AN EMERGING EPIDEMIC?

Emil Kraepelin, widely considered the leading psychiatric classifier of the late 19th century (Horwitz, 2002, p.38), was among the first to write a modern comprehensive nosology of psychiatric disorders¹. In his classification of mental disease, he focused primarily on two different types of disorders: psychotic disorders, including schizophrenia, and affective disorders, including bipolar disorder (Kraepelin & Diefendorf, 1912)². In Kraepelin's time and before, bipolar disorder and its recurrent states of mania, depression, and mixed mood episodes, was considered a serious and debilitating mental disease. Vincenzo Chiarugi in Tuscany (1759-1820) wrote: "Mania signifies raving madness. The maniac is like a tiger or a lion, and in this respect mania may be considered as a state opposite to true melancholia" (Angst & Marneros, 2001, p.7). In modern American psychiatry, bipolar disorder is conceptualized as consisting of two subtypes: bipolar-I and bipolar-II. The Diagnostic and Statistical Manual IV-TR (DSM-IV) estimates the prevalence of bipolar-I disorder to be between .4-1.6%, and the prevalence of bipolar-II disorder to be approximately .5% (American Psychiatric Association, 2000, p. 382-404).

If we fast-forward 100 years from the time Kraepelin first used the term manicdepressive insanity, and only 15 years from the time bipolar-II disorder was introduced into the DSM-IV in 1994 (American Psychiatric Association), estimates of the prevalence of disorders on the bipolar spectrum have risen dramatically. Leading bipolar researchers have recently estimated that the population incidence of bipolar-II disorder alone may be as high as 10.9% (Angst et al., 2003, p. 139). The occurrence of subthreshold variants on the bipolar spectrum, less clinically significant than bipolar-II, have been estimated at 9.4% (Angst et al., 2003, p. 139). Some researchers place the total community presence of most forms of bipolar illness as high as 24.2% (Angst et al., 2003, p.139). Secondary analyses of the US National Epidemiological Catchment Area Survey of Mental Disorders (ECA) provide evidence that subthreshold cases on the bipolar spectrum are at least four times more prevalent than diagnoses based purely on DSM-IV criteria (Judd & Akiskal, 2003, p. 127). Some bipolar researchers who argue for a revision of the diagnostic criteria for bipolar disorders suggest that, under the proposed criteria, up to 30% of patients currently diagnosed with major depressive disorder are actually misdiagnosed bipolar-II patients (Benazzi, 2006, p. 26). Another study found that 61% of outpatients seeking treatment for depression actually met the proposed revised criteria for bipolar-II disorder (Benazzi & Akiskal, 2003, p. 35).

The staggering increase in estimates of the prevalence of bipolar disorders in the community suggests an alarming epidemic of a family of serious mental disorders. The rapid ascendancy of the estimated prevalence of these disorders foretells a strain on the nations' mental health facilities and services. An epidemic of this scale would result in a marked increase in chronically disabled persons, with an estimated functional recovery rate reported to be as low as 37% over a 2-year period (Tohen et al., 2000, p.220). The 61% increased risk of death for persons with bipolar disorders (Angst et al., 1999 p.58) would devastate families, the economy, and the social structure. The gravity of this rapidly growing public health crisis inspires understandable apprehension, but there is more to the bipolar epidemic than is immediately apparent.

In this chapter, I argue that there are multiple interpretations of the rise in the estimated prevalence of disorders on the bipolar spectrum. I suggest that these

statistics do not in fact represent an increase in the number of people who experience the symptoms described by Kraepelin, and more recently classified as bipolar-I and bipolar-II in the DSM IV. I will explore the social processes and academic debates that have defined and re-defined bipolar disorders. Disease classifications are necessarily social products that adhere more or less closely to biological reality. When these classifications, or indeed, the very concept of epidemics, become reified as facts of nature, we lose sight of the origin of these concepts: human beings and the social worlds in which we live (Berger & Luckmann, 1967, p. 89). Human beings who manage competing social, political, economic, and intellectual interests³ create disease classifications, including those of bipolar disorders. In the rest of this chapter, I will first briefly outline the theoretical concept guiding this work, the social construction of mental illness, as well as the competing paradigm of the biological disease perspective of mental illness. After this quick overview of the intellectual debate that frames the current discussion about bipolar disorders and their ostensible epidemic proportions, I will narrow the discussion to focus primarily on two of the social causes, or 'culprits' for the apparent epidemic of these disorders.

SOCIAL CONSTRUCTIONS OR BIOLOGICAL ILLNESS?

There is no consensus regarding the "reality" of psychiatric illness categories. Two opposing points of view pit a social constructionist perspective against the disease model of psychiatric illness, which contends that diagnostic categories represent purely biomedical illness (Horwitz, 2002, pp.5-10). At it's most basic level, social construction theories contend that all systems of knowledge and ways of understanding are reflections of culturally specific processes. Our world is inseparable from the social

processes that allow us to comprehend and organize that world. Social constructionist scholars do not assume that taken-for-granted categories represent any natural reality, but instead they reflect and respond to shifting social forces (Berger & Luckmann, 1967). The pure social constructionist perspective on mental illness posits that diagnostic categories serve only to classify behavior as deviant or abnormal. Normality, therefore, is culturally created and there exists no universal normality in the same way as there exists no universal morality (Benedict, 1934, p. 73). Benedict was one of the first scholars to question psychiatric diagnostic categories as absolute, and asked, "In how far can we regard inability to function socially as diagnostic of abnormality, or in how far is it necessary to regard this as a function of the culture?" (1934, p. 60). Some scholars argue that the entire idea of mental illness is a fallacy, and that attributing problems with living to mental illness is similar to attributing problems to witchcraft, demons or fate (Szasz, 1960, p.117). Szasz (1960, p.118) contends that mental illness is a myth whose function is to disguise and obscure the moral conflicts in human relations. Addressing how the concept of mental illness became accepted and reified as a biological fact, Szasz writes:

"Mental illness, of course, is not literally a thing or physical object, and hence it can exist in only the same way that other theoretical concepts exist. Yet, familiar theories are in the habit of posing, sooner or later, at least to those who come to believe in them, as objective truths or facts" (1960, p.113).

Opposing the social constructionist perspective⁴ is the biological disease paradigm, which is currently in favor within the psychiatric profession. Psychiatric researchers, clinicians, and laypeople alike generally view mental illnesses as

biomedical diseases of the brain no different from other illnesses (Horwitz, 2002, p.5). American psychiatrists do not typically view the disease categories within the DSM as simply one, but not the only, way of viewing mental illness (Horwitz, 2002, p.5). In fact, most mental health professionals regard mental illness categories as defined in the DSM as natural entities and not as evolving social constructions (Leshner, 2001, pp.77-79). This perspective asserts that diseases, both physical and mental, presumably exist in nature as clearly defined entities, regardless of the social meaning attached to them (Horwitz, 2002, p.5). As diagnostic categories change with the release of new versions of the DSM, adherents to the disease model presume that these changes demonstrate that scientific and diagnostic knowledge is getting stronger, not that earlier conceptions or classifications were inaccurate or socially constructed.

Whether or not psychiatric disease categories are socially constructed, psychiatric diagnostic categories have a profound impact on clinical and community epidemiological studies that attempt to quantify the prevalence of specific mental illnesses. These epidemiological surveys necessarily rely on predetermined categories. However, by reifying and naturalizing these categories, epidemiological studies obscure the social processes that define and redefine what counts as a mental illness. I will now explore two of the social culprits that contribute to the illusion of a bipolar epidemic: the subthreshold and spectrum conception of bipolar disorders, and the expanding definition of mania. As the definition of illnesses expand or contract through social processes, epidemiological studies that quantify disease prevalence will necessarily reflect these social changes. In the case of bipolar disorders, the social redefinition of these disorders has created the impression of an epidemic.

CULPRITS: SUBTHRESHOLDS AND SPECTRUMS

The most recent version of the psychiatric disease classification manual that is used for the diagnosis and treatment of patients, as well as for epidemiological attempts to quantify the prevalence of mental illnesses, is the DSM-IV-TR. This manual tends to conceptualize mental illnesses as discrete, non-continuous categories. However, the newer psychiatric literature that is being used to create the anticipated DSM-V, scheduled to be released in 2012 (American Psychiatric Association, 2008), relies heavily on conceptions of mental illness as being on a continuum, or spectrum, of illness. This literature examines subthreshold conditions⁵ in which a person may meet some, but not all, of the DSM-IV diagnostic criteria for a particular disorder. A shift to a broader conception of mental disorders could lead to many more people potentially being placed on a spectrum of mental illness. This has important implications for epidemiological research that suggests that rates of particular illnesses are rising, creating the illusion of an epidemic.

Despite being different concepts, the subthreshold and spectrum movements within psychiatry have a similar impact on epidemiological studies that suggest an impending epidemic of bipolar disorders. Patients are said to have a subthreshold, or subclinical condition, when some, but not all, of the DSM-IV criteria for a specific diagnosis are met. A subthreshold presentation might also be identified if a patient meets modified, or reduced, versions of the diagnostic criteria. Including subthreshold symptoms as indicative of mental illness, and considering people with these symptoms for inclusion in epidemiological studies that ostensibly count people with the illness is a psychiatric analogue to counting everybody with freckles as having melanoma. For

adherents to the medical model of psychiatry, a patient with subthreshold presentation may represent the beginning of a more serious illness that requires treatment in order to prevent a worsening of the condition. Specifically in the case of bipolar disorders, the identification of subthreshold conditions is considered particularly important because pharmaceutical treatment for those with bipolar disorders, even at the subthreshold level, are typically different than treatment for those with anxiety or depressive disorders (Akiskal & Mallya, 1987, pp.69-70; Altshuler et al., 1995, p.1130). However, for those who subscribe to a social constructionist view of mental illness, the expanding definition of illness to include subthreshold conditions merely increases the number of individuals who qualify for a psychiatric diagnosis and treatment. The inclusion of subthreshold conditions in epidemiological studies may provide the appearance that the rates of particular illnesses are rising. However, prevalence rates rise as a direct result of the expansion of the definition of the illness that captures more people in its more inclusive net. For example, in a study that tested expanded definitions of 8 psychiatric disorders, looking at subthreshold variants of major depressive disorder, bipolar disorders, eating disorders, generalized anxiety disorder, alcohol abuse, substance abuse, conduct disorder, and ADHD, almost 53% of outpatient adolescents had at least one subthreshold disorder (Lewinsohn et al., 2004, p.620), far greater than the rates of these disorders when subthreshold conditions are not considered.

The spectrum conception of mental disorders goes one step further than the subthreshold movement, which primarily expands diagnostic categories to include more people with fewer or less severe symptoms. The spectrum model, on the other hand, sees mental illness and health on a continuous scale and not as sets of discrete

categories (Lewinsohn et al., 2000, p.345). Proponents of the spectrum model of mental disorders see both subthreshold and DSM-diagnosable conditions as being part of the same biopsychiatric process. From this perspective, the main difference between points on the spectrum is merely a matter of degree (Cox et al., 1999, p.20). In addition to bipolar-I and bipolar-II, which are described in the DSM-IV, such researchers propose adding a bipolar-1½ and 2½ (Akiskal & Pinto, 1999, p.519), bipolar-III (Akiskal & Mallya, 1987, p.69), as well as bipolar variants 3½, IV and V (Akiskal & Pinto, 1999, p.527-532). Still others suggest that certain forms of late-life dementia should be considered as part of the bipolar spectrum, as bipolar-VI (Ng et al., 2008, p.308). Although scholars disagree about the number of distinct disorders on the spectrum, they all agree that the spectrum includes distinct disorders in addition to subthreshold variants, and that even the distinct disorders seamlessly blend together on the continuum. Angst and Marneros (2001, pp.13-14) suggest a bipolar spectrum with 10 steps, as seen below in Figure 1.



Fig 1. Bipolar continuum. Adapted from Angst and Marneros, 2001, p.14.

The first node on Angst and Marneros' proposed spectrum (2001, p.14) consists

of non-pathological personality types; hyperthymic and cyclothymic temperaments.

Akiskal and Mallya (1987, p.71-72) suggest characteristics of the hyperthymic

personality to be:

*Chronic short sleeper (<6 hours, including weekends)

*Excessive use of denial

*Irritable, Cheerful, overoptimistic or exuberant

* Naïve, overconfident, self-assured, boastful, bombastic, or grandiose

*Vigorous, full of plans, improvident, and rushing off with restless impulse

*Over talkative

*Warm, people seeking, or extraverted

*Over involved and meddlesome

*Uninhibited, stimulus seeking, or promiscuous

In essence, this proposed bipolar spectrum includes a larger variety of human difference than has ever before been considered pathological. What was once normal is now mild disorder. We have certainly all known people with a "hyperthymic temperament" as described above, however, troublesome people have typically been just that—troublesome, but not disordered. Including personality types, even those that are arguably problematic for personal relationships, in the bipolar spectrum, changes the question from "are you bipolar?" to "to what degree are you bipolar?" As definitions of pathological mental illness get more and more broad, it is inevitable that epidemiological estimates of the prevalence of these disorders will rise.

The spectrum conception of mental illness presumes that health and illness are not categorically distinct, but instead lay on a continuum (Horwitz & Wakefield, 2007, p.126). In this way of thinking, health (or only very mild illness) is on the far left of the continuum, and severe mental illness lays on the far right of the continuum. Most individuals would lie somewhere in between these two extremes. Continuum notions of mental illness conceive virtually the entire population to be ill to some degree (Horwitz & Wakefield, 2007, p.126). However, this continuum notion does not allow for the concept of healthy and normal variations in mood. For example, disordered mood fluctuations give bipolar sufferers the extreme and dangerous highs of mania, and the profound depth of despair that characterizes depression and suicide. However, normal variation in mood certainly includes daily joy, even moments of euphoria, and sadness and grief. When normal and healthy fluctuations in mood are pathologized by being placed on the bipolar spectrum, the risk for false-positive bipolar diagnoses in clinical practice and

epidemiological studies increases. In fact, an alternative way to conceive of the bipolar spectrum is to imagine two continuums: one of healthy mood variation, and one of disordered mood variation (Horwitz & Wakefield, 2007, p.140). Imagining a spectrum of healthy variation as separate from the spectrum of disorder reduces the possibility that people with non-pathological mood states are considered in epidemiological estimates of the rate of bipolar disorders.

Although conceptions of the bipolar spectrum are currently in vogue among bipolar researchers, by no means is there clear agreement within the psychiatric profession that bipolarity should be considered a spectrum disorder. In his "Plea for the Integrity of the Bipolar Disorder Concept," Baldessarini (2000, p.5) writes:

"Widespread acceptance of increasingly broad definitions risks weakening or trivializing the core concept of Bipolar disorder, much as what has occurred in the past with schizophrenia, major depression and a growing number of other disorders whose academic and clinical popularity has waxed and waned." Critics of the spectrum concept of bipolar disorder worry that the inclusion of

subthreshold conditions on the bipolar spectrum pathologizes normal behavior as well as trivializes serious illness. In their report about the danger subthreshold variants on the bipolar spectrum pose to research on the causes and treatments of bipolar-I disorder, Soares and Gershon (2000, p.1) write:

"Increasingly broader definitions of Bipolar disorder are frequently justified under the assumption that these conditions have some resemblance in some of their symptoms and also appear to respond in various degrees to some of their treatments [...] The move towards inclusion of increasingly heterogeneous

groups of patients under this diagnostic category threatens to jeopardize clinical research and would be a disservice to the field."

Unfortunately for the integrity of epidemiological estimates of bipolar disorder, the voices of the spectrum critics do not appear to have the same persuasive power as those arguing for an increasingly inclusive spectrum. It is likely that the forthcoming DSM-V will even further promote the spectrum conception of bipolar illnesses. This will ensure that epidemiological studies will begin to record an increase in the prevalence of bipolar disorders, as the definitions are expanded and the spectrum becomes reified in the manual.

In their argument for a spectrum conception of psychiatric disorders, Lewinsohn et al., (2004, p.613) write:

"Under the DSM-IV, an individual is determined to have a particular disorder when he or she exceeds a cut-off of a diagnostic algorithm [....] While one hopes that the criteria and cut-off are based on sound empirical and clinical considerations, the fact that these systems are continuously revised and that few are entirely satisfied with them suggests that the determination of the caseness of a disorder is ultimately somewhat arbitrary."

It is this very arbitrariness that makes subthreshold and spectrum movements within psychiatry potential culprits for the appearance of an epidemic. If disease categories are rather arbitrarily constructed and re-constructed, then no epidemiological study that presumes to count the prevalence of a disease in the community can be seen as capturing an immutable, biological fact. Instead, as disease categories themselves are created through social processes, epidemiological estimates reflect only those social

processes that have defined the disorder in question. The impact of the definition of disorder on epidemiological studies is striking: estimates of the prevalence of bipolar-I disorder are 1.6% (Kessler et al., 1994, p.12) but up to 24.2% (Angst et al., 2003, p.139) for the entire bipolar spectrum.

CULPRIT: EXPANDING MANIA

Threshold and spectrum conceptions of bipolar illness expand the range of symptoms that are considered abnormal, encompassing more people. Subthreshold conditions and the bipolar spectrum work together with the second culprit of bipolar epidemics. The redefinition and weakening of the important symptom of mania also contributes to the illusion of a bipolar epidemic. The key feature that differentiates bipolar-I from bipolar-II and other forms of disorders on the bipolar spectrum is the type of mania experienced by individuals. Individuals with bipolar-I experience mania, which is characterized by nonstop activity, euphoria, racing thoughts, thoughts of grandiosity ("I am Don Juan") and frequently, psychotic delusions (Akiskal & Pinto, 1999, p.520). Manic episodes often require hospitalization to prevent dangerous and destructive behavior such as excessive gambling, reckless driving, sexual promiscuity, shopping, impulsive traveling, and drug and alcohol use. Individuals diagnosed with bipolar-II and other bipolar variants experience a less severe form of mania known as hypomania. The DSM-IV (American Psychiatric Association, 2000, p.368) characterizes hypomania as:

(a) Periods of elevated or irritable mood (mood changes), which must always be present and must last at least 4 days, different from the usual mood;

(b) 3 of the following 8 symptoms if mood is elevated, 4 if mood is irritable:

inflated self-esteem, decreased need for sleep, more talkativeness, racing thoughts, distractibility, increased goal-directed activity, psychomotor agitation, and excessive involvement in risky activities;

(c) Change in functioning;

(d) Observable mood and functioning change;

(e) No marked impairment of functioning, no psychotic symptoms; and

(f) Symptoms must not be caused by substances, drugs (including antidepressants), or medical disorders

The identification of hypomania as a psychiatric symptom, rather than a part of normal human experience, was one of the first stages in the broadening of bipolar disorder from a discrete, serious illness, to a spectrum disorder ranging from mild to serious distress. Due to its typically relatively benign nature, hypomania is often unrecognized by physicians, and as a result, patients who might meet the criteria for bipolar-II disorder are frequently diagnosed with major depressive disorder (Benazzi, 2006, p.26). For proponents of a spectrum conception of bipolarity, this represents a worrisome and problematic misdiagnosis (Bhargava et al., 2007, p.264). As result, some pro-spectrum researchers argue for a reduction in the number of days that hypomania must be present in order to qualify for a diagnosis of bipolar disorder. Currently, the DSM-IV (American Psychiatric Association, 2000, p.368) requires hypomania symptoms to be present for a minimum of four days at least once in a patient's life in order to qualify for a bipolar-II diagnosis. Some psychiatrists suggest that this is too conservative, and that a bipolar disorder may in fact be present if hypomania symptoms are present for two days or more (Benazzi, 2006, p. 26; Benazzi & Akiskal,

2003, p. 35; Cassano et al., 1992, p.131; Manning et al., 1997, p.105). In arguing for a lower threshold for a patient to meet the criteria for hypomania, Akiskal et al., (2000, p.5) suggest that most hypomania episodes last between 1-3 days, and they bemoan the arbitrary 4-day cutoff currently enshrined in the DSM-IV. However, other researchers have found that, at least in children, hypomanic episodes last for a minimum of 4 days, with a mean length of 12 days (Bhargava et al., 2006, p.264). The key feature that differentiates bipolar disorders from other psychiatric conditions—mania or hypomania—is still contended by leading researchers in the field. The final outcome of this academic debate will significantly affect epidemiological studies that attempt to quantify the number of people with these disorders. As the threshold for hypomania continues to drop, more and more people will potentially be considered as having a variant of bipolar disorder.

In addition to arguing for a reduction in the minimum number of days in which hypomania must be present in order to qualify for a bipolar diagnosis, researchers who are concerned about the under diagnosis of bipolar disorders propose that revised diagnostic criteria would better capture hypomanic symptoms. They suggest that hypomanic diagnostic criteria that require the presence of an elevated or irritated mood might inappropriately screen out people who may in fact have a bipolar spectrum disorder. In an article about the misdiagnosis of bipolar-II disorder, Benazzi (2006, p.26) writes, "Because the DSM-IV stem question requires remembering periods of elevated or irritable mood, the response to this question by patients with BP II is frequently "no," since these periods may be seen as normal mood fluctuations." In order to reduce the misdiagnosis of pseudo-unipolar depression (Angst et al. 2003, p.144), some

researchers have proposed new diagnostic criteria to screen for hypomania, which would presumably allow many more people to qualify for the diagnosis. In part, the proposed diagnostic criteria (Angst et al., 2003, p.144) requires:

Over the last 12 months and, without any special reason, have you, for any length of time,

- been much more energetic
- been more active
- been less easily tired
- needed less sleep
- been more talkative
- traveled around more
- been busier etc.?

Was this so evident that you had problems with it yourself, it caused you problems with others or it got you into financial difficulties?

Did other people (e.g., family members, partner, etc.) notice these states in you and come to the conclusion that something must be wrong with you?

Proponents of both the expansion of the bipolar spectrum and the redefinition of hypomania argue that the "misdiagnosis" of bipolar disorders, even subthreshold variants, as major depressive disorder can be extremely dangerous to patients. Psychoactive drugs that are prescribed for the treatment of depression may be ineffective, or even dangerous, in treating any bipolar spectrum disorder (Akiskal and Mallya, 1987, p.70). In this way, the impending bipolar epidemic can be conceived of as a shifting epidemic. By lowering bipolar criteria and expanding the bipolar spectrum, the immediate result will be the reclassification of patients currently diagnosed with depression or anxiety to be diagnosed instead with having bipolar disorders. This is turn will effect epidemiological estimates of the rates of both bipolar disorders and the rates of depression.

The proposed changes in criteria intend to classify behavior that the patient may see as "normal mood fluctuations" (Benazzi, 2006, p.26) as characteristic of an illness that requires medical treatment, specifically, mood-stabilizing drugs. Patients infrequently seek treatment for hypomanic symptoms because they are frequently seen as a period of improved functioning. Impairment from hypomania, when it occurs, is mild. Persons with hypomanic symptoms seldom complain of, or suffer from, their shifts in energy, activity and sleep behavior, but tend to experience them as positive. Most such changes, if noticed at all, are likely to be recognized by family and friends (Angst et al., 2003, p.134). In fact, patients experiencing hypomania function so well they don't recognize it as illness at all (Akiskal & Mallya, 1987, p.68). The result is that bipolar-II, and other bipolar spectrum disorders, are characterized primarily by a symptom, hypomania, that is generally perceived as nonproblematic and a part of normal behavior. This is in contrast to virtually every other disorder in the DSM-IV, which requires symptoms to either cause significant distress to the patient, or marked dysfunction. As the threshold for what qualifies as hypomania, in both duration and character, continues to drop, bipolar spectrum diagnoses will include even more symptoms that most individuals do not consider marked difficulties. This, in turn, will be reflected in epidemiological estimates of the prevalence of bipolar disorders.

CONCLUSION: WHAT ABOUT THE DRUG COMPANIES?

Frequently in public discourse about increasing rates of mental illness diagnoses, pharmaceutical companies and their aggressive marketing tactics (Thomson & Trotto, 2002, p. B1), as well as their ties to psychiatrists (Harris et al., 2007), are blamed. However, in this chapter I have not directly addressed the pharmaceutical industries' role in rising rates of diagnoses. I want to briefly explore the role of psychoactive drugs in the appearance of a growing bipolar epidemic, but I argue that these drugs have a dialectical relationship with the two main culprits described in this chapter, and are therefore secondary to them.

Trends in psychiatric diagnoses, or diagnostic fads which could contribute to the illusion of an epidemic, have historically followed the advent of new medication (Akiskal, 1983, p.271). For example, when Thorazine was invented in the 1950s, it preceded a spike in diagnoses of schizophrenia (Akiskal, 1983, p.271). Likewise, when lithium was approved for the treatment of bipolar disorder in 1970, rates of bipolar diagnoses rose over those of schizophrenia (Akiskal, 1983, p.271). In this same vein, when selective serotonin reuptake inhibitor (SSRI) drugs were made available for the treatment of depression, rates of diagnoses of clinical depression rose dramatically (Horwitz & Wakefield, 2007, p.183). With the advent of atypical antipsychotics and safe anticonvulsant medications for the treatment of bipolar-II and subthreshold variants of bipolar disorder, rates of these diagnoses have risen.

Pharmaceutical companies aggressively market drugs directly to consumers (Bell et al., 2000, p.329), and this affects how often patients are prescribed particular drugs. Also contributing to the rates of specific diagnoses, and therefore, drug treatment for these disorders, are ethically and professionally questionable relationships between

drug companies and prescribing physicians. For example, it has been widely reported that more than half of the taskforce members who are working on the newest edition of the DSM, the DSM-V, have ties to the pharmaceutical industry (Parker-Pope, 2008).

However, while the relationship between pharmaceutical companies and rising rates of psychiatric diagnoses is clear, the advent of new drugs alone does not directly increase the rates of diagnoses. Instead, an intervening factor, the redefinition of disorder, and the expansion of conditions that are considered disordered, and therefore eligible for drug treatment, are the direct causes of a spike in diagnoses. Psychiatric drugs have a dialectical relationship with the definition and redefinition of bipolar disorders. For example, of the 59 authors of bipolar papers written after 1990 referenced in this chapter, 15 disclosed ties to drug companies that manufacture psychiatric drugs- a rate of over 25%. Leading bipolar researchers who directly influence and create the definition of bipolar disorders may themselves be influenced by drug companies, but it is this redefinition and expansion of the bipolar spectrum that is the proximal cause of the purported bipolar epidemic. The creation of new drugs may influence the creation of a longer and more inclusive bipolar continuum, and a longer continuum spurs the development of new drugs. Certainly a bipolar spectrum that includes more and more people who can be treated with mood-stabilizing drugs is beneficial to the pharmaceutical industry, but the definition of bipolar disorders must first be expanded in order for the industry to accrue these benefits.

The processes described in this chapter of the social construction of bipolar epidemics applies equally to rates of diagnosis of epidemic proportions of other psychiatric conditions such as depression, ADHD, anxiety, autism spectrum disorders,

and borderline personality disorder. As human beings enmeshed in complex social environments define and redefine conceptions of the normal and abnormal, epidemiological estimates of the prevalence of these disorders will shift in the direction of these changes. If diagnostic criteria tighten, fewer people will be considered abnormal and epidemiological estimates of the prevalence of those disorders will shrink. However, if diagnostic criteria expand, as in the case of bipolar disorders, more people will be considered disordered, and epidemiological reports will reveal an increase in the rates of affected persons. Epidemiological estimates of disease, especially of psychiatric disorders, do not quantify natural immutable processes. Instead, they reflect the very human social processes that define disorder and that are subject to the social, political, economic, and intellectual whims of human beings.

NOTES

1. One of the earliest known classification systems of mental illness was written in the 10th century by Arabian physician Najab ud-din Unhammad, which included nine major categories of mental disorders (Millon, 2004, p.38).

2. Kraepelin's classification used the terms manic-depressive insanity and dementia praecox to refer to those conditions known today as bipolar disorder and schizophrenia. The term schizophrenia appeared in the first edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-I) in 1952 (American Psychiatric Association, 1952, pp.26-28). The term bipolar was first used in 1957 by German psychiatrist Karl Leonhard (Goodwin & Jamison, 2007, p.9). Throughout this chapter, I will use the modern nomenclature.

3. Not only mental disorders are socially defined; our understanding and classification of many physical diseases and assumedly biological "facts" are strongly influenced by social processes. Two recent papers on this general topic explore the social construction of Lyme disease (Aronowitz, 1991), and the appearance and activity of human sperm and egg cells (Martin, 1991).

 4. Not all sociologists use the social constructionist framework to understand the phenomenon of mental illness. Much sociological work in the study of mental illness presumes the biological disease paradigm to guide their work. Examples include work about the relationship between mental health and family status (Umberson & Williams, 1999), class (Muntaner et al., 2008), and race (Evans-Campbell et al., 2008).
5. See for example: Akiskal & Mallya, 1987; Angst et al., 2003; Goodwin & Jamison, 2007; Judd & Akiskal, 2003; Lewinsohn et al., 2004; Lewinsohn et al., 2000.

ACKNOWLEDGEMENTS

I am grateful to Alan Horwitz, Sarah Rosenfield, Dena Smith, and Eunkyung Song for stimulating discussions and thoughtful reviews that greatly enhanced the ideas in this chapter.

WORKS CITED

Akiskal, H.S., 1983. The bipolar spectrum: new concepts in classification and diagnosis. *Psychiatry Update: The American Psychiatric Association Annual Review,* 2, pp.271–291.

Akiskal, H.S. Bourgeois, M.L. Angst, J. Post, R. Moller, H.J. & Hirschfeld, R., 2000. Reevaluating the prevalence of and diagnostic composition within the broad clinical spectrum of bipolar disorders. *Journal of Affective Disorders*, 59, pp.5-30.

Akiskal, H.S. & Mallya, G., 1987. Criteria for the soft bipolar spectrum: treatment implications. *Psychopharmacology Bulletin*, 23(1).

Akiskal, H.S. & Pinto, O., 1999. The evolving bipolar spectrum-prototypes I, II, III, and IV. *The Psychiatric Clinics of North America*, 22(3).

Altshuler, L.L. Post, R.M. Leverich, G.S. Mikalauskas, K. Rosoff, A. & Ackerman, L., 1995. Anti-depressant-induced mania and cycle acceleration: a controversy revisited. *American Journal of Psychiatry*, 152(8).

American Psychiatric Association (APA), 1952. *Diagnostic and Statistical Manual of Mental Disorders.* Washington, DC: American Psychiatric Association.

-----1994. *Diagnostic and Statistical Manual of Mental Disorders.* 4th ed. Washington, DC: American Psychiatric Association.

------2000. *Diagnostic and Statistical Manual of Mental Disorders.* 4th ed, text revision. Washington, DC: American Psychiatric Association.

------2008. *DSM-V Prelude Project*. [Online] Available at: <u>http://dsm5.org/index.cfm</u> [Accessed on-line November 11, 2008].

Angst, J. Angst, F. & Stassen, H.H., 1999. Suicide risk in patients with major depressive disorder. *Journal of Clinical Psychiatry*, 60, pp.57-62.

Angst, J. Gamma, A. Benazzi, F. Ajdacic, V. Eich, D. & Rossler, W., 2003. Toward a redefinition of subthreshold bipolarity: epidemiology and proposed criteria for bipolar-II, minor bipolar disorders and hypomania. *Journal of Affective Disorders*, 73(1-2), pp. 133-146.

Angst, J. & Marneros, A., 2001. Bipolarity from ancient to modern times: conception, birth, and rebirth. *Journal of Affective Disorders*, 67, pp.3-19.

Aronowitz, R. 1991. Lyme disease: the social construction of a new disease and its social consequences. *Milbank Quarterly*, 69(1).

Baldessarini, R., 2000. A plea for integrity of the bipolar disorder concept. *Bipolar Disorders* 2 pp. 3-7.

Bell, R.A. Kravitz, R.L. & Wilkes, M.S., 2000. Direct-to-consumer prescription drug advertising, 1989-1998. *The Journal of Family Practice*, 49(4).

Benazzi, F. 2006. Bipolar II disorder: current issues in diagnosis and management. *Psychiatric Times*, 23(9), pp. 26-29.

Benazzi, F. & Akiskal, H. 2003. Refining the evaluation of bipolar-II: beyond the strict SCID-CV guidelines for hypomania. *Journal of Affective Disorders*, 73(1-2), pp. 33-38.

Benedict, R. 1934. Anthropology and the abnormal. *Journal of General Psychology,* 10, pp. 59-82.

Berger, P.L. & Luckmann, T., 1967. *The social construction of reality.* New York: Anchor.

Bhargava Raman R.P. Sheshardri, S.P. Janardhan Reddy, Y.C. Girimaji, S.C. Srinath, S. & Raghunandan, V.N.G.P., 2007. Is bipolar II disorder misdiagnosed as major depressive disorder in children? *Journal of Affective Disorders*, 98(3), pp. 263-266.

Cassano, G.B. Akiskal, H.S. Savino, M. Musetti, L. & Perugi, G. 1992. Proposed subtypes of bipolar II and related disorders: with hypomanic episodes (or cyclothymia) and with hyperthymic temperament. *Journal of Affective Disorders*, 26, pp.127-140.

Cox, B.J. Enns, M.W. Borger, S.C. & Parker, J.D.A., 1999. The nature of the depressive experience in analogue and clinically depressed samples. *Behavior Research and Therapy*, 37, pp.15-24.

Evans-Campbell, T. Lincoln, K.D. & Takeuchi, D.T., 2008. Race and mental health: past debates, new opportunities. In Avison, W.R., McLeod, J.D. & Pescosolido, B.A., *Mental Health, Social Mirror*. Springer. pp.169-189.

Goodwin, F.K. & Jamison, K.R., 2007. *Manic-depressive illness: bipolar disorders and recurrent depression*. 2nd ed. New York: Oxford University Press.

Harris, G. Carey, B. & Roberts, J., 2007. Psychiatrists, children, and drug industry's role *New York Times,* [Internet] 10 May 2007. Available at: http://www.nytimes.com/2007/05/10/health/10psyche.html [Accessed November 22, 2008].

Horwitz, A.V. 2002. Creating mental illness. Chicago: University of Chicago Press.

Horwitz, A.V. & Wakefield, J.C., 2007. *The loss of sadness*. New York: Oxford University Press.

Judd, L. & Akiskal, H.S., 2003. The prevalence and disability of bipolar spectrum disorders in the US population: re-analysis of the ECA database taking into account subthreshold cases. *Journal of Affective Disorders* 73(1-2), pp. 123-131.

Kessler R. McGonagle, K, Zhao, S. Nelson, C. Hughes, M. Eshieman, S. Wittchen, H. & Kendler, K., 1994. Lifetime and 12-month prevalence of DSM-IIIR psychiatric disorders in the United States: results from the National Comorbidity Survey. *Archives of General. Psychiatry*, 5, pp. 8-19.

Kraepelin, E. & Diefendorf, A.R., 1912. *Clinical Psychiatry: A Textbook for Students and Physicians*. London: Macmillan.

Leshner, A.I., 2001. Addiction is a brain disease. *Issues in Science and Technology*, 17(3), pp.75-80.

Lewinsohn, P. Shankman, S. Gau, J. & Klein, D., 2004. The prevalence and comorbidity of subthreshold psychiatric conditions. *Psychological Medicine*, 34, pp.613-621.

Lewinsohn, P. Solomon, A. Seeley, J. & Zeiss, A., 2000. Clinical Implications of Subthreshold Depressive Symptoms. *Journal of Abnormal Psychology*, 109(2), pp.345-351.

Manning, J.S. Haykal, R.F. Connor, P.D. & Akiskal, H.S., 1997. On the nature of depressive and anxious states in a family practice setting: the high prevalence of bipolar II and related disorders in a cohort followed longitudinally. *Comprehensive Psychiatry*, 38(2), pp.102-108.

Martin, E. 1991. The egg and the sperm: how science has constructed a romance based on stereotypical male-female roles. *Signs*, 16, pp.485-501.

Millon, T., 2004. *Masters of the Mind: Exploring the Story of Mental Illness from Ancient Times to the new Millennium*. Hoboken (NJ): Wiley.

Muntaner, C. Borrell, C. & Chung, H., 2008. Class relations, economic inequality and mental health: why social class matters to the sociology of mental health. In Avison, W.R., McLeod, J.D. & Pescosolido, B.A., *Mental Health, Social Mirror*. Springer. pp.127-141.

Ng, B. Camacho, A. Lara, D.R. Brunstein, M.G. Pinto, O.C. & Akiskal, H.S., 2008. A case series on the hypothesized connection between dementia and bipolar spectrum disorders: bipolar type VI? *Journal of Affective Disorders*, 107, pp.307-315.

Parker-Pope, T., 2008. Psychiatry handbook linked to drug industry. *New York Times,* [Internet] 6 May 2008. Available at: <u>http://well.blogs.nytimes.com/2008/05/06/psychiatry-handbook-linked-to-drug-industry/</u> [Accessed 19 November, 2008].

Soares, J, & Gershon, S., 2000. The Diagnostic Boundaries of Bipolar Disorder. *Bipolar Disorders*, 2, pp.1-2.

Szasz, T., 1960. The Myth of Mental Illness, American Psychologist, 15, pp. 113-118.

Thomson, S.C. & Trotto, S., 2002. Washington U hosts program on depression among students. *St Louis Post-Dispatch*, [Internet] 13 November 2002. Available at: http://aisweb.wustl.edu/alumni/atwu.nsf/depression [Accessed 18 November, 2008].

Tohen, M. Hennen, J. Zarate, C.M. Baldessarini, R.J. Strakowski, S.M. Stoll, A.L. Faedda, G.L. Suppes, T. Gebre-Medhin, P. & Cohen, B.M., 2000. Two-year syndromal and functional recovery in 219 cases of first-episode major affective disorder with psychotic features. *American Journal of Psychiatry*, 157(2), pp.220-228.

Umberson, D. & Williams, K., 1999. Family Status and Mental Health. In Aneshensel, C.S. & Phelan, J.C., eds. *Handbook of the Sociology of Mental Health*. New York: Plenum. pp.225-253.