comparatively measures physical and mental health conducted in the study with results taken from an earlier study conducted prior to the quakes. Not surprisingly, with many people in Christchurch displaced and under various pressures during the quake sequence, recruitment was 64% and necessarily selective, to some extent. However, this compares with rates discussed by Bryant (2014) of 16% in his study of the Black Saturday fires.

In the second study, a birth cohort of participants has been followed up for 30 years. As the time of the quakes, approximately half were living in Christchurch and half elsewhere. The response rate in the interviews conducted after the earthquakes was 78%. These results are published elsewhere (Fergusson et al., 2014). However, in this issue, Fergusson et al. (2014b) present data from the same study examining the psychosocial effects of unemployment. The high response rate at multiple time points (in this paper data from assessments at 18, 21, 25 and 30 years is reported) makes it possible to begin to examine causation in a meaningful way and to conclude that exposure of unemployment does have 'small but pervasive effects on psychosocial adjustment in adulthood and young adulthood'.

Finally, Meadows et al. (2014) engage in the much neglected area of transtholical clinical trials. Large pharmacological trials are almost always funded by the pharmaceutical industry, while psychotherapy trials are conducted by orgonizers of the therapy being examined. Results from these trials do not always translate into clinical practice. In the trial of Meadows et al., a pragmatic design was used in real clinical practice. Exclusion criteria were minimized and the trial studied patients with three or more episodes of depression, regardles of underlying diagnosis (unipolar or bipolar). Interestingly, in the context of the previous discussion on bipolar II disorder, no-one with this diagnosis was recruited (21 patients had bipolar I disorder). The study showed a large reduction in the number of days with major depression, in the 2-year period of the study, in the group that received mindfulness-based cognitive therapy. These and other papers in this month's issue represent a collection of data, reviews and viewpoints that explore areas of psychiatry that seem to receive scant attention in many psychiatric journals, reflecting ANZJP's continued commitment to highlight and discuss these sometimes neglected areas of psychiatric practice and research.

References


Inflation

To the befuddlement of physicists, the universe is expanding; in fact it is accelerating at such an unremarkable rate. The precise explanation for this is still a matter of debate, but dark forces in the fabric of the universe are thought to be in play (Perlmutter et al., 1999, Riess et al., 1998). While the implications of this are profound, the timeframe involved means there is little chance of any direct impact on human kind as we know it in contrast, another kind of expansion is affecting our lives here and now and also helps to see no end in sight: the diagnostic expansion of bipolarity.

A brief history of why polar, multi-polar and bipolar Why polar? In 1980, the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III) replaced manic-depressive illness with the term bipolar disorder (APA, 1980). This was the first time that DSM recognised the unipolar-bipolar distinction as proposed by Leonhard many decades earlier. However, there was one subtle difference: Leonhard had used the terms ‘monopolar’ (unipolar) and ‘bipolar’ to describe recurrent affective disorders, but in DSM-III recurrence was given less importance as a discriminating feature of affective illnesses, and polarity therefore gained salience, even though, arguably, recurrence provides a better means of grouping mood disorders (Leonhard, 1957; Perls, 1966). Two additional factors appear likely to have contributed to the dominance of polarity. First, a change in polarity (such as the occurrence of mania in the context of previous depression) is a readily identified differentiator; second, the incredibly low threshold for defining ‘recurrence’ of depressive disorders in DSM (two or more episodes) meant that, in practice, everyone qualified as having recurrent depression, thereby rendering this descriptor meaningless (APA, 1994).

Tri- and bi-polar? Bipolar II disorder was introduced in DSM-IV, after it was realised that not all presentations of mania qualified for bipolar I disorder and that manic symptoms of shorter duration were both common (APA, 1994). Bipolar II disorder has recently been reclassified in DSM-5 following nearly two decades of interest in defining its occurrence and mapping its prevalence (APA, 2013). Alongside this process, however, many additional questions have been asked about the possible existence of other kinds of bipolar disorder. These questions arise because studies of bipolar II disorder have revealed that many individuals either experience sufficient manic symptoms, for very short periods (fewer than 4 days), or have individual symptoms that meet the duration criteria but are too few in number to cross the threshold for diagnosis. A few of these myriad presentations have been captured in DSM-5, either as ‘other specified bipolar and related disorders’ or as ‘conditions for further study’ (APA, 2013). Another form of bipolarity that came to light during the DSM-IV era, termed ‘bipolar III disorder’, describes the occurrence of mania or hypomania in the context of antidepressant treatment (treatment-emergent mania) (Akiskal, 2007). It is thought to represent the unmasking of an underlying bipolar diathesis, although there is some debate as to whether treatment-induced manic symptoms are equivalent to spontaneous mania.

In this manner, bipolar disorder has been diversified and expanded and, whilst each step is inexplicable and apparently logical, the potential for bipolarity to permeate normality – facilitated by the recent changes in DSM – is a grave concern.

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Are ‘buy-polar’ forces and ‘try-polar’ thinking expanding bipolarity?
Bipolar II has problems too

There is no doubt that, outside bipolar I disorder, there are clinical changes in mood, energy and activity that debilitating a considerable number of psychiatric patients. In other words, individuals with bipolar II disorder, if this is taken to mean 'bipolarity that is not bipolar I disorder', certainly exist and they are often very unwell. The practical difficulties that ensue are how best to define these syndromes, to understand their evolution and trajectory, and to determine optimal treatment. Careful revision of bipolar II disorder in DSM-5 could have assisted with these matters, but instead bipolar II has remained much the same, with profound ongoing consequences.

Already, three major problems plague bipolar II disorder: first, its diagnostic expansion to the point that it encroaches on normal experience; second, its frequent misdiagnosis because of overlap with other disorders; and, third, its treatment lacks differentiation from that of bipolar I disorder.

Blurred lines

The diagnostic boundaries of bipolar II disorder are known to be arbitrary, and hence its detection remains open to subjective interpretation of hypomanic symptomatology (Malhi et al., 2010). This inability to characterise the disorder, and in particular a failure to identify a pristine lower boundary, has advanced the conflation of bipolar II disorder with normality and contributed to its over-diagnosis, particularly in young people, where it has seemingly given rise to an epidemic.

Comorbidity and misdiagnosis

The second problem, namely that of overlapping diagnoses, is both a conceptual problem and one of dependence on phenomenology for arriving at diagnoses. Clinical presentations of bipolar II disorder clearly 'overlap' with anxiety states, substance abuse and personality traits — all of which are usually captured diagnostically as comorbidities. This makes the demarcation of bipolar II disorder extremely difficult (Malhi et al., 2012). For example, it is often difficult to distinguish whether irritability and distractibility are features of bipolar II disorder or a consequence of heightened anxiety. Similarly, impulsivity and hedonic changes are common sequelae of substance abuse, which also generates a myriad of symptoms that can obfuscate bipolar phenomenology further. In a similar vein, personality traits and affective dysregulation mimic bipolar mood changes, making disambiguation based solely on phenomenology near impossible (Bassett, 2012; Coulston et al., 2012).

For these reasons, the creation of bipolar II disorder has more than doubled the problem of bipolarity, and it is important to note that this has not come at the expense of lowering the threshold for morbidity. Although technically bipolar II disorder causes less impairment than bipolar I disorder (hypomania versus mania), because manic symptoms are present for a shorter period of time and are less severe (no psychotic symptoms, for example (APA, 2013)), evidence suggests that the morbidity of depression in bipolar II disorder is equal to, if not greater than, that exacted by bipolar I disorder (Judd et al., 2002, 2003). Furthermore, subsyndromal symptoms are more common in bipolar II disorder and last longer (Judd et al., 2002, 2003).

Lack of therapeutic specificity

Interestingly, in terms of overall course, bipolar II disorder is possibly more similar to recurrent unipolar depression than it is to bipolar I disorder. However, despite this, the medications used to treat bipolar II disorder are virtually identical to those used for bipolar I disorder, and this is the third unsolved problem. In other words, bipolar II disorder has been unable to achieve sufficient therapeutic specificity. This is partly because all 'bipolar medications', with the notable exception of lithium, have actually been developed for other disorders. This is quite remarkable. The paradigm employed for the development of medications for the treatment of bipolar disorder has been to 'adopt' medications originally developed for different indications and to trial them in bipolar I disorder patients. Notably, medications trialled in one phase of acute bipolar illness (usually mania) have often been subsequently used informally across other phases of the disorder without much further investigation of putative benefits. As such, antidepressants have migrated from major depression, anti-convulsants from the treatment of epilepsy, and neuroleptics from the treatment of psychoses. This may explain why therapeutic subtyping within bipolar disorder has received little attention, and why in practice the management of bipolar II and bipolar I disorders is much the same. One problem this causes is that medications used in bipolar I disorder are often administered similarly in bipolar II disorder, despite a lack of evidence, and often in combinations for which there is virtually no evidence. Such extrapolation of practice and 'evidence' from bipolar I disorder to guide treatment in bipolar II disorder is widespread, undermines therapeutic differentiation of the two subtypes and renders diagnostic separation meaningless.

Try before you buy

Of the many potential drivers of the expansion of bipolar disorder, there are two that warrant specific consideration. First, academics and consumers have argued for better recognition and treatment of bipolar disorder, citing studies suggesting delayed diagnosis (mainly of bipolar I disorder) and the relative lack of efficacy of antidepressants in depression (Malhi, 2012). Second, the diagnosis of
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For these reasons, the creation of bipolar II disorder has more than doubled the problem of bipolarity, and it is important to note that this has not come at the expense of lowering the threshold for morbidity. Although technically bipolar III disorder causes less impairment than bipolar I disorder (hypomania versus mania), because manic symptoms are present for a shorter period of time and are less severe (no psychotic symptoms, for example APA, 2013), evidence suggests that the morbidity of depression in bipolar III disorder is equal to, if not greater than, that exacted by bipolar I disorder (Judd et al., 2002, 2003). Furthermore, subsyndromal symptoms are more common in bipolar I disorder and last longer (Judd et al., 2002, 2003).

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Curiously, in certain circumstances, bipolar II disorder may be seen as desirable (Chen and Singlet, 2010). From both a clinician's and a patient's perspective, this label can be easier to assign, and to accept, respectively, than that of schizophrenia, although in some cases the distinction is difficult and the actual clinical features may be more indicative of a psychotic process.

Sometimes the diagnosis of bipolar II disorder is itself 'trialled'. For example, mixed features that emerge in the context of bipolar I disorder present similarly. One problem this causes is that medications used in bipolar I disorder are often administered similarly in bipolar II disorder, despite the lack of evidence, and often in combinations for which there is virtually no evidence. Such extrapolation of practice and evidence from bipolar I disorder to guide treatment in bipolar II disorder is widespread, undermines therapeutic differentiation of the two subtypes and renders diagnostic separation meaningless.

Try before you buy
Of the many potential drivers of the expansion of bipolar II disorder, there are two that warrant specific consideration. First, academic and consumer consumers have argued for better recognition and treatment of bipolar II disorder, citing studies suggesting delayed diagnoses are invariably more likely for bipolar II disorder and the relative lack of efficacy of antidepressants in depression (Mahi, 2012). Second, the diagnosis of bipolar II disorder has been championed by Phraam and seen as a target for expansion and investment, and as an opportunity for expanding the use of existing medications, particularly antipsychotics. A combination of these two forces has propelled the diagnosis of bipolar II disorder to a point of being clinically recognized and diagnosed and almost always treated with medications. And yet it remains poorly defined, with likely diminished interest in identifying specific treatments.

In summary, bipolar II disorder is a diagnosis that has multitudinous problems.

1. There is concern over expansion of the diagnosis such that it introduces into normal mood, which allows access to 'bipolar medications'.
2. Psychiatrists have been encouraged to view bipolar II disorder as similar to bipolar I, rather than considering the similarities to recurrent depression, and again this has expanded the use of 'bipolar medications'.
3. Other psychiatric conditions are often misdiagnosed as bipolar II and then the patient is medicated and managed with bipolar treatment strategies, often for long periods of time, until they are crossed off the bipolar medications.
4. When there is diagnostic uncertainty (as is often the case in psychiatry) there is a tendency to 'try'

the diagnosis and commence medication. This often produces non-specific calming effects, which then confirm the diagnosis and justify the initiation of treatment, further reinforcing the 'try-polar' paradigm.

For the foreseeable future, and at least until DSM-5 is revised, or the International Classification of Diseases (ICD) offers some novel advance, the difficulties created by the diagnosis of bipolar II disorder will have to be addressed by psychiatrists in their clinical practice. To do this, the tendency to delineate bipolar diagnostic boundaries so as to assign certain diagnostic labels in order to access particular treatments has to be tempered, and executed with caution, if all diagnoses of bipolar II disorder past and present need to be frequently reviewed and revised as necessary and non-specific treatments should not be regarded as bipolar medications and used to confirm diagnostic hypotheses. The diagnosis of bipolar II extends beyond bipolar I disorder and that this moiety affects the lives of many individuals and therefore merits diagnostic differentiation and treatment; the key challenge is how to achieve this while avoiding mislabelling and mistreating those with other ailments, or with no illness at all.

Keywords
Bipolar disorder, bipolar II, diagnosis, mania-depressive illness

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