thought-out negative claims by one of its own very vigorously.

*David J Nutt, Guy M Goodwin, Dinesh Bhugra, Seena Fazel, Stephen Lawrie
Imperial College London, London, UK (DJN); University of Oxford, Oxford, UK (GMG, SF); Kings College London, London, UK (DB); and University of Edinburgh, Edinburgh, UK (SL)
d.nutt@imperial.ac.uk

DJN has received grants and personal fees from Lundbeck and GSK; and personal fees from Lilly, BMS, Otsuka, Servier, and Pfizer. GMG has received grants and personal fees from Servier and Lundbeck; personal fees from Teva, Otsuka, Takeda, Eli Lilly, Merck, GSK, and AstraZeneca; and grants from P1vital. DJN and GMG have a small number of stocks in P1vital, a CNS experimental medicine research consultancy company. SL has received research funding from Abbvie, Roche, and Pfizer in connection with genetic, brain imaging, and therapeutic studies of people with schizophrenia. He has also been paid by Janssen and Roche to speak at or chair educational meetings about schizophrenia, as well as to contribute to advisory boards about new antipsychotic treatments. The other authors declare no competing interests.

1 Wittchen HU, Jacobi F, Rehm J, et al. The size and burden of mental disorders and other disorders of the brain in Europe 2010. *Eur Neuropsychopharmacol* 2011; **21:** 655–79.
2 Mathers CD, Loncar D. Projections of global mortality and burden of disease from 2002 to 2030. *PLoS Med* 2006; **3:** e442.
3 Anderson IM, Ferrier IN, Baldwin RC, et al. Evidence-based guidelines for treating depressive disorders with antidepressants: a revision of the 2000 British Association for Psychopharmacology guidelines. *J Psychopharmacol* 2008; **22:** 343–96.
4 Leucht C, Huhn M, Leucht S. Amitriptyline versus placebo for major depressive disorder. *Cochrane Database Syst Rev* 2012; **12:** CD009138.
5 Leucht, S Hierl S, Kissling W, Dold M, Davis JM. Putting the efficacy of psychiatric and general medicine medication into perspective: review of meta-analyses. *Br J Psychiatry* 2012; **200:** 97–106.
6 Geddes J, Carney S, Davies C, et al. Relapse prevention with antidepressant drug treatment in depressive disorders: a systematic review. *Lancet* 2003; **361:** 653–61.
7 Office for National Statistics. Suicide rates in the United Kingdom, 2012 Registrations. http://www.ons.gov.uk/ons/rel/subnational-health4/suicides-in-the-united-kingdom/2012/stb-uk-suicides-2012.html (accessed May 20, 2014).
8 Fazel S, Grann M, Ahlner J, Goodwin G. Suicides by violent means in individuals taking SSRIs and other antidepressants: a post-mortem study in Sweden, 1992–2004. *J Clin Psychopharmacol* 2007; **27:** 503–6.
9 Isacsson G, Holmgren A, Osby U, Ahlner J. Decrease in suicides among the individuals treated with antidepressants: a controlled study of antidepressants in suicide in Sweden 1995–2005. *Acta Psychiatr Scand* 2009; **120:** 37–44.
10 Nutt DJ, Sharpe M. Uncritical positive regard? Issues in the safety and efficacy of psychotherapy. *J Psychopharmacol* 2008; **22:** 3–6.
11 Bridge J A, Barbe R P, Birmaher B, et al. Emergent suicidality in a clinical psychotherapy trial for adolescent depression. *Am J Psychiatry* 2005; **162:** 2173–75.
12 Stone A. Suicide precipitated by psychotherapy. *Am J Psychotherapy* 1971; **25:** 18–28.
13 Cooney GM, Dwan K, Greig CA, Lawlor DA, Rimer J, Waugh FR, McMurdo M, Mead GE. Exercise for depression. *Cochrane Database Syst Rev* 2013; **9:** CD004366.

Why I think antidepressants cause more harm than good

In *The Lancet Psychiatry*, David Nutt and colleagues[1] stated that headlines such as "Antidepressants do more harm than good" plumb a "new nadir in irrational polemic." I disagree and describe here the evidence that supports my argument so that readers can judge for themselves what they think about the defence of these drugs by Nutt and colleagues.

With regard to the benefits of antidepressants, in its large meta-analysis of 100 000 patients, half of whom were depressed, the US Food and Drug Administration (FDA) noted that 10% more patients responded on antidepressants than did those on placebo,[2] and the Cochrane review of depressed patients reported similar results[3] (ie, one patient might benefit for every ten patients treated).

I believe those results were exaggerated, however, for several reasons.[4] Most importantly, the trials were not effectively blinded. Antidepressants have conspicuous side-effects and many patients and their doctors will therefore know whether the blinded drug is active or placebo. A systematic review of 21 trials[5] in a variety of diseases that had both masked and non-masked outcome assessors, and which had mostly used subjective outcomes, found that the treatment effect was exaggerated by 36% on average (measured as odds ratio) when non-masked observers rather than masked ones assessed the effect. The effect of antidepressants is assessed on highly subjective scales (eg, the Hamilton scale), and if we assume that the blinding is broken for all patients in the trials and adjust for the bias, we will find that antidepressants have no effect (odds ratio 1·02).[4]

However, I do not believe that the blinding is always broken, only that the reported effect is highly likely to have been exaggerated. Many years ago, adequately blinded trials of tricyclic antidepressants were done, in which the placebo contained atropine, which causes dryness in the mouth like the active drugs do. These trials reported very small, clinically insignificant effects of tricyclic antidepressants compared with placebo (standardised mean difference 0·17, 95% CI 0·00–0·34).[6]

Another worrying finding in randomised trials is that as many patients stop treatment on SSRIs as on placebo for any reason.[7] After only 2 months, half the patients have stopped taking the drug.[8] This finding suggests that, overall, considering benefits and harms together, the patients find the drugs useless. More importantly, no research shows whether these drugs work for the outcomes that really matter, such as saving relationships and getting people back to work.

With respect to the harms of antidepressants, most patients who take these drugs will experience side-effects. The package inserts list many common side-effects, of which one of the most frequent is sexual problems. In a study[9] designed to assess this side-effect, sexual problems developed in 604 (59%) of 1022 patients who all reported no problems with sexual function before they started using an antidepressant. The symptoms include decreased libido (50% of patients on fluoxetine), delayed orgasm or ejaculation (also 50%), no orgasm or ejaculation (39%), and erectile dysfunction or decreased vaginal lubrication (22% for both combined).

Even when tapering off them slowly, half the patients have difficulty stopping the drugs because of withdrawal effects, which can be severe[10] and long-lasting.[4] We noted that withdrawal symptoms were described in similar terms for benzodiazepines and SSRIs and were very similar for 37 of 42 identified symptoms.[11] However, they were not described as dependence for SSRIs.[11] To define similar problems as "dependence" in the case of benzodiazepines and as "withdrawal reactions" in the case of SSRIs is irrational. For patients, the symptoms are just the same; it can be very hard for them to stop either type of drug.

Psychiatrists often argue, as did Nutt and colleagues,[1] that antidepressants protect against suicide. However, I believe that no good evidence in support of this idea exists. Good observational studies have refuted it,[12] and results from randomised trials[13] have shown that antidepressants are associated with increased risk of suicide attempts (5·6 more suicide attempts per 1000 patient-years of SSRI exposure compared with placebo). Antidepressants have not only been associated with suicide but also with homicide.[4,14-16] The FDA's analysis[2] showed that suicidal behaviour is increased with antidepressants until about the age of 40 years—but in fact, the situation is much worse than this. Suicides and suicide attempts were vastly underreported in the FDA's analysis for various reasons.[4] For example, only five deaths by suicide were recorded in 52 960 patients on antidepressants in the 2006 FDA analysis[2] whereas five deaths by suicide were recorded in 2963 patients on paroxetine alone in a meta-analysis from 1993.[17]

SSRIs are particularly harmful for elderly patients. Results from a carefully controlled cohort study[18] of people older than 65 years of age with depression showed that SSRIs led to falls more often than did older antidepressants or if the depression was left untreated. For every 28 elderly people treated for 1 year with an SSRI, there was one additional death, compared with no treatment.[18] SSRIs have also stimulant effects and might precipitate conversion to bipolar disorder in about 10% of children aged 10–14 years under the care of mental health services .[19]

SSRIs are very poor drugs and I doubt they are safe at any age. The first SSRI was fluoxetine, which the German drug regulator deemed "totally unsuitable for the treatment of depression".[14,20] I, and others,[4,21] have written about the controversy surrounding this drug and the process by which it nevertheless came to be approved and widely used.

I have written previously[4] that there has been heavy marketing and widespread crime committed by drug companies, including fraud, illegal promotion, and corruption of psychiatrists. In the USA, psychiatrists receive more money from the drug industry than any other specialty.[4,22] As a result, enough antidepressants are prescribed every year in Denmark to provide treatment for every person in the country for 6 years of their lives.[4] I believe this situation is not sound and that it also partly portrays the fact that many patients cannot stop these drugs because of intolerable withdrawal symptoms.

SSRIs have been shown to have minimal or non-existent benefit in patients with mild or moderate depression[23] and I think they might not even work for severe depression.[4] They should be used very sparingly, if at all, and always with a clear plan for tapering off them. The so-called maintenance studies, in which patients after successful treatment get randomly assigned to continue with the drug or a placebo, cannot be interpreted as showing that the patients still need the drug because withdrawal symptoms, which can include depression, are inflicted on the placebo group.

Nutt and two of his co-authors, Guy M Goodwin and Stephen Lawrie, have between them declared 22 conflicts of interest in relation to drug companies.[1] I wonder whether this declaration explains their dismissal of psychotherapy (although it is effective and recommended by NICE) and their description of my evidence-based views as a somewhat irrational polemic that is insulting to the discipline of psychiatry and is reinforcing stigma against mental illnesses. They also talk about anti-psychiatry, anti-capitalism, and a conspiracy theory. This is the language of people who are short of arguments.

Peter C Gøtzsche
Nordic Cochrane Centre, Dept 7811 Rigshospitalet, Copenhagen, Denmark
pcg@cochrane.dk

I declare no competing interests.

1 Nutt DJ, Goodwin GM, Bhugra D, Fazel S, Lawrie S. Attacks on antidepressants: signs of deep-seated stigma? *Lancet Psychiatry* 2014 ; **1:** 103–04.
2 Laughren TP. Overview for December 13 Meeting of psychopharmacologic drugs advisory committee (PDAC). 2006 Nov 16. http://www.fda.gov/ohrms/dockets/ac/06/briefing/2006-4272b1-01-FDA.pdf (accessed Oct 22, 2012).
3 Arroll B, Elley CR, Fishman T, et al. Antidepressants versus placebo for depression in primary care. *Cochrane Database Syst Rev* 2009; 3: CD007954.
4 Gøtzsche PC. Deadly medicines and organised crime: how big pharma has corrupted health care. London: Radcliffe Publishing, 2013.
5 Hróbjartsson A, Thomsen AS, Emanuelsson F, et al. Observer bias in randomised clinical trials with binary outcomes: systematic review of trials with both blinded and non-blinded outcome assessors. *BMJ* 2012; **344:** e1119.
6 Moncrieff J, Wessely S, Hardy R. Active placebos versus antidepressants for depression. *Cochrane Database Syst Rev* 2004; **1:** CD003012.
7 Barbui C, Furukawa TA, Cipriani A. Effectiveness of paroxetine in the treatment of acute major depression in adults: a systematic re-examination of published and unpublished data from randomized trials. *CMAJ* 2008; **178:** 296–305.
8 Serna MC, Cruz I, Real J, et al. Duration and adherence of antidepressant treatment (2003 to 2007) based on prescription database. *Eur Psychiatry* 2010; **25:** 206–13.
9 Montejo A, Llorca G, Izquierdo J, et al. Incidence of sexual dysfunction associated with antidepressant agents: a prospective multicenter study of 1022 outpatients. Spanish Working Group for the study of psychotropic-related sexual dysfunction. *J Clin Psychiatry* 2001; **62** (suppl 3)**:** 10–21.
10 Fava GA, Bernardi M, Tomba E, et al. Effects of gradual discontinuation of selective serotonin reuptake inhibitors in panic disorder with agoraphobia. *Int J Neuropsychopharmacol* 2007; **10:** 835–38.
11 Nielsen M, Hansen EH, Gøtzsche PC. What is the difference between dependence and withdrawal reactions? A comparison of benzodiazepines and selective serotonin re-uptake inhibitors. *Addiction* 2012; **107:** 900–08.
12 Zahl PH, De Leo D, Ekeberg Ø, et al. The relationship between sales of SSRI, TCA and suicide rates in the Nordic countries. *BMC Psychiatry* 2010; **10:** 62.
13 Fergusson D, Doucette S, Glass KC, et al. Association between suicide attempts and selective serotonin reuptake inhibitors: systematic review of randomised controlled trials. *BMJ* 2005; **330:** 396.
14 Healy D. Let Them Eat Prozac. New York: New York University Press; 2004.
15 Moore TJ, Glenmullen J, Furberg CD. Prescription drugs associated with reports of violence towards others. *PLoS One* 2010; **5:** e15337.
16 Lucire Y, Crotty C. Antidepressant-induced akathisia-related homicides associated with diminishing mutations in metabolizing genes of the CYP450 family. *Pharmgenomics Pers Med* 2011; **4:** 65–81.
17 Montgomery SA, Dunner DL, Dunbar GC. Reduction of suicidal thoughts with paroxetine in comparison with reference antidepressants and placebo. *Eur Neuropsychopharmacol* 1995; **5:** 5–13.
18 Coupland C, Dhiman P, Morriss R, et al. Antidepressant use and risk of adverse outcomes in older people: population based cohort study. *BMJ* 2011; **343:** d4551.
19 Martin A, Young C, Leckman JF, et al. Age effects on antidepressant-induced manic conversion. *Arch Pediatr Adolesc Med* 2004; **158:** 773–80.
20 Internal Eli Lilly memo. Bad Homburg. 1984 May 25 (available on request).
21 Virapen J. Side effects: death. College Station: Virtualbookworm.com Publishing, 2010.
22 Insel TR. Psychiatrists' relationships with pharmaceutical companies: part of the problem or part of the solution? *JAMA* 2010; **303:** 1192–93.
23 Fournier JC, DeRubeis RJ, Hollon SD, et al. Antidepressant drug effects and depression severity: a patient-level meta-analysis. *JAMA* 2010; **303:** 47–53.

Mental health services provision in Somaliland

Somaliland in northern Somalia has one of the highest prevalences of mental illness worldwide, with two out of every five people estimated to be living with severe mental health disorders.[1] People with mental disorders are highly marginalised in the Somali community and are often isolated at home with chains, abused, and sometimes even kept in prison for many years. Very few have access to modern treatment because of stigma, an insufficient number of people trained in mental health care, and the huge shortage of health-care providers[2] following two decades of civil war in Somalia. Abuse of the stimulant khat is prevalent, which exacerbates existing mental illness.[3] Training in psychiatry was first introduced to the medical curriculum in Somaliland universities in 2007[4] through a King's–THET Somaliland Mental Health Group programme linking Somaliland and the King's College London Global Health Center (London, UK)[5] in mental health service provision and training. Through this programme, several junior doctors[5] were recruited who are now involved in teaching and in the provision of limited mental health services in hospitals and in the community.[6] More recently, the Somali diaspora have become involved in building acute mental health clinics in some cities in Somaliland and providing financial support to existing ones.

Here, we describe a pilot project introducing a community-based mental health service in Borama, Somaliland. This community has 20 000 inhabitants living in poor conditions, mostly people displaced