

The Rights of Children and Parents In Regard to Children Receiving Psychiatric Diagnoses and Drugs

Peter R. Breggin

Center for the Study of Empathic Therapy, Private Practice of Psychiatry, Ithaca, New York, USA

Based on the author's extensive clinical, forensic and research experience, this article addresses the scientific and moral question of whether it is ever in the best interests of a child to be given a psychiatric drug. The focus is on the diagnosis Attention Deficit Hyperactivity Disorder (ADHD) and stimulant drugs, and on the diagnosis Bipolar Disorder and antipsychotic (neuroleptic) drugs. The conclusion is that we should work towards a prohibition against giving psychiatric drugs to children, and instead focus on safe and effective alternative ways of meeting the needs of children within their families, schools and society. © 2014 John Wiley & Sons Ltd and National Children's Bureau.

Keywords: children, mental health, rights.

Introduction: setting standards for the protection of children

This article deals with negative rights — that is, the right to be free of certain kinds of interferences in one's life. In regard to children, these rights are protected by society, often regardless of parental wishes, such as legal prohibitions against physical or sexual abuse. In making judgments about children, this analysis will, like in the USA courts, rely upon the standard of the child's best interests (Child Welfare Information Gateway, 2012; also see Gottstein, 2012), including the 'the physical, mental, emotional and moral well-being' of the child (FindLaw, undated: 1). However, I will argue that when it comes to the psychiatric drugging of children, which, I maintain, can be seen as a form of child abuse, this standard cannot be relied upon to protect children. Using the examples of stimulant drugs for Attention Deficit Hyperactivity Disorder (ADHD) and antipsychotic drugs for Bipolar Disorder, I ask, 'Is it ever in a child's best interest to be psychiatrically diagnosed and medicated?'

Effects of the ADHD diagnosis and stimulant drugs

ADHD is not a valid diagnostic category that meets the criteria for a medical syndrome (Baughman and Hovey, 2006; Breggin, 2008a; Whitely, 2010). Like all other psychiatric disorders, there is no evidence that it has a biological cause (Moncrieff, 2007a). With regard to the three ADHD behavioural categories of hyperactivity, impulsivity and inattention, sometimes these behaviours may be part of typical childhood behaviours. Other times, they may result from boring and poorly disciplined classrooms, lack of grade level educational skills, emotional problems generated from problems at home or in school, issues relating to poverty such as hunger or poor nutrition, or insomnia and fatigue and a variety of chronic illnesses, including diabetes and head injury (e.g. sports concussions) (Breggin and Breggin, 1998). In my clinical practice, all these causes have been evident.

Stimulants are the most commonly prescribed drugs for ADHD. Most are either amphetamines (e.g. Adderall or Dexedrine) or methylphenidate (e.g. Ritalin or Concerta). Atomoxetine has been promoted by manufacturer Eli Lilly & Co. as a 'non-stimulant' treatment for ADHD,

but it has been shown to cause dangerously stimulating symptoms in one-third of children (Henderson and Hartman, 2004) and carries a Black Box Warning about causing suicidality in children (Strattera, 2011). Black box warnings are labels placed on pharmaceuticals in the USA, required by the Food and Drug Administration, when there is sufficient scientific evidence for causality with regard to serious adverse or life threatening effects.

Amphetamine and methylphenidate belong to Schedule II of the Drug Enforcement Agency's (DEA) controlled substances list, which is the highest risk of addiction and abuse. A Black Box Warning at the top of the Adderall label states, 'Amphetamines have a high potential for abuse. Administration of amphetamines for prolonged periods of time may lead to drug dependence' (Adderall, 2011). Lambert (2005) conducted a 28-year prospective study of children-diagnosed ADHD. She found that children treated with methylphenidate were much more likely to abuse cocaine in young adulthood compared to those diagnosed with ADHD without drug exposure. This is not surprising, as stimulants are known to cause physical alterations in the reward centres of the brain (Carlezon and Konradi, 2004).

Amphetamine and methylphenidate produce persistent biochemical abnormalities in the brain (Breggin, 2008a). Children treated with stimulants often develop atrophy of the brain. At the NIH Consensus Development Conference on ADHD, Swanson (Swanson and Castellanos, 1998) reviewed available studies purporting to show biological bases for ADHD including brain atrophy (e.g. Castellanos and others, 1996; Giedd and others, 1994). My presentation at the same conference concluded that these brain scans were 'almost certainly measuring pathology caused by psychostimulants' (Breggin, 1998: 109). Proal and others (2011) found widespread brain atrophy in grown adults who had been diagnosed and treated for ADHD as children. Furthermore, there is evidence that these stimulants lead to growth suppression in children. A large-scaled federally funded study (the MTA) involving multiple centres reconfirmed that stimulants suppress growth (Swanson and others, 2007a,b). These stimulant-induced losses in growth are due to a disruption in growth hormone cycles (Aarskog and others, 1977) that could adversely affect other organs of the body.

Stimulants have also been found to induce depression and apathy in children (reviewed in Breggin, 1999). A study of children age 4–6 given methylphenidate found that two-thirds developed symptoms of depression and withdrawal (Firestone and others, 1998; see Breggin, 2008a; Table 11.1: 286). Older children also may become 'tired, withdrawn, listless, depressed, dozey, dazed, subdued and inactive' (Mayes and others, 1994; see Breggin, 2008a, Table 11.1: 286). When these adverse drug effects are mistaken for a worsening of the child's 'mental disorders', these children are often given more serious diagnoses and additional drugs, leading to chronicity.

Other adverse effects of stimulants include tics and other behaviours that are consistent with obsessive-compulsive symptoms. An NIMH study focused on stimulant-induced symptoms of obsessive-compulsive disorder (OCD) and found that 51 per cent of methylphenidate-treated children were afflicted with drug-induced OCD and 58 per cent with abnormal movements, usually tics (Borcherding and others, 1990).

Numerous animal studies confirm that stimulant drugs reduce overall spontaneous mental and behavioural activity (including social interest), causing apathy or indifference, plus enforcing compulsive meaningless behaviours (Arakawa, 1994; Bell and others, 1982; Hughes, 1972; Randrup and Munkva, 1967, 1970; Rebec and others, 1997; Schiorring, 1977, 1979; Wallach, 1974). Consistent with the brain-disabling principle of psychiatric drug effects (Breggin, 2008a; Moncrieff, 2007b), this reduction in spontaneity is the primary or 'therapeutic' effect. The child has diminished energy or motivation to act 'hyperactive' or 'impulsive', and a diminished fantasy life and the creativity to be distracted and 'inattentive'. As a result, we have 'less child' to get into trouble.

When children are told they have ADHD and need medications, they are given the idea that they cannot control their behaviour. The diagnosis of ADHD discourages personal responsibility and the stimulant drugs crush the ability to exercise it (Breggin, 1991, 2001, 2002).

No long-term benefit for children of any kind has ever been demonstrated for any stimulant drug — no improved behaviour, no improved socialisation skills, no improved academic skills and no improvement in learning (McDonagh and Peterson, 2006; Regier and Leshner, 1992; Despite six decades of research, the FDA-approved labels for stimulants remain required to state, 'Long-term effects of amphetamines in children have not been well established' (Adderall, 2013; Section 8.4 'Pediatric Use'). Even the pro-medication Multi-Modal Treatment Study (MTA) found at 36 months that medication treatment strategies were no better than any other behavioural and educational approaches, including a stay at a summer camp (Swanson and others, 2007b).

Effects of bipolar diagnosis and antipsychotic drugs

Moreno and others (2007) reported a 40-fold increase in the diagnosis of childhood bipolar disorder between 1994–1995 and 2002–2003. A remarkable 90.6 per cent of the children received psychiatric drugs and 47.7 per cent were prescribed antipsychotic drugs. Joseph Biederman, Thomas Spencer and Timothy Wilens from Harvard University fuelled this increase in diagnosing and drugging children while accepting funds from the pharmaceutical industry in return for promoting their products (Sarchet, 2011; Yu, 2011; also see Littrell and Lyons, 2010a,b).

Antipsychotic drugs include the older ones such as chlorpromazine (Thorazine or Largactil), haloperidol (Haldol) and perphenazine (Trilafon), as well as the newer 'atypicals' or 'novel' antipsychotic drugs such as olanzapine (Zyprexa), risperidone (Risperdal), aripiprazole (Abilify), ziprasidone (Geodon) and quetiapine (Seroquel). Moreover, there are four yet newer atypical antipsychotics: paliperidone (Invega), iloperidone (Fanapt), lurasidone (Latuda) and asenapine (Saphris). All of these drugs block dopamine neurotransmission to the frontal lobes (*Drug Facts and Comparisons*, 2012: 1627). As such, they will cause the same adverse effects as the older antipsychotic drugs, including lobotomy-like indifference and apathy, Parkinsonian symptoms, akathisia, dystonia, tardive dyskinesia, neuroleptic malignant syndrome, gynecomastia and other sexual dysfunctions. The atypicals also impact on numerous other neurotransmitter systems, including serotonin.

Tardive dyskinesia (TD) is a movement disorder caused by antipsychotic drugs (dopamine blockers). It can impair any muscle functions that are wholly or partially under voluntary control, including the face, eyes, tongue, jaw, neck, back, abdomen, extremities, diaphragm, oesophagus and vocal cords. Controlled clinical trials and epidemiological studies demonstrate that the rates for tardive dyskinesia are an alarming 5 per cent to 7 per cent cumulative per year (Chouinard and others, 1986; Glazer and others, 1993). Tardive akathisia, a variant of TD, causes a torture-like inner sensation that can drive patients into despair, psychosis, violence and suicide (American Psychiatric Association, 2000: 803). Tardive dystonia, another variant, causes painful and deforming spasms.

When study subjects are given equivalent doses of the older and newer antipsychotic drugs, there is little or no difference in the frequency of extrapyramidal effects or TD (Lieberman and others, 2005; Miller, 2009; Nasrallah, 2007; Rosebush and Mazurek, 1999; Woods and others, 2010). TD is a major threat to children (Breggin, 1983, 2008a; Mejia and Jankovic, 2010). In my clinical and forensic practice, I have evaluated many cases of childhood TD caused by newer antipsychotic drugs including risperidone, olanzapine, ziprasidone, aripiprazole and quetiapine. Even 'mild' cases of eye blinking or grimacing can humiliate, stigmatise and isolate a child. More severe cases disable children with painful spasms in the neck and shoulders, abnormal posture and gait, or constant agitated body movements and a

need to constantly, frantically pace. Although I did not personally evaluate the following cases, they illustrate some of the symptoms of severe TD (<http://www.youtube.com/watch?v=WIVxv5agOpQ>; <http://www.youtube.com/watch?v=v0GAJrUhNyk>).

In addition to these serious adverse effects, recent brain scan studies also demonstrate that exposure to antipsychotic drugs frequently causes brain shrinkage (atrophy) in patients (van Haren and others, 2011; Ho and others, 2011; Levin, 2011). There is long-standing evidence for antipsychotic drug-induced brain damage (Breggin, 1990, 1993, 2008a, 2011, 2013). Shrinkage of brain tissue has also been demonstrated in primates (Dorph-Petersen and others, 2005; Konopaske and others, 2007, 2008; Navari and Dazzan, 2009).

Other adverse effects include tardive psychosis and tardive dementia. Referring to both children and adults, Gualtieri and Barnhill (1988: 149) concluded, 'in virtually every clinical survey that has addressed the question, it is found that TD patients, compared to non-TD patients, have more in the way of dementia' (also see Myslobodsky, 1986, 1993). Patients withdrawn from antipsychotic drugs commonly become more disturbed and psychotic (tardive psychosis) than before they took the medications (Breggin, 2008a; Chouinard and Jones, 1980; Moncrieff, 2006). Children manifest tardive psychosis as a severe worsening of their behaviour beyond pre-treatment intensity (Gualtieri and Barnhill, 1988). Long-term patients can develop Neuroleptic-Induced Deficit Syndrome (NIDS) with cognitive and affective losses (Barnes and McPhillips, 1995), leading to a misdiagnosis of chronic schizophrenia.

Also of concern, the newer antipsychotics may cause a metabolic syndrome that predisposes children to heart disease and early death, including weight gain and obesity, elevated blood sugar and diabetes, elevated blood lipids and atherosclerosis, and high blood pressure (Lieberman and others, 2005). One-third or more of children and adolescents given antipsychotic drugs are at risk of developing metabolic syndrome (Splete, 2011; also see Goeb and others, 2010).

Patients diagnosed with serious mental disorders have a markedly shortened lifespan, as much as 13.8 years in the Veterans Administration and 25 years in state mental health systems (Kilbourne and others, 2009; Parks and others, 2006; Whitaker, 2010). Most of these patients have been exposed for years to antipsychotic drugs. Adults aged 20–34 on antidepressants have increased mortality when also taking antipsychotic drugs (Sundell and others, 2011). This increased mortality is not related to lifestyle but to polydrug treatment (Gill and others, 2007; Joukamaa and others, 2006).

Antipsychotic drugs have their 'therapeutic' effect by suppressing the frontal lobes and reticular activating system, producing relative degrees of apathy and docility (Breggin, 2008a). This effect occurs regardless of diagnosis and indeed regardless of species (Breggin, 1983). The National Institute of Mental Health conducted a long-term study ('CATIE') that compared several newer atypical antipsychotic drugs to an older one. The study gave a bleak picture of antipsychotic drug efficacy: 'In summary, patients with chronic schizophrenia in this study discontinued their antipsychotic study medications at a high rate, indicating substantial limitations in the effectiveness of the drugs' (Lieberman and others, 2005: 1218). Lieberman and Stroup concluded, 'By revealing the truth about the emperor's new clothes, CATIE has helped to refocus efforts on the need for truly innovative treatments and strategies that can make significant advances for persons with schizophrenia and related psychoses' (2011: 774). An overview of the problems associated with giving antipsychotic drugs to children can be found in Olfman and others (2012). Decades of research confirm the lack of efficacy of antipsychotic drugs (Whitaker, 2010, 2012; Breggin, 2008a). Moreover, no psychiatric drugs have been proven effective for children over the long term (that is, for many months or years).

In the limited space of this article, I have not presented personal narratives and clinical vignettes. They can be found in Kevin Miller's 2007 film, *Generation Rx*, and several of my

books (Breggin, 1991, 2001, 2002, 2008b), where I also examine the larger moral, social and political contexts of the drugging of children.

Concluding considerations

The question that I posed at the outset of this article is whether it is ever in the best interests of a child to be given a psychiatric drug. I first started working with children and adults in state psychiatric facilities 1954–1958 as a leader of the Harvard-Radcliffe Mental Hospital Volunteer Program (Breggin, 1991). I continued to work at times with psychiatric patients in medical school (1958–1962) and then did so fulltime for half my internship and fulltime for 3 years of psychiatric residency (1962–1966). I have had a private practice since 1967–1968. Based on my clinical experience and scientific research, spanning nearly 60 years, I conclude that children should not be exposed to psychiatric drugs. I have focused here on two diagnoses, ADHD and Bipolar Disorder, and on the drugs used to treat them. These two diagnoses in many ways cover the spectrum of psychiatric ‘disorders’ with their varied manifestations and degrees of impairment. Similarly, the two classes of drugs, stimulants and antipsychotics, also vary greatly and reflect the hazards associated with most psychiatric drugs. I believe that the lessons or conclusions drawn from these diagnoses and drugs may be applied to all childhood psychiatric diagnoses and the drugs used to treat them.

In my research and clinical experience, I have found children’s problems to be primarily psychosocial and/or educational in nature. Psychiatric diagnoses reduce these highly complicated contexts to narrow, unhelpful categories that fail to capture the richness and complexity, the human quality, of the child’s experiences. The psychiatric drugs used to ‘treat’ them do not address the underlying problems; at best they can only temporarily suppress their manifestations, while adding brain impairments. As I have demonstrated, children exposed to psychiatric diagnoses and drugs can suffer iatrogenic effects that impair, rather than improve, their physical, mental and emotional well-being. Prescribing drugs to children enforces physical dependency on psychoactive substances. The drugged child’s brain cannot physically develop in its intended manner but instead develops in response to a toxic internal environment. Furthermore, there is strong evidence that stigmatisation follows psychiatric diagnosis and treatment (Sartorius, 2002), contributing to loss of self-esteem and potentially bringing long-term disadvantages with regard to future opportunities. Children may learn to view themselves as physically or genetically disabled, adding to loss of self-esteem, impaired self-determination and increased feelings of helplessness.

There are circumstances when psychoactive substances have a legitimate medical purpose in the treatment of children, such as surgical anaesthesia, relief of physical pain and control of seizures. These *medical* drugs (in contrast to *psychiatric* drugs) are not intended for the control of behaviour and emotions, or the treatment of psychiatric disorders. Nonetheless, even in these cases, grave caution should be exercised if and when children are exposed to chemicals that affect the brain and mind. This distinction between medical drugs and psychiatric drugs is similar to one that I first made in the early 1970s when delineating the difference between genuine *neurosurgery* for the treatment of physical disorders such as seizures and *psychosurgery* or *psychiatric surgery* for the control of emotions and behaviour and the treatment of psychiatric disorders (see Breggin, 1973, 1975, 1977, 1981). Making this distinction proved very helpful in framing legislation and it informed the judicial opinion in *Kaimowitz v. Department of Mental Health*, a landmark psychosurgery case in which I offered these distinctions during my testimony (Breggin, 1975; Kaimowitz, 1973). The Kaimowitz decision contributed to ending psychosurgery in USA state hospitals, the Veterans Administration and the National Institutes of Health (NIH).

We can, and in my view should, apply these distinctions with regard to the epidemic psychiatric drugging of children in North America (and spreading throughout the world) in order to curtail, and eventually to end, the use of psychoactive substances to control emotions and behaviour in children. Drugs may be used effectively and ethically to treat genuine medical conditions such as epilepsy, but should not be used to control the *behaviour* of children or to *change their personalities and attitudes and to increase conformity*. Psychiatric diagnoses and drugs have not proven effective in helping children and they distract from confronting or locating the source of the child's problem(s) and from finding better solutions to the distress they experience, including better family, social and educational approaches. It is on these grounds that I have concluded that it is the right of every child to be protected from psychiatric drugs.

However, there is another more complex issue: 'What if parents *wish* to give their children psychiatric drugs such as stimulants or antipsychotic drugs?' Except when one parent resists another parent giving psychiatric drugs to their child, I am not aware of any circumstances in which a parent has been stopped from following a prescriber's directions to medicate a child. I have been an expert in family court legal actions in the USA where one parent has resisted another parent's desire to prescribe psychiatric medications for their child. Although I have been a medical expert in cases that led to judges supporting the child's removal from drugs, the courts tend to take a conventional psychiatric viewpoint and to side with the parent or doctors who wish to give the medications (Breggin, 2008b). Should a parent, especially when uncontested by the other parent, be allowed to give legally prescribed psychiatric drugs to their children? In the USA, the law responds with a resounding 'yes'. On clinical, moral and scientific grounds, I believe it is time to reconsider this.

Coppock (2002) points out that society, psychiatry and medicine now medicalise the inevitable and varied conflicts that arise between parents and children. In the process, psychiatry lends medical authority to the enforcement of parental control over children who rebel or fail to meet expectations, even when the control is arbitrary or abusive. While many parents are well meaning but misled by psychiatry, some parents do exercise this medically endorsed and abusive control over their children. Coppock points out that the majority of children and youth undergo psychiatric diagnosis and drug treatment without giving consent and, indeed, without any active participation in the decision. She challenges the concept that adults can and should be relied upon to make judgments in the 'best interests' of children. She warns that this mistaken viewpoint has enabled the current widespread psychiatric drugging.

When society, psychiatry and parents reach a consensus, as has now occurred, that it is in the best interests of children to psychiatrically drug them into more submissive and socially acceptable states, the usual legal safeguards cannot be relied upon. In this case, medical, legal and parental judgments in regard to the child's best interest can no longer be trusted. Just as physically beating children into submission was once widely accepted, the psychiatric drugging of unruly children is now considered the norm. However, in neither case was it ever scientifically, psychologically or morally right. For these reasons, a ban on psychiatrically drugging children becomes a legitimate goal, similar to other bans on child abuse.

We are a long way from changing the current positive attitude towards psychiatrically diagnosing and drugging children. In addition, any significant reduction in the widespread drugging of children will also cut deeply into the authority, power and profits of the entire psychopharmaceutical complex from drug companies and medical societies to individual researchers and prescribers (Breggin, 1991, 2008a). Rather than prematurely seeking a legal ban on psychiatric drugs at this time, we should view this as an ideal and an ultimate goal as we work towards a future when society, including healthcare providers and parents, will view psychiatric drugs as an abuse of children, and be ready to prohibit it the same way

it prohibits other forms of child abuse. This should become a goal for children's rights advocates as well as for those of us in the medical profession who remain concerned about the moral, psychological and physical well-being of children.

Meanwhile, individual parents should avoid putting their children on psychiatric drugs and, if already on drugs, parents should seek help in withdrawing them as soon and safely as possible (Breggin, 2013). Physicians and other prescribers should resist pressure to put children on psychiatric medications and instead work towards withdrawing them as soon and safely as possible. It is my hope that society will learn to view the psychiatric diagnosing and drugging of children as a huge and tragic mistake, and instead turn attention towards psychological, social, family and educational approaches that meet the genuine needs of children.

References

- Aarskog D, Fevang F, Klove H, Stoa K, Thorsen T. 1977. The effect of stimulant drugs, dextroamphetamine and methylphenidate, on secretion of growth hormone in hyperactive children. *Journal of Pediatrics*, 90: 136–139.
- Adderall. 2011. Complete Prescribing Information and Medication Guide. Available at <http://www.shire.com/shireplc/en/investors/investorsnews/irshirenews?id=544jj> [Accessed 20 January 2013].
- Adderall XR. 2013. *Physicians' Desk Reference*. PDR Network: Montvale, NJ; 2273–9.
- American Psychiatric Association. 2000. *Diagnostic and Statistical Manual of Mental Disorders*, 4th edn. Text Revision: Washington, DC.
- Arakawa O. 1994. Effects of methamphetamine and methylphenidate on single and paired rat open-field behaviors. *Physiology and Behavior*, 55: 441–446.
- Barnes T, McPhillips M. 1995. How to distinguish between the neuroleptic-induced deficit syndrome, depression and disease-related negative symptoms in schizophrenia. *International Clinical Psychopharmacology*, 10(Suppl. 3): 115–121.
- Baughman F, Hovey C. 2006. *The ADHD Fraud: How Psychiatry Makes "Patients" of Normal Children*. Trafford Publishing: Victoria, BC, Canada.
- Bell R, Alexander G, Schwartzman R, Yu J. 1982. The methylphenidate-induced stereotypy in the awake rat: local cerebral metabolism. *Neurology*, 32: 377–381.
- Borcherding B, Keysor C, Rapoport J, Elia J, Amass J. 1990. Motor/vocal tics and compulsive behaviors on stimulant drugs: is there a common vulnerability? *Psychiatric Research*, 33: 83–94.
- Breggin P. 1973. Psychosurgery. *Journal of the American Medical Association*, 226: 1121.
- Breggin P. 1975. Psychosurgery for political purposes. *Duquesne Law Review*, 13: 841–862.
- Breggin P. 1977. If psychosurgery is wrong in principle? *Psychiatric Opinion* Nov–Dec: 23.
- Breggin P. 1981. Psychosurgery as brain-disabling treatments. In *Divergent Views in Psychiatry*. Dongier M, Wittkower D. (Eds.). Harper & Row: Hagerstown, MD; 302–326.
- Breggin P. 1983. *Psychiatric Drugs: Hazards to the Brain*. Springer Publishing Company: New York.
- Breggin P. 1990. Brain damage, dementia and persistent cognitive dysfunction associated with neuroleptic drugs: evidence, etiology, implications. *Journal of Mind and Behavior*, 11: 425–464.
- Breggin P. 1991. *Toxic Psychiatry*. St. Martin's Press: New York.
- Breggin P. 1993. Parallels between neuroleptic effects and lethargic encephalitis: the production of dyskinesias and cognitive disorders. *Brain and Cognition*, 23: 8–27.
- Breggin P. 1998. Risks and mechanism of action of stimulants. In *NIH Consensus Development Conference Program and Abstracts: Diagnosis and Treatment of Attention Deficit Hyperactivity Disorder*. National Institutes of Health: Rockville, MD; 105–120.
- Breggin P. 1999. Psychostimulants in the treatment of children diagnosed with ADHD: risks and mechanism of action. *International Journal of Risk and Safety in Medicine*, 12: 3–35.
- Breggin P. 2001. *Talking Back to Ritalin*. Springer Publishing Company: New York, NY.
- Breggin P. 2002. *The Ritalin Fact Book*. Perseus Books: Cambridge, MA.

- Breggin P. 2008a. *Brain-disabling Treatments in Psychiatry*, 2nd edn.. Springer Publishing Company: New York.
- Breggin P. 2008b. *Medication Madness: The Role of Psychiatric Drugs in Cases of Violence, Suicide, and Crime*. St. Martin's Press: New York.
- Breggin P. 2011. Psychiatric drug-induced chronic brain impairment (CBI): implications for long-term treatment with psychiatric medication. *International Journal of Risk & Safety in Medicine*, 23: 193–200.
- Breggin P. 2013. *Psychiatric Drug Withdrawal: A Guide for Prescribers, Therapists, Patients and their Families*. Springer Publishing Company: New York.
- Breggin P, Breggin G. 1998. *The War against Children of Color: Psychiatry Targets Inner City Children*. Common Courage Press: Monroe, ME.
- Carlezon W, Konradi C. 2004. Understanding the neurobiological consequences of early exposure to psychotropic drugs: linking behavior with molecules. *Neuropharmacology* 47(supplement Suppl. 1): 46–60.
- Castellanos F, Giedd J, Marsh W, Hamburger S, Vaituzis A, Dickstein D, Sarfatti S, Vauss Y, Snell J, Lange N, Kaysen D, Krain A, Ritchie G, Rajapakse J, Rapoport J. 1996. Quantitative brain magnetic resonance imaging in attention-deficit hyperactivity disorder. *Archives of General Psychiatry*, 53: 607–616.
- Child Welfare Information Gateway. 2012. Administration for children & families, U.S. Department of Health and Human Services. Determining the best interests of the child: summary of State Laws. Available at: https://www.childwelfare.gov/systemwide/laws_policies/statutes/best_interest.cfm [Accessed 20 January 2013].
- Chouinard G, Jones B. 1980. Neuroleptic-induced supersensitivity psychosis: clinical and pharmacologic characteristics. *American Journal of Psychiatry*, 137: 16–21.
- Chouinard G, Annable L, Mercier P, Ross-Chouinard A. 1986. A five year follow-up study of tardive dyskinesia. *Psychopharmacology Bulletin*, 22: 259–263.
- Coppock V. 2002. Medicalising children's behaviour. In *The New Handbook of Children's Rights: Comparative Policy and Practice*. Franklin B (ed.). Routledge: London and New York.
- Dorph-Petersen KA, Pierri J, Perel J, Sun Z, Sampson A, Lewis D. 2005. The influence of chronic exposure to antipsychotic medications on brain size before and after tissue fixation: a comparison of haloperidol and olanzapine in macaque monkeys. *Neuropsychopharmacology*, 30: 1649–1661.
- Drug Facts and Comparisons. 2012. Wolters Kluwer Health: St. Louis.
- FindLaw. undated. Terminating parental rights. Available at <http://family.findlaw.com/parental-rights-and-liability/terminating-parental-rights.html> [Accessed 20 January 2013].
- Firestone P, Musten L, Pisterman S, Mercer J, Bennett S. 1998. Short-term side effects of stimulant medication are increased in preschool children with attention-deficit/hyperactivity disorder: a double-blind placebo-controlled study. *Journal of Child and Adolescent Psychopharmacology*, 8: 13–25.
- Giedd JN, Castellanos FX, Casey BJ, Kozuch P, King AC, Hamburger SD, Rapoport J. 1994. Quantitative morphology of the corpus callosum in attention deficit hyperactivity disorder. *American Journal of Psychiatry*, 151: 665–669.
- Gill S, Bronskill S, Normand SL, Anderson M, Sykora K, Lam K, Bell C, Lee P, Fischer H, Herrmann N, Gurwitz J, Rochon P. 2007. Antipsychotic drug use and mortality in older adults with dementia. *Annals of Internal Medicine*, 146: 775–786.
- Glazer W, Morgenstern H, Doucette J. 1993. Predicting the long term risk of tardive dyskinesia in outpatients maintained on neuroleptic medications. *Journal of Clinical Psychiatry*, 54: 133–139.
- Goeb JL, Marco S, Duhamel A, Kechid G, Bordet R, Thomas P, Delion P, Jardri R. 2010. Metabolic side effects of risperidone in early onset schizophrenia. *Encephale*, 36: 242–52.
- Gottstein J. 2012. Legal issues surrounding the psychiatric drugging of children and youth. In *Drugging Our Children. How Profiteers are Pushing Antipsychotics on Our Youngest and What We Can Do to Stop It*. Olfman S, Robbins B, Berk L, Gottstein J, Meyers A, Olsen G, Shanker S, Sparks J, Stanton T, Stone G, Whitaker R. Praeger: Santa Barbara, California; 99–118.
- Gualtieri C, Barnhill L. 1988. Tardive dyskinesia in special populations. In *Tardive Dyskinesia: Biological Mechanisms and Clinical Aspects*. Wolf ME, Mosnaim AD (eds). American Psychiatric Press: Washington, DC; 135–154.

- van Haren N, Schack H, Cahn W, van den Heuvel M, Lepage C, Colloings L, Evans A, Pol J, Kahn R. 2011. Changes in cortical thickness during the course of illness in schizophrenia. *Archives of General Psychiatry*, **68**: 871–880.
- Henderson T, Hartman K. 2004. Aggression, mania, and hypomania induction by associated with atomoxetine. *Pediatrics*, **14**: 895–896.
- Ho B-C, Andreasen N, Ziebell S, Pierson R, Magnotta V. 2011. Long-term antipsychotic treatment and brain volumes. A longitudinal study of first-episode schizophrenia. *Archives of General Psychiatry*, **68**: 128–137.
- Hughes R. 1972. Methylphenidate induced inhibition of exploratory behavior in rats. *Life Sciences*, **11**: 161–167.
- Joukamaa M, Heliovaara M, Knekt P, Aromaa A, Raitasalo R, Lehtinen V. 2006. Schizophrenia, neuroleptic medication and mortality. *British Journal of Psychiatry*, **188**: 122–127.
- Kaimowitz v. Department of Mental Health for the State of Michigan. 1973. No 73-19434-AW (Mich. Cir. Ct. Wayne County, July 10, 1973). Available at <http://www.toxicpsychiatry.com/psychosurgery/> [Accessed 26 January 2013].
- Kilbourne A, Ignacio R, Kim H, Blow F. 2009. Are VA patients with serious mental illness dying younger? *Psychiatric Services*, **60**: 589.
- Konopaske G, Dorph-Petersen KA, Pierri J, Wu Q, Sampson A, Lewis A. 2007. Effect of chronic exposure to antipsychotic medication on cell numbers in the parietal cortex of macaque monkeys. *Neuropsychopharmacology*, **32**: 1216–1223.
- Konopaske G, Dorph-Petersen KA, Sweet R, Pierri J, Zhang W, Sampson A, Lew D. 2008. Effect of chronic psychotic exposure to astrocyte and oligodendrocytic numbers in macaque monkeys. *Biological Psychiatry*, **63**: 759–765.
- Lambert N. 2005. The contribution of childhood ADHD, conduct problems, and stimulant treatment to adolescent and adult tobacco and psychoactive substance abuse. *Ethical Human Psychology and Psychiatry*, **7**: 197–221.
- Levin A. 2011. Brain volume shrinkage parallels rise in antipsychotic dosage. *Psychiatric News* **47**: 1.
- Lieberman J, Stroup T. 2011. The NIMH-CATIE schizophrenia study: what did we learn? *American Journal of Psychiatry*, **168**: 770–775.
- Lieberman J, Stroup T, McEvoy J, Swartz M, Rosenheck R, Perkins D, Keefe R, Davis S, Davis C, Lebowitz B, Severe J, Hsiao J. 2005. Effectiveness of antipsychotic drugs in patients with chronic schizophrenia. *New England Journal of Medicine*, **353**: 1209–1223.
- Littrell J, Lyons P. 2010a. Pediatric bipolar disorder: Part I—Is it related to classical bipolar disorder. *Children and Youth Services Review*, **32**: 965–973.
- Littrell J, Lyons P. 2010b. Pediatric bipolar disorder. An issue for child welfare. *Children and Youth Services Review*, **32**: 965–973.
- Mayer S, Sanderson D, Bixler F, Humphrey F, Mattison R. 1994. Methylphenidate and ADHD: influence of age, IQ, and neurodevelopmental status. *Developmental Medicine and Child Neurology*, **36**: 1009–1007.
- McDonagh M, Peterson K. 2006. Drug class review on pharmacological treatments for ADHD: Final Report 2006. Oregon Evidence-based Based Practice Center, Oregon Health and Science Center: Portland, Oregon. www.ohsu.edu/drugeffectiveness.
- Mejia N, Jankovic J. 2010. Tardive dyskinesia and withdrawal emergent syndrome in children. *Expert Review of Neurotherapeutics*, **10**: 893–901.
- Miller K. 2007. Generation Rx. Available at: <http://kevinpmiller.blogspot.com/2007/10/generation-rx-examines-rise-in.html> [Accessed 14 April 2013].
- Moncrieff J. 2006. Why is it so difficult to stop psychiatric treatment? It may be nothing to do with the original problem. *Medical Hypotheses*, **67**: 517–523.
- Moncrieff J. 2007a. *The Myth of the Chemical Cure: A Critique of Psychiatric Drug Treatment*. Basingstoke, Hampshire, UK: Palgrave Macmillan.
- Moncrieff J. 2007b. Understanding psychotropic drug action: the contribution of the brain-disabling theory. *Ethical Human Psychology and Psychiatry*, **9**: 170–179.

- Moreno C, Laje G, Blanco C, Jiang H, Schmidt A, Olfson M. 2007. National trends in the outpatient diagnosis and treatment of bipolar disorder in youth. *Archives of General Psychiatry*, **64**: 1032–1039.
- Myslobodsky M. 1986. Anosognosia in tardive dyskinesia: “Tardive dysmentia” or “tardive dementia”? *Schizophrenia Bulletin*, **12**: 1–6.
- Myslobodsky M. 1993. Central determinants of attention and mood disorder in tardive dyskinesia (“tardive dysmentia”). *Brain and Cognition*, **23**: 88–101.
- Nasrallah H. 2007. The roles of efficacy, safety, and tolerability in antipsychotic effectiveness: practical implications of the CATIE schizophrenia trial. *Journal of Clinical Psychiatry*, **68**(Suppl. 1): 5–11.
- Navari S, Dazzan P. 2009. Do antipsychotic drugs affect brain structure? A systematic and critical review of MRI findings. *Psychological Medicine*, **39**: 1763–1777.
- Olfman S, Robbins B, Berk L, Gottstein J, Meyers A, Olsen G, Shanker S, Sparks J, Stanton T, Stone G, Whitaker R. 2012. *Drugging Our Children. How Profiteers are Pushing Antipsychotics on Our Youngest and What We Can Do to Stop It*. Praeger: Santa Barbara, California, USA.
- Parks J, Svendsen D, Singer P, Foti M. 2006. *Morbidity and Mortality in People with Serious Mental Illness*. Alexandria, VA: National Association of State Mental Health Program Directors.
- Proal E, Reiss P, Klein R, Mannuzza S, Gotimer K, Ramos-Olagast M, Lerch J, He Y, Zijdenbos A, Kelly C, Milham M, Castellanos X. 2011. Brain gray matter deficits at 33-year follow-up in adults with attention-deficit/hyperactivity disorder established in childhood. *Archives of General Psychiatry*, **68**: 1122–1134.
- Randrup A, Munkva I. 1967. Stereotyped activities produced by amphetamine in several animal species and man. *Psychopharmacologia*, **11**: 300–310.
- Randrup A, Munkva I. 1970. Correlation between specific effects of amphetamines on the brain and on behavior. In *Current Concepts on Amphetamine Abuse: Proceedings of a Workshop, Duke University Medical Center, June 5–6, 1970*. Ellinwood EH, Cohen S (eds.). National Institute of Mental Health: Rockville, MD.
- Rebec G, White I, Puotz J. 1997. Responses of neurons in dorsal striatum during amphetamine-induced focused stereotypy. *Psychopharmacology (Berl)*, **130**: 343–341.
- Regier A, Leshner A. 1992. *Request for Applications: Cooperative Agreement for a Multi-site, Multimodel Treatment Study of Attention-deficit Hyperactivity Disorder (ADHD)/Attention-deficit Disorder (ADD)*. (Rep. MH-92-03). Department of Health and Human Services; Public Health Service; Alcohol, Drug Abuse and Mental Health Administration; and NIMH: Washington, DC.
- Rosebush P, Mazurek M. 1999. Neurologic side effects in neuroleptic-naïve patients treated with haloperidol or risperidone. *Neurology*, **52**: 782–785.
- Sarchet P. 2011. Harvard scientists disciplined for not declaring ties to drug companies. *Nature News Blog* July 11. Available at http://blogs.nature.com/news/2011/07/Harvard_scientists_disciplined.html [Accessed 4 July 2011].
- Sartorius N. 2002. Iatrogenic stigma of mental illness: begins with behaviour and attitudes of medical professionals, especially psychiatrists. *British Medical Journal*, **324**: 1470–1.
- Schiorring E. 1977. Changes in individual and social behavior induced by amphetamine and related compounds in monkeys and man. In *Cocaine and Other Stimulants*. Ellinwood EH, Jr., Kilbey MM (eds.). Plenum: New York; 481–522.
- Schiorring E. 1979. Social isolation and other behavioral changes in groups of adult vervet monkeys (*Cercopithecus aethiops*) produced by low, nonchronic doses of d-amphetamine. *Psychopharmacology (Berl)*, **64**: 297–302.
- Splete H. 2011. Antipsychotics linked to metabolic syndrome spike in children. *Clinical Psychiatry News*. Available at <http://www.clinicalpsychiatrynews.com/single-view/antipsychotics-linked-to-metabolic-syndrome-spike-in-children/934df78c22.html> [Accessed 11 September 2011].
- Strattera. 2011. *Physician's Desk Reference*. PDR Network: Montvale, NJ.
- Sundell K, Gissler M, Petzold M, Waern M. 2011. Antidepressant utilization patterns and mortality in Swedish men and women aged 20–34 years. *European Journal of Clinical Pharmacology*, **67**: 169–178.
- Swanson J, Castellanos F. 1998. Biological bases of attention deficit hyperactivity disorder: neuroanatomy, genetics, and pathophysiology. In *NIH Consensus Development Conference Program and*

- Abstracts: Diagnosis and Treatment of Attention Deficit Hyperactivity Disorder.* National Institutes of Health: Rockville, MD; 37–42.
- Swanson J, Elliott G, Greenhill L, Wigal T, Arnold L, Vitiello B, Hechtman L, Epstein J, Pelham W, Abikoff H, Newcorn J, Molina B, Hinshaw S, Wells K, Hoza B, Jensen P, Gibbons R, Hur K, Stehli A, Davies M, March J, Conners C, Caron M, Volkow N. 2007a. Effects of stimulant medication on growth rates across 3 years in the MTA follow-up. *Journal of the American Academy of Child and Adolescent Psychiatry*, 46: 1015–1027.
- Swanson J, Hinshaw S, Arnold L, Gibbons R, Marcus S, Hur K, Jensen P, Vitiello B, Abikoff H, Greenhill L, Hechtman L, Pelham W, Wells K, Conners C, March J, Elliott G, Epstein J, Hoagwood K, Hoza B, Molina B, Newcorn J, Severe J, Wigal T. 2007b. Second evaluation of MTA 36-month outcomes: propensity score and growth mixture model analyses. *Journal of the American Academy of Child and Adolescent Psychiatry*, 46: 989–1002.
- Wallach MB. 1974. Drug-induced stereotypical behavior: similarities and differences. In *Neuropsychopharmacology of Monoamines and Their Regulatory Enzymes*. Usdin E (ed.). Raven: New York; 241–260.
- Whitaker R. 2010. *Anatomy of an Epidemic*. Crown: New York.
- Whitaker R. 2012. Weighing the evidence: what science has to say about prescribing atypical antipsychotics to children. In *Drugging Our Children. How Profiteers are Pushing Antipsychotics on Our Youngest and What We Can Do to Stop It*. Olfman S, Robbins B, Berk L, Gottstein J, Meyers A, Olsen G, Shanke S, Sparks J, Stanton T, Stone G, Whitaker R (eds.). Praeger: Santa Barbara, California, USA; 3–16.
- Whitely M. 2010. *Speed Up and Sit Still: The Controversies of ADHD Diagnosis and Treatment*. UW A Publishing: Crawley, Western Australia.
- Woods S, Morgenstern H, Saksa J, Walsh B, Sullivan M, Money R, Hawkins K, Gueorguieva R, Glazer W. 2010. Incidence of tardive dyskinesia with atypical versus conventional antipsychotic medication: a prospective cohort study. *Journal of Clinical Psychiatry*, 71: 463–474.
- Yu X. *Three Professors Face Sanctions Following Harvard Medical School Inquiry*. 2011, June 2. Harvard Crimson. Available at <http://www.thecrimson.com/article/2011/7/2/school-medical-harvard-investigation> [Accessed 16 July 2013].

Correspondence to: Peter R. Breggin, MD, 101 East Street, No. 112, Ithaca, New York, 14850, USA, Tel.: 607 272 5328; Fax: 607 272 5329. E-mail: EmpathicTherapy@hotmail.com

Accepted for publication 17 June 2013