Brain-Disabling Treatments in Psychiatry

8 - Lithium and Other Drugs for Bipolar Disorder

Drugs, Electroshock, and the Psychopharmaceutical Complex


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Chapter 8

Lithium and Other Drugs for Bipolar Disorder

Lithium for the treatment of manic episodes or bipolar disorder was originally promoted to the public and to the mental health profession as the ultimate example of a specific biochemical treatment for a specific psychiatric disorder. To bolster this claim, it was said that lithium lacks any brain-disabling effects on either patients or normal volunteers. This view lithium directly challenges the concept of medication spellbinding and brain-disabling principle of psychiatric treatment. Although a number of new drugs have now been added to the mood stabilizer armamentarium, lithium remains the prototype.

8.1 Claims of Lithium Specificity for Mania

In 1970, a booklet published by the National Institute of Mental Health (NIMH) \(^{[71]}\) and intended for public consumption claimed that lithium produces “no unwanted effects on mood and behavior” and “only the symptoms are leached out while the rest of the personality remains unaffected”. The NIMH report concludes that “the drug is unique among psychopharmaceuticals in that it rarely produce any undesirable effects on emotional and intellectual functioning”. It calls the substance “the first specific chemical treatment for a mental disease”.

Five years later, the American Psychiatric Association (APA, 1975) \(^{[5]}\) published “The Current Status of Lithium Therapy: Report of the APA Task Force.” Without citing evidence, the authors stated, “The task force has concluded that lithium is a more specific anti-manic agent than neuroleptics and that its therapeutic results are achieved in a unique pharmacologic effect rather than nonspecific calming action.”

Ronald Fieve became one of the leading advocates of lithium. In his book Moodswing (1989) \(^{[29]}\), he stated, “I have not found another treatment in psychiatry that works so quickly, so specifically, and so permanently as lithium for recurrent manic and depressive mood states” (p. 4). He describes this extraordinary therapeutic effect as occurring with no discernible adverse effects. The evidence will reveal that instead that lithium is neither quick nor specific nor permanent in its impact. Nor is lithium relatively free of adverse effects. It is one of the more deactivating, disabling drugs in the psychiatric armamentarium.
8.2 Brain-Disabling Effects on Animals, Infants, Patients, and Volunteers

8.2.1 Subduing Effects on Animals

Cade (1949)\(^{[16]}\) discovered the potential therapeutic value of lithium accidentally while experimenting with guinea pigs and immediately decided to try administering it to human beings. In his own words, here is the deductive leap he made:

“A noteworthy result was that after a latent period of about two hours the animals, although fully conscious, became extremely lethargic and unresponsive to stimuli for one to two hours before once again becoming normally active and timid.”

“It may seem a long distance from lethargy in guinea pigs to the excitement of psychotics, but as these investigations had commenced in an attempt to demonstrate some possibly excreted toxin in the urine of manic patients, the association of ideas is explicable.”

Cade’s leap from producing a toxic lethargy in animals to “treating” human beings shows his intuitive recognition of the central role deactivation in psychiatric treatment. As reviews by Schou (1957\(^{[86]}\), 1968\(^{[82]}\), 1976\(^{[83]}\)) indicated, no large studies on primate behavior were conducted before the widespread use of lithium in humans. One reason for this be indicated in Schou’s summary of how lithium affected mice and rats. In a 1957 review, he noted, “A certain apathy and slowness of reaction have been frequent symptoms in the experimental animals.” Or, as he remarked in a later review (Schou, 1976\(^{[83]}\)), there is “decreased spontaneous exploratory activity”.

This suppression of “spontaneous and exploratory activity,” as well as the suppression of other expressions of volition and vitality, are the hallmarks of most biopsychiatric treatments and helped to inspire my concept of deactivation and the brain-disabling principles of psychiatric drugs. In studies of lobotomy and in the early and most forthright early dies of neuroleptic drugs, the primary or essential effect was identified the production of indifference. In the antidepressant literature, this same effect is gaining recognition in regard to how these drugs produce apathy in long-term use. Stimulant advocates have failed to recognize these same effects in regard to Ritalin, Adderall, and other drugs used for the control of behavior in children; but the scientific literature will confirm that their primary effect is the crushing of spontaneity with a loss interest in autonomously generated, imaginative, creative, and social activities.

Lithium is toxic in rats at the same serum concentrations as in humans (Schou, 1976\(^{[83]}\)). In a rat study by Smith and Smith (1973)\(^{[14]}\), lithium was administered in the low therapeutic range for a period of only 1 week. The authors summarized, “The most consistent effect of lithium was to decrease the voluntary activity of the rats.”

The consistent finding of generalized behavioral suppression in animals undermines the claim that lithium is a specific magic bullet for mania. Suppression of voluntary or spontaneous activity is perhaps the lost concise description of the primary impact of all brain-disabling therapies on animals and humans alike.

8.2.2 Subduing Effects on Normal Infants

If a drug subdues the human fetus or infant, it is likely that its effect is not specific for a particular psychiatric disorder. Lithium freely crosses the placental barrier in utero and can be passed through breast milk (An3nth, 1978). The effects of lithium in producing lethargy and hypotonia (loss of muscle function) in babies at relatively low serum levels has been thoroughly documented (Rane et
al., 1978 [77]; Strothers et al., 1973 [97]). Hollister (1976) [41] noted that lithium causes “lethargy, cyanosis, poor suck and Moro reflexes”. Lethargy in an infant describes the primary brain-disabling effect. As in animal studies, clinical reports concerning newborn and nursing babies demonstrate that lithium suppresses, and even disables, the central nervous system.

8.2.3 Disabling Effects on Normal Volunteers

Because they considered lithium to be disease-specific for mania, advocates of the drug initially claimed that it had little or no effect normal individuals (Dempsey et al., 1977 [25]; Hollister, 1976 [40]). Even van Putten (1975a) [101], usually a keen observer of drug effects, stated that “lithium prophylaxis does not affect normal mental functioning or deprive a pane of normal human sorrow or elation”.

Claims that lithium has no effect on normal volunteers are often based on a study by Schou et al. (1968) [84], who stated: “The most striking observation seems to be how little lithium affects normal mental functions: in prophylactic dosage not at all and in higher therapeutic dosage only moderately.”

However, Schou et al.’s (1968) [84] own data do not support this view. It is true that the researchers found no impact in six volunteers when the drug was given at low doses for only 1 week. However, the authors also administered lithium to themselves within the therapeutic dose (1.0 mEq/L) for 1-3 weeks. The authors, who now became the subjects of the experiment, experienced the common initial somatic side effects, including “transient nausea, diarrhea, slight tremor of the hands, etc”. In addition, they suffered from a straitjacketing effect: “A feeling muscular weakness or heaviness was prominent in all the subjects. They had to overcome a certain resistance against rising and moving and also had a feeling that mental effort was needed to undertake any physical task.”

The most remarkable effects were subjective. Keep in mind that Schou et al. (1968) [84] are trying to substantiate how little effect lithium on normal mental function when they described the following effects themselves:

“Psychological effects were, on the whole, subtle and ill defined. There was no consistent change of the mood level, but irritability or emotional lability could at times be noted. There might be hypersensitivity to everyday sights and sounds. On other occasions responsiveness to environmental stimuli was diminished; this was in one of the cases welcomed by the family (‘Dad is much easier and nicer than usual’), while the families of the two other subjects complained about their being so dull. The subjective experience was primarily one of indifference and slight general malaise. This led to a certain passivity. The subjects often had a feeling of being at a distance from their environment, as if separated from it by a glass wall. The subjective feeling of having been altered by the treatment was disproportionately strong in relation to objective behavioral changes. The subjects could engage in discussions and social activities but found it difficult to comprehend and integrate more than a few elements of a situation. One of the subjects noted, for example, that whereas he had unaltered ability in a game such as chess with only two participants, he was less good at bridge with its four players. Intellectual initiative was diminished, and there was a feeling of lowered ability to concentrate and memorize; but thought processes were unaffected, and the subjects could think logically and produce ideas. The assessment of time was often impaired; it was difficult to decide whether an event had taken place recently or some time ago.”

References to diminished “responsiveness to environmental stimuli,” diminished “intellectual initiative,” “indifference and a slight general malaise,” and “a certain passivity” definitively describe the deactivating, in-disabling effects of lithium (chapter 1). The language used is identical that used to describe lobotomy effects.

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Most interesting, perhaps, the authors, in writing about themselves, seem medication spellbound. That is, they fail to recognize how much harm the drugs are doing to their mental capacities, even as they report them. They used their study as the basis for their widely publicized claim that lithium has little or no effect on normal volunteers. Their study was published in such an obscure foreign-language journal that it was not even available in the National Library of Medicine, and therefore other researchers and professionals had to rely upon their claims concerning their results.

That one of the author's children thought he was improved by deactivation confirms the brain-disabling principles. At least from this child's viewpoint, it was a relief to have her father become subdued and withdrawn.

Small et al. (1972) \(^{93}\) examined the mental effects of lithium on 11 normal volunteers in a more systematic fashion. Three had such serious reactions that there were "objective indications of impairment in work and school performance". A fourth developed a "severe, precipitous toxic delirium on the tenth day of taking lithium". A fifth volunteer dropped out of the study in the first week with "severe muscle weakness, confusion, and depression," which, the authors argue, without evidence, was "more likely" related to psychological factors than to the drug.

Limonella et al. (1974) \(^{58}\) focused on behavioral reactions in simulated automobile driving and found lithium-induced impairment in response and reaction times, and in judgment.

Judd et al. (1977a\&b \(^{46}\) \& \(^{47}\)) also studied the reactions of normal volunteers to lithium (mean, 0.9 mEq/L) over a 2-week period. In one study (Judd et al., 1977 \(^{46}\)) they reported the effects of lithium on mood and personality in 23 subjects. They expressed surprise at their findings, which included a decreased "sense of well-being" among their volunteers and a "large number of spontaneous complaints". The authors described their results in no uncertain terms:

"These subjective changes are not mood elevating, but rather mood lowering. In general, these feeling-tone alterations are dysphoric and characterized by lassitude, lethargy, and feelings of negativism and depression. In addition, feelings of agitation, anxiety, tension, and restlessness are related to lithium carbonate maintenance. There is also some evidence that subjects indicated they did not want to have to deal with the demands of interacting with their human environments. Finally, there are consistent self-reports of inability to concentrate, mental confusion, feeling muddled, and a loss of clear-headedness."

Although not as picturesque as Schou et al.'s self-described lithium effects \(^{86}\), the impression of brain-disabling effects is similar. In 1979 \(^{49}\), Judd summarized the results of studies with 42 healthy young men. He concluded that lithium produces a "general dulling and blunting of various personality functions" and a "generalized subjective dysphoria". Consistent with the brain-disabling principles, he attributed the therapeutic effect of lithium to a general slowing of cognitive processes.

An especially interesting aspect of Judd's research confirms the trained independent observers are not likely to report adverse drug effects, even when they are apparent to those who administered the drug and to those personally associated with the persons receiving the drug (Judd et al., 1977a \(^{46}\)):

"It was of interest to find that the effects of lithium carbonate in normal subjects were not perceptible to trained independent observers in the experimental situation. We initially speculated that these changes, although profound to the individual experiencing them, were not such that they were easily discernible, even to trained observers. In contrast to this was the fact that the 'significant other,' an individual who had a much more extensive interpersonal experience with the subject, was able to identify alterations in behavior and mood during the time the subjects were being maintained on lithium carbonate. Further, their observations were completely consistent with qualitative changes obtained from the self-rating data from the subjects.

\(^{1}\) I obtained a translation of the original article from one of the authors.
themselves. Thus, these changes due to lithium carbonate are not just subjectively experienced, but are apparent to independent observers who are well acquainted with the normal range of behavior of each of the subjects."

The adverse effects most frequently noted by personal associates of the subjects included "increased levels of drowsiness and lowered ability to work hard and to think clearly" (Judd, 1979 [49]). The group who reported these changes in the subjects consisted of "friends, roommates, girlfriends, etc." The background of the "trained independent observers" not described, but presumably they are mental health professionals.

It is striking that the trained observers were "unable to detect any behavioral changes in the subjects induced by lithium" when they were apparent to personal associates and could be measured on testing. Judd (1979 [49]) attributed their failure to a lack of familiarity with the subjects in their normal surroundings. But various findings in this book confirm that this failure to observe adverse drug effects is characteristic of the vast majority of research reports and review articles in the drug literature. It the doctor’s part iniatrogenic denial: the tendency to deny the brain disabling effects of psychiatric treatments (chapter 18).

Studies have continued to demonstrate adverse effects of lithium on normal subjects (Glue et al., 1987 [33]; Kroph et al., 1979 [52]; Muller-Oerlinghausen et al., 1977 [69]; Weingartner et al., 1985 [104]). Schatzberg and Cole (1991) [80] appropriately warned that the patient’s subjective experience of mental dysfunction should be taken seriously:

"Some patients on lithium complain of slowed mentation and forgetfulness and, on testing, a memory deficit has been found. Although such patients are often suspected or accused of 'using' such symptoms to avoid necessary lithium therapy, our impression is that these complaints are often real and constitute a basis for lowering the dosage or trying another therapy." (p. 159)

Jefferson (1993) [43] summed up the deactivating effect of lithium,

"Neurologic adverse effects of lithium include reduced reactivity, lack of spontaneity, intellectual insufficiency, memory problems, difficulty in concentration, dysphoria. Some of these effects may be related to the therapeutic action of lithium in reducing hypomania. However, hypothyroidism, weakness and fatigue due to hypercalcemia, and breakthrough depression must be considered in the presence of these symptoms."

The production of thyroid disorders by lithium is common and requires constant concern throughout the treatment. Lithium-induced hypothyroidism can produce depression and other mental dysfunction, greatly confusing and complicating the patient’s clinical picture.

In a review of the literature concerning the impact of psychiatric drugs on cognition in normal subjects, Judd et al. (1987) [48] found the following:

"In summary, lithium often induces subjective feelings of cognitive slowing together with decreased ability to learn, concentrate and memorize. In addition, controlled studies have consistently described small but consistent performance decrements on various cognitive tests, including memory tests. The available data suggest that the slowing of performance is likely to be secondary to a slowing in the rate of central information processing." (p. 1468)

Studies of normal volunteers should lay to rest the claim that lithium only affects a disease process. It should also put an end to the claim that lithium has a specific antimanic effect, rather than a generalized brain-disabling, deactivating effect. This effect may at times reduce the occurrence of manic episodes, but it does so by reducing overall brain function. Even in regard to reducing the frequency of manic episodes, its efficacy is doubtful and it causes manic withdrawal reactions (see following sections).
8.2.4 Turning Down the Dial of Life

Confirming the brain-disabling principle, lithium has the same subduing effects on psychiatric patients as on normal volunteers. Speaking of individuals successfully treated with lithium, Dyson and Mendelson (1968) \(^{26}\) observed the following:

“It is as if their ‘intensity of living’ dial had been turned down a few notches. Things do not seem so very important or imperative; there is greater acceptance of everyday life as it is rather than as one might want it to be; and their spouses report a much more peaceful existence.”

As a demonstration of the brain-disabling concept of psychiatric treatment, the reference to the spouse’s report of a more peaceful existence’ reminiscent of Schou et al.’s (1968) \(^{84}\) observation that one of the children preferred it when Dad’s “responsiveness to environmental stimuli was diminished”. The comparison to neuroleptic deactivation and to lobotomy again seems apparent.

According to Dyson and Mendelson (1968) \(^{26}\), even on effective maintenance therapy, the dial of life remains turned down. They quoted some of their patients:

“I just don’t get irritated and upset at things as I used to”. “Things that used to bother me don’t seem so important anymore”. “I don’t have any energy, can’t accomplish what I used to be able to.

Schlagenhauf et al. (1966) \(^{81}\) found that “when improvement was first noted the patients complained of feeling internally ‘curbed,’ a subjective experience that all of them had considerable difficulty in describing very precisely”. The patients felt “unable to talk, think or move as fast as they would like”. Again, lithium is obviously and grossly disabling the brain mind.

Demers and Davis (1971) \(^{24}\) examined the attitudes of spouses toward patients treated with lithium. Without intending to emphasize the point, the study made clear that there is an overall reduction in all forms of lively expression or vitality:

“An apparent unfavorable result of lithium treatment was a reduction in enthusiastic behavior, as well as sexual responsiveness in the manic-depressive. Hypomanic joviality, enthusiasm, and spontaneity are often regarded as social pluses; and manic-depressives and their spouses complain about the loss of these valued attributes. When pressed to discuss the sexual compatibility of the marriage, frequently they will say it is worse since lithium treatment started, as the lithium-treated spouse has less libidinal strivings.”

This excerpt illustrates the brain-disabling principle that the evaluation treatment success depends upon the observer’s attitude toward the g-induced mental disability. In these instances, the spouses are described as missing their partners’ vitality and sexuality. On the other hand, the doctors label these valued attributes “hypomanic” in order to justify the brain-disabling effect of their treatments.

8.2.5 Crushing Creativity

Ronald Fieve, of the New York State Psychiatric Institute, achieved national attention (“New Old Treatment,” 1973) in newspapers and magazines when he presented theatrical producer-director Joshua Logan at the annual meeting of the American Medical Association, where Logan ve a testimonial for lithium.

The entire question of testimonials for various treatments is a difficult and complex one. Quack cures, for example, often have avid supporters. Logan (1976) \(^{60}\), in his autobiography, described
his many contacts with psychiatric treatment over the years, including earlier public testimonials for psychiatry. He expressed surprise that people are critical of electroshock treatment, which he found to be very “benign”.

Logan’s own psychiatrist, Fieve, coauthored an article (Polatin et al., 1971) describing three individuals (rare cases, in the authors’ opinion) who rejected maintenance lithium, two of whom did so specifically on the grounds that it interfered with their creativity as writers of bestsellers: “These patients report that lithium carbonate inhibits creativity so that an individual is unable to express himself, drive is diminished, and there is no incentive.”

Despite their claim that lithium does not interfere with creativity Schou and Baasstrup (1973) described its inhibiting, flattening effect:

“It is not always the elation that is missed. An undertaker’s customers, mistaking depressive sadness for compassion, complained about his appearance of indifference when he was in lithium treatment. Another patient regretted that in discussions he was unable to attain the level of excitement he considered necessary: ‘Doctor, I am a communist and I must get excited when I discuss.’ There are also patients who feel that lithium treatment makes life ‘flat’ and less colorful, ‘curbs’ their activity, and prevents them from going as fast as they would like. 10 most cases these complaints disappear when the patients become used to the stable life course.”

Whether these complaints do in fact disappear in most cases has never been carefully investigated. Even if the complaints become less frequent, there may be many unfortunate reasons for this, including the extremely spellbinding effect of lithium. In my clinical experience, child and adults exposed to any psychiatric drug for a lengthy period of time lose their ability to perceive their emotionally subduing effects; but spellbinding effect of lithium is especially potent.

Jefferson (1993) and Goodwin and Jamison (1990) also confirmed that loss of creativity is experienced by some patients on lithium; but it did not daunt their advocacy for the drug.

8.2.6 Cade Supports the Brain-Disabling Hypothesis

There is a particular irony in the date of the first publication on the use lithium in mental patients: Cade’s article [16] appeared in 1949, the same year that Corcoran et al. [21] published “Lithium Poisoning From the Use of S. Substitutes” in the Journal of the American Medical Association.

In regard to neuroleptics, we found that pioneers in their use WI most straightforward about its brain-disabling effects. We find the same phenomenon with lithium. Cade (1949) indicated that lithium, when used for other medicinal purposes, produced “actual mental depression in a variety of patients, not just those suffering from mania or manic depression. The drug enforced a so-called quieting effect on persons considered schizophrenic (dementia praecox, in his nosology):

“An important feature was that, although there was no fundamental improvement in any of them, three who were usually restless, noisy and shouting nonsensical abuse . . . lost their excitement and restlessness and became quiet and amenable for the first time in years.”

Cade (1949) preferred lithium to lobotomy on “restless and psychopathic mental defectives” in order “to control their restless impulses and ungovernable tempers”.

8.3 Spellbinding and Iatrogenic Helplessness and Denial

The previously cited research by Judd demonstrates how professionals utterly fail to see lithium-induced disabilities that are obvious to friends and detectable with psychological testing. Due to
medication spellbind” patients themselves have difficulty evaluating their mental status on lithium. Toxicity often creeps up slowly over many days or weeks so that their judgment is impaired in an almost imperceptibly gradual manner. In fact, patients cannot be relied on to notice when they are becoming severely toxic, even though the symptoms include marked gastrointestinal disturbances, tremor, and disturbed mental functions. Instead of relying on the perceptions of patients, blood levels must be carefully monitored and the patients carefully watched.

In keeping with this medication spellbinding effect, normal volunteers on small doses suffer impairments of their reflexes but do not realize acknowledge the impairment (Linnoila et al., 1974 [58]). Lithium patients who report no side effects often have grossly obvious tremors. The failure of patients on maintenance therapy to notice their own neurologic defects clearly demonstrates that long-term treatment with lithium is medication spellbinding.

8.4 Toxicity to the Central Nervous System

8.4.1 The Production of Cognitive Deficits

It is now generally accepted that lithium can impair intellectual function. For example, Shaw et al. (1987) [87] found impairments of memory and hand motor speed on lithium. In Manic-Depressive Illness, a book written wholly from a biopsychiatric perspective, Frederick Goodwin and Kay Jamison (1990) [134] nonetheless concluded that lithium does cause serious cognitive impairments. They summarized much of the literature up that time and declared,

"Since the drug's primary action is mediated through the central nervous system, it is not surprising that lithium can cause cognitive impairments of varying types and degrees of severity. Indeed, memory problems are among the side effects of lithium treatment that patients report most frequently. Although affective illness itself contributes both to cognitive deficits and complaints about such deficits, it is important to bear in mind that impairment of intellectual functioning caused by lithium is not uncommon and, in many patients, leads to noncompliance. Creativity can also be affected." (p. 706)

More recently, Stip et al. (2000) [95] summarized the literature on lithium-induced memory problems: “Several studies have shown cognitive impairment in short-term memory, long-term memory and psychomotor speed in bipolar patients taking lithium.” Their study aimed at testing the effect of lithium in normal subjects in a double-blind, 3-week study. They found that lithium-treated volunteers had long-term memory deficits on recalling words compared to the placebo group.

8.4.2 Acute Organic Brain Syndromes

Considering how vigorously lithium is promoted as relatively free of overpowering mental effects, it is surprising how many cases of toxic delirium during routine lithium therapy were reported soon after the drug came into use (Johnson et al., 1968 [45]; Mayfield et al., 1966 [64]; Prien et al. 1972 [75]; Shopsin et al., 1971 [89]; Strayhorn et al., 1977) [96]. Prien et al. (1972) [75] found that almost one-third of the patients in their highly active category suffered “severe” reactions, including several with toxic confusion described as “disorientation, confusion, lack of continuity of thought, and reduced comprehension”. Lithium is highly neurotoxic.
8.4.3 SILENT: Irreversible Lithium-Induced Neurotoxicity

In 1987 [1], Adityanjee discussed so-called lithium poisoning and made an observation that remains true today: “There is a general lack of awareness about irreversible and untreatable complications of lithium treatment despite evidence to the contrary.”

Originally, it was thought that, except in extreme cases, lithium induced neurotoxicity was reversible. However, it eventually became apparent that many patients develop irreversible brain damage and dysfunction, often involving the cerebellum (Grignon et al., 1996 [36]). In the last two decades, researchers have defined a syndrome of irreversible lithium-effectuated neurotoxicity (SILENT). Adityanjee et al. (2005) [2] viewed the literature from 1965 to 2004 for cases of lithium neurotoxicity with the persistence of sequelae for at least 2 months after cessation of treatment. They found 90 cases of SILENT, with persistent cerebellar dysfunction as the most commonly reported persistent aftereffect. These chronically disabled patients may need “physical rehabilitation for gait ataxia, speech training for dysarthria, and cognitive training for dementia memory impairments” (p. 47). The most likely cause, according to the authors, is “demyelination caused by lithium in multiple sites in the nervous system, including the cerebellum”. Not surprisingly, lithium toxicity also cause chronic neuropsychological changes, including impaired memory, attention, executive control functions, and visuospatial deficits (Brumm et al., 1998 [13]).

Irreversible neurotoxicity can occur at relatively low serum doses. Lang and Davis (2002) [54] described “the case of a 44 year old man who presented with a two-month history of dysarthria, ataxia and leg weakness whilst on maintenance lithium for bipolar disorder”. He had significant cerebellar and pyramidal dysfunction. His serum lithium was 1.5 mmol/L, a moderate elevation for this patient. His recovery was only partial, leaving him mainly with cerebellar ataxia. The authors warned about the insidious onset of persistent neurotoxicity during routine treatment.

8.5 Neurotoxic Effects in Low-Dosage Maintenance Therapy

Branchev et al. (1976) [13] published a follow-up of patients on long-term lithium maintenance (6 months to 7 years). Only 10 of 36 were “free of neurologic symptoms,” even with the low maintenance doses employed. Four 6 patients had parkinsonian symptoms at a “low level of severity”.

8.5.1 Abnormal Brain Waves Produced Routine Lithium Therapy

From early on, the electroencephalogram (EEG) was found to demonstrate significant pathologic response to lithium therapy, confirming the intoxicating effect of the drug (Baldessarini, 1977 [7]; Corcoran et al., 1949 [21]; Mayfield et al., 1966 [64]; Peach, 1975 [72]; Schou, 1957 [86]; Small et al., 1972 [93]). Consistent with the brain-disabling principle, Mayfield and Brown (1966) [64] correlated EEG abnormalities with the therapeutic response to treatment. Muller-Oerlinghausen et al. (1977) [69] reported grossly abnormal brain wave patterns in patients and normal volunteers. These persisted in the volunteers at the final testing 7 days after the withdrawal of lithium therapy.

Two review articles confirmed reports of persistent brain wave changes in patients treated with lithium (Friedman et al., 1977 [30]; Reisberg et al., 1979 [78]). Reisberg and Gershon (1979) declared, wholly without proof, that “the evidence is that these effects are benign”.

Because some studies had shown changes in functional imaging in patients diagnosed with bipolar disorder during cognitive testing, Bell et al. (2005) [10] sought to separate out the influence of
medication. conducted a double-blind study of volunteers taking lithium or valproate using functional MRI. Both medication groups showed a significant decrease in the magnitude of the blood-oxygen-level-dependent (BOLD) signal. The authors linked these changes to the cognitive dysfunction measured in many studies of lithium.

8.5.2 Lithium Disruption of the Compromised Brain

In combination with neuroleptics, especially haloperidol, there is an increased likelihood of severe encephalopathic syndromes that are sometimes irreversible (Baldessarini, 1978 [10]; Cohen et al., 1974 [20]). There is a case report of a similar reaction from combining lithium with the newer neuroleptic, risperidone (Swanson et al., 1995 [99]).

Lithium administered in combination with electroshock produces more severe organic brain syndromes (Weiner et al., 1980 [103]). Remick (1978) [79] and Hoenig and Chauk (1977) [39] reported single cases of an acute severe delirium resulting from this combination. Mandel et al. (1980) [62] reported on two more cases of this nature. In 1980 [92], Small et al. reviewed 25 patients given electroshock while being treated with lithium a found that the patients had more severe memory loss, more severe confusion, and occasional neurologic dysfunctions. The authors recommend, against the use of electroconvulsive therapy (ECT) in patients receiving lithium therapy.

The literature concerning lithium administration to individuals with preexisting brain disease is sparse but indicates the expected increase, brain disability, including in the elderly (Baldessarini, 1978 [6]).

Beitman (1978) [9] described a case of reactivation of tardive dyskinesia as a result of lithium therapy; the tardive dyskinesia had been quiescent for many years. Crews and Carpenter (1977) [22] also described a case which lithium aggravated a preexisting tardive dyskinesia.

8.6 Brain Damage As Treatment

8.6.1 General Toxicity to Neurons and Other Cells

Writing from the viewpoint of the pharmacologist, rather than the psychiatrist, Peach (1975) [72] observed:

"The accumulation of lithium in the intracellular environment could be envisioned to perturb any event that is modulated by monovalent cations, e.g., sodium or potassium. These possible interactions signify the enormous magnitude of the task of determining precise mechanisms of action of the lithium ion."

Lithium disrupts almost every measurable cellular activity pertaining nerve transmission as well as many other vital functions. In addition, its distribution is fairly uniform throughout the central nervous m, with no known areas of specific concentration. It produces what Wilson et al. (1975) [106] called a nonselective diminution in neuronal activity. The neurophysiology of lithium, even without supporting clinical data, renders absurd the notion of a specific biochemical treatment for a specific disease and confirms the brain-disabling effect.

Because of its neurotoxic impact, lithium appears to increase the of tardive dyskinesia for patients taking neuroleptics (Ghadirian et al., 1996 [31]). Consistent with this, there have been reports of
extrapyramidal symptoms in patients taking lithium without neuroleptic exposure, ding parkinsonism (Lecanwasam et al., 1994 [53]), chore a (Podskalny et al., 1996 [73]), tardive parkinsonism (Muthane et al., 2000 [70]), tardive dystonia (Chakrabarti et al., 2002 [18]), and tardive dyskinesia (Meyer-Lindenberg et al., 1997 [66]). The existence of extrapyramidal side effects on maintenance lithium has been found in numerous studies (e.g., Kane et al., 1978 [51]; Shopsin et al., 1975 [89]). Shopsin and Gershon’s (1975) [89] patients, like those of Branchey et al. (1976) [13], did not complain about their neurologic symptoms, suggesting further mental impairment and a profound medication binding effect.

Lithium also impairs the function of the peripheral nervous system, reducing motor nerve conduction velocity (Faravelli et al., 1999 [28]). It causes many metabolic adverse effects, resulting in hypothyroidism, hyperthyroidism (rare), hyperparathyroidism, and diabetes insipidus (Livingston et al., 2006 [59]).

Psychiatry has gone from denying that lithium causes kidney damage to trying to ignore it. The threat is very real. Lepkifker et al. (2004) [56] viewed the files of 140 patients exposed to lithium for at least 4 years and found that 20% developed creeping creatinine (a laboratory test for ney malfunction) and renal insufficiency. Overall, lithium is very toxic cells (Yao et al., 1999 [107]).

8.6.2 The “Protective” and Therapeutic Effects of Poisoning Brain Cells

An increasing number of psychiatric drugs have been shown to cause abnormal proliferations of brain cells. The process is abnormal, first, because it is caused by the toxic impact of a drug; second, because the drugs are already known to cause many clinically obvious toxic effects on brain cells and many organs of the body; and third because the number and morphology of the cells are abnormal. Yet researchers are dependent on the psychopharmaceutical complex, both emotionally and economically, that many persist in seeing these abnormalities as evidence of a specific therapeutic mechanism. Lagace and Eisch (2005) [53] review the so-called neuroprotective effects of mood-stabilizing agents, including lithium, valproic acid, carbamazepine, and neuroleptics. Two separate effects were studied: neuroprotective and neurogenic changes caused by mood stabilizers.

First, these drugs exert a so-called protective effect on cell cultures, preventing cell death from occurring in response to certain trauma. For example, a rat is stressed by immobilizing it in a glass tube (Lim et al., 2005 [56]). This causes changes to take place in the responsiveness of brain cells to electrical stimulation, as measured in the decapitated animal postmortem brain. If 1 hour after death, slices of the animal’s brain are bathed in lithium, the brain changes in response to stimulation do not occur. Unbelievably, this laboratory finding in animal brain slices has been leaned on by researchers, including Lagace and Eisch (2005) [53], as an indication that this postmortem protection may have something to do with the clinical effect of these drugs in living human beings. Never mind that lithium, for example, is extremely toxic to the human central nervous system and peripheral nervous system, a virtual poison to brain cells; this quirk in a Petri dish may nonetheless show that these drugs protect brain cells.

Second, these drugs produce abnormal cell growth. The researches call this process neurogenesis as if it were benign; but the neurons are not normal in appearance. According to Lagace and Eisch (2005) [53],

“In general, these studies have assessed neuron proliferation, neurite [axon] outgrowth, regeneration, and differentiation. In sensory neurons, lithium, valproic acid, and carbamazepine have a common effect of increasing growth of cone formation, leading to a spreading of the neuron and a shorter neuronal axon . . . . Recently, lithium has been shown to induce proliferation and neuronal differentiation of rap hippocampal progenitor cells . . . . Like lithium, valproic acid
treatment has been shown to induce neurogenesis in vitro, specifically inducing neurite growth, cell reemergence, and the formation of mature neurons in embryonic cortical cells.”

These authors are a little more skeptical than others; they do want to make the complete leap to clinical, therapeutic effects. But they are hoping: “To determine if the clinical efficacy of mood-stabilizing drugs is dependent on the neuroprotective or neurogenic properties of these medications, greater strides need to be made in relating findings from cell culture and animal models to human imaging and pathology.” The obvious brain-disabling, mood-flattening effects of lithium are ignored in the interest of promoting a more benign effect based on the most flimsy experimental grounds.

Chen et al. (2000) gave lithium to rats in their chow, achieving blood levels comparable with human treatment, and found a proliferation of brain cells in the hippocampus. They made the leap to claim that this neurotrophic effect may make lithium “of use in long-term treatment of other neuropsychiatric disorders”. In other words, stimulating the brain to make abnormal brain cells is likely to be good for a variety of psychiatric disorders. This kind of giant leap, utterly ignoring the obvious toxic effects of lithium, has become common in the literature.

Not all researchers are so quick to assume that any drug-induced abnormal growth in brain cells will be beneficial to human beings. Harada et al. (1996) set out to “understand the mechanism underlying the neurotoxicity of lithium”. They found that lithium impaired the function of nerve growth factor in rat cells. In doing so, it caused some of the abnormalities seen in lithium treated cells, including attenuated neurite growth.

Meanwhile, it does not occur to these researchers that lithium causes demonstrable memory dysfunction and that the hippocampus plays a major role in memory processes, suggesting instead that they were looking at how lithium harms the brain—and not how it might help it. Indeed, there is research that addresses the effect of lithium on biochemical processes that specifically affect mental functions such as memory and spatial discrimination. Banchaabouchi et al. (2004) gave rats lithium for weeks to reach a typical human therapeutic serum level. This resulted in suppression of a biochemical factor in the hippocampus associated with cognitive processes (Nurr 1) and also resulted in impairment of spatial discrimination in the animal. (Nurr 1 also plays a role in dopamine cell function and perhaps in the development of parkinsonism, Zetterstrom et al., 1997; lithium-induced dysfunction in Nurr I may be associated with the drug’s capacity to cause dopamine-related neurological disorders, such as parkinsonism.)

The finding of abnormal cell growth stimulated by mood stabilizers is consistent with research showing that bipolar patients taking lithium and valproic acid have increased hippocampal regions measured on MRI. Beyer et al. (2004) found that this increase in hippocampal size correlated with the use of lithium. They also related it to the laboratory studies of neurogenesis.

There are, of course, many contradictory findings in the literature, but it is apparent that exposure to mood stabilizers, especially lithium, profoundly impairs the function of the brain, even causing abnormal cell proliferation in some cases, and cell loss in others (Blumberg et al. 2003). The distorted thinking in the psychiatric sciences is so rampant that none of the studies view these recently documented abnormalities in cell growth and brain size as a cause for alarm. Instead, they are automatically promoted as evidence of benefit and cause for hope.

8.7 The Relative Ineffectiveness of Lithium in Acute Mania

The myth of lithium specificity is shattered in exactly that arena in which one would expect to find the most support: clinical use as described by its advocates. Early on, it became generally accepted that the neuroleptics, not lithium, are most effective in stopping acute mania (Baldessarini, 1978;
Juhl et al., 1977 \cite{50}). Even with the development of combine, neuroleptic-lithium therapy, some authorities advocate ECT, as well, as the control of especially severe cases (Hollister, 1976 \cite{40}).

The clinical preference for the neuroleptics as the treatment for acute mania was based on the single most comprehensive, controlled study which was conducted by Prien et al. (1972) \cite{75}. They specifically contradicted the thesis that lithium has any specificity for mania or the “underlying manic process”. They cautioned that “unfortunately, these observations have been all but lost in the vast number of unqualified endorsements of lithium carbonate therapy that have deluged the literature”. Alexander et al. (1979) \cite{3} and Growe et al. (1979) \cite{37} also opined that lithium is not disease-specific for mania.

In the past, a great deal was written about the use of lithium for the control of violence (Fieve, 1989 \cite{29}; Marini et al., 1977 \cite{63}; Micer et al., 1974 \cite{67}; Morrison et al., 1973 \cite{68}; Sheard et al., 1976 \cite{88}, reviewed in Breggin, 1983b \cite{14}). While these claims have not been confirmed, they focus once again on the tendency to use or advocate lithium for a variety of purposes.

8.8 How Effective Is Lithium in Preventing the Recurrence of Manic Episodes?

Lithium has been promoted so strongly within psychiatry and to the public as a method of preventing recurrences of mania that few practitioners or consumers doubt its efficacy. In reality, lithium’s effectiveness in this regard remains questionable. At the height of lithium’s popularity, Prien et al. (1974) \cite{76} reviewed the literature and found that studies showed a relapse rate as high as 50% over 2 years during lithium prophylactic treatment. Lithium did reduce the number of manic episodes in patients who had a history of infrequent attacks. But in patients with a high rate of past manic episodes, lithium did no better than placebo, and all patients in this group eventually relapsed. If lithium were a disease-specific treatment, it surely would have performed better than this.

Continuing research has been even more discouraging. Gitlin et al. (1995) \cite{32} conducted a prospective study of patients treated with lithium bipolar disorder. The patients were carefully monitored for effective drug treatment. Despite this, 73% of the patients relapsed into mania depression within 5 years. Of those who relapsed, two-thirds had multiple episodes. Even among those patients who did not completely relapse, many suffered serious emotional difficulties. The authors concluded, “even aggressive pharmacological maintenance treatment does not prevent relatively poor outcome in a significant number of bipolar patients” (p. 1635).

8.9 Mania and Depression As Lithium Withdrawal Reactions

Although little notice was given of the phenomenon within the profession, I recall my own patients telling me about painful emotional reactions that they suffered during lithium withdrawal. The evidence is now substantial in regard to serious adverse psychiatric effects caused by lithium withdrawal.

Suppes et al. (1991) \cite{98} analyzed 14 studies and found that the rate of relapse into mania increased following the discontinuation of lithium. e patients, who tended to cycle into mania about once a year (mean .6 months), developed a new episode less than 2 months (mean 1.7 months) after stopping their medication. In other words, discontinuation treatment with lithium produced a much more rapid onset of mania than the untreated patients would have endured.

Numerous studies have now confirmed that withdrawal from lithium causes adverse psychiatric
reactions. Cavanagh et al. (2004) \[17\], in a 7-year follow-up, found that lithium withdrawal caused both mania and depression. They concluded, “These results confirm that acute discontinuation of lithium leads to a high immediate relapse rate.” However, they did not find that this justified the continuation of lithium. To the contrary, “outcome was not worsened by discontinuation”.

Unfortunately, patients who relapse soon after taking lithium are rarely, if ever, told that their relapse was probably caused by lithium withdrawal. Instead, they are told that the new manic episode proves the need to take the medication for the rest of their lives.

Many psychiatrists advise patients who are diagnosed bipolar manic that they must take lithium for many years, or even for the rest of their lives. They are told that it is irresponsible for them not to do so. Families and psychotherapists are pressured to urge or coerce patients to take their lithium. The data do not confirm this strong advocacy for drug.

On the basis of the general observation that the brain tends to fight back against psychoactive interferences in the brain, any medication used to control mania should be viewed as having the potential to cause mania during withdrawal. For example, Jess et al. (2004) \[44\] described a case of rebound mania during withdrawal from carbamazepine.

### 8.10 Other Adverse Reactions to Lithium Withdrawal

Swartz and Jones (1994) \[100\] reviewed the literature and presented three cases concerning severe and often persistent adverse reactions to the abrupt withdrawal of lithium in patients suffering from elevated serum levels during routine treatment. One of the patients became severely demented. In their review of 50 cases obtained from the Lithium Information Center of the University of Wisconsin, they found that many patients became demented or otherwise deteriorated severely when abruptly withdrawn from lithium. Patients subjected to kidney dialysis for lithium toxic often deteriorated mentally with a rapid drop in lithium levels. Neurologic sequelae persisted in 30% of the 50 patients. The authors found substantial neurotoxic risks in rapidly withdrawing patients from high lithium levels.

If rapid withdrawal from high lithium levels can produce mania and disable neurologic reactions, then it is probable that rapid withdrawal from lower levels may produce more subtle adverse reactions.

### 8.11 Lithium in Your Drinking Water

In 1970 \[23\], Dawson et al. tried to support a fantastic thesis: Increased rainfall dilutes certain minerals in reservoirs, including lithium, producing a correlation between areas of lesser rainfall, higher lithium levels in drinking water, and a lower incidence of mental illness as measured by hospital admissions. In Psychiatric Drugs (1983b), I examined and debunked the study and its various supporters (see Fieve, 1989 \[29\]; “Texas,” 1971). The researchers recommended putting lithium in the drinking water, much like drinking water has been fluoridated. Perhaps this is the logical extension of absurd claims that psychiatric treatments correct biochemical imbalances without adversely affecting the brain.

### 8.12 Other So-Called Mood Stabilizers

Three antiepileptic drugs have now been FDA approved as mood stabilizers for the prevention of recurring episodes of mania: divalproex sodium (Depakote), extended-release carbamazepine
(Equetro), and lamotrigine (Lamictal). Many of these drugs are prescribed to children for the control of epilepsy and, increasingly, for bipolar disorder. A critical question is their effect on the developing mental and emotional function of children, but there is little research on the subject (Loring, 2005 [61]).

Valproic acid (Depakene), sodium valproate (Depakene syrup), and divalprox sodium (Depakote, enteric-coated combination of the other two) are forms of an antiepileptic agent that has been approved by the DA for the treatment of bipolar disorder. The drug can be hepatotoxic, especially in children. From the brain-disabling perspective, it can cause sedation, tremor, and ataxia. More rarely, it can cause adverse changes in mood and behavior, including behavioral automatisms, aggression, and confusion. Somnolence or delirium can develop, especially when combined with other sedatives (Silver et al., 1994 [91]). There may be “mild impairment of cognitive function with chronic use” (Hyman et al. [42], 1995, p. 127). Like lithium, valproic acid causes delirium in a significant percentage of older patients (Shulman et al., 2005 [90]). It also causes a variety of endocrine disorders and metabolic changes (Verrotti et al., 2005 [102]). Clinically, I have seen this drug cause depression and hostility.

Of as yet unknown consequence to the brain and nervous system, here are many studies indicating that valproic acid promotes a variety of potentially dangerous viruses (e.g., Fan et al., 2005 [27]). Both valproic acid and carbamazepine cause a small increase in the rate of major congenital malformations in infants (Wide et al., 2004 [105]). Acute and potentially fatal pancreatitis has been reported with valproic acid (e.g., Grauso-Ely et al., 2003 [35]). Liver failure is a known problem as well. Valproic acid is known to cause hyperammonemia with encephalopathy (e.g., McCall et al., 2004 [63]). Severe and even lethal skin disorders can occur with all of the antiseizure medications now used as mood stabilizers. The various adverse effects of valproic acid and other mood stabilizers are not nearly as benign as physicians believe in their eagerness to switch patients from lithium.

Carbamazepine (Tegetrol) is closely related to the tricyclic antidepressants. In neurological medicine, its principal uses are as an anticonvulsant for partial complex seizures and in the management of tic douloureux, a facial pain syndrome. It causes similar brain-disabling effects to the older antidepressants, including sedation, tremor, confusion, depression, psychosis, and memory disturbances (chapter 1). Cognitive disturbances are more common with concomitant use of neuroleptics, with preexisting brain damage, and with aging (Hyman et al., 1995 [42]). In addition, it poses the threat of potentially lethal agranulocytosis or aplastic anemia. Carbamazepine can cause hypotension (low serum sodium), leading to a syndrome that includes lethargy, confusion or hostility, and stupor.

Clonazepam (Klonopin), a benzodiazepine tranquilizer, has been used to treat both acute mania and as prophylaxis. It has all the many, sometimes severe, problems associated with the other benzodiazepines, including sedation, rebound and withdrawal syndromes, addiction, and behavioral abnormalities (chapter 7). Neuroleptics remain the mainstay for controlling acute manic reactions.

Verapamil (Calan and others) is a calcium channel blocker used for the treatment of cardiac disorders that has also been used off-label as mood leveler. It can produce a variety of cardiovascular side effects.

Clonidine, an antihypertensive drug, also has been used in the treatment of mania. Sudden withdrawal can produce a rebound hypertensive crisis. Consistent with the brain-disabling principles, it can produce a variety of psychiatric symptoms, including sedation, vivid dreams or nightmares, insomnia, restlessness, anxiety, and depression. More rarely, it can cause hallucinations. Unfortunately, this drug is too commonly used as so-called mood stabilizer in children. When mistakenly prescribed with stimulants, it causes an elevated risk of cardiac arrhythmia and cardiac arrest in children.

Some clinicians will add a variety of antidepressants, including SSRIs like Prozac, to the treatment
of patients with bipolar disorder. Nearly a antidepressants can cause or worsen mania (chapter 12). Nonetheless, Eli Lilly managed to obtain FDA approval for Symbyax, a combination Zyprexa and Prozac, for the treatment of depressive episodes associate, with bipolar disorder. In reality, Prozac should not be prescribed to patients with bipolar disorder, given the frequency with which SSRIs cause and exacerbate manic reactions.

The lengthy list of attempts to substitute for lithium suggests, 001 again, that it is hardly a specific magic bullet for mania or bipolar disorder.

8.13 Why So Many "Bipolar" Patients?

When I was in my psychiatric training, we rarely saw a patient undergoing a florid manic episode. When a case was admitted, it would become a subject for grand rounds for everyone to see and evaluate. I can remember only a handful of such cases during nearly 4 years working in psychiatric hospitals. Nowadays, the diagnosis of bipolar disorder has become a fad, and many patients are given it without meeting the diagnostic criteria. But many other cases do involve patients who have undergone manic-like episodes. Why the increase? As we saw in chapters 7 and 6, the newer antidepressant drugs commonly cause mania.

When a patient develops a manic-like adverse drug reaction, the correct diagnosis, according to the official American Psychiatric Association (2000) [4] diagnostic manual, is substance-induced mood disorder. Yet I cannot recall a single patient who was properly diagnosed in this manner in either my clinical or forensic experience (Breggin, in press). Doctors do not want to admit to their own mistakes, and they do not want to disease the mistakes of their colleagues, so it is so much easier to diagnose patient as having a manic episode or bipolar disorder than as having adverse drug reaction with manic features.

Even when the drug is such an obvious culprit that its role cannot be denied, the typical health care provider is likely to tell the patient and the family that the drug merely unmasked an underlying disorder. Instead of withdrawing the patient from the offending agent, the health care provider is likely to increase the dose or to add another drug, ultimately worsening the patient’s condition. But as the research in chapters 7 and 6 shows, many people with no past history of manic episodes are driven into manic-like states by antidepressant medication.

Chapter 7 will examine one of the great shames of my profession psychiatry: the increasing numbers of children diagnosed with bipolar order and medicated with adult mood stabilizers and neuroleptics.

8.14 Conclusion

Lithium is a highly neurotoxic substance with a generally suppressive effect on neuronal function and mental function in the commonly prescribed therapeutic range. It is poisonous to brain cells. The much promoted concept that lithium and other "mood stabilizers" are somehow "protective" of brain cells is fantastical.

Lithium has no specific therapeutic effect on mania or other states of overexcitement. Its brain-disabling effect is not specific for patients diagnosed as manic or bipolar. Lithium will subdue or suppress the mental and physical functioning of animals, newborn infants and nursing infants of mothers who take lithium, and normal volunteers, as well as people diagnosed with psychiatric disorders. Lithium-treated volunteers suffer devastating effects on their ability to relate and to function intellectually. Animals show similar taming effects.
Lithium is highly spellbinding. Normal volunteers fail to perceive how impaired they have become, and patients given therapeutic doses easily become severely toxic without perceiving their deteriorating clinical condition. Patients treated long term with lithium typically fail perceive how subdued they have become or how impaired their memories have become.

The various alternatives to lithium have their own brain-disabling effects, and none of the drugs is specific for mania.

Although lithium possesses these suppressive properties, it is not as effective in controlling mania as the neuroleptics, especially in acute mania or in severe, recurrent mania. This is partly because lithium is too overwhelming in toxicity in doses sufficient to subdue severely disturbed or rebellious individuals.

The claim that lithium is a disease-specific therapy for mania or manic-depressive (bipolar) disorder has no basis in fact; it is a brain disabling agent. Its efficacy has been exaggerated, and its adverse effects on the brain and mind, as well as the body as a whole, have been too frequently minimized.
Bibliography


