions as beneficial, sometimes even linking them to reduced depression They don't consider that the changes might be negative or might be characterised as brain damage. To do so would revert to the original 1940s theory that ECT works *because* it causes brain damage.

A similar line of argument was resurrected, 70 years later, by researchers who reported that ECT reduces the 'functional connectivity' of the brain (Fosse & Read, 2013). Our neutral observer might assume the researchers would be concerned by this rather worrying side effect. Instead, they celebrated having discovered how ECT works. They positing a supposed 'hyperconnectivity' that somehow causes depression, and which is somehow corrected by ECT (Perrin et al., 2012). Other ECT advocates, meanwhile claim to have found the opposite; that ECT works because it *increases* functional connectivity (Fosse & Read, 2013; Wei et al., 2018).

Still other ECT proponents, meanwhile, acknowledge that we simply don't know what brain changes lead to the temporary lift in mood that some people experience, or what biological deficits are being corrected. Max Fink, a very famous ECT advocates, admitted,

83 years after the first ECT: ‘Studies to decode the mechanism by which such interventions improve serious illnesses are sorely needed’ (Fink, 2021, p. 151).

A recent audit of 36 ECT patient information leaflets in England found that 22% acknowledged that it is not known how ECT works. Nevertheless, 78% claimed it corrects some deficit in the brain (Harrop, Read, Geekie, & Renton, 2021).

Freeman’s old idea of ‘brain damaging therapeutics’ does receive some support from contemporary evidence. A 2013 review concluded:

> The temporarily improved scores on depression instruments following ECT reflect the combination of frontal and temporal lobe functional impairments and activation of the HPA axis and the mesocorticolimbic dopamine system. These effects as well as other detailed changes observed in structures such as the hippocampus appear consistent with those typically seen after severe stress-exposure and/or brain trauma. (Fosse & Read, 2013, p. 6)

Furthermore, bilateral ECT causes more memory loss/brain damage than unilateral (Sackeim et al., 2007), and is more effective in the short-term (Read & Arnold, 2017), suggesting that cognitive impairment and ‘improvement’ result from the same brain process.

So, as is the case for ‘antidepressants,’ the story of ECT appears to be one of a biological intervention being claimed to correct biological deficits, but in reality having negative effects on healthy brains, some of which are misconstrued as signs of improvement.

Medical intervention or expectancy effect?

Like antidepressants, the story of ECT is also the story of the power of placebo effects (Rasmussen, 2009; Read & Bentall, 2010). Positive expectations affect prescribers as well as patients. They influence perceptions of recovery as well as recovery itself. Neurologist John Friedberg (1976, p. 31) pointed out that the rapid spread of ECT across Europe and the USA in the 1940s took place despite their being no studies comparing recipients and non-

recipients, and that ‘the influence of ECT was on the minds of the psychiatrists, producing optimism and earlier discharges.’

The standard placebo in ECT studies, known as ‘sham ECT’ {SECT}, is the administration of the general anesthetic but not the electricity or subsequent convulsion. A review of the literature on placebo responses to ECT concluded: ‘Rigorously defined endogenously depressed patients did exceptionally well with sham ECT, just as well as with real ECT’ (Rasmussen 2009, p. 59).

In the 83 years since the first ECT there have only been 11 randomized placebo-controlled studies (RCTs) for its target diagnosis, depression, all before 1986 (Read & Bentall, 2010). A recent review, involving Dr Irving Kirsch, Associate Director of Placebo Studies at Harvard Medical School, highlighted the poor quality of the 11 studies (Read et al., 2019, p. 64):

> Only four studies describe their processes of randomization and testing the blinding. None convincingly demonstrate that they are double-blind. Five selectively report their findings. Only four report any ratings by patients. None assess Quality of Life. The studies are small, involving an average of 37 people.

Furthermore:

> Four of the 11 found ECT significantly superior to SECT at the end of treatment, five found no significant difference and two found mixed results (including one where the psychiatrists reported a difference but patients did not).’

No studies showed that ECT outperforms placebo beyond the end of the treatment period (Read et al., 2019). Two of the least flawed studies reported follow up data. One produced a near-zero effect size (.065) in favour of ECT (Johnstone et al., 1980), the other a small effect size (.299) in favour of SECT (Lambourn & Gill, 1978).

Nevertheless, all five meta-analyses of these flawed studies somehow conclude that ECT is effective. They pay little or no attention to the shortcomings of the studies, fail to comment

on the high response to sham ECT, and don't identify any evidence on long-term effects (Read et al., 2019). The Food and Drug Administration (2020) in the US mandates that ECT machines have signs next to them stating: 'The long-term safety and effectiveness of ECT treatment has not been demonstrated.'

The meta-analyses fail to identify any evidence that ECT prevents suicide, as often claimed. Numerous studies have found ECT recipients are *more* likely than other patients to kill themselves (Munk-Olsen et al., 2007; Read, Bentall, Johnstone, Fosse, & Bracken, 2013). In a recent study 14,810 ECT patients were 16 times more likely to try to kill themselves than a matched control group of 58,369 (Peltzman, Shiner, & Watts, 2020). Even after controlling for 'demographic, clinical, and service use characteristics,' including psychiatric diagnoses and inpatient admissions, the ECT patients were 1.3 times more likely to have killed themselves (a non-significant difference). A study using the Danish National Patient Registry also found an increased risk of suicide in patients who received ECT compared to equally depressed non-ECT patients (Jorgensen, Rozing, Kellner, & Osler, 2020),

Another recent study, using the Swedish national registry, claimed its findings 'support the continued use of ECT to reduce suicide risk in hospitalized patients who are severely depressed' (Ronnqvist, Nilsson, & Nordenskjold, 2021). The overall difference in suicides over 12 months between the ECT group (1.1%) and the non-ECT group (1.6%) was small. The difference was significant, but only for patients who were psychotic, not for those who were depressed but not psychotic. Nor did the difference hold for people under 45. Furthermore, at three months (at which point any difference might more reasonably be attributed to ECT than at 12 months) there had been no significant overall difference. More importantly, 'Suicide was defined as death caused by intentional self-harm (*ICD-10* codes X60-X80) or by an event of undetermined intent (*ICD-10* codes Y10-Y35).' Emails to the

authors failed to determine how many of the events they counted as suicide had, instead, been 'events of undetermined intent'.

The most recent study found that 1,524 homeless US veterans who received ECT had made significantly more suicide attempts, at 30 days follow up, than 3,025 matched homeless veterans who hadn't had ECT. The difference remained significant at 90 days and one year (Tsai, Peltzman, Watts & Shiner, 2021).

In the only recent meta-analysis, from the Institute of Psychiatry in London (Mutz et al., 2019), just one sham ECT study (Brandon et al., 1984) contributed to their 'network meta-analysis' (involving comparisons of different types of treatments). Thus, after more than 80 years, only one placebo study was considered robust by the Institute. Strangely, another relatively high quality study, the well-known Northwick Park study (Johnstone et al., 1980), which found much smaller effects of ECT, was excluded because, according to the authors, it 'cannot be obtained'. It had been published in the Lancet. Furthermore, the one study they did include (Brandon et al., 1984) was classified, by the reviewers themselves, as having a 'high risk' of bias. Nevertheless, they announced that two of the four types of ECT they claimed to have assessed are more effective than placebo (and two are not), even though the only sham-ECT study they included assessed only one of those four types (bilateral ECT) (Read et al., 2019, pp. 88,89).

The 2019 review concluded:

> Given the high risk of permanent memory loss and the small mortality risk, this longstanding failure to determine whether or not ECT works means that its use should be immediately suspended until a series of well designed, randomized, placebo controlled studies have investigated whether there really are any significant benefits against which the proven significant risks can be weighed. (Read et al., 2019, p. 64)

Esteemed British Clinical Psychologist, Professor Richard Bentall (2020) commented:

> I believe that Read and his colleagues have done an important service in pointing out the parlous state of ECT research . . . ECT is a classic failure of evidence-based medicine.

A subsequent discussion regarding the ethics of ECT concluded:

> Although most of the medical literature states that ECT is an effective and safe technique, there is no conclusive evidence of long-term effectiveness (González-Pando et al., 2021).

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The six defenses against having no robust evidence

There are six standard responses used to try to counteract the absence of any robust evidence that ECT is better than placebo (see Table 2).

The first is that it has been used for so long that it must be effective. Unfortunately, the history of psychiatry is littered with treatments considered, by intelligent, well-intentioned doctors, to be safe and effective, which turned out to be neither, such as lobotomies.

The second is that it is unfair to apply today's standards of evidence-based medicine to studies conducted 40 or 50 years ago. Perhaps so, but this acknowledges that there is no robust evidence It also begs the question: why have none been conducted since 1985?

The third defense, an attempt to answer the above question, is that it is unethical to conduct studies that withhold a treatment which we believe works from severely depressed, suicidal patients. Arguing that we can't find out whether X actually *does* work because withholding X is unethical because we *believe* it works, renders ECT proponents beyond the realms of normal science and evidence-based medicine. (It also implies that 39 colleagues, the authors of the 11 RCTs, who did engage in the scientific process, were unethical).

TABLE TWO ABOUT HERE

The fourth is to argue that RCTs aren't essential because other types of studies can be relied upon, such as comparisons with antidepressants or between different types of ECT,

along with clinical impressions. The sham-ECT trials clearly demonstrate a powerful placebo effect, however, that will confound other types of evidence. Furthermore, a review of these non-placebo studies found that '89% produced no meaningful follow-up data beyond the end of treatment, and none investigated whether ECT prevents suicide.' (Read & Arnold, 2017).

The fifth is to acknowledge the absence of evidence of any benefits beyond the end of the treatment period but argue that this doesn't matter because you can maintain the short-term effects with antidepressants. The claim requires forgetting that ECT is primarily recommended for 'treatment resistant depression' , i.e. for people for whom the drugs have proved ineffective.

The sixth defense is to shoot the messenger. Researchers and ECT recipients who question the efficacy and highlight the adverse effects of ECT, are often publicly denigrated, by ECT advocates, as 'anti-psychiatry ideologues', 'extremists' 'Scientologists' and 'non-medical zealots', whose work is 'biased polemics written masquerading as science', 'garbage', 'dangerous misinformation' or part of a 'guild war' between professions. See, for example, comments from psychiatrists in response to *Medscape*'s coverage of the Read et al. (2019) review (Vlessides, 2020).The President and Chair of the *International Society for ECT and Neurostimulation* recently accused authors (including two ECT recipients) who had published some inconvenient findings (Read, Hancock and Cunliffe, 2020) of being 'ideologically driven' of 'spreading misinformation' and of having 'questionable motives' (Coffey & Kellner, 2021).

The actual effects of ECT

The fact that the temporary lift in mood experienced by some ECT recipients is primarily a placebo effect, would not matter if it weren't for the fact that repeatedly passing sufficient electricity through the brain to cause a seizure does have effects on the brain. As already

discussed, these effects are sometimes acknowledged but presented not as damage but as a beneficial correction of an imagined pathology (Perrin et al., 2012). Many of the changes, however, are the same as those documented after brain trauma (Fosse & Read, 2013, p. 6).

As well as the short-term memory loss, which is widely acknowledged, between 12% (Sackeim et al., 2007) and 55% (Rose et al., 2003) of ECT recipients suffer persistent or permanent memory loss (typically defined as six months or longer) (Read & Bentall, 2010; Read et al., 2019). The American Psychiatric Association (2001) acknowledges that 'ECT can result in persistent or permanent memory loss.' An ECT machine manufacturer in the USA recently added 'permanent brain damage and permanent memory loss' to its list of risks (Somatics, 2018). For example:

> My long-term memory was destroyed. Memories of childhood friends, memories of major events I attended, memories of my training as a psychiatric registrar. I started struggling with simple spelling and calculations. …. I never told colleagues about this, as I felt ashamed. But I started talking to other people who had ECT and realized I am not alone. (Bink, 2020)

Furthermore, ECT causes adverse psychological and emotional effects (Johnstone, 2009). It also carries a small risk of mortality (Read, Bentall, Johnstone, Fosse, & Bracken, 2013; Read et al., 2019) and cardiac complications, the leading cause of ECT-related deaths. A review of 82 studies found that one in 50 ECT patients experience 'major adverse cardiac events' (Duma et al., 2019).

The recent audit of information sheets in England found that people were not well informed about the risks of ECT. 28% failed to acknowledge the risk of long-term/persistent/permanent memory loss (Harrop et al., 2021) and none informed women and older people, the two demographic groups most likely to receive ECT, that they are at particularly high risk (Sackeim et al., 2007). Few leaflets presented clear information on mortality and cardiac risks (Harrop et al., 2021).

Two audits of how ECT is administered in England (Read, Harrop, Geekie, & Renton, 2018, 2021) found inconsistent but generally poor practice, including little evidence of adequate assessment of cognitive damage. The more recent of the two concluded:

> Given the apparent failure of current monitoring and accrediting of ECT clinics in England, by the Royal College of Psychiatrists' ECT Accreditation Service (ECTAS), an independent government sponsored review is urgently needed.

A campaign for such a review (Johnstone & Cunliffe, 2020; Read, 2020) has broad support, including Mind (England's largest mental health charity), the Royal College of Nursing, the Association of Clinical Psychologists, Headway (the brain injury association), and cross-party MPs including Dr Rosena Allin-Khan, the Shadow Mental Health Minister.

Alternative approaches

We are suggesting that antidepressants and ECT can change an individual's mental state by modifying normal brain activity. In someone with depression, these mental changes are superimposed onto pre-existing depressed feelings, which may temporarily obscure them. Although this situation is routinely understood as an improvement of the depression itself, this is because the brain and mind-altering properties of these procedures are ignored. Temporarily dampening down depressed feelings with brain manipulations may sound helpful for some serious situations, but the long-term consequences of these interventions have not been adequately researched. Procedures that change normal brain functions should be expected to have adverse effects, some of which may be long-lasting, and this seems to be the experience of numerous people who undergo ECT or take antidepressants long-term. We need much more information on long-term consequences before we deem it safe to continue prescribing these techniques

Furthermore, believing you have a brain disease requiring medical intervention can be profoundly disempowering. It encourages people to view themselves as the victims of their biology, to adopt pessimistic views about recovery, increases self-stigma and discourages

people from taking active steps to improve their situation (Deacon & Baird, 2009; Kemp, Lickell, & Deacon, 2014; Read, Haslam, & Magliano, 2013). Amongst the general population and mental health professionals biological causal beliefs (genetic, biochemical imbalance etc.) about 'mental health problems' have been consistently linked to negative attitudes (Kvaale, Haslam, & Gottdiener, 2013; Lebowitz & Ahn, 2014; Read et al., 2013).

So, if antidepressants and ECT are not helpful and are potentially unsafe, how should we help people when they feel depressed or distressed? First, understanding depression and anxiety as emotional reactions to life circumstances, rather than the manifestations of supposed brain pathology, demands a combination of political action and common sense. There is longstanding evidence on how deprivation and social adversity make people vulnerable to depression (Bjorkenstam et al., 2017; Brown & Harris, 1978; Cacioppo et al., 2006; Hoebel et al., 2017; Postmes et al., 2018). Social prescribing is now a recognised intervention for depression, currently being tested in trials (Lewellyn, 2019). For example, psychotherapy (Cuijpers et al., 2021), exercise (Schuch et al., 2016) and mindfulness (Reangsing et al., 2020) have all been shown to be beneficial. However, helping someone in distress is not primarily a scientific activity - it is an essentially human one. Common sense suggests that the conditions needed to lead an emotionally balanced and fulfilling life, relatively free of major ongoing worry and distress, include a dependable income, housing, secure and rewarding employment, engaging social activities, and opportunities to form close relationships. Some people may need relationship counselling or family therapy, others support with employment or finances. People who feel severely depressed for a long time may simply need to be cared for, reassured with kindness and hope, reminded of times when they have felt good, and kept safe until their condition improves, which it often does with time.

There is no scientific evidence for some of these suggestions. We learn how to support our fellow humans through our life experience, through being cared for ourselves, and sometimes through art and literature. Classifying anxiety, depression and other emotional reactions as mental diseases or disorders obscures the relation between our moods and our circumstances. It leads society to believe that social structures are unchangeable. Instead, we need to listen carefully to the message that people's emotional reactions convey, and endeavour to create a society in which all people can flourish. This approach to understanding and helping people with 'mental health problems' is increasingly endorsed by professionals and political bodies, as well as service user organisations. The United Nations Special Rapporteur, Dr Dainius Pūras, a Lithuanian psychiatrist, recently wrote:

> Current mental health policies have been affected to a large extent by the asymmetry of power and biases because of the dominance of the biomedical model and biomedical interventions. This model has led … to the medicalization of normal reactions to life's many pressures, including moderate forms of social anxiety, sadness, shyness, truancy and antisocial behaviour. (Puras, 2019)

The World Health Organization (2021) echoed these sentiments in its 'Guidance on Community Mental Health Services' which argues that social determinants of mental health are being neglected, resulting in 'an over-diagnosis of human distress and over-reliance on psychotropic drugs to the detriment of psychosocial interventions'. The document offers 22 examples of alternatives to drugs and electricity (Read, 2021).

The British Psychological Society has published reports on depression (Bowden, Shankar, Cooke & Kinderman, 2020) and mental health more generally (Cooke, 2017; Johnstone et al., 2018) that suggest that brain-based understandings and treatments rest on false assumptions, and call for alternatives to uninformative and potentially stigmatising diagnoses, and for treatments that address the role of trauma, power structures and social adversity (Johnstone et al., 2018; Read & Harper, 2021).

A plethora of international organisations representing people who have been harmed by psychiatric treatments, including ECT and antidepressants, also call for a different approach to understanding and treating depression. James Moore, founder of Mad in the UK (www.madintheuk.com) and the Let's Talk Withdrawal podcast (www.letstalkwithdrawal.com), came to realise that he had not been adequately informed about the risks and benefits of antidepressants, and was 'coerced and led on a merry dance, ultimately to the detriment of my health, my confidence, my family and my social life'. He concludes:

> Psychiatric drugs can't address isolation, poverty, inequality, racism, intolerance, hatred, bigotry, sexism, etc., but they can mask those things. Perhaps that is why they are so successful. The blame is placed on us, the patient, for being broken because it obviates the need for powers that be to take any action to address those underlying causes of distress and suffering (Moore, 2018).

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References

American Psychiatric Association. (2001). *The practice of ECT: A Task Force Report* (2nd edn). Washington, DC: A.P.A.

American Psychiatric Association. (2021a). *What is depression*? Retrieved from: https://www.psychiatry.org/patients-families/depression/what-is-depression

American Psychiatric Association. (2021b). Retrieved from: https://www.psychiatry.org/patients-families/what-is-psychiatry-menu. Accessed 15th Jan 2021.

Bala, A., Nguyen, H., & Hellstrom, W. (2018). Post-SSRI sexual dysfunction: A literature review. *Sexual Medicine Reviews, 6*(1), 29-34. doi:10.1016/j.sxmr.2017.07.002

Bentall, R. (2020). ECT is a classic failure of evidence-based medicine. *Council for Evidence-Based Psychiatry, June 4.* http://cepuk.org/2020/06/04/guest-blog-by-richard-bentall-ect-is-a-classic-failure-of-evidence-based-medicine/

Bink, S. (2020). Comment, August 7. In response to M. Vlessides (2020) Experts call for immediate suspension of ECT; Others push back. *Medscape,* July 24. Retrieved from: https://www.medscape.com/viewarticle/934536

Bowden, G., Shankar, R., Cooke, A., & Kinderman, P. (2020). *Understanding depression: Why adults experience depression and what can help*. Leicester: British Psychological Society.

Bjorkenstam, E., Pebley, A., Burstrom, B., & Kosidou, K. (2017). Childhood social adversity and risk of depressive symptoms in adolescence in a US national sample. *Journal of Affective Disorders, 212,* 56-63. doi:10.1016/j.jad.2017.01.035

Brandon, S., Cowley, P., McDonald, C., Neville, P., Palmer, R., & Wellstood-Eason, S. (1984). ECT results in depressive illness from the Leicestershire trial. *British Medical Journal, 288*(6410), 22–25. https://doi.org/10.1136/bmj.288.6410.22

Breggin, P. (2008). *Brain-disabling treatments in psychiatry* (Vol. 2nd). New York: Springer.

Brown, G., & Harris, T. (eds.)(1978). *The social origins of depression: a study of psychiatric disorder in women.* . London: Tavistock.

Cacioppo, J., Hughes, M., Waite, L., Hawkley, L., & Thisted, R. (2006). Loneliness as a specific risk factor for depressive symptoms: cross-sectional and longitudinal analyses. *Psychology of Aging, 21*(1), 140-151. doi:10.1037/0882-7974.21.1.140

Chen, J., Papakostas, G., Youn, S., Baer, L., Clain, A., Fava, M., & Mischoulon, D. (2011). Association between patient beliefs regarding assigned treatment and clinical response: reanalysis of data from the Hypericum Depression Trial Study Group. *Journal of Clinical Psychiatry, 72*(12), 1669-1676. doi:10.4088/JCP.10m06453

Coffey, C., & Kellner, C. (2021). A spirited defense of ECT. *Psychiatric Times,* June 7. Retrieved from: https://www.psychiatrictimes.com/view/spirited-defense-ect

Cooke, A. (2017) *Understanding psychosis and schizophrenia*. Leicester: British Psychological Society.

Cromby, J., Harper, D., & Reavey, P. (2013). *Psychology, mental health and distress.* New York: Palgrave Macmillan.

Cuijpers, P., Quero, S., Noma, H., Ciharova, M., Miguel, C., Karyotaki, E., … Furukawa, T. (2021). Psychotherapies for depression: a network meta-analysis covering efficacy, acceptability and long-term outcomes of all main treatment types. *World Psychiatry, 20,* 283-293. doi.org/10.1002/wps.20860

Davies, J., & Read, J. (2019). A systematic review into the incidence, severity and duration of antidepressant withdrawal effects: Are guidelines evidence-based? *Addictive Behaviours, 97,* 111-121. doi:10.1016/j.addbeh.2018.08.027

Deacon, B., & Baird, G. (2009). The chemical imbalance explanation of depression: Reducing blame at what cost? *Journal of Social and Clinical Psychology, 28*(4), 415-435. doi: https://doi.org/10.1521/jscp.2009.28.4.415

Duma, A., Maleczek, M., Panjikaran, B., Herkner, H., Karrison, T., & Nagele, P. (2019). Major adverse cardiac events and mortality associated with Electroconvulsive Therapy A systematic review and meta-analysis. *Anesthesiology, 130,*(1), 83-91. doi:10.1097/ALN.0000000000002488.

Eveleigh, R., Speckens, A., van Weel, C., Oude Voshaar, R., & Lucassen, P. (2019). Patients' attitudes to discontinuing not-indicated long-term antidepressant use: barriers and facilitators. *Therapeutic Advances in Psychopharmacology, 9,* 2045125319872344. doi:10.1177/2045125319872344

Faria, V., Gingnell, M., Hoppe, J. Hjorth, O., Alaie, I., Frick, A., . . . Furmark, T. (2017). Do you believe it? Verbal suggestions influence the clinical and neural effects of escitalopram in social anxiety disorder: a randomized trial. EBioMedicine, 24, 179-188. doi:10.1016/j.ebiom.2017.09.031

Fink, M. (2021). Random controlled trial of sham electroconvulsive therapy and other novel therapies. *Journal of ECT, 37*(3), 150-151. doi:10.1097/YCT.0000000000000759

Fink, M. & Sackeim, H. (1996). Convulsive therapy in schizophrenia? *Schizophrenia Bulletin, 22*(1), 27-39. doi.org/10.1093/schbul/22.1.27

Food & Drug Administration (2020). CFR - Code of Federal Regulations Title 21; Part 882 – Neurological Devices. Retrieved from: https://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfcfr/cfrsearch.cfm?fr=882.5940

Fosse, R., & Read, J. (2013). Electroconvulsive treatment: Hypotheses about mechanisms of action. *Frontiers in Psychiatry, 4,* 94-103. doi:10.3389/fpsyt.2013.00094

Framer, A. (2021). What I have learnt from helping thousands of people taper off antidepressants and other psychotropic medications. *Therapeutic Advances in Psychopharmacology, 11*, 2045125321991274. doi:10.1177/2045125321991274

Freeman, W. (1941). Brain-damaging therapeutics. *Diseases of the Nervous System, 2*, 83.

Friedberg, J. (1976). *Shock treatment is not good for your brain*. San Francisco: Glide.

Gibbons, R., Hur, K., Brown, C., Davis, J., & Mann, J. (2012). Benefits from antidepressants: Synthesis of 6-week patient-level outcomes from double-blind placebo-controlled randomized trials of fluoxetine and venlafaxine. *Archives of General Psychiatry, 69*(6), 572-579. doi:10.1001/archgenpsychiatry.2011.2044.

Goldsmith, L., & Moncrieff, J. (2011). The psychoactive effects of antidepressants and their association with suicidality. *Current Drug Safety, 6*(2), 115-121. doi:10.2174/157488611795684622

González-Pando, D., González-Menéndez, A., Aparicio-Basauri, V., Sanz de la Garza, C., Torracchi-Carrasco, J., & Pérez-Álvarez, M. (2021). Ethical implications of electroconvulsive therapy: a review. *Ethical Human Psychology and Psychiatry, 23*(1), 1-14. http://dx.doi.org/10.1891/ EHPP-D-21-00002

Guy, W. (1976). The Clinical Global Impression Scale. In *ECDEU Assessment Manual for Psychopharmacology- Revised* (pp. 218-222). Rockville, MD: US Department of Education, Health and Welfare.

Haddad, P., Lejoyeux, M., & Young, A. (1998). Antidepressant discontinuation reactions. *British Medical Journal, 316*(7138), 1105-1106. doi:10.1136/bmj.316.7138.1105

Hagmayer, Y. & Engelmann, L. (2014). Causal beliefs about depression in different cultural groups. *Frontiers in Psychology, 5*, 1303. https://doi.org/10.3389/fpsyg.2014.01303

Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry, 23*(1), 56-62. https://doi.org/10.1136/jnnp.23.1.56

Harrop, C., Read, J., Geekie, J., & Renton, J. (2021). How accurate are ECT patient information leaflets provided by mental health services in England and the Royal College of How Psychiatrists? An independent audit. *Ethical Human Psychology and Psychiatry*, Published online: https://doi.org/10.1891/EHPP-D-21-00003

Henderson, D., & Gillespie, R. D. (1962). *Henderson and Gillespie's textbook of psychiatry* (6th ed.). Oxford, Oxford University Press.

Hengartner, M. (2020). How effective are antidepressants for depression over the long term? A critical review of relapse prevention trials and the issue of withdrawal confounding. *Therapeutic Advances in Psychopharmacology, 10*, 2045125320921694. doi:10.1177/2045125320921694

Hengartner, M., Schulthess, L., Sorensen, A., & Framer, A. (2020). Protracted withdrawal syndrome after stopping antidepressants: a descriptive quantitative analysis of consumer

narratives from a large internet forum. *Therapeutic Advances in Psychopharmacology, 10*, 2045125320980573. doi:10.1177/2045125320980573

Henry, J., & Antao, C. (1992). Suicide and fatal antidepressant poisoning. *European Journal of Medicine, 1*(6), 343-348.

Hoebel, J., Maske, U. E., Zeeb, H., & Lampert, T. (2017). Social Inequalities and Depressive Symptoms in Adults: The Role of Objective and Subjective Socioeconomic Status. *PLoS One, 12*(1), e0169764. doi:10.1371/journal.pone.0169764

Horowitz, M., & Taylor, D. (2019). Tapering of SSRI treatment to mitigate withdrawal symptoms. *Lancet Psychiatry, 6*(6), 538-546. doi:10.1016/S2215-0366(19)30032-X

Hunter, A., Cook, I., Tartter, M., Sharma, S., Disse, G., & Leuchter, A. (2015). Antidepressant treatment history and drug-placebo separation in a placebo-controlled trial in major depressive disorder. *Psychopharmacology (Berl), 232*(20), 3833-3840. doi:10.1007/s00213-015-4047-2

Johnstone, E., Deakin, J., Lawler, P., Frith, C., Stevens, M., McPherson, K., & Crow, T. (1980). The Northwick Park ECT trial. *Lancet, 316*(8208-8209), 1317-1320. https://doi.org/10.1016/S01406736(80)92393-4

Johnstone, L., Boyle., M., with Cromby, J., Dillon, J., Harper, D., Kinderman, P., Longden, E., Pilgrim, D. & Read, J. . (2018). *The Power Threat Meaning Framework: Towards the identification of patterns in emotional distress, unusual experiences and troubled or troubling behaviour, as an alternative to functional psychiatric diagnosis.* Leicester: British Psychological Society.

Johnstone, L. (1999). Adverse psychological effects of ECT. *Journal of Mental Health, 8*(1), 69–85. https://doi.org/10.1080/09638239917652

Johnstone, L. & Cunliffe, S. (2020). Call for an independent review into the practice of ECT. *Council for Evidence Based Psychiatry*, September 7. http://cepuk.org/2020/09/07/blog-call-for-an-independent-review-into-the-practice-of-ect/

Jorgensen, M., Rozing, M., Kellner, C., & Osler, M. (2020). Electroconvulsive therapy, depression severity and mortality data from the Danish National Patient Registry. *Journal of Psychopharmacology, 34*(3), 273-279. doi:10.1177/0269881119895518

Kalinowsky, L. (1986). History of convulsive therapy. *Annals of the New York Academy of Sciences, 462*(1), 5-11.

Kemp, J., Lickel, J., & Deacon, B. (2014). Effects of a chemical imbalance causal explanation on individuals' perceptions of their depressive symptoms. *Behaviour Research and Therapy, 56*, 47-52. doi:10.1016/j.brat.2014.02.009

Kirsch, I., Huedo-Medina, T., Pigott, H., & Johnson, B. (2018). Do outcomes of clinical trials resemble those 'real world' patients? A reanalysis of the STAR-D antidepressant dataset. *Psychology of Consciousness: Theory, Research and Practice., 5*(4), 339-345. doi.org/10.1037/cns0000164

Kirsch, I., & Moncrieff, J. (2007). Clinical trials and the response rate illusion. *Contemporary Clinical Trials, 28*(4), 348-351. doi:https://doi.org/10.1037/cns0000164

Kirsch, I., Moore, T., Scoboria, A., & Nicholls, S. (2002). The emperor's new drugs: An analysis of antidepressant medication data submitted to the US Food and Drug Administration. *Prevention and Treatment, 5(23).* doi:10.1037/1522-3736.5.1.523a

Kvaale, E., Haslam, N. & Gottdiener, W. (2013). The 'side effects' of medicalization: A meta-analytic review of how biogenetic explanations affect stigma. *Clinical Psychology Review, 33*(6), 782-794. doi:10.1016/j.socscimed.2013.07.017

Lambourn, J., & Gill, D. (1978). A controlled comparison of simulated and real ECT. *British Journal of Psychiatry, 133,* 514-519. doi.org/10.1192/bjp.133.6.514

Lebowitz, M., & Ahn, W. (2014). Effects of biological explanations for mental disorders on clinicians' empathy. *Proceedings of the National Academy of Sciences, 111,* 17786-17790. doi:10.1073/pnas.1414058111

Leucht, S., Fennema, H., Engel, R., Kaspers-Janssen, M., Lepping, P., & Szegedi, A. (2013). What does the HAM-D mean? *Journal of Affective Disorders, 148*(2-3), 243-248. doi:10.1016/j.jad.2012.12.001

Lewellyn, M. (2019). A Mental Health Social Prescribing Trial (British Red Cross) (PROSPECT). *ClinicalTrials.gov*. Retrieved from https://www.clinicaltrials.gov/ct2/show/NCT04099095

Loughman, A., & Haslam, N. (2018). Neuroscientific explanations and the stigma of mental disorder: a meta-analytic study. *Cognitive Resear*ch, 3, 43. doi.org/10.1186/s41235-018-0136-1

Maund, E., Dewar-Haggart, R., Williams, S., Bowers, H., Geraghty, A., Leydon, G., . . . Kendrick, T. (2019). Barriers and facilitators to discontinuing antidepressant use: A systematic review and thematic synthesis. *Journal of Affective Disorders, 245,* 38-62. doi:10.1016/j.jad.2018.10.107

Moncrieff, J. (2008). *The myth of the chemical cure: A critique of psychiatric drug treatment*. Basingstoke, UK: Palgrave Macmillan.

Moncrieff, J. (2009). *A straight talking introduction to psychiatric drugs.* Ross-on-Wye, UK: PCCS Books.

Moncrieff, J. (2020). “It was the brain tumor that done it!” Szasz and Wittgenstein on the importance of distinguishing disease from behavior and implications for the nature of mental disorder. *Philosophy, Psychiatry & Psychology, 27*, 169-181. doi:10.1353/ppp.0.0023

Moncrieff, J., Stockmann, T., Amendola, S., Hengartner, M., & Horowitz, M. (2021). The serotonin theory of depression: an umbrella review of the evidence. *Molecular Psychiatry, submitted*.

Moncrieff, J., & Cohen, D. (2005). Rethinking models of psychotropic drug action. *Psychotherapy and .Psychosomatics, 74*(3), 145-153. doi.org/10.1159/000083999

Moncrieff, J., & Kirsch, I. (2015). Empirically derived criteria cast doubt on the clinical significance of antidepressant-placebo differences. *Contemporary Clinical Trials, 43*, 60-62. doi: 10.1016/j.cct.2015.05.005

Moore, J. (2018). *We’ve backed the wrong horse – It’s time for our love affair with psychiatric drugs to end.* Retrieved from https://www.madintheuk.com/2018/09/its-time-for-our-love-affair-with-psychiatric-drugs-to-end/. Accessed 13th July 2021

Munk-Olsen, T., Laursen, T., Videbech, P., Mortensen, P., & Rosenberg, R. (2007). All-cause mortality among recipients of electroconvulsive therapy: register-based cohort study. British Journal of Psychiatry, 190, 435-439. doi:10.1192/bjp.bp.106.026740

Mutz, J., Vipulananthan, V., Carter, B., Hurlemann, R., Fu, C., & Young, A. (2019). Comparative efficacy and acceptability of non-surgical brain stimulation for the acute treatment of major depressive episodes in adults: Systematic review and network meta-analysis. *British Medical Journal*, *364*, l1079. https://doi.org/10.1136/bmj.l1079

Myerson, A. (1942). Fatalities following ECT. *Transactions of the American Neurological Association, 68,* 39.

Organisation for Economic Co-Operation and Development (OECD) (2020). Statistics Health Status. Retrieved from: https://stats.oecd.org/index.aspx?DataSetCode=HEALTH_STAT#. Accessed 16th May 2020

Olfson, M., Wang, S., Wall, M., Marcus, S., & Blanco, C. (2019). Trends in serious psychological distress and outpatient mental health care of US adults. *JAMA Psychiatry, 76*(2), 152-161. doi:10.1001/jamapsychiatry.2018.3550

Padala, P., Padala, K., Majagi, A., Garner, K., Dennis, R., & Sullivan, D. (2020). Selective serotonin reuptake inhibitors-associated apathy syndrome: A cross sectional study. *Medicine (Baltimore), 99*(33), e21497. doi:10.1097/MD.0000000000021497

Peltzman, T., Shiner, B., & Watts, B. (2020). Effects of Electroconvulsive Therapy on short-term suicide mortality in a risk-matched patient population. *Journal of ECT, 36*(3), 187-192. doi: 10.1097/YCT.0000000000000665

Perrin, J., Merz, S., Bennett, D., Currie, J., Steele, D., Reid, I, & Schwarzbauer, C. (2012). Electroconvulsive therapy reduces frontal cortical connectivity in severe depressive disorder. *Proceedings of the National Academy of Sciences, 109*(14), 5464-5468. doi:10.1073/pnas.1117206109

Pigott, H. (2015). The STAR*D Trial: It Is Time to Reexamine the Clinical Beliefs That Guide the Treatment of Major Depression. *Canadian Journal of Psychiatry, 60*(1), 9-13. doi:10.1177/070674371506000104

Pigott, H., Leventhal, A., Alter, G., & Boren, J. (2010). Efficacy and effectiveness of antidepressants: current status of research. *Psychotherapy and Psychomatics, 79*(5), 267-279. https://doi.org/10.1159/000318293

Pilkington, P., Reavley, N., & Jorm, A. (2013). The Australian public's beliefs about the causes of depression: Associated factors and changes over 16 years. *Journal of Affective Disorders, 150*(2), 356-362. doi:10.1016/j.jad.2013.04.019

Price, J., Cole, V., & Goodwin, G. (2009). Emotional side-effects of selective serotonin reuptake inhibitors: qualitative study. *British Journal of Psychiatry, 195*(3), 211-217. doi: 10.1192/bjp.bp.108.051110.

Postmes, T. Wichmann., L. van Valkengoed, A., & van der Hoef, H. (2018). Social identification and depression: A meta-analysis. *European Journal of Social Psychology, 49,* 110-126. doi.org/10.1002/ejsp.2508

Puras, D. (2019). *Report of the Special Rapporteur on the right of everyone to the enjoyment of the highest attainable standard of physical and mental health.* United Nations Human Rights Council, 24 June-12 June. Retrieved from: https://www.un.org/en/ga/search/view_doc.asp?symbol=A/HRC/41/34

Rasmussen K. (2009). Sham ECT studies in depressive illness. *Journal of ECT, 25*(1), 54-59. doi:10.1097/YCT.0b013e3181719b23.

Read, J. (2021). W.H.O. and U.N. join calls to transcend medical model. *Psychology Today,* June 22. Retrieved from: https://www.psychologytoday.com/gb/blog/psychiatry-through-the-looking-glass/202106/who-and-un-join-calls-transcend-the-medical-model

Read, J. (2020). Electroshock therapy: From research to action. *Psychology Today,* August 24. Retrieved from: https://www.psychologytoday.com/gb/blog/psychiatry-through-the-looking-glass/202008/electroshock-therapy-research-action

Read, J., & Arnold, C. (2017). Is electroconvulsive therapy for depression more effective than placebo? A systematic review of studies since 2009. *Ethical Human Psychology and Psychiatry, 19*(1), 5–23. https://doi.org/10.1891/1559-4343.19.1.5

Read, J., & Bentall, R. (2010). The effectiveness of electroconvulsive therapy: A literature review. *Epidemiology and Psychiatric Sciences, 19*(4), 333-347. https://doi.org/10.1017/S1121189X00000671

Read, J., Bentall, R., Johnstone, L., Fosse, R., & Bracken, P. (2013). Electroconvulsive Therapy. In J. Read & J. Dillon (Eds.), *Models of madness: Psychological, social and biological approaches to psychosis* (2nd ed., pp. 90–104). Hove, UK: Routledge. https://doi.org/10.4324/9780203527160

Read, J., Cartwright, C., Gibson, K., Shiels, C., & Haslam, N. (2014). Beliefs of people taking antidepressants about causes of depression and reasons for increased prescribing rates. *Journal of Affective Disorders, 168,* 236-242. doi:10.1016/j.jad.2014.06.010

Read, J., Gibson, K., Cartwright, C., Shiels, C., Dowrick, C., & Gabbay, M. (2015). Understanding the non-pharmacological correlates of self-reported efficacy of antidepressants. *Acta Psychiatrica Scandinavica, 131*(6), 434-445. doi:10.1111/acps.12390

Read, J., Hancock, S., & Cunliffe, S. (2021). ECT: Dangerous on either side of the pond. *Psychiatric Times, 38*(4), 4-7. Retrieved from: https://www.psychiatrictimes.com/view/ect-dangerous-either-side-pond

Read, J., & Harper, D. (2020). The Power Threat Meaning Framework: Addressing adversity, challenging prejudice and stigma and transforming services. *Journal of Constructivist Psychology.* Published online: doi.org/10.1080/10720537.2020.1773356.

Read, J., Harrop, C., Geekie, J., & Renton, J. (2018). An audit of ECT in England 2011-2015: Usage, demographics, and adherence to guidelines and legislation. *Psychology and Psychotherapy: Theory, Research and Practice, 91*(3), 263-277. doi: 10.1111/papt.12160.

Read, J., Harrop, C., Geekie, J., Renton, J., & Cunliffe, S. (2021). A second independent audit of ECT in England: Usage, demographics, consent, and adherence to guidelines and legislation in 2019. *Psychology and Psychotherapy: Theory, Research and Practice.* Published online: doi:10.1111/papt.12335

Read, J., Haslam, N., & Magliano, L. (2013). Prejudice, stigma and 'schizophrenia': the role of bio-genetic ideology. In J. Read & J. Dillon (Eds.), *Models of madness: Psychological, social and biological approaches to psychosis* (2nd ed., pp. 143-156). Hove, UK:

Routledge. https://doi.org/10.4324/9780203527160

Read, J., Kirsch, I., & McGrath, L. (2019). Electroconvulsive Therapy for depression: A Review of the quality of ECT vs sham ECT trials and meta-analyses. *Ethical Human Psychiatry and Psychology, 21(2),* 64-103. doi:10.1891/EHPP-D-19-00014

Read, J., & Sanders, P. (2010). *A straight talking introduction to the causes of mental health problems.* Ross-on-Wye, UK: PCCS Books

Read, J., & Williams, J. (2018). Adverse effects of antidepressants reported by a large international cohort: Emotional blunting, suicidality, and withdrawal effects. *Current Drug Safety, 13*(3), 176-186. doi:10.2174/1574886313666180605095130

Reangsing, C., Rittiwong, T., & Schneider, J. K. (2021). Effects of mindfulness meditation interventions on depression in older adults: A meta-analysis. *Aging Mental Health, 25*(7), 1181-1190. doi:10.1080/13607863.2020.1793901

Ronnqvist, I., Nilsson, F., & Nordenskjold, A. (2021). Electroconvulsive therapy and the risk of suicide with major depressive disorder. *JAMA Network Open, 4*(7), e2116589. doi:10.1001/jamanetworkopen.2021.16589.

Rush, A., Trivedi, M., Wisniewski, S., Nierenberg, A., Stewart, J., Warden, D., . . . Fava, M. (2006). Acute and longer-term outcomes in depressed outpatients requiring one or several treatment steps: A STAR*D Report. *American Journal of Psychiatry, 163*(11), 1905-1917. Retrieved from PM:17074942

Rutherford, B., Sneed, J., & Roose, S. (2009). Does study design influence outcome?. The effects of placebo control and treatment duration in antidepressant trials. *Psychotherapy and Psychosomatics, 78*(3), 172-181. doi:10.1159/000209348

Sackeim, H., Prudic, J., Fuller, R., Keilp, J., Lavori, P., & Olfson, M. (2007). The cognitive effects of ECT in community settings. *Neuropsychopharmocology, 32*(1), 244-254. doi:10.1038/sj.npp.1301180.

Schuch, F. B., Vancampfort, D., Richards, J., Rosenbaum, S., Ward, P. B., & Stubbs, B. (2016). Exercise as a treatment for depression: A meta-analysis adjusting for publication bias. *Journal of Psychiatric Research, 77,* 42-51. doi:10.1016/j.jpsychires.2016.02.023

Simonsen, A., Danborg, P., & Gotzsche, P. (2016). Persistent sexual dysfunction after early exposure to SSRIs: Systematic review of animal studies. *International Journal of Risk & Safety in Medicine, 28*(1), 1-12. doi:10.3233/JRS-160668

Somatics. (2018). *Regulatory update to Thymatron system IV instruction manual*. Retrieved from: http://www.thymatron.com/downloads/System_IV_Regulatory_Update.pdf

Speed, E., Moncrieff, J., & Rapley, M. (Eds). (2014). *De-medicalising misery II*. Basingstoke, UK: Palgrave Macmillan.

Sugarman, M., Loree, A., Baltes, B., Grekin, E., & Kirsch, I. (2014). The efficacy of paroxetine and placebo in treating anxiety and depression: a meta-analysis of change on the Hamilton Rating Scales. *PLoS.One. 9*(8), e106337. doi:10.1371/journal.pone.0106337

Taylor, S., Annand, F., Burkinshaw, P., Greaves, F., Kelleher, M., Knight, J., . . . Marsden, J. (2019). *Dependence and withdrawal associated with some prescribed medicines: an evidence review.* Public Health England. Retrieved from: https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/940255/PHE_PMR_report_Dec2020.pdf

Tsai, J., Peltzman, T., Watts, B., & Shiner, B. (2021). Effects of Electroconvulsive Therapy on suicidal behavior and emergency department use among homeless veterans: a propensity score-matched study. *Journal of Clinical Psychiatry, 82(6),* 21m13935. doi:10.4088/JCP.21m13935.

Turner, E., Matthews, A., Linardatos, E., Tell, R., & Rosenthal, R. (2008). Selective publication of antidepressant trials and its influence on apparent efficacy. *New England Journal of Medicine, 358*(3), 252-260. doi:10.1056/NEJMsa065779

Valenstein, E. (1988). *Blaming the brain.* New York: Free Press.

Viola, S., & Moncrieff, J. (2016). Claims for sickness and disability benefits owing to mental disorders in the UK: trends from 1995 to 2014. *BJPsych Open, 2*(1), 18-24. doi:10.1192/bjpo.bp.115.002246

Vlessides, M. (2020). Experts call for immediate suspension of ECT; Others push back. *Medscape,* July 24. Retrieved from: https://www.medscape.com/viewarticle/934536

Wampold, B., Minami, T., Baskin, T., & Callen Tierney, S. (2002). A meta-(re)analysis of the effects of cognitive therapy versus 'other therapies' for depression. *Journal of Affective Disorders, 68*(2-3), 159-165. doi:10.1016/s0165-0327(00)00287-1

World Health Organisation (2021). *Guidance on community mental health services: Promoting person-centred and rights-based approaches.* https://www.who.int/publications/i/item/9789240025707

Wei, Q., Bai, T., Chen, Y., Ji, G., Hu, X., Xie, W., . . . Tian, Y. (2018). The changes of functional connectivity strength in Electroconvulsive Therapy for depression: A longitudinal study. *Frontiers in Neuroscience, 12,* 661. doi:10.3389/fnins.2018.00661

Zahodne, L., Bernal-Pacheco, O., Bowers, D., Ward, H., Oyama, G., Limotai, N., . . . Okun, M. (2012). Are selective serotonin reuptake inhibitors associated with greater apathy in

Parkinson's disease? *Journal of Neuropsychiatry and Clinical Neurosciences, 24*(3), 326-330. doi:10.1176/appi.neuropsych.11090210

Table 1. Alternative models of drug action (adapted from Moncrieff, 2009; Moncrieff & Cohen, 2005)

Disease centred model	**Drug centred model**
Drugs help correct an abnormal brain state	Drugs create an altered brain state
Drugs as disease treatments	Psychiatric drugs as *psychoactive drugs*
Therapeutic effects derive from drugs' effects on underlying biological mechanisms that produce symptoms	Therapeutic effects derive from the impact of the drug-induced state on behavioural and emotional problems
Paradigm: insulin for diabetes	Paradigm: alcohol for social anxiety

Table 2. The six defenses against the continuing absence of any evidence of efficacy from adequate randomized, placebo-controlled studies (RCTs) of ECT

1	ECT has been used for a long time so we know it works
2	It's unfair to critique the pre-1986 RCTs using today's scientific standards
3	It's unethical to conduct RCTs that involve withholding a treatment from very ill people
4	RCTs aren't necessary; non-placebo studies are sufficient
5	ECT *is* effective long-term, if you use antidepressants after ECT ends
6	Denigrate the people raising the issue, or scientific/media outlets publishing their critiques