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For Psychology of Mental Health Disorders module at Oxford Brookes University

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CASE STUDY ON CASE 2 - RB

Diagnosis

RB meets the criteria for a diagnosis of **bipolar I disorder (BD-I)**

DSM-5 Criteria

Criterion A states criteria for at least one manic episode must be met and criterion B states the manic and major depressive episode(s) must not be better explained by another disorder (American Psychiatric Association (APA), 2013; Black & Grant, 2014). Diagnosis of a **manic episode** requires the following criteria A-D be met (Black & Grant, 2014; APA, 2013). Criterion A: Persistently elevated/expansive/irritable mood and increased activity for at least one week, present most of and nearly every day (or any duration if hospitalization is necessary). Criterion B: During this period three or more of the following symptoms present to a significant degree (or four or more if mood is more irritable than elevated), representing a departure from normal behaviour: Inflated self- esteem/grandiosity (B1), decreased sleep (B2), overly talkative (B3), flight of ideas/racing thoughts (B4), distractibility (B5), increased goal-directed activity (B6), and excessive involvement in potentially damaging activities (B7). Criterion C: The episode causes severe impairment in social/occupational functioning that may require hospitalization to prevent harm to self or others or includes psychotic features. Criterion D: The episode is not caused by substance or medication use or by another medical condition.

Patient Symptoms

Criterion A for a manic episode was met by RB presenting a persistently elevated, expansive, and irritable mood lasting two weeks prior to hospitalization with six symptoms within criterion B. RB presented grandiosity and inflated self-esteem (B1), evidenced by an abnormal belief he was the most gifted surgeon in the county with a mission to benefit many people, and his existing equipment was not good enough for him. One-week post-mania onset RB's wife noticed he looked 'haggard, wild-eyed and run-down' and during interview he

seemed ‘overwrought’ and ‘hyperactive’, suggesting decreased sleep need (B2). RB was overly talkative, losing his voice through excessive shouting at his wife and talking constantly during interview (B3), this continuous speech suggesting a flight of ideas (B4). RB showed increased goal-directed activity (B6) by planning for the enlargement of his dental practice through drawings and phone-calls, and through pacing and restlessness during the interview. His excessive involvement in the former activity led to catastrophic consequences (B7); he smashed his dental practice walls and equipment and turned away his patients, his continued recklessness led to disinhibited sexual and physically aggressive behaviour toward his family resulting in his arrest and subsequent hospitalization meeting criterion C, also met by his psychotic belief that family members had ‘double crossed’ him.

Diagnostic Considerations

There is no suggestion of substance misuse/other medication or a medical condition that could explain RB’s mania therefore criterion D for a manic episode is met. RB’s manic episode is not better explained by another mental health disorder therefore criteria B for BD-I is met. This diagnosis is strengthened by RB’s three previous hospital admissions due to (assumed) previous manic episodes, there is no suggestion of these occurring within one year, therefore the bipolar disorder (BD) is not rapid cycling (cyclothymic) and there is no suggestion of major depression. The BD is not type II (BD-II), a less severe form in which both mania and depression are present, but mania is hypomanic not requiring hospitalization. RB’s resistance to treatment, physical aggression and rapid shifts into anger further support the diagnosis (Black & Grant, 2014).

Case Formulation

Amongst disorders, BD (type I, type II or cyclothymic), is one of the most heritable (Kring & Johnson, 2018), with estimates around 80% in monozygotic twins (De Pradier, Gorwood, Beaufils, Ades & Dubertret, 2010). Although RB’s familial history is not stipulated it is likely BD exists in his family. Heritability is specific for BD and not a general disposition for psychiatric disorders (Edvardsen et al., 2008), however, genome-wide association studies (GWAS) have found considerable overlap in genetic markers between BD and schizophrenia, so the genetic basis for specific BD heritability remains unclear (Gordovez & McMahon, 2020) and many scholars view the two disorders as a continuum rather than distinct disease entities (Moller, 2003). GWAS reveal BD to be multi-factorial; a small effect of each gene within multiple high-risk genes combine to increase risk (Lasky-Su, Faraone, Glatt &

Tsuang, 2005; Misiak et al., 2018). Scholars, therefore, group functionally related genes into genetic pathways, of which the calcium signalling pathway in BD is most evidence based (Gordovez & McMahon, 2020) with around 30% elevated calcium levels in BD (most prominent in BD-I) whether manic, depressed or medication free. However, the genetic mechanism of cycling into acute symptoms remains unresolved (Gordovez & McMahon, 2020).

A gene in the serotonergic pathway (known to affect mood, emotion, and cognition) coding for the neurotransmitter serotonin transporter (5-HT) is the 5-HTT, modulated by a polymorphism, which produces a long or short allele (s-allele); the s-allele impairs 5-HTT functioning, decreases activity (Lasky-Su et al., 2005; Lesch et al., 1996) and increases the risk of BD (Lasky-Su et al., 2005). Maddaloni et al.'s (2018) rodent study found 5-HT depletion caused mania and abnormal hippocampal neural plasticity. Specific environmental risk factors act upon specific genes, for example, BD risk increases in males but not females when the presence of the TPH1 allele coincides with winter births (Misiak et al., 2018).

64 polymorphisms associated with BD have been found so far (Mullins et al., 2020); these, along with other forms of genetic variation and various known environmental factors give a complex picture of the gene x environment interactions causing BD (see figure 1; Robinson & Bergen, 2021).

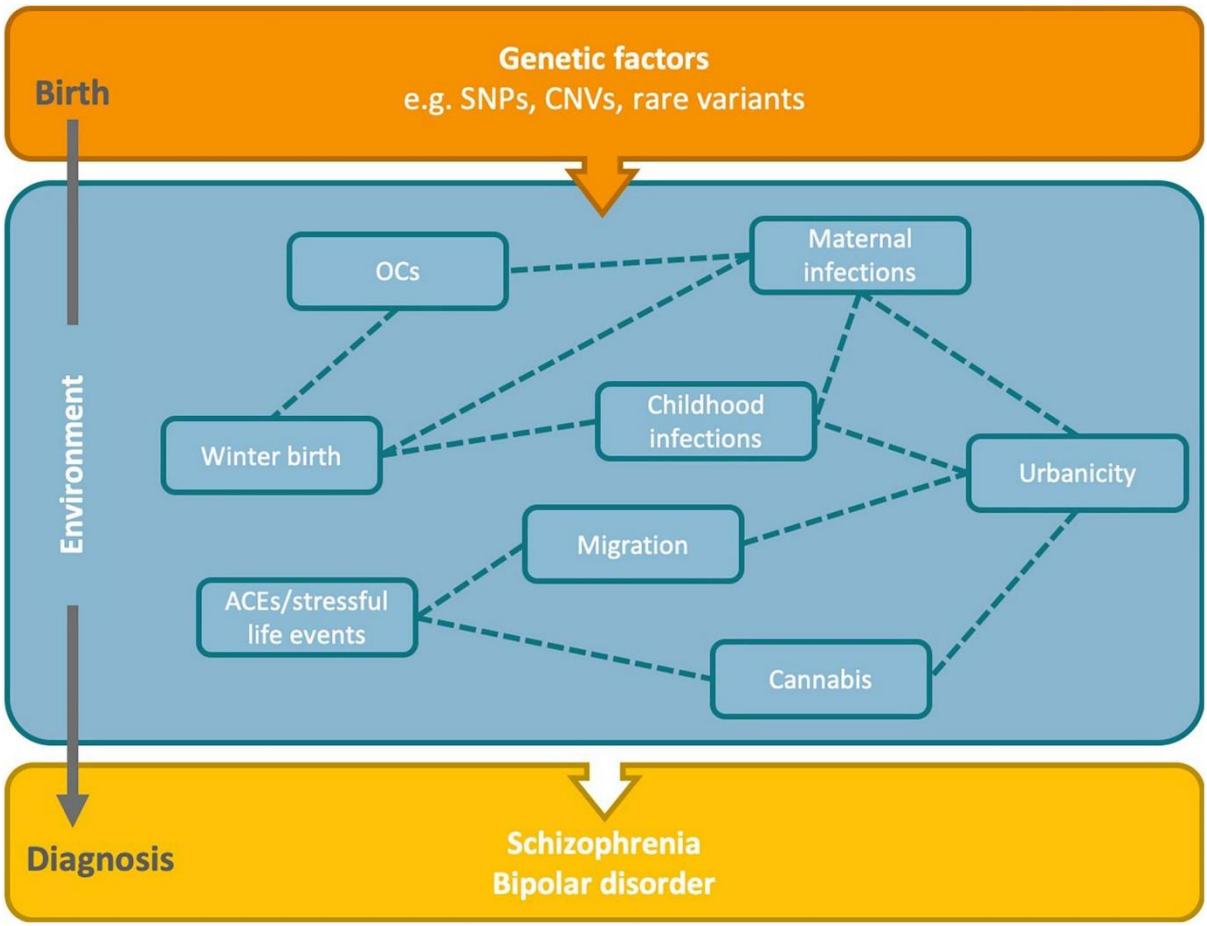

Figure 1.

Hypothesized relationships between genetics and environmental risk factors in the development of BD and schizophrenia. Also shown are potential complex interrelationships between the identified environmental risk factors: OCs, obstetric complications; ACEs, adverse childhood experiences; SNPs, single nucleotide polymorphisms; CNVs, copy number variants. (Robinson & Bergen, 2021).

Whilst several neurotransmitters are implicated in bipolar, serotonin (mentioned above), and dopamine are most well researched (Moller, 2003). The highly regarded dopamine hypothesis stipulates increased dopaminergic neurotransmission leads to mania whilst reduced dopaminergic function leads to depression (Ashok et al., 2017). Despite increased dopaminergia during mania, hypoconnectivity in the salience network causes dysfunction, increasing psychosis risk (Karcher, Rogers & Woodward, 2019) and this lack of equilibrium potentially underlies BD (Ashok et al., 2017). Hengartner and Moncrieff (2018) counter antipsychotic medication impacts dopaminergic pathways, suggesting dopaminergic

dysfunction is a confounding factor rather than the cause of psychosis. Stress alters dopamine function (Hengartner & Moncrieff, 2018) and stressful life events can trigger mania in BD (Saraf et al., 2021). RB may have had work related stress, potentially exacerbated by dopaminergic dysfunction caused by medication.

Reward sensitive dopamine neurons in the limbic system and frontal cortex underlie the behavioural approach system (BAS) dysregulation theory (Alloy & Abramsom, 2010). BD patients - Alloy and Abramson (2010) state - are vulnerable to excessive activation (caused by events involving rewards, goal striving and attainment) or deactivation (caused by failures or non-attainment of goals) of the BAS leading to mania or depression respectively (see figure 2).

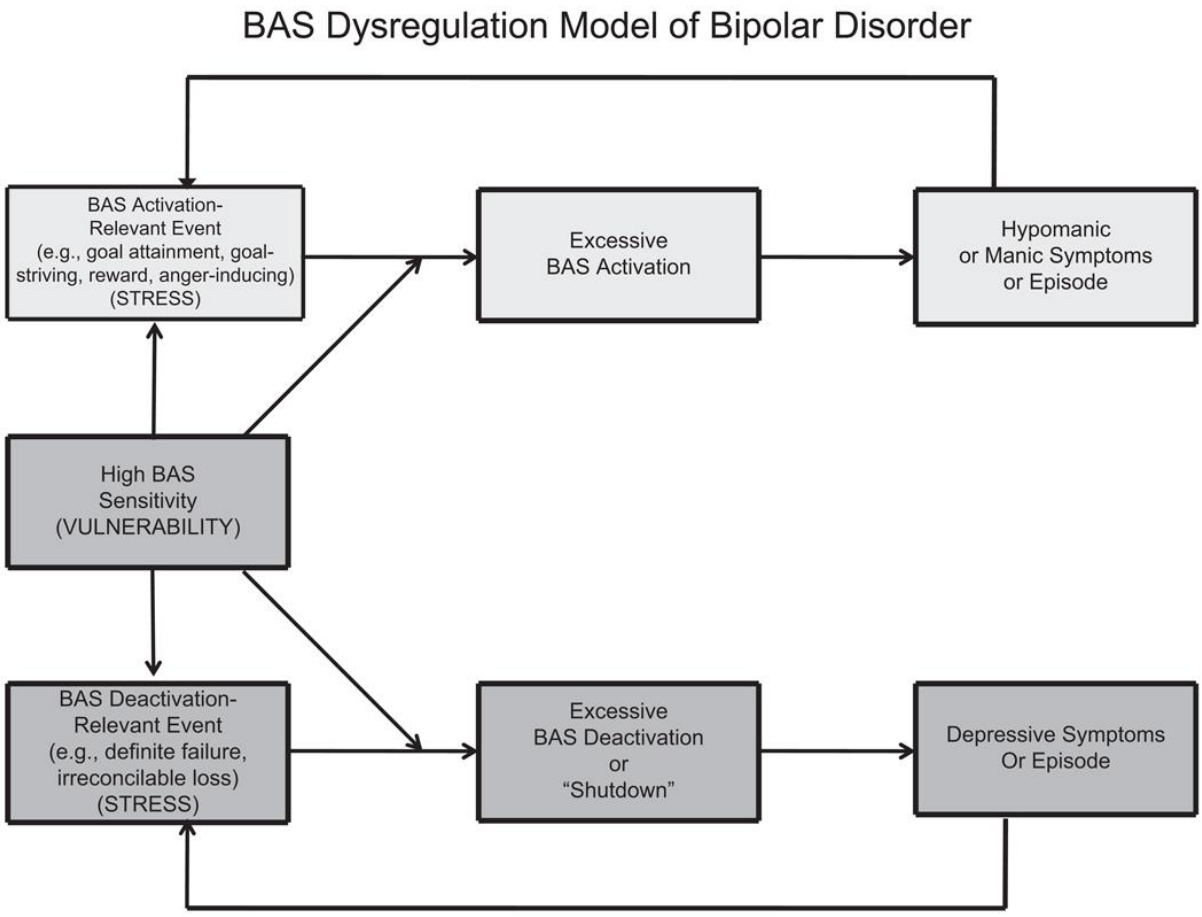

Figure 2.

The behavioral approach system (BAS) dysregulation model of BD (Alloy & Abramsom, 2010).

BD patients self-reporting higher vs lower BAS sensitivity experienced increased manic symptoms and were more likely to have another manic episode quicker (Alloy, Abramson, Urosevic, Bender & Wagner, 2009). Johnson (2005) supports this, stating excessive goal pursuit (described as behavioural expression of genetic vulnerability) triggers mania. Johnson (2005) furthers this behavioural expression overlays a stable (inc. inter-episode) background of high ambitions which is typical in people with a history of mania. Indeed, those with high vs low BAS sensitivity had higher academic achievement, but only when impulsiveness was low (Nusslock, Alloy, Abramson, Harmon-Jones & Hogan, 2008). RB is a dentist with his own practice, suggesting a highly educated and ambitious man, excessive pursuit of work-related goals may have triggered his mania. Excessive goal pursuit is related to longer working hours and higher caffeine intake which disrupts sleep pattern, exacerbating mania (Naidoo, 2020).

Treatment Plan

RB's acute mania needs to be urgently addressed to stabilize his presentation (Harrison, Geddes & Tunbridge, 2018). Although mood stabilizers (such as lithium) are preferred as a monotherapy to avoid side effects/drug interactions, RB demonstrated violent and risky behaviour needing urgent treatment with an anticonvulsant such as sodium valproate to reduce mania (Maddaloni et al., 2018) and/or an anti-psychotic such as olanzapine (Atagün & Oral, 2021). In Niufan et al's. (2008) randomized, double-blind study, Olanzapine was shown to be more effective than lithium in treating acute mania however it is standard practice to treat severe mania with both lithium and olanzapine so underlying mood-stabilization can begin (Harrison et al., 2018). Before lithium can be administered, a full-blood count and liver and kidney function needs assessment, the initial dosage depends on various patient factors (Atagün & Oral, 2021). Risperidone or quetiapine are alternative atypical antipsychotics (Moller, 2003) that can be tried if olanzapine doesn't reduce symptoms. However, if RB becomes acutely delirious/agitated, resisting medication, he may need forced injections of haloperidol, chlorpromazine or zuclopenthixol which act fast, however, these first-generation medications have increased side effects and should be ceased as soon as possible, with a return to an orally administered atypical antipsychotic (Atagün & Oral, 2021). If RB becomes agitated at points during the day a benzodiazepine (e.g., clonazepam), which has a calming effect, can also be prescribed (Battaglia, Lindborg, Alaka, Meehan & Wright, 2003). B12 deficiency (Moller, 2003) and excess gluten, caffeine intake and omega 3 fatty acids (Naidoo,

2020) are linked to mania and RB's diet should be adapted accordingly during hospitalization.

All antipsychotics and benzodiazepines with a high dosage have a sedative effect enabling sleep and recovery speeds up upon sleep improvement (Plante & Winkelman, 2008). However, once RB stabilizes, excessive sleep will impact his quality-of-life (Miller, 2004). Upon stabilization RB should be discharged and managed under the community mental health team where a medication review can take place (Pons et al., 2020).

Olanzapine (or alternative anti-psychotic) dosage should be decreased (to omit sedation) but continued as olanzapine promotes 5-HT functioning, including working with 5-HT_2 receptors to lengthen slow wave sleep; improving sleep quality (Miller. 2004). Despite medication, 70% of bipolar patients suffer disturbed sleep and 55% are insomniacs inter-episode (Kanady, Soehner, Klein & Harvey, 2017). This disrupted sleep-wake cycle increases chance of relapse to either BD pole (Leibenluft & Suppes, 1999). If RB experiences sleep disturbance, CBTI-BD (cognitive behavioural therapy for insomnia in BD) might improve sleep and cognition; in Kanady et al's (2017) BD study, working memory improved when total wake time decreased, and verbal learning improved when sleep time variability reduced.

Lithium is the gold standard in BD-I medication and should be continued for RB with dosage in accordance with blood lithium levels (Volkmann, Bschor & Köhler, 2020). AC et al. (2021) found patients on lithium compared to other medication presented greater volume of the right precentral gyrus, thalamus, and right amygdala, associated with enhanced executive function. And Kato's (2019) review of MRI studies showed patients on lithium had a larger cerebral cortex than those not on lithium. Lithium has other long-term benefits; protecting against suicide, the frequency and severity of mood episodes (Gordovez & McMahon, 2020), and in Harrison, Hall, Mould, Al-Juffali & Tunbridge's (2019) systematic review, lithium appeared to normalise intracellular calcium levels. Despite Lithium's many benefits Milienne-Petiot et al. (2017) argue it still impairs learning and memory compared to healthy subjects, but RB should remain on lithium long-term due to the severity of his illness. Both lithium and sodium valproate increase stress resilience; Saraf et al. (2021) found no difference between euthymic (inter-episode) bipolar patients on lithium/valproate and healthy participants in stress induced dopamine release, showing these drugs stabilize the dopaminergic system. Due to RB's high work stress valproate should continue.

Because medication has serious side effects, they should be used alongside psychological treatment which may assist in counteracting side effects, bringing dosage down, and preventing relapse (Harrison et al., 2018). CBTI-BD has been mentioned above, also particularly for RB, CBT from a BAS dysregulation perspective would help him recognize and challenge increases or decreases in goal striving to prevent a relapse to either pole (Alloy & Abramson, 2010).

The gut microbiome in BD is impaired therefore nutrition is an important part of maintaining stability (Naidoo, 2020). A Mediterranean (high in fresh fruit, vegetables, and wholefoods) vs Western (high in sugar, 'bad' fats, and 'bad' carbohydrates) diet is recommended, the latter which increases anxiety, depression and obesity compared to the former (Naidoo, 2020). Some specific foods will help, for example, spirulina will increase serotonin synthesis (Demelash, 2018) and gluten-free foods protect against anxiety and mania (Naidoo, 2020).

Prognosis

BD is an incurable, debilitating, lifelong illness (Milienne-Petiot et al., 2017) with prevalence of 2% (Carvalho, Firth & Vieta, 2020) and the typical onset age for BD-I is around 20 (Kawa et al., 2005). RB is fifty-six, experiencing a fourth hospitalization, suggesting he cycles into mania (at least) once-per-decade (comparatively low).

RB has maintained married life (married patients have less episodes than single patients; Özerdem, Tunca & Kaya, 2001), provided well for his family and managed his dental practice for over two decades, despite BD, which showcases an optimistic degree of stability. His high level of education is also associated with better outcomes (Özerdem et al., 2001) and his family are clearly very supportive; family support is associated with better compliance in taking medication and better prognosis (Özerdem et al., 2001).

Turvey et al.'s (1999) longitudinal study on BD-I, found that monophasic (exclusively manic or depressive) as opposed to polyphasic (at least two switches in polarity) patients had a better prognosis as they experienced longer periods of emotional stability inter-episode. RB may experience monophasic BD-I whereby he spirals into mania but stabilizes post-episode, not switching to the depression pole, explaining his comparatively stability. However, Turvey et al. (1999) found lengthier episodic duration dictated poorer prognosis (rather than increased cycle frequency), so although RB experiences relatively low-frequency cycling this isn't a marker of prognosis. The duration of RB's episodes are unclear, but they are potentially short enough, with a rapid return to emotional stability gifting RB a good

prognosis. However, each subsequent episode leads to increased cognitive impairment (Rosa et al., 2009) which persists inter-episode regardless of episodic frequency (Harrison et al., 2018) and worsens with age (Rosa et al., 2009).

Rates of comorbidity in bipolar is high, for example, most bipolar patients suffer anxiety, which predicts suicide (Soreca, Frank & Kupfer, 2009), shorter duration of euthymia, co-occurring substance misuse (Cazard & Ferreri, 2012) and is a feature of the depression pole (Soreca et al., 2009). If RB is manic monophasic this doesn't apply which reduces his chance of dying by suicide (suicide rate in BD is 15%; Gordovez & McMahon, 2020) and means his first episode was mania vs depression, the latter which predicts increased obesity and a higher prevalence of comorbidity (Soreca et al., 2009).

To conclude, RB potentially has many factors working in his favour which has enabled him to lead a comparatively stable life within a disorder often much more severe and debilitating. Due to his older age, he should expect increased cognitive impairment and due to his dental career, he should implement more support at his practice and consider reducing work demand (particularly as work stress may have triggered his recent episode). It is highly likely he will cycle into another manic episode, but with continued family support, adherence to medication and sustaining the correct diet his prognosis will continue to be the best it can be.

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