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ORIGINAL PAPER

Rational Principles of Psychopharmacology for Therapists, Healthcare Providers and Clients

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Abstract Because the epidemic dispensing of psychiatric drugs is based on misinformation, it is important for all health professionals, consumers, and most citizens (including patients and their family members) to have a more rational understanding of how psychiatric drugs actually "work." Instead of enforcing authoritarian "medication compliance" in obedience to the prescriber's orders, informed therapists and healthcare providers have an ethical duty to provide scientific information about the real effects of psychiatric drugs. Instead of naively accepting whatever the doctor prescribes to them, consumers need to educate themselves about all medications, but especially about psychiatric ones, which are consistently misrepresented and oversold. This review focuses on three principles of rational psychopharmacology. The first is *the brain-disabling principle,* which states that all psychoactive substances work by causing dysfunctions of the brain and mind. It further observes that no psychiatric drugs work by improving or correcting biochemical imbalances or any other presumed biological malfunctions. The second principle is *intoxication anosognosia* (*medication spellbinding*) which states that all psychoactive substances tend to cause a subjective over-estimation of their positive effects while masking their harmful ones, sometimes resulting in extremely harmful behaviors such as mania, violence and suicide. The third principle is *chronic brain impairment* (*CBI*)—that exposure to psychoactive substances, especially long-term, results in impairments of the brain or mind that can become persistent or permanent, including atrophy (shrinkage) of brain tissue. Not only are psychiatric drugs likely to do more harm than good, there are more effective and infinitely safer proven psychosocial approaches for treating the whole spectrum of "psychiatric disorders" from "ADHD" and "major depressive disorder" to "schizophrenia."

Keywords Psychopharmacology · Psychiatric drugs · Adverse psychiatric drug effects · Psychosocial alternatives to psychiatric drugs · Ineffectiveness of psychiatric drugs · Critical analysis of psychiatric medication

Introduction

What is the need for an article titled "*Rational* Principles of Psychopharmacology"? Theory and practice in the entire field of mental health is now dominated by what I have called the psychopharmaceutical complex (Breggin 1991, 2008a). Fueled by billions of dollars of drug company money, and supported by organized psychiatry and medicine, the psychopharmaceutical complex exerts influence or control over medical and psychological associations, medical schools, researchers, journals, state and federal governments, insurance companies, the media, prescribers, and nearly all healthcare providers (Breggin 1991, 2008a). Psychologists, counselors, and social workers have been trained that it is their duty to refer their more distressed clients for psychiatric drugs. A huge portion of the general population accepts that psychiatric drugs are the answer to everyday problems from fatigue and a broken heart to conflicts in the family between parents and their children. The drugging of children has become an epidemic of medical child abuse, leading me to call for a halt to

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giving psychoactive substances to children for the control of their minds and behavior (Breggin 2014a).

Since my early work (Breggin 1983, 1991), a growing, well-documented literature continues to describe the tragic results of this avaricious complex led by the drug companies, psychiatry and organized medicine (Abramson 2005; Angell 2004; Baughman and Hovey 2006; Caplan 1996; Cosgrove et al. 2006; Kirsch 2010; Gøtzsche 2013, 2015; Moncrieff 2013; Watters 2011; Whitaker 2002, 2010; Whitaker and Cosgrove 2015). The drug companies have been especially successful in transforming the discipline of *psycho*pharmacology—mostly the science of psychiatric drugs—into a marketing arm of the industry (Cosgrove et al. 2006; Decker 2013; Gøtzsche 2013, 2015; Watters 2011; also, Breggin 1983, 1991, 2008a). The committees that write the most important book in psychiatry, the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*), are predominantly drug-company flacks (Caplan 1996; Cosgrove et al. 2006; Cosgrove 2010; Kutchins and Kirk 2003; Watters 2011). Even the American *Psychological* Association, along with its attempts to gain prescription privileges for psychologists, has been increasingly associating itself with the pharmaceutical industry (Pachter et al. 2007). Allen Frances (2014), editor of the Fourth Edition of the *DSM*, has come out with scathing criticism of the new Fifth Edition, without any shame about his own long-time enjoyment of drug company largesse (Caplan 2015).

Drug company employees or consultants ghostwrite many scientific peer-reviewed articles (Wilson et al. 2009) and nearly all research in the field is driven by pharmaceutical company money. For a legal analysis of the self-serving manipulations by one drug company, Johnson & Johnson and its subsidiary Janssen, see David Rothman's report (Rothman 2010). I have evaluated how Ely Lilly foisted Prozac onto the professions and the public against all scientific evidence (Breggin 2008a; Breggin and Breggin 2004).

The need for a rational psychopharmacology is undeniable.

The Myth of the Biochemical Imbalance

Many professionals and the public have been falsely convinced that biochemical imbalances in the brain drive mental suffering, such as the serotonin theory of depression or the dopamine theory of so-called schizophrenia. Yet the evidence for any biological basis for "psychiatric disorders" is utterly lacking (Glenmullen 2000; Healy 2015; Kirsch 2010; Lacasse and Leo 2005; Moncrieff 2007a, b, 2013; also Breggin 1983, 1991, 2008a).

Smarting under so much criticism for promoting a false theory, one prominent psychiatrist has gone so far as to deny that psychiatrists ever held so an absurd belief: "In the past 30 years, I don't believe I have ever heard a knowledgeable, well-trained psychiatrist make such a preposterous claim except perhaps to mock" (Pies 2011, p. 1). This, of course, will seem dismaying to the millions of patients who have been misled into believing that they or their children must take dangerous psychoactive drugs to correct their biochemical imbalances.

Three Basic Rational Principles of Psychopharmacology

There are three basic principles of psychopharmacology without which any practitioner, student of the subject or drug recipient will remain in the dark. These three principles throw light on the current confusion and distress experienced by well-meaning clinicians in the field and by consumers of psychiatric medication, as well as their families. These principles apply to all psychoactive agents, but are especially important in respect to psychiatric drugs, which are the object of so much false marketing by drug-company sponsored experts in the field.

The First Basic Principle: The Brain-Disabling Principle of Psychopharmacology

> *All drugs that impact on the brain and mind "work" by partially disabling the brain and mind. No psychoactive substance corrects biochemical imbalances or any other real and presumed defects, deficits or disorders of the brain and mind, and none improve the function of the brain or mind. The so-called therapeutic effect is always a disability* (Breggin 2007, 2008a, b, 2013).

The brain-disabling principle is fundamental to understanding psychopharmacology, including the supposed therapeutic effects of psychiatric drugs and the inevitable serious adverse effects on the brain and mind (see Breggin 1980, 1983, 1991, 2008a; Moncrieff 2007b).

According to the brain-disabling principle, a psychiatric drug is evaluated as "working" or having a "therapeutic" effect when the evaluator approves or values the drug-induced mental and emotional disability. Opinions about the helpfulness of the drug will often vary depending on conflicts among interested parties, including the drug recipient, the family, the prescriber, and the hospital or clinic staff. For example, patients frequently reject antipsychotic drugs because of their adverse effects, including mental dulling, while others will value the docility and emotional indifference induced in the previously difficult person. On the other hand, patients frequently seek increasing amounts of benzodiazepine tranquilizers and sleeping pills, while caring

family members view the patient as seriously impaired with a diminished quality of life.

The shared or common capacity of all psychiatric drugs to compromise brain and mind function helps to account for the current practice of using psychiatric drugs off label and combining multiple drugs into "cocktails." It is a matter of increasing the disability of the brain and mind until the required effect is achieved, such as docility and passivity, indifference to self and others, emotional numbness or anesthesia, robotic behavior, or reticence about emotional distress.

All psychoactive drugs specifically impair the frontal lobes because they are among the most vulnerable areas in the brain and because the widespread disruption of neurotransmitters inevitably has a negative impact on them. As we examine the remaining categories of psychiatric drugs, keep in mind that all of them over time will impair frontal lobe function and produce a degree of apathy and indifference, with a related loss in quality of life.

Most recently, drug advocates having been discussing the capacity of various drugs (e.g., Malberg et al. 2000; University of Rochester 2011) and even electroconvulsive therapy (Madsen et al. 2000) to produce neurogenesis or the generation of new neurons in the brain, along with the production of various growth factors. These studies usually claim this indicates that the drugs are useful, but neurogenesis is a direct response to brain injury (Parent 2003; Wang et al. 2011). These studies do not demonstrate that psychiatric drugs and ECT improve mental function by causing neurogenesis; they demonstrate that these treatments cause brain damage and dysfunction. Once again, psychiatry and pharmaceutical industry research redefine brain injury as a therapeutic effect.

How Antipsychotic Drugs Disable the Brain

Antipsychotic or neuroleptic drugs disable the brain by disrupting multiple neurotransmitter systems, usually by suppressing them, and have no specific effect on "symptoms" such as psychosis, hallucinations and delusions (Breggin 2008a). Delay and Deniker compared the new drugs to lethargic encephalitis, an epidemic brain disease that was prevalent a few decades earlier, which induced profound indifference in its victims. The virus, like the drugs, also caused bizarre, permanent neurological impairments (Deniker 1970; also see Breggin 1990, 1993). Lehmann and Hanrahan (1954) who introduced the first drug (Thorazine or chlorpromazine) into North America had no qualms about describing its lobotomy-like effect of "indifference," so that the patients "display a lack of spontaneous interest in the environment [and] they tend to remain silent and immobile when left alone and to reply to questions in a slow monotone." The pioneers of the first "antipsychotic drugs" were well aware that they had moved from surgical lobotomy to chemical lobotomy; and they never claimed that they had a specific antipsychotic effect. Calling them "antipsychotics" was a later and highly misleading approach to promoting the drugs.

With the exception of clozapine, all antipsychotic drugs cause a functional lobotomy by blockading dopamine neurotransmission, which is the main conduit to the frontal lobes. This fact is confirmed in the FDA-approved labels (package inserts) for antipsychotic drugs. For example, the Risperdal label (Janssen 2014, p. 13) under Mechanism of Action and under Pharmacodynamics indicates that the drug is a receptor antagonist (blocker of neurotransmission) of "dopamine Type 2 (D_2)" and that it is very potent in this regard, e.g., has a "high affinity" for blocking the receptors. This blockade is what causes chemical lobotomy with the sought-after effects of indifference, apathy and docility.

Anyone who argues that antipsychotic drugs have a specific impact on psychosis must answer these questions: "Then why do these drugs also 'work' everywhere that social control is sought—in nursing homes, in children's institutions, in jails in the US, and in psychoprisons in the old USSR (Podrabinek 1979). Why do they work on children accidentally treated with them?" I was a medical expert in a malpractice case in which a pharmacy accidentally dispensed Zyprexa (an antipsychotic drug) instead of Zyrtec (an antihistamine), causing a child to experience overall mental and behavioral suppression and apathy.

The drug effect is independent of the recipient's mental condition, or even species, and so veterinarians use the drugs for controlling obstreperous mammals such as dogs and pigs (Read 2002). The theory of "neuroleptic threshold"—that the therapeutic effect begins with the onset of adverse neurological effects such as Parkinsonism—continues to be mentioned in the psychiatric literature (Miller 2009), reconfirming the brain-disabling principle.

In part due to their brain-injurious effects, antipsychotic drugs shorten the lifespan by a decade or more (Joukamaa et al. 2006; Whitaker 2010; also Breggin 2008a, 2011).

How Stimulant Drugs Disable the Brain

The addictive stimulant drugs given to children disrupt numerous neurotransmitter systems. Does this effect take place only if a child has ADHD? No, the same effect has been studied for many years in normal children, animals, and stimulant addicts (Grahame-Smith and Aronson 1992, p. 141; Randrup and Munkva 1967; reviewed in Breggin 1999, 2008a, pp. 303–307). At clinical doses of stimulants in monkeys, all spontaneous behavior is reduced and sometimes crushed (Schiorring, 1977, 1979). The monkey's repertoire of spontaneous behaviors diminishes or disappears including socializing, mutual grooming,

playing, and exploring. At the same time, probably due to impact on the basal ganglia, the monkeys develop perseverative behaviors, defined as the repetition of meaningless activities. (In children on stimulants, perseverative behavior manifests as obsessive–compulsive behavior.) Instead of grooming, the monkeys will pick at their own skin; instead of socializing, they will stay by themselves; instead of playing, they will do boring things like chewing on the bars of their cage or fingering pebbles; and instead of exploring, they will pace a corner or stare out their cage. Stimulants make seemingly good caged animals and they do the same thing to children, making them good caged children at school or at home. They stop their annoying socializing, lose their overall spontaneity, become more docile, and finally show willingness to perform behaviors that to them otherwise seem rote and meaningless.

How Benzodiazepine Drugs Disable the Brain

The addictive benzodiazepines used to quell anxiety and induce sleep produce their brain-disabling effect by abnormally over-stimulating the neurotransmitter system GABA, found throughout the brain. Increasing the effect of GABA produces a dose-dependent suppression of all central nervous system (CNS) neurons throughout the brain (Ballenger 1995), leading to a dose-dependent gradient of sedation, sleep, generalized anesthesia, and coma (Breggin 1998, 2008a, 2013). One anesthesiologist researcher described the overall effect as "pulling out the plugs at the switch board" (Orser 2007, p. 255 ff.). These drugs have no specific anti-anxiety effect and any one of them could produce general anesthesia. Because anxiety is a higher mental and emotional function, it is one of the first reduced by the drug; but in the process, overall mental life is suppressed.

Because the individual taking the drug is focused on reducing the anxiety, its reduction is more apparent than the more subtle diminution in cognitive processes. Furthermore, the knowledge that taking a pill will eventually dampen the anxiety gives individuals a sense of control, but through pills rather than through personal mental effort. The reliance on pills will eventually prove futile as the brain resists the sedative effect. Furthermore, the use of pills also undermines the individual's confidence and practice in learning to manage feelings of emotional helplessness.

How Antidepressant Drugs Disable the Brain

The so-called antidepressant drugs have no specific impact on depression and in fact are used off label to treat everything imaginable from physical pain to anxiety and ADHD (Breggin 2003/2004, 2008a, b, 2013). Initially the antidepressants sometimes cause euphoria, which is a very abnormal state that precedes mania, but is often mistaken for an "improvement" by prescribers and their patients. Almost inevitably, the euphoria is short-lived; but when it does persist it can it lead to bad judgment and impulsivity that ruins lives (Breggin 2007, 2008b).

Mostly the antidepressants produce an anesthesia of feelings that dulls emotional life. Some patients develop what one textbook calls an apathy syndrome (Marangell et al. 2003; also see Barnhart et al. 2004). Long term, they also produce a dysphoria syndrome (El-Mallakh et al. 2011). These drugs suppress both sexuality and love, often without full recovery when the drugs are stopped (Csoska and Shipko 2006). In my clinical experience, most people stay on antidepressants because they fail to perceive their loss of quality of life (see medication spellbinding, below). Also, when they try to stop their drugs the withdrawal syndrome produces such horrendous emotional and physical torture that they mistakenly believe that they are experiencing a return or worsening of their "mental illness" and that they need to stay on their drugs for the rest of their lives.

How Mood Stabilizers Disable the Brain

The so-called mood stabilizers in reality are mood flatteners. These drugs impair the individual's capacity to experience all emotions. Lithium—once touted by advocates as a "magic bullet" for mania—in fact floods the brain with a toxin that disrupts multiple neurotransmitter systems, as well as overall propagation of electrical impulses along the axons (Breggin 2008a). The result is an overall mental sluggishness that is anything but specific for a disorder, and which occurs in all mammals given the drug. The brain-disabling effect was discovered by chance during lab research on guinea pigs when the drug made them sluggish. The researcher immediately went across the street to try it on incarcerated mental patients (Cade 1949). Yet it took a few decades for the drug companies to decide it was worth marketing a substance so readily available in nature that it was difficult to patent, and also so highly toxic in effective doses. The long-term impact of lithium can lead to a syndrome of neurological dilapidation (Adityanjee et al. 2005).

The brain-disabling principle is key to understanding psychopharmacology and the unfortunate plight of so many patients. Because of the toxic effects of psychiatric drugs on the brain and body, they are vastly reducing the quality of life, health, and lifespan of millions of people (Breggin 2008a, 2013; Whitaker 2010).

The Second Basic Principle: Intoxication Anosognosia (Medication Spellbinding)

> *Intoxication anosognosia or medication spellbinding occurs when a psychoactive drug prevents the recipients from fully knowing or grasping that they are experiencing adverse drug effects upon their brain and mind* (Breggin 2007, 2008a, b, 2013).

Intoxication anosognosia or medication spellbinding is closely related to the brain-disabling principle of psychopharmacology and can be viewed as a specific disability that renders the drugged individual relatively unable to perceive the harmful emotional or psychological impacts of the medication. Even if the drug is prescribed for something other than its psychoactive effects, it can still cause medication spellbinding. As examples, diphenhydramine (Benadryl) for allergic reactions, isotretinoin (Accutane) for acne, or the statins for high cholesterol can at times have potent psychoactive effects, such as irritability, anxiety or depression, that can emotionally disable the person without the individual realizing that the drug is the culprit.

All psychoactive drugs inhibit or distort the capacity to recognize or understand the drug's adverse effects on the individual's thoughts, feelings or conduct. Intoxication anosognosia helps to explain how and why some people abuse alcohol and street drugs or routinely take psychiatric drugs without recognizing their untoward consequences. The drug-induced brain dysfunction impairs their ability to appreciate how the drug is impairing their mental life.

Individuals drunk on alcohol often fail to appreciate the degree of their intoxication and impairment, even when their conduct becomes offensive at a social gathering or when they commit crimes such as domestic violence or vehicular homicide. Individuals who chronically use marijuana often do not perceive the flattening of their emotions and the gradual onset of cognitive impairments. Similarly, individuals on psychiatric drugs rarely appreciate how their mental functioning and the quality of their lives are being compromised.

Even when individuals have some recognition that they "don't feel right" or "don't feel like myself" on a psychiatric drug, they are likely to under-estimate the actual impairment. Typically, the prescriber reassures the patient that the drug could not be causing the adverse effect, or that it will wear off shortly, or that the new symptoms indicate a worsening of their mental disorder rather than a harmful drug effect. Unfortunately, during a few weeks more of exposure, medication spellbinding sets in more strongly, and the individual becomes unable to appreciate his or her impairment, even when appearing stupefied, emotionally flattened or euphoric to a therapist, coworkers, friends or family members. Often, everyone seems to recognize the drug disaster except the prescriber and the patient.

The medicated individual is likely to blame the adverse effects on something other than the drugs. When made irritable by the drug effect, the individual is likely to blame it on something a family member or even a stranger is doing. When made depressed by the drug, patients are likely to feel that they are being realistic. When made psychotic by the drug, individuals are most likely to deny that they are psychotic, and continue to take the offending medication because the doctor told them to do so and out of fear that they would feel even worse without it. In several forensic cases, I have seen individuals perpetrate murder a few days or weeks after starting an antidepressant and then insist on needing it their first day in jail (Breggin 2008b).

Here are some familiar examples of medication spellbinding from routine clinical practice:

Individuals taking antipsychotic drugs always undergo a loss of interest or apathy because this is the drug's primary effect; but very few specifically complain about this change in their overall mental life. They are more likely to mention dry mouth or feeling fatigued or ill. They commonly develop symptoms of Parkinsonism and even bizarre abnormal movements from tardive dyskinesia without recognizing or reporting them (Myslobodsky 1985, 1993; also, Breggin 2008a). Patients who are obviously toxic on these drugs will often seem wholly indifferent to their condition.

Patients taking antidepressants will develop a diminished interest in their activities, including sexuality and love, without noticing or reporting the change unless specifically questioned (Csoska and Shipko 2006, Opbroek et al. 2002). In my clinical experience, even when asked specific questions they will often fail to grasp how indifferent they have become to activities that previously engaged them, such as their marriage, children, work, or recreational activities. If interviewed as couple, the drugged individual might say "The marriage is going better now that I'm on the medication," but their partner replies "He used to want to make love more often than I did. Now he hardly notices me. He rarely plays with the children anymore."

Patients taking benzodiazepines may have a vague sense of how much their memory and other cognitive functions have been impaired over many months or years. However, if they partially or wholly withdraw from the medication and lose the effect of medication spellbinding, they are likely to feel devastated at the destruction of their memories for large blocks of time and feel despair at their continuing difficulties with memory, learning and other cognitive functions.

Children on stimulant drugs may not like "the feeling" they get without being able to explain it, but they continue to take the medications because they are hungry for the approval they receive for their more subdued behavior. The children will have no awareness that their overall spontaneity has been blunted or even crushed, even when some of them are drugged into a robotic state.

Individuals taking lithium need to have their lithium blood levels checked regularly because they can become toxic to the point of neurological collapse without realizing what is happening to them. They may not notice or react strongly to the gradual erosion of their overall cognitive ability over the years, and continue to believe that the drug is helping them when it is ruining their brain function.

The concept of intoxication anosognosia or medication spellbinding is new, but the actual phenomena have been recognized as long ago as the descriptions of King David's drunken behavior in the Bible. The concept is central to understanding why patients continue to take their psychiatric medications even when the psychoactive chemicals are obviously doing more harm than good.

The Third Basic Principle: Chronic Brain Impairment (CBI)

> *The continued use of psychoactive substances leads to chronic brain impairment and in worst cases to irreversible mental deficits, shortened lifespan, and dementia* (Breggin 1983, 1990, 1991, 2008a, 2011, 2013).

Chronic brain impairment (CBI) is a result of the brain-disabling effects of psychoactive substances. Repeated or chronic exposure to substances that change brain functions is highly likely to cause persistent brain dysfunctions. Signs of chronic brain impairment include (1) cognitive dysfunction, (2) apathy and indifference, (3) emotional worsening (affective dysregulation), (4) diminished quality of life, and finally (5) intoxication anosognosia which prevents the victim from fully recognizing these widespread drug-induced impairments. During withdrawal or within a short time afterward, the symptoms almost always begin to diminish, with increasing recovery over a period of weeks or months. Sometimes, but not always, recovery seems complete.

A careful interview with anyone on long-term psychiatric drugs, especially if bolstered by discussions with family members or friends, will commonly indicate that the individual is more impaired than he or she can appreciate or communicate.

Cognitive dysfunction initially manifests as short-term memory dysfunction, impaired new learning, inattention, and difficulties concentrating. This can progress to the whole array of symptoms of generalized or global mental dysfunction, including loss of executive functions, abstract reasoning, judgment, and insight, along with "fogginess" or mental sluggishness.

Apathy or indifference manifests itself as "not caring" and not feeling engaged in life. Artistic activities and spiritual practices require the highest and most subtle brain function and therefore are often completely lost over a period of months or years. Empathy is reduced, along with the quality of all relationships. Frustrated and sad family members have told me, "He doesn't even care about the dog anymore."

Emotional worsening is a broad category that reflects the dysregulation of emotional life. Individuals become impatient, impulsive, irritable, hostile, and labile. Occasionally they become euphoric but far more often they lapse into depression. The deterioration usually develops insidiously over months or years, so that it is mistaken for "normal" or is attributed to "stress," "mental illness," or "getting old."

In my clinical experience, drug-free people can learn through therapy to transcend the effects of chronic brain injury. They will of course at times regret their mental deficits or resent the hard work required to maintain emotional stability. But when they learn to love even more fully than before their injury, they can actually reach new levels of creativity, love and happiness. Put simply, we are more than our brains, and we can often transcend brain injury through the increased exercise of reason and ethics, and through learning to become a source of love (Breggin 2014b).

Ineffectiveness of Psychiatric Drug Treatment

It is very difficult to demonstrate the effectiveness or usefulness of psychiatric drug treatment. So many patients quit because of ineffectiveness or adverse effects, that the drug companies keep their trials very short. For example, the drug trials for Adderall XR (an extended-release amphetamine mixture) for children lasted only 3 weeks (see below). Then it was unleashed on America's children as something to take for months, years, or a lifetime.

As another example, the placebo-controlled clinical trials for Risperdal in schizophrenia lasted 4–8 weeks in adults and 6–8 weeks in adolescents (Janssen 2014, p. 14). Even worse, I seem to be the only one to have observed that by testing their drugs on schizophrenia rather than on schizophreniform disorder, antipsychotic drug manufactures avoid trying to prove that their drugs are useful during the first six months of an initial psychotic episode (see official criteria for schizophreniform disorder in American Psychiatric Association 2013, p. 319). Because of this ruse, the antipsychotic drugs are never FDA-tested or approved

for the truly critical period of the first 6 months of the first episode and therefore this widespread use of these drugs is off label and unproven.

In fact, as in the case of Risperdal, testing the drugs on chronic patients only proves that the drugs ameliorate withdrawal symptoms in patients who have been removed from their existing drugs for the study. Their current antipsychotic drugs were abruptly stopped ("cold turkey") for 3 days (called the placebo washout). Then the patients were placed on Risperdal or a placebo (Janssen Research Foundation 1992a, p. 3). The study patients were already hospitalized, averaged 38 years of age, and averaged 15.9 years of psychotic disorder (Janssen Research Foundation 1992b, pp. 6–7), so it is inevitable that most or all were thrown into severe antipsychotic drug withdrawal when their ongoing treatment was stopped for three days. (The Risperdal was so ineffective that 46 % of medicated patients did not complete the study and the average length of treatment was only 5.5 weeks). This has nothing to do with actually treating the underlying disorder.

This is so hard to believe that it bears restating: antipsychotic drugs are approved by the FDA after being tested on chronically psychotic patients during withdrawal from similar drugs without ever being tested on drug-naïve patients undergoing their first psychotic episode. In actual clinical practice, every patient undergoing an initial psychotic break is given these drugs, but they have never been FDA-tested or approved for that purpose. The entire edifice of antipsychotic drug treatment is built on deception.

On the other hand, it is relatively easy to demonstrate how much harm psychiatric drugs do. Any placebo-controlled double-blind clinical trial will easily demonstrate many more adverse effects for patients on the psychiatric drug than on the placebo (i.e., see Janssen 2014). The array of adverse effects usually includes potentially serious and sometimes life-threatening ones, such as insomnia or hypersomnia, abnormal dreams, stimulation or sedation, emotional lability, irritability, agitation, anxiety, aggression, suicidality, neurological impairment, and psychosis.

The studies required by the FDA to prove safety and efficacy are extremely flawed and do not represent normal use of the drug in clinical practice (Breggin 2008a; Leber 1992). The studies are too short (typically lasting 3–8 weeks) and usually exclude high-risk patients who are potentially suicidal or violent. They involve only one drug instead of the usual multiple drugs prescribed in actual practice. Furthermore, the FDA studies involve weekly supervision of the patient, examination by experienced clinicians, encouragement, and a belief instilled in the patients that they may be taking a "miracle" drug—factors that reduce the risk of serious adverse effects and encourage improvement. In addition, the studies are planned by the drug companies, conducted by trusted allies, and ultimately evaluated by the drug companies rather than by the researchers.

Antidepressants Lack Effectiveness, but Psychosocial Therapies are Helpful

According to FDA regulations, drug companies need to produce two studies to demonstrate the effectiveness of a drug; but they can try as many times as they wish. Kirsch et al. (2008) reviewed all the antidepressant controlled clinical trials conducted during the FDA approval process and found that overall the antidepressants do not work (also see Antonuccio et al. 1999). The drugs were not better than placebo except in a relatively small group of the most extremely depressed patients, and even then the difference was marginal and probably not clinically significant. More recently, Kirsch (2010) reviewed the literature and found that psychotherapy is most effective, particularly in long-term follow-ups, and of course it avoids the devastating adverse effects of the drugs.

The STAR*D study, the largest antidepressant trials ever conducted, found that a mere 2.7 % of patients (108 of 4041) had an initial remission that lasted or could be followed up for 12 months (Pigott 2011; also, Pigott et al. 2011). Neal et al. (2011) with random assignment found that patients taking antidepressants are more likely to relapse than those who take no medication. Meanwhile, research continues to show the superiority of psychotherapy compared to antidepressants (Cuijpers et al. 2013a, b).

In December 2011, the Director of the National Institute of Mental Health (NIMH), Thomas Insel, made a startling public confession about antidepressants. He wrote in his official blog, "The bottom line is that these medications appear to have a relatively small effect in patients broadly classified as having depression." He meant a small *therapeutic* effect; the *adverse effects* are numerous and potentially devastating.

Meanwhile numerous studies find that psychotherapy is useful, even in psychotic depression (reviewed in Karon 2005). Therapy and guided self-help are both effective in depression (Cuijpers et al. 2010). In my clinical experience, psychotherapy with a hopeful, enthusiastic and encouraging therapist is most effective in helping people get over depression.

Stimulants Lack Effectiveness but Psychosocial Therapies are Helpful

Stimulant drugs for hyperactivity, including methylphenidate and amphetamine, have been on the market since the 1950s. The FDA-approved labels for these drugs continue to admit that they their long-term effectiveness (beyond a few weeks) has never been demonstrated. One of the more

recently approved stimulants, Adderall XR (2015), has the following statement in its FDA-approved label:

Long-Term Use

> The effectiveness of Adderall XR for long-term use, i.e., for more than 3 weeks in children and 4 weeks in adolescents and adults, has not been systematically evaluated in controlled trials. (Section 1, p. 3)

Of what use is a 3–4 week trial of a stimulant? Almost no use at all. It is too short to test for addiction, growth suppression, depression, psychosis, permanent tics, cardiovascular problems, drug withdrawal, and other adverse drug effects. It is too short to indicate any effectiveness in routine use, which usually lasts months or years. So, why don't the drug companies do longer-term studies? Because they know that these drugs do not work after the first few weeks of behavioral suppression.

In a last ditch effort to finally show effectiveness, NIMH funded the most ardent advocates of stimulant drugs to carry out the Multi-Modal Treatment Study (MTA). Overall, stimulant drugs were no more effective than behavioral and educational approaches, including a brief stay at a summer camp (Swanson et al. 2007b). Meanwhile, the medicated children experienced the growth stunting impact of these drugs with reductions in height and weight (Swanson et al. 2007a). They were also exposed to the many other risks, including depression, psychosis, obsessive–compulsive disorder, permanent tics, and cardiovascular problems.

Even more serious, many children diagnosed with ADHD and treated with stimulants go on to develop a long-term career as a mental patient that leads to an overall decline in quality of life with increased suicides, premature deaths, drug addiction, incarceration and mental hospitalization, and atrophy of the brain (reviewed in Breggin 2013, p. 78 ff.; Breggin 2014a).

Children diagnosed with ADHD typically reflect a continuum of normal child development. Often their parents or teachers lack the skills to teach them discipline and focus (see the work of Corrigan 2012, 2014). Clinical trials without medication have found that Cognitive Behavioral Therapy (CBT) is helpful in adults (Weiss et al. 2012). In my clinical practice, nearly all children diagnosed with ADHD quickly respond to improved parenting or a more disciplined and interesting classroom. Sometimes a male teacher makes all the difference. Those children who do not respond almost inevitably have parents who find it difficult to exert rational, consistent discipline or to spend sufficient time with their children. Often a mother feels overwhelmed trying to raise one or more boys without meaningful help from the father.

Antipsychotic Drugs Lack Effectiveness, but Psychosocial Therapies are Useful

As documented earlier in this report, the FDA does not require antipsychotic drugs to be tested within the first 6 months of a psychotic episode. Amazingly and tragically, their widespread use in millions of patients as an acute or initial treatment for schizophrenic-like psychosis is untested.

Furthermore, as also documented earlier, FDA-approval for new antipsychotic drugs in patients diagnosed with schizophrenia is based on demonstrating that the new drug can reduce the severe withdrawal symptoms caused in test subjects by abruptly removing them from their long-term ongoing treatment with existing antipsychotic drugs. *Thus, FDA approval for a drug for schizophrenia does not mean it is useful in treating either an acute episode or a chronic disorder*. It only means that the drug will partially reduce the suffering experienced by chronic patients who have been cruelly removed "cold turkey" from their long-term medications. Antipsychotic drug treatment is a massive sham.

The Clinical Antipsychotic Trials for Intervention Effectiveness (CATIE) was intended to finally demonstrate longer-term effectiveness for antipsychotic drugs in chronic patients (Lieberman et al. 2005). Despite drug-company funding and a lead author with more than a dozen drug company financial ties, the study found that "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" (p. 1218). Two authors of the CATIE study admitted, "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" (Lieberman and Stroup 2011, p. 774).

Harrow and Jobe (2007) have followed long-term patients diagnosed with schizophrenia and find that recovery is positively related to less drug exposure. As Whitaker (2010, pp. 99–104) and this author (Breggin 2008a, 2013) have observed after extensive reviews of the scientific literature, even the most disturbed patients tend to do better with little or no exposure to these highly toxic chemicals, while there are much more effective psychotherapeutic family-oriented interventions available.

There is a long history of research demonstrating the effectiveness and superiority of psychotherapeutic individual and family interventions compared to drugs in patients diagnosed with schizophrenia (Karon 2003; Karon and Vandenbos 1981; Mosher 1996; Mosher and Bola 2004; also see Whitaker 2010; Breggin 1991, 2008a for overviews). CBT has proven useful without drugs with

patients diagnosed with schizophrenic spectrum disorder (Morrison et al. 2012, 2014). A largely nondrug family intervention approach in Lapland, Finland offers the most encouragement; it has nearly eradicated so-called "schizophrenia" in that city with multi-professional interventions into the family at the first signs of psychosis in a member (Seikkula et al. 2003, 2006).

Benzodiazepines Lack Effectiveness but Psychosocial Approaches are Helpful

Sedative, hypnotic and so-called anxiolytic drugs are so highly addictive that they should not be used in a continuous fashion beyond a very few weeks. For occasional sedation to control insomnia or to take the edge off anxiety, they work best if used rarely (or not at all). Alprazolam (Xanax) is the most widely used of this class of drugs. In studies used for FDA approval, patients were worse off at 8 weeks than at baseline at the start of the trials (Marks et al. 1989; reviewed in Breggin 2008a, pp. 341–344). In these short eight-week trials, the number of patients unable to withdraw from brief drug exposure varied from a low of 7 % to a high of 29 % (Xanax XR, 2011, p. 6). Very few clinicians seem to realize that the addiction process is already in full swing after less than two months exposure.

There is a vast clinical literature on the psychological treatment of anxiety, which I will not attempt to review here. My newest book, *Guilt, Shame and Anxiety* (Breggin 2014a, b) offers a new scientific theory that roots guilt, shame and anxiety in biological evolution as nature's anger management solution to conflict within the intimate family and tribe. It shows how to identify these emotions, along with resultant chronic anger and numbness, and then how to overcome them with reason and love.

Mood Stabilizers Lack Effectiveness

The efficacy of mood stabilizers, like all psychiatric drugs, is seriously in doubt (Lagace and Eisch 2005). The Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD) (Perlis et al. 2006) used the "best treatment available" approach employing every category of psychiatric drug, including the mood stabilizers, such as lithium and Depakote. A total of 1500 patients were followed for 2 years. Of 58 % who recovered, nearly half of those (48 %) relapsed. The study concluded, "Recurrence was frequent and associated with the presence of residual mood symptoms at initial recovery" (p. 217). In other words, even the initial recovery was not nearly a full recovery. Another study focused exclusively on lithium and found that 73 % relapsed within 5 years (Gitlin et al. 1995). More dismally, two-thirds of the relapses were worse than at baseline with multiple episodes. In my clinical experience, which now spans many decades, so-called "rapid cycling" was nonexistent before aggressive drug therapy began in recent decades.

If you do not prescribe stimulants and antidepressants to patients, you will rarely have to deal with a dangerous manic-like episode. In my clinical experience, it is sometimes possible with therapy to prevent spontaneous manic episodes from evolving by focusing on the underlying feelings of depression and helplessness, and encouraging emotional self-control; but after a manic episode becomes full-blow the person needs protection and a drug-free safe place while recovering. Unfortunately, institutions that provide drug-free safety no longer exist as they once did during the era of moral therapy inspired by the Quakers and the Tuke family in the 18th and 19th centuries (Bockoven 1963; Tuke (1996) [1813]; also see Breggin 1991).

Discussion

Psychiatric Drug Withdrawal

Withdrawing from psychiatric drugs can be emotionally and sometimes physically dangerous. It should be done carefully with experienced supervision (Breggin 2013).

Despite what many clinicians believe, every psychoactive substance, including every psychiatric drug, poses a risk of serious withdrawal reactions. This is because the brain has compensatory mechanisms that resist every psychoactive intrusion (Breggin 2008a). When the drug intrusion stops, the over-compensated brain displays the abnormal functioning that it has generated in its flawed attempt to balance out the drug effects. In addition, while taking the drug, individuals are rendered unable to appreciate the degree of their impairment. After drug withdrawal, when the "brain fog" starts to lift, the mental dysfunction becomes apparent to the victim leading to increased emotional distress.

Sometimes withdrawal effects can be lasting. When they continue for years, it is more accurate to describe them as lasting toxic effects due to drug-induced brain injury rather than to describe them as persistent drug withdrawal effects, which implies potential recovery (Breggin 2013). Every class of psychiatric drugs can produce lengthy withdrawal periods, potentially leaving the individual with long-term brain injury (Breggin 2008a, 2011, 2013).

Legal and Ethical Risks for Psychologists and Therapists

Psychologists and therapists are often taught that they must promote medication compliance—that is, they must convince their patients or clients to be blindly obedient to the

doctors' orders. Ironically, psychologists are supposed to maintain this posture of ignorance about what is good for their clients even when they see them for a full session every week while the prescriber may see them every 1–6 months for 10 minutes.

Compliance is antithetical to respect for the autonomy and freedom of our patients and clients. It harkens back to the old days when the doctor was always right. The concept reeks of bullying and manipulation. Instead of enforcing "compliance," health professionals including psychologists and therapists will achieve a higher ethical standard when they become able to recognize and to inform clients and their families about the potential risks associated with psychiatric drugs, as well as their lack of efficacy.

Psychologists, therapists and other non-medical healthcare professionals sometimes worry that they will be subject to malpractice suits if they communicate their knowledgeable concerns about psychiatric medications to their clients. To the contrary, the prescribers of drugs are commonly sued for the harm they do. By contrast, in my decades of forensic work I cannot recall a malpractice suit against a non-medical therapist for voicing or documenting concerns about excessive drugging, overlooked adverse drug effects, or related issues. However, I have on a few occasions been asked to defend health professionals, including psychiatrists and psychologists, against their own professional associations or institutions for voicing opposition to medication, and have done so successfully.

The real danger, however, is neither from lawsuits or from professional ethics committees—it is from outraged prescribers within the clinic or school in which the therapist works. Professionals are fired from jobs for criticizing medication practices. I recommend that ethical professionals whenever possible maintain a part-time back up private practice, because most contemporary institutions—universities, public schools, hospitals, and clinics—are likely to turn against anyone who speaks truthfully about psychiatric drugs. This is nothing new in human history. It has always required ethics and courage to stand up on public issues, in this case, the damaging impact of the pharmaceutical industry and its medical minions on individuals and society. We can helplessly lament this or refuse to be cowed.

Time to Revive the Study of Human Nature, Human Mind and Psychotherapy

Within the field of psychology and psychotherapy, and to some extent within psychiatry, there once thrived a field of in-depth biopsychosocial clinical research and theorizing, now largely marginalized and even crushed by the psychopharmaceutical complex. My own roots begin with William James and surprisingly Charles Darwin whose influence I have more recently recognized (Breggin 2014a, b, 2015). Innumerable others include Sigmund Freud, Alfred Adler, Karon Horney, Carl Rogers, Abraham Maslow, William Glasser, Thomas Szasz, and R. D. Laing writing with Aaron Esterson in *Sanity, Madness and the Family*. Each contributed from unique perspectives.

My own interests have become increasing *bio*psychosocial, including a refreshing revisit to the monumental work of John Bowlby. Like Bowlby, the "bio" in my work reflects genuine evolutionary and biological science (Breggin 2014a, b, 2015), and not the fabricated "medical model" and corrupt neuroscience funded by the pharmaceutical industry. It is time to revive our tradition of serious thinking about human nature and human life. As my wife, Ginger, recently said to me, "Human beings are more than a chemistry project."

Conclusion

Psychologists, therapists and other healthcare providers who read this article will have a more accurate understanding of drug effects than the vast majority of prescribers. By selecting and reading a few other key articles and books from the bibliography, professionals and non-professionals alike will probably have more understanding than *any* of the prescribers they are ever likely to meet. That is because physicians and other prescribers who acquire this kind of knowledge tend to stop prescribing! Informed mental health practitioners can educate their clients about the hazards and ineffectiveness of psychiatric drugs, while providing them the best psychosocial and educational services available.

We need to put biological psychiatry into proper perspective as a failed institution and realize that psychiatric drugs commonly do more harm than good. We also need to reconfirm the usefulness of psychosocial therapies. Ultimately, we need to re-inspire interest in the kind of meaningful biopsychosocial research, theory and practice that once flourished in our field before the takeover by the psychopharmaceutical complex.

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