

DEPRESSION: WHY DRUGS AND ELECTRICITY ARE NOT THE ANSWER

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Abstract

The dominant ideology within mental health services and research, sometimes simplistically described as ‘the medical model’, tends to argue that feeling depressed is a kind of medical illness caused by various forms of biological deficits which are somehow corrected by psychiatric interventions such as drugs and electroconvulsive therapy. This article discusses the evidence for these claims and concludes that it is, at best, very weak. We argue that this flawed understanding of the causes of human distress and misery has led to equally flawed, and sometimes dangerous, interventions. An alternative understanding recognises depression as an emotional response to unwanted circumstances that requires human support and understanding, rather than technical, physical manipulations. It also demands that we address the social conditions that make depression likely. Such alternative approaches, increasingly endorsed around the world, including by the United Nations, the World Health Organisation and service users, are briefly described.

Introduction

Before discussing biological psychiatry’s two major attempts to help people when they feel depressed, 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, we are

asked to believe, are the causes of a ‘mental illness’ or ‘psychiatric condition’ with a set of symptoms that is referred to by a medical sounding name, like ‘Major Depressive Disorder’. We shall show that no biological dysfunction that can be corrected by current treatments has been demonstrated.

Even when the biological dysfunction is not referred to directly, it is assumed that depression is a condition that somehow *causes* 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 influential American Psychiatric Association (2021a) unequivocally 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 also advised:

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

Although we will focus primarily 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 deeply flawed from the outset. Viewing depression as a medical disorder that originates in the brain and responds to brain-based interventions is fundamentally inconsistent with understanding it as a human emotion, a meaningful reaction to troubling circumstances (Moncrieff, 2020). It cannot begin to address the issues underlying women being about twice as likely as men to be administered either ‘treatment’, for example. Any approach that fails to acknowledge the meaning of depression, and address the common social causes of misery and sadness (Cromby, Harper, & Reavey, 2013; Johnstone et al., 2018; Read & Sanders, 2010), is doomed to failure.

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. Evidence from hundreds of placebo-controlled trials suggests that antidepressants are marginally better than placebo at reducing depressive symptoms as measured by common 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 (Moncrieff & Kirsch, 2015; Leucht et al., 2013). Indeed, 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 even 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. Although most drugs are assumed to work according to a ‘disease-centred’ model of drug action, which assumes that they act on underlying biological abnormalities, an alternative, ‘drug-centred’ model suggests that drugs change mental states and behaviour through the modification of normal physiological processes (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 leading to amplified placebo effects among those taking active drugs (Moncrieff & Cohen, 2005). Following this theory, it appears that almost any drug with psychoactive properties has been found to have equivalent

effects to antidepressants in depression in one study or another, including benzodiazepines, stimulants, opiates, buspirone and antipsychotics (Moncrieff, 2008).

TABLE ONE ABOUT HERE

The alterations themselves may also reduce depressed feelings or other so-called symptoms of depression. SSRIs and some other antidepressants appear to 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 *and anxiety* rating scales. The fact that differences from placebo are so small suggests these effects are not particularly useful, however.

Other artefacts of research 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 then withdrawn, may also account for the differences between drugs and placebos in randomised trials. It has been found, for example, that a difference between antidepressants and placebo only occurs in those who have formally been treated with antidepressants and not in those who have no prior exposure (Hunter et al., 2015). This may be because those who have received treatment previously are more likely to be unblinded because they are familiar with the effects of medication, or it may be that those who are allocated to placebo are precipitated into a state of antidepressant withdrawal by the removal of their prior medication, and that this state masquerades as symptoms of depression.

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. Only 108 participants out of a total of over 4000 recovered, remained well and completed the study (Pigott, Leventhal, Alter, & Boren, 2010). For 14 years no data was published on the primary outcome, the change on the Hamilton rating scale scores after 12 weeks., When an independent group of researchers eventually obtained and published 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 responses.

Do antidepressants correct an underlying biological abnormality?

Despite widespread 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 depression and people without have been found, these are likely to be explained by prior use of antidepressants and other medications, but in most areas no 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 by 2018 (Taylor et al., 2019), and 145 of US adults by 2015 (Olfson et al., 2019). Yet the number of people seeking help and going onto long-term disability due to depression is increasing (Viola & Moncrieff, 2016; Olfson et al., 2019).

Antidepressants alter normal mental activity and behaviour

As well as 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 available. We do not know the full implications and long-term consequences of taking these drugs, therefore. 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 their well-recognised impairment of sexual function (Goldsmith & Moncrieff, 2011; Read & Williams, 2018; Padala et al., 2020; 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 sexual dysfunction persists after they stop the drug (Bala, Nguyen, & Hellstrom, 2018). This is consistent with research with young animals that finds 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 numbers, i.e. hundreds of millions, 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 started to receive serious attention in recent years. Recent evidence suggests that around 56% of people experience withdrawal effects after discontinuing antidepressants, and for 46% of those the effects are severe and impairing (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 persist for months or years after the drugs have finally been stopped (Hengartner et al., 2020; Framer, 2021).

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 (Maund et al., 2019; Eveleigh, Speckens, van Weel, Oude Voshaar, & Lucassen, 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 drugs that supposedly treat depression, the various biological deficits that are supposedly corrected by ECT have never been demonstrated. The first two bio-medical claims about how ECT works, and what it was supposedly correcting, are interesting.

The first was that there was a ‘biological antagonism’ between schizophrenia and epilepsy (Fink & Sackeim, 1996). If you had one you couldn’t have the other. While some doctors treated epilepsy with injections of the blood of ‘schizophrenics’ (Kalinowsky, 1986), others were using a range of approaches, 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 or simplifying thought processes. In 1941, Walter Freeman, who exported ECT from Europe to the United States, wrote:

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).

The paper was entitled ‘Brain Damaging Therapeutics’. 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 its use in depression, at least until the 1960s. Authors of 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).

Although intentionally damaging brains is now considered unethical, a similar line of argument was resurrected 70 years later by researchers who reported that ECT reduces the ‘functional connectivity’ of the brain. A neutral observer might assume the researchers would be concerned by this finding. Instead, they celebrated having finally discovered how ECT works, by correcting a supposed ‘hyperconnectivity’ that somehow causes depression (Perrin et al., 2012). Other ECT advocates, meanwhile claim to have found the opposite; that ECT works because it *increases* ‘functional connectivity’ (Wei et al., 2018).

Many ECT proponents acknowledge that we don’t know what brain changes ECT causes that lead to the temporary lift in mood that some people experience, or what biological deficits are being corrected (sometimes arguing this doesn’t matter because the same is true of some treatments in general medicine). A recent audit of 36 ECT information leaflets for patients in England found that 22% acknowledged that it is not known how ECT works.

Nevertheless, 78% claimed it corrects some kind of deficit in the brain (Harrop, Read, Geekie, & Renton, 2021).

The idea of ‘brain damaging therapeutics,’ however, receives some support from contemporary evidence. A recent review, with the advantage of neuroimaging not available in Freeman’s time, 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)

So, as is the case for so-called ‘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 (Read & Bentall, 2010; Rasmussen, 2009). 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 the absence of any 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 the placebo response to ECT concluded that ‘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 ever been 11 randomized placebo-controlled studies (RCTs) for its primary target diagnosis, depression, conducted between 1956 and 1985 (Read & Bentall, 2010). A recent review, involving one of the current authors (JR) and Professor Irving Kirsch, Associate Director of Placebo Studies at Harvard Medical School, highlighted the poor quality of the 11 studies (Read, Kirsch, & McGrath, 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.

In terms of results:

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 relatively high quality studies reported follow up data. One produced a near-zero effect size (.065) in the direction of ECT (Johnstone et al., 1980), and the other a small effect size (.299) in favour of sham treatment (Lambourn & Gill, 1978).

Nevertheless, all five meta-analyses that depend on these flawed studies still conclude that ECT is effective. They paid little or no attention to the methodological shortcomings of the studies, failed to comment on the high response to sham ECT in most of the studies, and did not identify any evidence on long-term effects (Read et al., 2019). The Food and Drug

Administration (2020) in the US mandates that every ECT machine has a sign next to it stating: ‘The long-term safety and effectiveness of ECT treatment has not been demonstrated.’

The meta-analyses also failed to identify a single study finding that ECT prevents suicide, as often claimed. Numerous studies have found that ECT recipients are more likely than other patients to kill themselves (Read, Bentall, Johnstone, Fosse, & Bracken, 2013). For example, 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 other ‘mental health patients’ (Peltzman, Shiner, & Watts, 2020). Such findings may be confounded by the fact that people receiving ECT have severe depression, but Peltzman and colleagues found that after controlling for ‘demographic, clinical, and service use characteristics,’ including psychiatric diagnoses and inpatient admissions, members of the ECT group were still 1.3 times more likely to have killed themselves.

In the only recent meta-analyses, 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’ regarding efficacy (which involves comparisons of a range of different types of treatments). Thus, after more than 80 years, only placebo study was considered robust enough to meet the Institute’s inclusion criteria. However, 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’, even though it was 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. (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.

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. Why else would psychiatrists keep using it? Unfortunately, the history of psychiatry is littered with treatments considered by 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 and begs the question: why have none been conducted since 1985?

The third defense, and an attempt to answer the above question, is that it would be unethical to conduct studies that involve withholding a treatment which we know works from severely depressed and acutely 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, perhaps unintentionally, that 39 colleagues, the authors of the 11 RCTs, who had at least tried to engage in the scientific process, were unethical).

TABLE TWO ABOUT HERE

The fourth is to argue that RCTs aren't necessary, and that other types of studies can be relied upon, such as comparisons with antidepressants or between different types of ECT, as well as clinical impressions. The sham 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. As we have seen above, however, it is doubtful that antidepressants have useful effects in depression. Moreover, 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 the device of shooting the messenger. Researchers and ECT recipients who question the efficacy and highlight the adverse effects of ECT (summarised below), are often publicly denigrated, by psychiatrists who advocate for ECT, 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, potentially enhanced by post-treatment amnesia and cognitive impairment, would not matter so much if it weren’t for the fact that passing sufficient electricity through the brain to cause a seizure has effects on the brain. These effects are, as is the case with antidepressants, sometimes acknowledged as existing but then presented not as damage but as a beneficial correction of an imagined deficit (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, that is well recognised, between 12% (Sackeim et al., 2007) and 55% (Rose et al., 2003) of ECT recipients suffer persistent or permanent memory loss (Read & Bentall, 2010; Read et al., 2019). The American Psychiatric Association (2001) has acknowledged 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 some people, this can be devastating:

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)

ECT can also cause adverse psychological and emotional effects (Johnstone, 2009). It also carries a small risk of mortality and cardiac complications, the leading cause of ECT-related

deaths. A recent review of 82 studies, including more than 100,000 patients, found that 1 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. Only 72% acknowledged 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 an independent review (Johnstone & Cunliffe, 2020; Read, 2020) has broad support, including from 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 virtue of the way they modify 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 have been 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. Any procedure that changes 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 some people who undergo ECT or take long-term antidepressants. On top of this, believing that you have a brain disease requiring medical intervention can be profoundly disempowering, is likely to decrease self-confidence, can increase feelings of vulnerability and passivity and discourage people from taking active measures that might help improve their mental state and quality of life. Therefore, we need much more information on long-term consequences before we deem it safe to continue prescribing these techniques.

So, if antidepressants and ECT are not helpful and potentially unsafe, how should we help people who feel depressed or distressed? First, understanding depression and anxiety as emotional reactions to life circumstances, rather than the manifestations of brain pathology demands that we address the social conditions that provoke them. To lead an emotionally balanced and fulfilling life, largely free of major worry and distress, people need a dependable income, secure and rewarding employment, engaging social activities, and the opportunity to form close and supportive relationships, relatively unburdened by personal and social insecurities. Exercise, healthy eating and anything else that promotes good physical health are likely to be helpful. Psychological therapy may be useful in some circumstances, although we should remember that it is the quality of the therapeutic relationship, rather than specific therapeutic techniques, that is helpful (Wampold, Minami, Baskin, & Callen Tierney, 2002). 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 most of these suggestions. Helping someone in distress is not primarily a scientific activity - it is an essentially human one. We learn how to support our fellow humans through life experience, through being cared for ourselves, and sometimes through art and literature, following a 'tradition of looking for the origins of suffering in the conditions of existence, and of understanding the sighs of despair and the ravings of madness as a commentary on the failings of the society that fostered them' (Speed, Moncrieff, & Rapley, 2014). Although lip service is paid to the social precipitants of depression, classifying anxiety, depression and other emotional reactions as mental diseases or disorders obscures the relation between our moods and our circumstances. It encourages people to view themselves as the victims of their biology, and society to believe that current 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

international examples of alternatives to drugs and electricity for helping people with mental health problems (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 calling 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 before he started them, 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 points out that:

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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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 <i>psychoactive drugs</i>
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 <i>is</i> effective long-term, if you use antidepressants after ECT ends
6	Denigrate the people raising the issue, or scientific/media outlets publishing their critiques