Prevalence of Type II Bipolar among Young People

Introduction

A simple search for "the most common misdiagnosed mental disorders" on the internet places bipolar related disorders high on the list. This prevalence is not only because the highly overlapping conditions between bipolar and other psychiatric disorders (Strakowski et al., 2011) but also because the symptoms per se could be sometimes viewed as simply being a human (Frances, 2013).

Bipolar disorder was once called manic depressive psychosis (Singh & Rajput, 2006). The term bipolar disorder (BD) emerged in 1980 and was written in the third edition of the DSM (Whitaker, 2015). Fourteen years later, in the DSM-IV, a new type of BD, named bipolar II, emerged. Different from the type I, instead of requiring a manic episode, the heightened mood of BD-II is so-called hypomania, which was considered to be a less serious version of mania (DSM-IV). Since the publication of the DSM-5, bipolar II is no longer to thought of as a "milder" version of bipolar I based on the belief of the DSM-5 editors that the impairment of BD-II could be as severe as BD-I (American Psychiatric Association, 2013). Although the intention behind these changes is good, some effects of this change have gone contrary to some experts' wishes.

Immediately after the publication of the DSM-5, the chairman of the DSM-IV, Dr. Allen Frances published a confession in the form of his book, Saving Normal, to disclose the dark side of psychiatric diagnosis. This further served as a warning not only to health services but also to the general public in the hope of stopping the artificial epidemic. (Frances, 2013) In this book, there is a chapter completely dedicated to his worries toward the misdiagnosis of bipolar disorder, especially bipolar II.

This essay aims to divide and evaluate every component behind the phenomenon of the recent high prevalence of BD-II among young people. The following sections will begin with the reason why BD-II was determined to be a distinct disorder. Meanwhile, discourse on the importance of diagnosis and the risk factors of over-diagnosing will evaluated. It is followed by an analysis of the necessity and downside of the recent medication commonly prescribed. Lastly, this paper will look at different developmental stages of humans alongside the criteria of BD-II to see if there are any potential elements which would cause a misdiagnosis.

Bipolar II: the Rise and Fad

Since the fourth edition of Diagnostic and Statistical Manual, DSM-IV, was published in 1994, a new category of bipolar disorder (BD) called type II bipolar has been introduced to the public. The reason why the editors added this new category is because the boundary between bipolar and unipolar depression is blurred, but the impact on patient prescriptions with ambiguous symptoms is huge (Frances & Jones, 2012; Frances, 2013). However, this has led to a concern of increasing prevalence and misdiagnosis of BD. Moreover, not so long ago, bipolar disorder among younger groups was considered to be rare (Parens & Johnston, 2010). Until the 1960s, there was not even a single case of juvenile bipolar disorder reported in the Journal, and less than 20 cases were "found" before DSM-IV (Carlson, 2011).

Following this, a significant forty-fold increase in youth bipolar (aged 0-19) diagnoses within merely eight years from 1994 was seen (Frances, 2013). This could be attributed to the DSM-IV's adoption of diagnostic criteria in childhood bipolar (Parens & Johnston, 2010; Moreno et al., 2007).

Even worse, during the editing period of the DSM-5, the working groups had once tried to lower the threshold of bipolar II by changing the hypomanic episode from four days to two days (Frances & Jones, 2012) because some experts declared the average duration of hypomania is 1 to 3 days (Singh & Rajput, 2006). They worried that patients who do not meet the four-days criteria could be misdiagnosed with major depressive disorder and be left without an appropriate prescription of antipsychotics and mood-stabilizers (Frances & Jones, 2012). Fortunately, under pressure from all sides, the final version of the DSM-5 kept the four-days rule and slightly restricted the criteria by adding an additional qualifier (Substance Abuse and Mental Health Services Administration, 2016). However, the changes were too minor to stop the fad, and they coincided with the fashionable notion of the efficacy of preventive medication. This could be seen as a forecast of another tide of misdiagnosis, whose victims will be our precious next generation.

Diagnosis: the Importance and Risks

To understand why diagnosis is so effective and why the DSM has moved into today's dominant position, we should trace back the historic path of psychiatric diagnosis. Before 1980, psychoanalysis still occupied the field of mental issues (Healy, 2008). At that time, the practical value of the first and second DSM was swept under the carpet. During the 1970s, the problems of psychoanalysis came to the fore. The public was shocked by research that showed a wide variety of diagnoses done by different psychiatrists within a simple case. At the same time, it became apparent how easy a psychiatrist could be fooled into sending a normal person to an asylum. Shortly after, Robert Spitzer led his team in editing the DSM-III to provide a clear and easy to follow manner to solve the prevailing problems within psychiatry (Frances, 2013). Moreover, the reason why this new system was so accepted by the public is because it offered a quick explanation and release for both patients and their families (Frances, 2013; Siegel, 2015).

However, in the past four decades, instead of advancing, the situation seems to have gotten worse. Certainly, sometimes diagnosis leads to positive outcomes, but a lot of the time, it causes harm. Perkins and colleagues conducted a systematic review of the experience of psychiatric diagnosis in 2018. In the review, the positive experiences include: helping patients to understand their experiences, providing a sense of relief, control, and hope for recovery, improving relationships with service, and reducing uncertainty. On the other hand, the negative outcomes could be: increased individual and social burden; feelings of hopelessness and frustration; stigma, and discrimination; exacerbated symptoms; disengagement from services (Perkins. et al., 2018). Frequently, positive and negative experiences coexist, or they sometimes alternate. An individual could be once happy with the diagnosis but face the misery of regret for the rest of their life. Many patients don't realize the power of stigma. Once a label is placed, it lasts forever, but relationships and society shift constantly.

In the long run, diagnosis causes negative identity (Perkins. et al., 2018). It could eventually become a self-fulfilling cycle. Especially in patients with BD, the symptoms are highly associated with people's moods, but having emotions is an essential aspect of the human experience. However, being diagnosed with BD could make patients frustrated by being forced to over-analyze their every single mood, and further exacerbate the illness. As a 25-year-old female bipolar patient said, "I feel like everything I do is now somehow connected to me being sick. If I'm happy it's because I'm manic; if I'm sad it's because I'm depressed. I don't want to think that every time I have an emotion, every time I get angry at somebody it's because I'm ill… some of my feelings are justified. People say I'm a different person everyday, but that's me! I've never been a stable person (cited in Miklowitz & Goldstein, 1997, p.157)."

Individual's feeling of diagnosis aside, two main reasons contribute to the importance of diagnosis: a) it provides a clear guideline toward a proper treatment, b) each case represents a data point which helps researchers devise a better treatment (Strakowski et al., 2011). If we look at the diagnosis of BD-II from the standpoint of the first argument, whether a patient should be diagnosed as having major depression or BD-II is merely a red herring. Controlled studies suggest that the typical response to antidepressant (both fluoxetine and venlafaxine) monotherapy is good (Amsterdam et al., 2010; Amsterdam & Brunswick, 2003; Strakowski et al., 2011). This means no matter if a potential BD-II patient falls into the category of major depression, simply treating him with antidepressants is acceptable. Opposite from the minor effect of the first factor, the second one is what we should evaluate cautiously. Changing the definition of diseases can cause the demographic landscape of patients to shift. The issues of the same diagnosis with different criteria could be poles apart. This could make previous studies become less reliable or lead clinical psychiatrists to provide the wrong treatment. Further, it leads to chaos by providing inconsistent results or add "noise" in the data pool (Strakowski et al., 2011).

Medication of Bipolar

"Outcomes for bipolar disorder have dramatically worsened in the pharmacotherapy era."
—Robert Whitaker (2015, p.177)

Last century, before the use of psychiatric medicine, the long-term course of manic depression had a much better prognosis than present BD. During that time, more than half of the patients would recover within a year and never had another attack. And most of them came back to the workforce (Whitaker, 2015). Nowadays, bipolar has become one of the leading causes of disability over the world, and it is highly associated with suicide and fatal medical comorbidities (Rowland & Marwaha, 2018). Can we make a bold assumption that it is the psychiatric drugs that worsen the disease? Generally, prescriptions of BD includes antidepressants for depressive episode, antipsychotics or mood-stabilizers (or sometimes both) for the mania (Frances, 2013; Parens & Johnston, 2010; Whitaker, 2015). An investigation of psychotropic practice showed a two to three fold rise of psychotropic medication for youth between 1987 to 1996. Among these, the category of mood stabilizers was the one which increased dramatically (Zito et al., 2003). In the UK, antidepressant prescriptions to children doubled from 2006 to 2015 (Sarginson et al., 2017). In 2010, four percent of American teenagers used antidepressant (Frances, 2013). In other words, at least

one adolescent in a typical high school classroom is beholden to a daily dose of psychiatric medicine.

Thus far, the aetiology of BD remains unclear (Rowland & Marwaha, 2018). Scientists have found a few potential risk factors evoking BD. Nevertheless, none of them contributed to be sufficient conditions. The present medical intervention is based on a hypothesis of "chemical imbalance" in the brain. The idea of "chemical imbalance" came from our interest in neurotransmitters (Frances, 2013). Although the whole concept has been proved thoroughly unfounded (Whitaker, 2015), the impact is long-lasting.

In the domain of neurotransmitters, almost every tiny aspect can be altered due to experiences (Sapolsky, 2018). It is easy to come to a conclusion that the efficacy of selective serotonin reuptake inhibitors (SSRIs) comes from a prolongation of the neurotransmitter lingering around the synapses. In fact, the real mechanism is much more complex than this owing to homeostasis and synaptic resilience. Generally, SSRIs need several weeks to work. When serotonin accumulates in the synapses, the autoreceptor, which detects the serotonin levels, projects signals to the presynaptic neurons to limit the release of serotonin in order to keep balance. Meanwhile, the serotonin receptors of postsynaptic neurons start to decline. A few weeks later, this feedback loop fatigues and becomes less active than normal. This is the time that serotonin can really pile up in the synapses and drugs begin working. Thus, the truth is SSRIs does not fix any existing chemical imbalance. It creates an imbalance (Whitaker, 2015).

No one can doubt that it is difficult to manage an accurate diagnosis of bipolar disorder. A survey done in 2003 calculated the average time it takes a patient to get a final diagnosis of BD from the initial onset of illness is 5.7 years (Morselli & Elgie, 2003; Singh & Rajput, 2006). In 2014, the editor of the book, Clinical Insights: Mental Health in Adolescents: Bipolar Disorder, even provided a more remarkable number of 8-10 years in the forward (Duffy, 2014). The most common misdiagnosis of BD is unipolar depression (DSM-IV) (Singh & Rajput, 2006; El-Mallakh & Karippot, 2002). The crucial criteria to separate bipolar from unipolar disorder is an episode of mania or hypomania. The symptoms of a manic episode are obvious and easy to recognize. In contrast, BD-II is difficult to be identified because the border between hypomania and "feeling good" is not clear (Frances, 2013), and most of the time the occurrence of hypomania does not cause any damage or loss. As a result, patients do not report it to their doctors (Singh & Rajput, 2006). However, everything could be viewed from a different standpoint. About sixty percent of the patients with BD reported their initial problem was depression. Yet, after taking antidepressants, they had been diagnosed as bipolar (Whitaker, 2015; El-Mallakh & Karippot, 2002). Similar to this, in a 1995 study done by Lewinsohn and colleagues, within 1,709 adolescent BD cases, 61 percent of their first onset episode was depression. A follow-up survey of children with major depression indicated a similar phenomenon. Almost half of the patients met the criteria of BD (33.3% with BD-I; 15.3% with BD-II) (El-Mallakh & Karippot, 2002). In 2008, at the American Psychiatric Association's (APA) annual meeting, a controversial topic was brought to the table: "Do depressants worsen the long-term course of bipolar disorder? And notably so? (Whitaker, 2015)" More and more researchers suggest that it is antidepressants themselves that worsen the course of BD (El-Mallakh & Karippot, 2002). A large group study about the rate of patients with depression who were converted to BD conducted by Martin and his colleagues found that the number of patients taking antidepressants is 3 fold more than those who were never exposed to the drugs. More specifically, the researchers

highlighted the fact that young depressed patients aged 15-19 showed the highest ratio to convert to bipolar (Martin et al., 2004). The debate between whether the first diagnoses of those patients was wrong or whether it was the drugs causing bipolar is contentious. And the debate is likely to last for a period because neither side can provide conclusive evidence to support their argument. Yet, the thing we know is that with long-term use of SSRIs, the patients' brain does become different from normal.

Excluding antidepressants, the adverse effects of mood-stabilizer and antipsychotics are undeniable (Frances, 2013/2012; Parens & Johnston, 2010). These side effects range from simple weight gain to severe heart attack (Morrison et al., 2012). There is clear evidence showing that taking antipsychotics increases the risk of cardiovascular problems and related death (Morrison et al., 2012). Similar to this, lithium, a mood stabilizer, was once banned in 1949 because it causes cardiovascular problems (Whitaker, 2015). Even the seemingly minor side effect of gaining weight should not be neglected. It is common sense that obesity leads to numerous health problems. Besides, in the present fitness-pursuing culture, being overweight and the judgment attached to that is predictably adding even more shade upon the patients. Apart from the issues above, antipsychotics will also influence the patients by changing the structure of some areas of the brain (Morrison et al., 2012). Brain image studies in patients with schizophrenia show that, after taking drugs, the volume of the basal ganglia and the thalamus have increased but the frontal lobe shrunk (Whitaker, 2015).

Furthermore, neither antipsychotics nor antidepressants performed notably superior to placebo (Moncrieff, 2018; Morrison et al., 2012; Whitaker, 2015). Only one in four patients with psychotic disorder responded to antipsychotics (Morrison et al., 2012). In 2008, a clinical survey of antidepressant benefits showed, compared to placebo treatment, there was no significant improvement in most of the patients with depression (Kirsch et al. 2008; Whitaker, 2015). It is clear that psychiatrists overestimated the efficiency of medication and underestimated the risks of the side effects.

The Immature Brain

A behavior could be seen as bizarre in adults but totally normal in certain developmental phases. Diagnosis of BD among the younger group is controversial because many symptoms of mania or hypomania are just common features in the developmental context (Blader, 2014). A Japanese slang term, Chūnibyō, explains this concept well. "Chūni" means the second year of middle school; the word "byō" indicates illness. This term is used to describe teenagers who always feel good about themselves, live in a world of themselves as the center or always speak and act arrogantly (Liú, 2016). This could be overly interpreted to be euphoria, elation or grandiosity—which are the symptoms of mania or hypomania. Referring to "Increase in goal-directed activity", who doesn't have a young relative or remember themselves being so obsessed with one thing? Having unrealistic dreams or being sentimental is the beauty of being young (Blader, 2014). Another beautiful feature among young people is their bravery and passion for adventure. These are also important for the society since it provides a chance to subvert the tradition (Sapolsky, 2018). No wonder students are always in the first line of social movements. However, many conventional grown-ups see these as "activities that have a high potential for painful consequences" (American Psychiatric Association, 2013, p.124).

From biological perspectives, all of these incomprehensible conducts which drive adults crazy could be due to their unformed brain. Not until our mid-twenties is the brain fully mature, and the frontal cortex is the last area to develop. The frontal cortex is central to regulating emotion. This can explain why youngsters are irritable. One experiment showed that when adults see an emotional face, it will first activate the amygdala, followed by the responding frontal cortex. However, offering the same image to an adolescent, the frontal cortex activates less, but the amygdaloid activation increases (Sapolsky, 2018). Yet, this is nothing related to disease. Worst of all, as mentioned, antipsychotics make the frontal cortex shrink. In addition, teenagers are famed for their poor attentiveness. Thus, is this a clue of "Distractibility"? Apparently not. Concentration is ruled by another region of the cortex, the parietal cortex, which is also a late-developing area. It helps humans focus on one task at one time by inhibiting irrelevant thought (Jensen, 2015). Based on the conditions described above, adolescents in this period naturally fulfill the criteria of BD based on their normal human development. They perfectly fit in the framework.

Apart from manic and hypomanic features, being low can be also commonly seen among youths. The most usual factor behind youth depression is stress. Nowadays young people are constantly under pressure. Stressors such as schoolwork, peer recognition, expectations from teachers and parents, all can make teens blue. At the same time, young people crave social connections. This makes them more easily encounter rejection and further feel lonely. From an evolutionary perspective, loneliness forces humans to pay attention to their social needs (Cacioppo & Patrick, 2009). It is a process of socialization which everyone must encounter. The pity is when teenagers encounter social rejection, they cannot regulate the negative emotions as well as adults because of their immature frontal cortex (Sapolsky, 2018). A proper amount of stress is essential. It encourages people to strive (Jensen, 2015; Sapolsky, 2018). But, if the stress is overloaded, it causes harm. Besides, an over-abundance of stress leads to attention deficit (Jensen, 2015), which can link back to "Distractibility". The proper way to support young people to move past a low period is to remove a certain amount of burden instead of dosing them.

Another reason why youngsters could present such a bipolar-likely extreme high and low mood could be attributed to their reward system, dopamine. Researchers conducted an fMRI experiment to test the dopaminergic activity of reward in three different groups—adults, adolescents and children. They found that, compared to the older and younger group, teenagers responded much more to an unexpected reward. However, if the reward was lower than their expectations, they became extremely low (Sapolsky, 2018).

Take the metaphor of describing the human mind as a lively shrub. It responds to the environment blossoming or falling by seasons. The nerves in the brain are the branches. Since the day we are born, the world as a gardener started to shape it by pruning the branches in our brain. Although we all prefer flowers and greens, winter is unavoidable. Professional gardeners know that winter is the best time to prune. If the seedling is shaded by other bushes, the best way isn't providing a flashlight, it is better to trim some branches above and patiently wait for the day the seedling grows as tall as the other bushes. In accordance with this, adversity makes people grow. And medication provides just as little value as the flashlight. Regrettably, people nowadays have a lower capacity to endure negative emotions and tend to view them as mental disorders rather than a part of life. And the recent approach of psychiatry deprives young people's opportunities for growth through the application of careless labels.

Conclusion

One aspect of human nature is putting things into categories. It helps us to remember things better and to make decisions. However, it restricts our ways of thinking (Sapolsky, 2018). The more complex issues are, the more easily people would be confined in their categorical thinking. There is no doubt that mental problems are involved. Nowadays, psychiatry dominates the field and neuroscience has become fashionable. People have started to believe that all of our mental issues are caused by an imbalance in the brain and can be treated by medicine. Some scholars have even gone so far to believe in so-called "biological determinism" (Siegel, 2015). Nowadays, because psychiatrists and researchers labor hard to define the biological mechanism of mental issues, they incidentally neglect the fact that people are suffering and calling for help (Frances, 2013). It is careless to underestimate the distress of individuals who have no psychiatric diagnosis while the burden of diagnosis is blurred. As a professor of biology and Neurology at Stanford University, Robert Sapolsky said, "It actually makes no sense to distinguish between aspects of a behavior that are biological and those that would be described as, say, psychological or culture. Utterly intertwined (2018)."

Taking an accurate diagnosis is difficult. The immature brain can easily fool psychiatrists to misdiagnose a young person with BD. They will then prescribe a huge amount of drugs with tons of side effects. Most of the medication intervention are palliative, and from a long-term perspective, it has worsened the disease. Indeed, diagnosis per se could cause harm. However, the most condemning issue is the compelling bond between psychiatric diagnosis and medication. Many scholars have said that the onset of BD usually arrives during youth. However, by connecting the evidence and arguments above, we can skeptically assume that perhaps "the recent prevalence of BD is caused by misdiagnosing normal people when they were young. They then become a chronic patients owing to the adverse effects of medication." As Frances said, "The impact of the diagnostic system is not in the words as written, it's in the way words come to be used (2013)." These days, in health service, there is too much polypharmacy but too little psychotherapy. In the long run, non-medical therapy has more beneficial effects with less harm than medication. It is regretful that the percentage of visits to psychiatrists who offer some form of psychotherapy decreased 15% from 1996 to 2005 (Frances, 2013). It seems that the current psychiatric diagnosis and medication system has come to its conundrum and the next step is unclear. Nevertheless, the authorities should curate the present error as soon as possible, meanwhile, avoid falling into the same dilemmas psychoanalysis faced in the past.

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