Bipolar or borderline?

In this issue, PL examines the classic debate about whether patients have bipolar illness or borderline personality. There are proponents that one or the other condition is underdiagnosed relative to its opponent. Some claim that the two can’t be distinguished in many cases, or that comorbidity is very common. The PL review concludes that they can be distinguished, as long as we focus on what differentiates them, as opposed to their overlap. Specifically sexual trauma, self-harm and dissociative states occur with borderline personality but much less so with bipolar illness. True comorbidity is much less common than what is claimed using DSM definitions.

The Article of the Month describes a new study which finds that stress in older persons leads to cognitive decline, possibly increasing the risk of dementia. The Drug of the Month is duloxetine, an SRI used for pain syndromes as well as depressive states. A curbside consult question is discussed regarding the use of low-dose SRIs for anxiety in persons with stable bipolar illness.

Last week, the July CME course was completed with excellent discussion and interaction in Cape Cod. There is still time to register for the late August course in Martha’s Vineyard, which will provide a complete review of psychopharmacology. The October Santa Fe course will be broader and aimed at advanced clinician skills in scientifically sound diagnosis and treatment, with a special emphasis on existential psychotherapies. Both courses are taught by me, and will provide an extensive opportunity for PL readers to interact with me and with other clinicians about many of the ideas discussed in PL, as well as a good opportunity to receive extensive CME credits.

Nassir Ghaemi MD, Editor

New truths begin as heresies and end as superstitions - T. H. Huxley
**Special Article: Bipolar or Borderline**

Ignore mood swings and anger

**Introduction**

The differential diagnosis of bipolar illness and borderline personality is important, controversial, and difficult. Some claim that bipolar illness is overdiagnosed and that borderline personality is underdiagnosed. Others claim the reverse. Those who argue that bipolar illness is overdiagnosed often assert that those patients instead have borderline personality. Others claim the reverse. The most common approach to this controversy is to focus on the overlap between the syndromes and then to assert that one merely represents the other. It also would seem to make sense to focus on areas of difference, if indeed these are different conditions.

Borderline personality and bipolar illness have features in common, just as schizophrenia and anxiety conditions can also have symptoms in common (e.g., insomnia). One question is whether the similarities between borderline personality and bipolar illness are central to those conditions, or peripheral and secondary features.

In this clinical overview, PL gives a clinical and conceptual examination of how they are similar and how they differ.

**Diagnostic validators**

The classic diagnostic validators used in psychiatric nosology research are: symptoms, genetics, course, treatment response, and biological markers. The scientific literature on borderline personality and bipolar illness can be examined for these features. These are summarized in the table and are presented based on the diagnostic validators of symptoms (mood lability, impulsivity, parasuicidal self-harm, mania), genetics, course (sexual abuse), treatment response, and neurobiology.

<table>
<thead>
<tr>
<th></th>
<th>Bipolar</th>
<th>Borderline</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Symptoms</strong></td>
<td>Euphoric mood, increased activities</td>
<td>Dissociative symptoms, Parasuicidal behavior</td>
</tr>
<tr>
<td><strong>Genetics</strong></td>
<td>Very strong</td>
<td>Nonspecific</td>
</tr>
<tr>
<td><strong>Course</strong></td>
<td>Severe recurrent mood episodes</td>
<td>High prevalence of sexual abuse</td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>Biological cure in 1/3</td>
<td>Modest drug effects</td>
</tr>
<tr>
<td><strong>Biology</strong></td>
<td>Hippocampal atrophy, Amygala enlargement</td>
<td>Nonspecific</td>
</tr>
</tbody>
</table>

**Mood lability**

The diagnostic validator of symptoms or phenomenology can also be examined regarding the most common psychopathology discussed in the literature on bipolar versus borderline differential diagnosis: mood lability. Mood lability is here defined as rapid alternations or fluctuations in mood over minutes to hours. It should be noted that mood lability is a DSM-5 criterion for borderline PD, but not for bipolar illness. It is common in bipolar illness, however.

One study of 29 subjects reported being able to distinguish mood lability between these two
conditions, based on type and intensity of mood shifts; it was small and based on self-report, however. Another study of 55 subjects did not have a bipolar illness alone group (only one comorbid with personality disorders) and thus could not address the question.

In sum, small differences in intensity or frequency may exist, but mood lability is common in both conditions, and is not a strong distinguishing feature between them.

**Impulsivity**

The symptom of impulsivity is closely allied to mood lability, and is often seen as manifesting as sexual impulsivity in these two conditions, although it can also be physical or aggressive or financial. There are many studies demonstrating high impulsivity in both conditions separately, but very few have directly compared the two conditions, and in most of those studies, adequate comparison was not made between subjects who met only criteria for one or the other condition, as opposed to comparing one diagnosis versus a comorbid control group. Thus, as with mood lability, impulsivity seems to be common in both conditions and has not been shown to be distinctly different in one condition versus the other.

**Parasuicidal self-harm**

Another key differentiating feature is parasuicidal self-harm. A recent systematic review of 51 articles found that self-mutilation is common in borderline personality (50 to 80% of cases) and is frequently repetitive (41% of patients have more than 50 self-mutilation acts). The largest study of parasuicidal behavior in bipolar illness, and the only such study in an unselected non-clinical population (i.e., a general population sample) is found in the National Comorbidity Survey (n =5,877). In that study, the prevalence of self-harm among patients with type I bipolar illness was only 0.9%. The importance of the NCS data is that they are epidemiological, not clinical. They are based on determining prevalence of parasuicidal behavior in persons with bipolar illness who are in the community, not those who seek treatment in clinicians’ offices. By using clinically-selected samples, higher rates of parasuicidal behavior are seen, even in bipolar type I illness, in some studies, but these samples involve a selection bias of those who seek help and do not generalize to the entire bipolar population. Even with this limitation, the highest parasuicidal behavior rate reported in clinical studies is 36%, which remains two-fold less frequent than in borderline personality. In contrast, the NCS study does generalize to the whole bipolar population and is probably the most valid data on which to base judgments about parasuicidal self-harm prevalence. In the general population, self-mutilation is reported to occur in only about 4% of non-clinical samples, compared with 21% of clinical samples (patients with psychiatric diagnoses of varied types). Hence, the rates of parasuicidal behavior in the NCS non-clinical sample of bipolar illness is similar to the general population, even slightly lower, and comparable to other clinical samples of psychiatric conditions (excluding borderline personality).

In sum, compared to bipolar illness, borderline personality involves at least a two-fold increased relative risk of parasuicidal self-harm in clinical samples. In the general population, this difference is immense, about a 50-80 fold higher rate in borderline personality. This difference compares favorably to the classic association of cigarette...
smoking and lung cancer, in which a 5-8 fold effect size has been considered to be quite large and convincing. Given the literature reviewed above, similar judgments would seem to be reasonable in the case of parasuicidal self-harm and borderline personality.

**Manic symptoms**

The most straightforward diagnostic validator may be symptoms. A few studies have assessed whether the presence of manic symptoms or manic/hypomanic episodes can distinguish bipolar illness from borderline personality. The largest study (n= 5035), which assessed an unselected mood population with depressive episodes, examine mixed states, defined as the presence of three or more DSM-IV defined manic symptoms, of any duration, along with a major depressive episode. This definition of “mixed depression” differs from the DSM-IV definition of a full manic episode lasting at least one week in duration, co-occurring with a full depressive episode. This definition of “mixed depression” included patients who would meet DSM-IV defined bipolar disorders, type I or II, or DSM-IV defined major depressive disorder (MDD). In other words, “mixed depression” reflects a mood disorder population. The question was whether manic symptoms in this mood disorder population were also found in subjects who met DSM-IV criteria for borderline personality disorder. Using this broad “mixed depression” definition, DSM IV-defined manic symptoms occurred much more frequently in “mixed depression” than in borderline personality. In contrast, borderline personality had more of the following DSM-IV features than did patients with “mixed depression”: fears of abandonment, identity disturbance; recurrent suicidal or self-mutilating behavior, and dissociative symptoms.

In sum, the literature on presence or absence of manic symptoms supports the view that manic symptoms distinguish bipolar illness from borderline personality.

**Genetics**

The most definitive review of the immense genetic literature on bipolar illness compared to personality disorders and other conditions is a systematic review of twin studies of genetic heritability. In that systematic review, bipolar illness was found to be one of the two very heritable mental illnesses, along with schizophrenia, both having about 80% heritability, similar to Alzheimer's dementia. This rate is about twice as much as found in that systematic review for borderline personality or other personality traits or disorders, which tend to have about 40% heritability. For instance, in a study of 2794 Norwegian twins, the genetic heritability of borderline personality was only 37%; in fact, all DSM-IV defined personality disorders fell into the 20-41% heritability range, with the highest being for antisocial personality disorder. Environmental heritability, in contrast, was 63-79%, indicated that it was the major causative feature for these conditions. These studies use personal clinical interviews to assess personality disorders using DSM-IV criteria. It has been reported, using the same Norwegian sample, that the addition of self-report evaluations leads to higher heritability assessments, reaching 69% for borderline PD, and generally in the 60-70% range for all personality disorders. Whether it is legitimate to add self-report to clinical interview in assessing personality disorders is a methodological question that remains to be
answered. But if one accepts clinical interviews as the gold standard, and thus compares personality disorders using that method which is the standard approach in genetic twin research, then the genetic heritability of borderline PD is much lower than bipolar illness.

It should be noted that 40% heritability is not zero; it indicates a modest genetic effect. But it is half as much as 80%, which is similar to the heritability of traits that are widely accepted to be mostly genetic, such as physical height.

In sum, using currently accepted standards of genetic twin research, bipolar illness is almost completely genetic in causation, with a small environmental component. In contrast, borderline personality is mostly environmental in causation, with a small genetic component.

**Course of illness**

A key course feature that potentially could differentiate bipolar illness from borderline personality is a history of sexual abuse. In a commonly cited meta-analysis of 21 studies, 50–76% of patients with borderline personality disorder had experienced sexual trauma. In contrast, sexual abuse occurs in less than 30% of bipolar subjects. These prevalence rates are based on a number of different studies with large samples, including systematic reviews. For instance, a recent systematic review including 3407 bipolar patients found a 24% prevalence of sexual trauma in bipolar illness. According to one of the most extensive national studies of the topic, the US Department of Health and Human Services reports a prevalence of childhood sexual abuse in the general population of 9.2%.

In sum, there is a consistent frequency of sexual abuse in bipolar illness that is similar to the general population rate in some studies, or possibly higher than the general population. Yet the frequency of childhood sexual abuse is consistently at least two-fold higher in borderline personality than in bipolar illness or the general population.

It has been noted the average effect size for the association between childhood sexual abuse and borderline personality is only moderate ($r = 0.279$), but this way of assessing the data does not contradict the frequency noted above. It merely addresses the fact that other causative factors also exist for borderline personality. In this setting, the question is whether this specific risk factor is common in borderline personality, not whether other risk factors also might be common.

**Neurobiology**

In bipolar illness, a number of consistent neurobiological changes are reported in dozens of studies. Although a range of abnormalities is found, two of the most consistent abnormalities found are hippocampal atrophy and amygdalar enlargement. These differences are shown in studies which compare bipolar illness both to normal controls and often to other psychiatric conditions, like schizophrenia.

In contrast, there are fewer studies of neurobiological changes in borderline personality, and some abnormalities found compared to normal controls, such as deficits in integration between cognition and emotional processing stimuli, are not unique to borderline personality but are also found in other neuropsychiatric
syndromes, including schizophrenia and bipolar illness.

**Treatment response**

Treatment response has generally been seen as the most nonspecific diagnostic validator, as medications can affect varied diagnoses. However, certain treatment effects may be diagnostically specific. Regarding bipolar illness versus borderline personality, there is a strong consensus after a century of practice and research, that psychotherapies alone are not effective in bipolar illness. They may be effective adjunctively with medications, but not by themselves. In contrast, there is a similar strong clinical consensus for decades that psychotherapies are central to the treatment of borderline personality; many experts in borderline personality see medications as adjunctive treatments for that condition. Many randomized clinical trials (RCTs) of bipolar illness exist, and demonstrate good efficacy with various agents, like lithium, in prophylaxis of that condition, sometimes with complete remission. In contrast, fewer RCTs exist of treatment with medications for borderline PD, and they tend to demonstrate modest symptomatic benefits with psychotropic medications.

In sum, it would seem to be a fair reflection of a long-held clinical consensus that treatment response in bipolar illness versus borderline personality tends to be inverse: in bipolar illness, appropriate psychotropic medications are necessary, with psychotherapies being adjunctive; in borderline personality, appropriate psychotherapies are seen as necessary, with psychotropic medications being adjunctive.

**Discussion**

Reviewing the empirical evidence on classic diagnostic validators, it appears that bipolar illness and borderline personality are distinguishable and different. The most clear differentiating features appear to be a family history of bipolar illness, parasuicidal self-harm, and past sexual abuse, each of which is at least twice more frequent in one condition versus the other. Treatment response also appears to differ markedly, with medication efficacy much stronger and central to treatment of bipolar illness, whereas psychotherapies are not effective alone; in borderline personality, medication effects are modest at best, while psychotherapies are central to its treatment. Symptom features do not differentiate between these conditions as clearly as the above genetic, course, and treatment validators. Mood lability and impulsivity not different in any clearly proven and replicated manner between these conditions; manic symptoms and manic episodes may differentiate between them but direct comparative studies are few. A large neurobiological literature also exists that finds abnormalities in bipolar illness to a more consistent and definitive degree than in borderline personality. A number of direct comparisons exist which demonstrate more neurobiological abnormalities, and often of different kind, in bipolar illness than borderline personality.

In sum, these conditions appear to be different in a number of major diagnostic validators. To summarize: bipolar illness is a very genetic condition with a large amount of neurobiological abnormalities that requires medication treatment as central to its management. In contrast, borderline personality is a mostly environmental
condition with fewer neurobiological abnormalities that requires psychotherapies as central to its management.

One way of summarizing the proposed interpretation given in this review is that while there are superficial similarities between bipolar illness and borderline personality, there are profound differences. The comparison is between red apples and red skies; redness is shared, but these are two very different entities.

“Comorbidity”?

Even if borderline personality and bipolar illness can be distinguished, it may sometimes be the case that they can occur together. For instance, since bipolar illness is a genetic and biological condition, persons who are genetically predisposed to it are also likely at higher risk of sexual impulsivity being present in family members with bipolar illness; sometimes this may lead to sexual trauma that can derail personality development and eventually lead to borderline personality. This kind of co-occurrence of different conditions is what was meant by the introduction of the term “comorbidity” by Feinstein in 1970. In contrast, as the leadership of DSM-IV has explicitly stated, the DSM psychiatric nosology has been set up allowing for extensive overlap in diagnostic criteria so as to encourage diagnosis of multiple conditions at once. This “comorbidity” could rationally be attributed to our DSM system, rather than nature. Hence the application of DSM criteria and report of high comorbidity rates between bipolar disorder and borderline personality disorder does not imply that those conditions are highly similar.

A final historical point is that bipolar illness is derived from manic-depressive illness, which has been well-defined in the scientific literature for over a century, if not much longer dating to ancient Rome. Borderline personality, in contrast, was first clearly defined in the psychoanalytic literature in the late 1960s, and thus is a more recent construct. Bipolar illness is based on standard medical methods: observation of signs and symptoms and course of illness. Borderline personality is partly based on such standard medical observation, and DSM criteria are not specifically psychoanalytic in nature, but the most prominent borderline experts emphasize that this condition is also centrally determined by certain psychoanalytic concepts, such as splitting and projection and countertransference. Hence, the evaluation of the empirical literature here is consistent with historical and conceptual differences in how these conditions have been came to be conceived and continue to be understood.

Affective temperaments misinterpreted as borderline personality

Historically, it should be noted that the concept of manic-depressive illness as used by Kraepelin included depressive and manic episodes as part of the same illness, merely as gradations of severity, rather than as two separate illnesses. It can further be added that Kraepelin and Kretschmer held the view that even milder versions of mania and depression, namely hyperthymic and cyclothymic temperaments, were also part of manic-depressive illness. Recent clinical and genetic studies find high rates of these temperaments in persons with bipolar illness and their relatives. Since hyperthymia and cyclothymia are seen as mood temperaments, part
of personality, they can appear to be similar to clinical features that are often viewed as borderline personality traits, such as mood lability and impulsivity, which this review found to be nonspecific diagnostically. If the concept of mood temperaments is scientifically valid, it would require even more caution in making the borderline personality diagnosis in such persons who may not have bipolar illness type I or type II (i.e., full manic or hypomanic episodes) but have hyperthymic or cyclothymic temperament along with other features of manic-depressive illness (such as a family history of bipolar illness and severe recurrent depressive episodes).

Often it is emphasized that bipolar illness involves severe episodes. If symptoms are constant or chronic, borderline personality is presumed. But this conclusion ignores the concept of affective temperament. If sexual trauma and self-cutting are absent, then cyclothymia often is misdiagnosed as borderline PD, since DSM-criteria easily diagnose the latter in persons with constant mood swings and anger.

**The PL Bottom Line**

- Borderline personality and bipolar illness are distinguishable clinically and diagnostically.
- Bipolar illness can be seen as a genetically-based biological disease, while borderline personality can be interpreted as a psychosocially-caused clinical picture.
- The two illnesses should be treated distinctly, with appropriate medications emphasized for bipolar illness and psychotherapies emphasizes in borderline personality.
- Borderline personality may be misdiagnosed in persons with cyclothymic or hyperthymic affective temperaments.

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**PL Reflection**

Psychiatry's motto is not the one Dante put over the gates of Hell: “Abandon all hope ye who enter here.” Psychiatry abounds in hope…But the proper words to put over psychiatry's door are still not ones to quicken every pulse. For we must write there: “Accept uncertainty within, or do not stay”…Psychiatry is the part of medicine furthest away from settled maturity, as strong as any in observation (perhaps even the richest, considering that social observations are much better accepted in psychiatry than in most of medicine), and weakest in correlations and calculations. It is like an adolescent, disputatious, alternately arrogant and humble, strong, full of promise, inconsistent, able to see what others can’t, even preoccupied with sex.

*Leston Havens*

*A Safe Place*
Current Study of the Month: Does stress cause dementia?

Perceived stress and cognitive decline in different cognitive domains in a cohort of older African Americans. AD Turner et al, American Journal of Geriatric Psychiatry. 2017; 25:25-34

It seems so

Does stress cause dementia? We know that stress is bad for the body and the brain. When you are stressed, and in your fight and flight mode, the hypothalamic pituitary adrenal (HPA) axis is in overdrive. Steroids get produced, go directly into the brain, directly into the neurons, stimulate neurons excessively, and kill them (excitotoxicity). Stress is bad.

But does it cause dementia directly? Or is the effect mediated by depression or inflammation or some other mechanism.

In this study, part of the Minority Aging Research Study, older African Americans without dementia (mean age 73 years) were given a battery of 19 cognitive tests every year for a mean of 4 years (up to 9 years maximum). Stress was measured using a 4 item scale (Cohen’s Perceived Stress Scale, PSS).

Higher stress on the PSS correlated with decline in global cognition as well as episodic memory and visuospatial ability. This effect was present even after correcting for age, gender, education, presence of depression, and vascular risk factors (like diabetes or hypertension).

In sum, if you follow healthy older African Americans in their early 70s, they are more likely to have cognitive problems by their mid to late 70s if they have a lot of stress.

This finding is stronger than might otherwise be the case because they have corrected for other possible reasons for developing cognitive impairment, like depression or hypertension or diabetes. There could be other confounding factors that were not measured in this observational study, which could be the real explanation for the changes observed. But, it could be that stress does have an independent effect on cognitive decline in older persons.

The PL Bottom Line

• Stress is an independent predictor of cognitive decline in older persons in this study.

• Besides depression or hypertension or other risk factors, stress is an important remediable risk factor for dementia.

PL Reflection

We haven’t got the money, so we will have to think.

Lord Ernest Rutherford
physicist
Drug of the Month: Duloxetine (Cymbalta)
Souped-up Prozac

**Biological mechanism**

Duloxetine is similar to fluoxetine, from which it is derived, having serotonin and norepinephrine reuptake blockade. Both agents share the same mechanism; the only difference is in potency. While fluoxetine has mild norepinephrine reuptake inhibition and strong serotonin reuptake inhibition (and thus has more SRI than NRI effects), duloxetine has moderate norepinephrine reuptake inhibition and strong serotonin reuptake inhibition. They are thus more similar than different, and duloxetine still is primarily an SRI (not a “NRI”, contrary to its marketing).

**Clinical efficacy**

Duloxetine is FDA indicated for acute depressive episodes in major depressive disorder (MDD). It also has FDA indications for generalized anxiety disorder, fibromyalgia, chronic pain, and diabetic neuropathy. It has been shown to help pain in depression, and tends to be used by clinicians for pain syndromes. It has never been shown to be more effective than any other monoamine agonist (antidepressant) for depressive episodes.

**Dosing**

The standard dose is about 20-60 mg/d, with a maximum of 120 mg/d, given once daily at night. For MDD, doses above 60 mg/d have not been shown to be more effective. The half-life is about 12 hours.

**Side effects**

Nuisance side effects involve sexual dysfunction, diarrhea, and apathy, as with all SRIs. Medically important side effects are uncommon, though, as with all SRIs, there is some risk of osteoporosis and gastrointestinal bleeding (due to inhibition of blood clotting).

The main problem with this agent is that it has a very severe serotonin withdrawal syndrome. This syndrome tends to happen after about one year of constant use. Since this agent is used long-term in many persons for chronic pain, the serious withdrawal syndrome means that if clinicians give this medication long-term, they are committing patients to indefinite treatment, as it will be very difficult to come off it ever. If needed, direct taper is almost impossible. In that case, cross-taper with fluoxetine is the best option.

**The PL Bottom Line**

- Duloxetine is a SRI with somewhat stronger norepinephrine reuptake blockade than its cousin, fluoxetine.
- It is effective in pain syndromes, like fibromyalgia.
- It has typical SRI side effects like sexual dysfunction.
- It has terrible serotonin withdrawal syndrome, complicating long-term treatment.
Curbside Consults

Questions and cases from you

Question: Sometimes, patients have severe anxiety all their lives, which persists even when the bipolar illness is under control and offenders like amphetamine stimulants are discontinued. I’ve seen a person who was a complete nervous wreck all her life until Viibryd 40 mg was added. Lithium, Viibryd, Latuda 120 mg and Ativan 1 mg bid have gotten rid of 90% of her anxiety. If successfully treating the bipolar disorder still leaves severe anxiety, do you think sometimes it is okay to use SSRI medicines in that the pros and cons lean towards the pros of using SSRIs and then there will be cases where it will not destabilize the person’s mood? I usually use 1/4 of the depression dose of the SSRIs to treat the anxiety so as not to destabilize mood. 20 or 40 of Viibryd is my go-to medicine now for severe anxiety and I have 40 people who every month say this is very helpful.

PL: The March 2015 PL issue described how anxiety is a symptom, not usually a disease or “disorder” itself. If someone has bipolar illness, and they are adequately treated such that they no longer have definable mood episodes of any notable severity, then the continued presence of anxiety would rule out the idea that the bipolar illness itself caused the anxiety. However, two other options exist.

One possibility is that the anxiety is part of the personality trait of neuroticism, as described in the May 2015 PL issue; the person just is highly anxious as part of her personality, just as some people are tall or short, and some people are introverted or extroverted. That’s not a disease that can be taken away with medication. It’s not an illness to improve. However, you could make the argument that such personality traits can be modified modestly with medications, and that one can “take the edge off” the anxiety as part of the personality trait, and that some benefit would ensue functionally or subjectively. Another possibility, which is often ignored, is that the mood episodes may not be causing anxiety, but the person may have an affective temperament which could be causing the anxiety.

As reviewed in the June 2016 PL issue, affective temperaments are quite common, occurring in about 50% of persons with bipolar illness or unipolar depressive illness, and in many of their relatives. In other words, in between mood episodes, these persons have constant mild mood symptoms. They are either always manic (hyperthymia), always depressed (dysthymia), or always both (cyclothymia). Especially in the cases of the manic temperaments (hyperthymia and cyclothymia), it is common to have a good deal of anxiety (and often distractibility), leading to misdiagnoses of GAD or adult ADD.

These possibilities need not be mere speculations. Affective temperaments can be measured using the 50 item TEMPS scale. On the PL website, we will make available the TEMPS scale along with a scoring sheet for it. The cut-off PL recommends is that if 75% or more of the items are endorsed, then an affective temperament is present. Sometimes, 50% or more of items may be sufficient given the clinical history. If someone has cyclothymia or hyperthymia, then those temperaments could explain constant anxiety, even if full mood episodes are managed with the standard mood stabilizers. In that case, low dose SRIs would not be recommended by PL, since those agents can worsen the manic symptoms of hyperthymia or the mood lability of cyclothymia.

If the TEMPS scale is negative, the NEO scale can be used to measure neuroticism. If the score is high, then in that case, it may be that the
person has the personality trait of high neuroticism. Low dose SRIs can “take the edge off” anxiety in that case. Such low doses may not destabilize bipolar illness in terms of causing mood episodes, especially if the personality is otherwise normothymic (i.e., no affective temperaments). In that case, such judgments as our colleague makes here may be relevant. However, even there, one is committing the patient to lifelong treatment without the option of coming off SRIs, due to severe serotonin withdrawal syndrome. One wonders whether low dose benzodiazepines might not be a better alternative. There is withdrawal there too, but usually not as severely as with SRIs. There is tolerance and a very small addiction risk of course, which is higher in persons with substance abuse. Either way, whether with SRIs or benzodiazepines, the treatment of neuroticism with medications is a low risk, low yield proposition. Such symptomatic treatment has modest benefit at best, so even with small risks, the benefit-risk ratio is questionable, especially long-term. In the prior PL issue on Hippocratic psychopharmacology, such symptom oriented treatment was discouraged since in the long run, more harm than good occurs.

Upcoming Courses/Seminars
by PL Editor Nassir Ghaemi MD

August 2017 - Martha’s Vineyard, MD
Clinical Psychopharmacology: Principles and Practice,
Harvard Medical School CME series
August 21-25, 2017, Martha’s Vineyard, MA
www.capecodsummerseminars.com

October 2017 - Santa Fe, New Mexico
Becoming a Master Clinician: Diagnosis, Drugs and Existential Psychotherapy,
New England Educational Institute,
October 25 - 28, 2017, Santa Fe, NM.
www.neei.org