Acta Neuropsychiatrica

cambridge.org/neu

Paediatric bipolar disorder and its controversy

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Perspective

Cite this article: Connors MH. (2023) Paediatric bipolar disorder and its controversy. *Acta Neuropsychiatrica* **35**:96–103.
doi: 10.1017/neu.2022.28

Received: 25 March 2022
Revised: 3 October 2022
Accepted: 3 October 2022
First published online: 14 November 2022

Key words:
bipolar and related disorders; bipolar disorder; child; diagnosis; mood disorders

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Abstract

Objectives: Paediatric bipolar disorder – bipolar disorder occurring in prepubertal children – is a diagnosis subject to considerable controversy. Whilst historically considered to be very rare, proponents since the 1990s have argued that mania can present differently in children and, as such, is much more common than previously thought. Such proposals raise questions about the validity of proposed phenotypes and potential risks of iatrogenic harm. ***Methods:*** I critically examine the construct of paediatric bipolar disorder using Robins and Guze's (1970, *American Journal of Psychiatry* **126**, 983–987) influential criteria for the validity of a psychiatric diagnosis. I review, in turn, evidence relating to its clinical description, delimitation from other conditions, follow-up studies, family studies, laboratory studies, and treatment response. ***Results:*** Across domains, existing research highlights significant challenges establishing the diagnosis. This includes significant heterogeneity in operationalising criteria for children; variable or poor inter-rater reliability; difficulty distinguishing paediatric bipolar disorder from other conditions; large differences in rates of diagnosis between the United States of America and other countries; limited evidence of continuity with adult forms; and a lack of evidence for proposed paediatric phenotypes in children at genetic high-risk of the condition. Laboratory and treatment studies are limited, but also do not provide support for the construct. ***Conclusions:*** Evidence for the more widespread existence of paediatric bipolar disorder and its various proposed phenotypes remains weak. The ongoing popularity of the diagnosis, most evident in America, may reflect social pressures and broader limitations in psychiatric nosology. The uncertainty around the diagnosis highlights the need for careful longitudinal assessment of children potentially affected.

Summations

- Paediatric bipolar disorder – bipolar disorder in prepubertal children – remains a controversial construct on both conceptual and empirical grounds and is associated with risk of iatrogenic harm.
- The ongoing popularity of the diagnosis seems to outstrip evidence for it and may reflect broader social pressures and current limitations of psychiatric nosology.
- Reliably establishing the diagnosis appears to be challenging and seems to require longitudinal assessment, informant reports, and careful consideration of both the rarity of the diagnosis and other potential contributory factors.

Perspectives

- The possibility of bipolar disorder having its first onset in childhood is generally not disputed. Debates have instead focused on its relative prevalence – previous evidence suggested it was extremely rare – and the validity of proposed phenotypes claimed to be alternative prepubertal manifestations of bipolar disorder.
- There remain significant international differences in opinions on the construct, particularly between authors from the United States of America and the rest of the world.
- Research on paediatric bipolar disorder is limited by the heterogeneity of diagnostic criteria and difficulties establishing inter-rater reliability. There is likely to be significant variation across research samples depending on the criteria used.

Introduction

Bipolar disorder has long been thought to have its onset in adolescence and early adulthood. In an early study of over 900 patients, Kraepelin (1921) found that the first episode of what he termed 'manic-depressive insanity' was most common between the ages of 15 and 40 (69.1% of patients). A small proportion had symptoms between the ages of 10 and 15 (2.5%) but

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episodes prior to 10 were very rare (0.4%). Studies over subsequent decades replicated this pattern of findings and highlighted that depressive symptoms, rather than mania, typically develop first (Angst *et al.*, 1973; Angst, 1986; Goodwin & Jamison, 1990). This view, however, has been challenged since the mid-1990s by several influential American researchers, who have argued that manic symptoms occur much earlier and are relatively common in prepubertal children. According to this view, mania in childhood can manifest differently to adults, taking the form of multiple mood swings each day ('ultradian cycling' or 'narrow phenotype'; Geller *et al.*, 1995) or chronic irritability ('broad phenotype'; Wozniak *et al.*, 1995). Such views remain highly controversial on both conceptual grounds and from concerns around potential iatrogenic harm. In this paper, I examine the construct of paediatric bipolar disorder (PBD) – bipolar disorder occurring in prepubertal children – using the framework of Robins and Guze's (1970) criteria for the validity of a psychiatric diagnosis. I review evidence relating, in turn, to its clinical description, delimitation from other conditions, follow-up studies, family studies, laboratory studies, and treatment response (for further clarity, I summarise cited sources in a table as an online appendix). From this, I argue that, rare cases notwithstanding, evidence for the more widespread existence of PBD remains weak. I also discuss broader trends in psychiatry that have contributed to the controversy.

Clinical description

A first step in defining a psychiatric condition is characterising its clinical presentation. This is particularly challenging during childhood due to the developmental changes, communication barriers, and extrinsic factors that can affect clinical presentations and confound assessment. In the case of PBD, proposed defining features of mood lability and chronic irritability overlap with normative features of childhood (Malhi *et al.*, 2020). They also represent a significant broadening of existing categories, removing requirements for mania to be restricted to episodes lasting at least several days or be considered abnormal relative to a person's usual mental state (Malhi, 2016; Duffy *et al.*, 2020). Such changes raise questions about whether the condition can be reliably distinguished from normative behaviour and other disorders and whether it is related to adult bipolar disorder.

These issues are particularly relevant given challenges applying existing diagnostic criteria for bipolar disorder in adults. Several studies, for example, indicate limited diagnostic stability (Baca-Garcia *et al.*, 2007; Cegla-Schvartzman *et al.*, 2021) and frequent over-diagnosis, with more than 50% of adults diagnosed later found not to have the condition with more careful assessment (Zimmerman *et al.*, 2008; Zimmerman *et al.*, 2010; Mitchell, 2012; Zimmerman, 2016). Missed diagnosis, by contrast, appears to be much less common (Zimmerman, 2016). Contributing to over-diagnosis, people in the general population often endorse manic symptoms on initial questioning (Das *et al.*, 2005), sometimes more frequently than patients at genetic risk of or who actually develop bipolar disorder (Goodday *et al.*, 2017). Likewise, inter-rater reliability for bipolar disorder type I in the Diagnostic and Statistical Manual of Mental Disorders (DSM) field trials was only moderate (intraclass kappa 0.56; Regier *et al.*, 2013) representing agreement of less than 35% (McHugh, 2012) for highly trained psychiatrists under study conditions, with significant variability across sites.

Proponents of PBD have adapted DSM criteria, relying on checklists of symptoms and signs. Such an approach, however, can be particularly prone to error in a paediatric context if based on a cross-sectional assessment without consideration of contextual factors and informant reports (Duffy *et al.*, 2020). Many features of mania can also be difficult to operationalise across age groups. Mood lability and irritability, for example, are non-specific and can arise from both situational factors (e.g. unfamiliar environment, discomfort) and other clinical conditions (e.g. neurodevelopmental and externalising disorders, trauma). Other signs, such as distractibility, flight of ideas, risky behaviour, and increased goal-directed activity, may likewise be less reliable in childhood. Consistent with this, the reported characteristics of childhood mania and hypomania vary considerably both across and within studies (Van Meter *et al.*, 2016; Ryles *et al.*, 2017). In a similar way, scales and interviews used to diagnose PBD vary considerably in their criteria, including in how mood criteria are conceptualised, whether symptoms need to differ from a child's baseline state, requirements for features to co-occur, and general administration and scoring (Galanter *et al.*, 2012). As such, there is likely to be significant variation across research samples depending on the criteria used.

Due to the controversy around PBD, DSM 5 introduced 'disruptive mood dysregulation disorder' as an alternative diagnosis for children with chronic irritability (American Psychiatric Association, 2013), the so-called broad phenotype of PBD (Pavuluri *et al.*, 2005). This diagnosis, however, had poor inter-rater reliability (intraclass kappa 0.25; Regier *et al.*, 2013), representing agreement of less than 15% (McHugh, 2012) and indicating limited utility. The alternative narrow phenotype – mood lability or ultradian cycling – has been more difficult to assess because of variation in how it is defined and categorised. Many studies classed it within 'bipolar disorder not otherwise specified' using DSM IV criteria (Axelson *et al.*, 2011), which can be met in various ways and so is more ill-defined. Other studies (Wozniak *et al.*, 2017) diagnosed children with ultradian cycling as having 'bipolar I' despite this not being standard DSM criteria, representing a further blurring of diagnostic boundaries (Parry *et al.*, 2021). Adopting strict DSM criteria, however, does not solve these problems: DSM 5 field trials were unsuccessful for the diagnosis of bipolar disorder in children and unable to obtain an accurate estimate of inter-rater reliability due to large variability (Regier *et al.*, 2013). Another study found very weak inter-rater reliability between PBD experts diagnosing mania, hypomania, and most associated features in children (most interclass correlations < 0.50 with very large variability; Larios *et al.*, 2018a, b).

A further challenge to defining the condition is the fact that PBD has been disproportionately diagnosed within the United States of America (USA). Over the decade after PBD was first proposed, in the USA, visits involving the diagnosis increased 40 times in primary care (Moreno *et al.*, 2007) and PBD became the most common discharge diagnosis for children from inpatient units (Blader & Carlson, 2007). This is in sharp contrast to much of the rest of the world, where diagnoses of bipolar disorder prior to mid-adolescence remain rare (James *et al.*, 2014). The hospital discharge rate, for example, of PBD in the USA between 2000 and 2010 was 96 per 100,000 population – 8 times higher than Australia, 15 times higher than New Zealand, 64 times higher than Germany, and 106 times higher than England (Clacey *et al.*, 2015). By contrast, in adults, discharge rates were still higher in the USA, though more comparable: between 1.1 and 5.1 higher than the other countries (Clacey *et al.*, 2015). This marked disparity between the USA and other countries specifically for PBD suggests corresponding differences in diagnostic practice. Consistent with

https://doi.org/10.1017/neu.2022.28 Published online by Cambridge University Press

this, when presented with vignettes of emotionally dysregulated children, psychiatrists from the USA were much more likely to diagnose bipolar disorder than psychiatrists from the United Kingdom (Dubicka *et al.*, 2008).

Proponents of PBD vary in their response to these findings. Some have claimed that the condition is common worldwide and disputed that there are differences in actual prevalence. One meta-analysis by proponents of the condition reported that the global prevalence of PBD was 1.8% – later updated to 3.9% – and that the USA did not differ from other countries (Van Meter *et al.*, 2011; Van Meter *et al.*, 2019). Almost all included studies, however, were of adolescents, where the diagnosis is less controversial. A separate meta-analysis focusing on children aged 12 or younger in community samples identified only a single case of bipolar disorder worldwide, yielding a prevalence of <0.02% and challenging claims of its ubiquity (Douglas & Scott, 2014). Re-examination of Van Meter *et al.*'s data focusing on children and younger adolescents similarly showed very low rates, albeit slightly higher in the USA (Parry *et al.*, 2018, 2021). A further limitation of the Van Meter *et al.* papers was that analyses grouped different types of bipolar disorder together, despite the diverse diagnostic criteria used across studies, likewise complicating interpretation of their findings (Carlson, 2018; Parry *et al.*, 2018; Duffy, 2019; Parry *et al.*, 2019a, 2021).

Other proponents of PBD, by contrast, have accepted a higher prevalence in the USA and proposed possible aetiological explanations, including correspondingly greater rates of genetic vulnerability, childhood adversity, obesity with associated inflammation, and use of stimulant and antidepressant medication in the USA compared to other countries (Reichart *et al.*, 2000; Post *et al.*, 2017). Others have suggested that clinicians in the USA have appropriately greater awareness of PBD than other countries, which, in turn, explains the higher rates of diagnosis (Stringaris & Youngstrom, 2014). Such international differences in diagnosis rates are reflected in views expressed in publication: American authors are more likely to support the construct, whereas authors from other countries are more likely to be sceptical (Parry *et al.*, 2019b). Given such divergences, there appears to be considerable uncertainty about the diagnosis's clinical description.

Delimitation from other disorders

A second criterion for diagnostic validity is the ability to distinguish the condition from other disorders. From the outset, a challenge has been to distinguish PBD from attention deficit hyperactivity disorder (ADHD). Studies on PBD have consistently reported high rates of comorbid ADHD, up to 98% in initial descriptions (Wozniak *et al.*, 1995). A systematic review reported a mean prevalence of 48% across children and adolescents with PBD, with significantly higher rates in those diagnosed in childhood (Frías *et al.*, 2015). This level of comorbidity appears to be greater than what is found in adults: around 16% of adults diagnosed with bipolar disorder also have a lifetime diagnosis of ADHD (Schiweck *et al.*, 2021). Longitudinal studies following children at high risk of bipolar disorder into adulthood show similar (Lau *et al.*, 2018) or lower rates of ADHD, usually approximating rates in the general population after adjusting for family and socioeconomic factors (Duffy, 2012).

The high co-occurrence of PBD and ADHD, particularly in children, may reflect limitations of relying on symptom checklists, where one symptom may be counted as a feature of both disorders, and the challenges of applying criteria in developmentally appropriate ways (Carlson & Klein, 2014). More critically, it raises questions about their distinctness and whether supposed mania may instead be due to ADHD. Indeed, there is strong evidence that emotional dysregulation is a core feature of ADHD, even if not captured in DSM criteria (Barkley, 2015; Faraone *et al.*, 2019). Rates of oppositional defiant disorder are also elevated in PBD (around 31%; Frías *et al.*, 2015), raising analogous questions. Clinicians, moreover, appear to have difficulty in reliably distinguishing bipolar disorder II from chronic irritability in children, including what would otherwise be termed oppositional defiant disorder or disruptive mood dysregulation disorder in current nomenclature (Evans *et al.*, 2021).

A further challenge has been to distinguish the symptoms of PBD from those associated with developmental trauma. Disrupted attachment and various forms of abuse and neglect can manifest as emotional dysregulation, irritability, and challenging behaviour, mimicking many of the outward features of PBD. Trauma-related symptoms, however, have a very different aetiology and are unlikely to respond to pharmacotherapy alone. Despite this, relatively few studies on PBD have assessed attachment or abuse and, of those that have, many did so in a cursory or dismissive fashion (Parry, 2012, 2021). Some proponents, for example, reported implausibly low rates of trauma relative to the general population (Geller *et al.*, 2000); others found significantly elevated rates but suggested that mania was likely to be an antecedent, rather than a consequence, of trauma (Biederman *et al.*, 2000). Studies since then, however, have tended to more consistently find high rates of trauma in patients diagnosed with PBD (Axelson *et al.*, 2011), including many later found to have been misdiagnosed (Carlson *et al.*, 2009).

Potential misinterpretation of trauma-related symptoms as bipolar disorder appears to occur in adults. Studies in adults reveal frequent misdiagnosis of borderline personality disorder, a condition associated with childhood trauma, as bipolar disorder (Zimmerman *et al.*, 2010). Consistent with this, across age groups, clinicians in the USA diagnose bipolar disorder more frequently, but borderline personality disorder less frequently, compared to other countries (Clacey *et al.*, 2015). Patients with post-traumatic stress disorder in adulthood may likewise be misdiagnosed with bipolar disorder due to similar features between the two disorders (e.g. increased irritability, poor sleep, and difficulty concentrating; Cogan *et al.*, 2021). Altogether, when combined with the issues of heterogeneous criteria and limited inter-rater reliability, there would seem to be significant challenges in consistently distinguishing PBD from other conditions.

Follow-up studies

A third criterion for validity is the stability of the diagnosis and homogeneity in clinical outcomes. Given the proposal that PBD is a prepubertal form of the adult condition, longitudinal studies would seem to provide a direct test. For the broad phenotype, chronic irritability in childhood appears to be unrelated to bipolar disorder later in life (Brotman *et al.*, 2006; Leibenluft *et al.*, 2006; Stringaris *et al.*, 2009; Althoff *et al.*, 2010; Stringaris *et al.*, 2010). Chronic irritability instead seems to predict other mood and anxiety disorders (Brotman *et al.*, 2006; Leibenluft *et al.*, 2006; Stringaris *et al.*, 2009; Althoff *et al.*, 2010; Stringaris *et al.*, 2010). As a result, many proponents of PBD now advise against accepting irritability alone as the basis for diagnosis and recommend the need for further corroborating evidence (Goldstein *et al.*, 2017). The

https://doi.org/10.1017/neu.2022.28 Published online by Cambridge University Press

notion that irritability provides a strong suggestion of potential PBD, however, remains influential (Patino & DelBello, 2021).

For the narrow phenotype, evidence is more contentious, in part because of variation in diagnostic criteria. One influential study, for example, reported that 44% of patients with PBD had formal bipolar disorder after eight years (Geller *et al.*, 2008). The study, however, accepted bipolar diagnoses across time points based on ultradian cycling, arguably indicating persistence of affective lability, rather than true transition to bipolar disorder (Parry, 2021). Nevertheless, other studies report that around 45% of children with subthreshold symptoms, including ultradian cycling, develop bipolar disorder after five years (Axelson *et al.*, 2011), and around 60% of children meeting DSM criteria for bipolar disorder have a recurrence of depression or mania within two-to-four years (Birmaher *et al.*, 2006; Birmaher *et al.*, 2009; Stringaris *et al.*, 2010; Cirone *et al.*, 2021). Whilst providing stronger support for continuity, the findings also potentially undermine the initial diagnosis in around half of patients without further follow-up. Indeed, diagnosis in early adulthood is not definitive given the issues of diagnostic reliability already noted and evidence from population-based American databases suggesting that many people diagnosed with bipolar disorder prior to the age of 25 might not continue to meet criteria for the disorder beyond this (Cicero *et al.*, 2009). The findings also diverge from longitudinal studies of children at genetic risk of bipolar disorder, to which I now turn.

Family studies

A fourth criterion for validity is family studies. Bipolar disorder is highly heritable (Gordovez & McMahon, 2020) and children of a parent with bipolar disorder have an 8–10-fold risk of developing the condition relative to the general population (Duffy *et al.*, 2000, Duffy *et al.*, 2020). This risk reaches 52-fold if both parents are affected (Gottesman *et al.*, 2010). As such, children of parents with bipolar disorder are an important high-risk group to study the condition's onset (Duffy *et al.*, 2020). They would also seem most likely to exhibit early symptoms (Duffy *et al.*, 2020). To this end, multiple independent longitudinal studies have followed high-risk children through childhood into early adulthood.

As reviewed in detail elsewhere (Duffy, 2007; Duffy *et al.*, 2011; Duffy, 2012; Duffy *et al.*, 2017; Lau *et al.*, 2018; Duffy *et al.*, 2020), these studies indicate that bipolar disorder most commonly first presents with depression and sometimes intermittent subthreshold manic symptoms in adolescence or early adulthood. Onsets prior to this, however, appear very rare. Symptoms in childhood that predict bipolar disorder include anxiety and sleep difficulties (Duffy *et al.*, 2017). ADHD and other externalising disorders, by contrast, which are common in PBD, do not seem overly represented after controlling for family and socioeconomic factors (Duffy *et al.*, 2017). Most critically, proposed phenotypes of irritability and mood lability have not been observed in the vast majority of studies (Duffy *et al.*, 2020).

Laboratory studies

A fifth criterion for diagnostic validity is laboratory research, including physiological, radiological, and anatomical findings. Such findings, according to Robins and Guze (1970), are likely to be more reliable and precise than clinical descriptions. Across psychiatry, however, the existence of such biomarkers remains elusive. In bipolar disorder in adults, approaches using molecular biology, immunology, genetics, and neuroimaging have offered promising findings, though all remain too tentative and preliminary for translational application so far (Kennedy *et al.*, 2015; Carvalho *et al.*, 2020; McIntyre *et al.*, 2020). As such, there are currently no biomarkers to validate potential paediatric forms.

Several studies have compared adolescents with chronic irritability to those with a formal bipolar diagnosis (Rich *et al.*, 2007; Rich *et al.*, 2011). These revealed differences in neuroimaging and electroencephalography, further undermining the notion that chronic irritability constitutes bipolar disorder, though leaving unexamined the diagnosis's validity in the control group. Other studies have compared youths diagnosed with PBD and healthy controls. These suggest that patients diagnosed with PBD have deficits in cognition (Elias *et al.*, 2017), social cognition (Halac *et al.*, 2021), and emotion regulation (Khafif *et al.*, 2021) and show increased amygdala reactivity to emotional stimuli relative to controls (Simonetti *et al.*, 2022), though without any reliable structural differences in neuroimaging (Simonetti *et al.*, 2022). Such studies, however, do not clearly address questions of aetiology or nosology. A core challenge for basic research remains the heterogeneity and limited inter-rater reliability of the clinical diagnosis. Without first addressing this, samples recruited for research are likely to be similarly heterogeneous, limiting the generalisability of any resulting findings.

Treatment

Although not specified in Robins and Guze's (1970) original criteria, Kendler (1980) proposed response to treatment as a further criterion for diagnostic validity. In the case of PBD, antipsychotics – sometimes with concurrent stimulant medication – appear to be more effective in managing manic symptoms than lithium or anticonvulsants (Duffy *et al.*, 2018). These findings seem to indicate further discontinuity with adult forms of the condition, where lithium shows comparable effectiveness in treating acute mania to most antipsychotics (McKnight *et al.*, 2019). Evidence for the long-term benefits of interventions in children – a key justification for diagnosing PBD – also remains unclear (Yee *et al.*, 2019). The longitudinal study arguably providing strongest evidence for potential continuity into adulthood found no long-term benefit of pharmacological treatment in children and that psychosocial treatments predicted worse outcomes (Axelson *et al.*, 2011). By contrast, early interventions and psychosocial treatments in adolescents and adults with a more established diagnosis of bipolar disorder show significant promise (Berk, 2007; Castle *et al.*, 2009; Vieta *et al.*, 2018).

Broader controversies

Altogether, evidence for the widespread prevalence of bipolar disorder in prepubertal children appears weak. There appears to be difficulty in reliably distinguishing it from other conditions and there is limited evidence of continuity with adult forms. The diagnosis is also associated with three significant potential harms. First, treatment usually entails psychotropic medication, including antipsychotics and lithium. These place children at risk of cerebral atrophy (Fusar-Poli *et al.*, 2013; Bastiampillai *et al.*, 2018) and other well-known side effects such as weight gain, diabetes, dyslipidaemia, thyroid dysfunction, movement disturbances, renal failure, cardiac arrhythmias, and death (Taylor *et al.*, 2018). Second, misdiagnosis can have destructive psychological effects during a critical period of development. The diagnosis invokes altered conceptions of identity and agency – revolving around supposed

https://doi.org/10.1017/neu.2022.28 Published online by Cambridge University Press

biological illness and an external locus of control – and can lead to diminished expectations and stigma from others (Parry, 2021). Finally, misdiagnosis can lead to overlooking and failing to address the actual causes of the emotional and behavioural difficulties. These could include neurodevelopmental, externalising, and learning disorders; attachment difficulties; family dynamics; and trauma (Parry, 2021).

Despite this, the diagnosis continues to be highly influential, particularly within the USA. Questions remain as to why its influence so outstrips evidence for it. Perhaps most importantly, the diagnosis appears to fill a clinical need, namely severe emotional dysregulation in children. This undoubtedly causes significant distress and burden to those affected (Perez Algorta *et al.*, 2018; Vaudreuil *et al.*, 2019). Diagnostic options, however, may be limited for children, including many who otherwise meet criteria for ADHD (Barkley, 2015; Carlson & Klein, 2018; Faraone *et al.*, 2019). Particular features of the U.S. healthcare system may also contribute to the diagnosis' influence, including 'diagnostic upcoding' – whereby more serious diagnoses attract greater insurance reimbursement, thereby incentivising them – and the high costs of medical care, leading clinicians to prioritise brief reviews focused on medication (Parry *et al.*, 2015; Parry, 2021). The diagnosis, moreover, provides a large potential market for the pharmaceutical industry, which has used well-worn practices, such as funding supportive research, 'key-opinion leaders', medical education programmes, advocacy groups, and 'disease awareness' campaigns, to promote it (Healy & Le Noury, 2007; Parry *et al.*, 2015; Parry, 2021). Further support may come indirectly through the influence of particular high-profile researchers, journal editors, and institutions promulgating the construct; the effect of training and socialisation on clinicians and researchers; and the highly competitive nature of grant applications and journal publication that tend to favour claims of biological determinacy over the perceived relative intangibility of psychosocial factors.

More fundamentally, the phenomenon reflects limitations in current psychiatric nosology (Carlson & Klein, 2014; Parry *et al.*, 2015; Duffy *et al.*, 2020; Parry, 2021). In the absence of established biological mechanisms, classification continues to rely on observable features to distinguish disorders (American Psychiatric Association, 2013; World Health Organization, 2018). Such an approach is inherently prone to error as similar features can arise from distinct mechanisms. This approach has also resulted in narrowing assessment to the relevant checklist and reifying criteria, such that disorders are taken to be the criteria themselves (Hyman, 2010). When combined with social pressures in favour of a diagnosis and current trends toward biological reductionism, a hypothesised collection of features can take on the appearance of a distinct and widespread biologically determined entity, despite evidence of unclear boundaries, heterogeneity across patients, and limited continuity over time. Similar forces have the potential to shape research by influencing the beliefs and assumptions that guide how data are collected and interpreted. Faced with this uncertainty in the clinic, longitudinal assessment with careful consideration of biological, psychological, and social influences would seem to be the most effective way to protect patients' and their families' best interests.

Supplementary material. To view supplementary material for this article, please visit https://doi.org/10.1017/neu.2022.28.

Acknowledgements. None.

Author contributions. MHC conceptualised the paper, drafted the manuscript, revised it critically for important intellectual content, and approved the final version

Financial support. This research received no specific grant from any funding agency, commercial, or not-for-profit sectors.

Conflict of interest. None.

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