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Misconceptions about paediatric bipolar disorder

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Letter to the Editor

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Introduction

Professor Malhi et al. (2023) accept my overall critique of the construct of paediatric bipolar disorder and endorse the substantive content of my article (Connors, 2023). They nevertheless raise concerns with certain points. In particular, they criticise the final sentence of my article that recommended a biopsychosocial formulation of patients, suggest somewhat paradoxically that I might hold a latent belief in biological determination, and fault my limited discussion of the historical reasons for the introduction of disruptive mood dysregulation disorder (DMDD) as a diagnosis. Malhi et al. proceed to propose an alternative terminology for paediatric bipolar disorder that they argue better captures developmental changes. Malhi et al.'s criticisms reflect misinterpretations of my article. I appreciate the opportunity to respond and expand on points not possible in my original article. I address their criticisms and consider their proposal in turn.

Diagnosis versus formulation

Malhi et al.'s first criticism was with my concluding sentence that proposed the need to carefully consider biological, psychological, and social influences on children during assessment. Malhi et al. describe this as 'customary' and suggest that it implies an endorsement of current diagnostic practice. Such an interpretation, however, constitutes a considerable mischaracterisation of my position. Significantly, Malhi et al. omit the first part of the quoted sentence – that this suggestion was for clinical practice and include longitudinal assessment. They also ignore the context, namely the detailed analysis showing the invalidity and lack of reliability of the diagnosis; my discussion of the diagnosis' considerable harms; and the preceding sentences in which I highlight the contribution of brief cross-sectional assessments, checklist diagnostic criteria, and biological reductionism to the construct's continuing influence.

Contrary to Malhi et al.'s response, I consider both longitudinal assessment and the consideration of biological, psychological, and social factors as important and often missed in clinical practice. Brief cross-sectional assessments focused on whether patients meet diagnostic checklists are common and contribute to overdiagnosis (Carlson and Klein, 2014; Parry *et al.*, 2015; Duffy *et al.*, 2020). Such an approach is prone to missing contextual factors and can be distorted by the inclusion of alternative diagnostic criteria without strong evidence, such as proposed alternative paediatric phenotypes. The reliance on diagnostic checklists also contributes to reification of the disorder, such that a disorder can be defined entirely by the criteria themselves (Parry *et al.*, 2015). As a result, patients with features resembling those on a checklist can be diagnosed as having an enduring condition with a supposed biological diathesis – even if the patients' features are transitory or due to situational, psychological, or social factors – with risks of inappropriate long-term pharmacotherapy and altered concepts of self-identity (Parry, 2021).

My concluding sentence was thus intended as a modest practical suggestion for clinical practice that might serve as counterpoints to such trends: ensuring longitudinal, rather than cross-sectional, assessment and considering formulations of patients, rather than relying solely on diagnostic checklists. Contrary to Malhi et al., I contend that this represents an important shift in clinical thinking, moving beyond description – whether patients meet diagnostic checklists – to considering potential explanations for patients' presentations within formulations – including the biological, psychological, and social factors that could account for clinical features (Owen, 2023). This shift from a descriptive focus to an analytical and evaluative one thus might help broaden differentials beyond diagnostic labels and loosen the hold of checklists, biological reductionism, and system-level pressures that encourage overdiagnosis. As such, it may offer some protections against iatrogenic harm, particularly when accepting other aspects of my article. The approach may also be pertinent given other forces that sustain the diagnosis despite weak evidence – including dissenting opinions from prominent academics, influence from the pharmaceutical industry, insurance and reimbursement incentives, peer practice, parental advocacy, and so forth – and the repeated failure of previous attempts at diagnostic diversion and proscription.

Supposed aetiological assumptions

Malhi et al.'s second criticism of my article is for what they speculate might be my latent belief in biological aetiologies and biomarkers in bipolar disorder. They propose that this might account for why I failed, in their interpretation, to advocate for more radical nosological change in my final sentence. This criticism, however, overlooks sections of my article that discussed the errors and harms of biological reductionism. There is also an apparent contradiction in simultaneously critiquing my recommendation for a biopsychosocial conceptualisation and suggesting that I might hold latent beliefs in biological determination.

The only evidence that Malhi et al., provide for my supposed latent biological beliefs is a statement I made about the possibility of bipolar disorder having its first onset in childhood being generally not disputed – a statement referring to the previous literature, rather than my own views, and indicating how debate has instead focused on rates of diagnosis and proposed alternative phenotypes. Later, however, Malhi et al. state, 'we agree with Connors that theoretically, mania may in some instances, start in childhood . . . ', which appears to be somewhat inconsistent and to undermine the basis of their concerns. To add to this confusion, Malhi et al. recommend clarifying nosology for research 'to identify potential early markers', raising questions about their own belief in biomarkers in childhood. Indeed, their conclusion restates a point I made in my article, namely that without addressing the poor validity and reliability of the diagnosis, it is unlikely that basic research can progress.

Contrary to Malhi et al.'s claims, the possibility of bipolar disorder having its onset in childhood is supported by research, rather than any commitment to a biological orientation. Kraepelin's (1921) study of over 900 patients found a very small proportion of cases with pre-pubertal onset, as have other studies and case reports since then (Anthony and Scott, 1960; Weller *et al*., 1986; Goodwin and Jamison, 2007; Douglas and Scott, 2014). More recently, several prospective longitudinal studies with selective recruitment report pre-pubertal cases with continuity into later adolescence (Birmaher *et al*., 2009; Stringaris *et al*., 2010). In addition, a number of pre-eminent critics of paediatric bipolar disorder have reported encountering or being aware of rare cases (Carlson *et al*., 2009; Parry, 2021) or cited studies that have (Duffy *et al*., 2020). This last point supports the other aspect of my claim about the focus of the debate being about prevalence and the validity of alternative phenotypes, rather than the absolute existence of bipolar disorder in children. In any case, such converging sources of evidence would seem to suggest that a childhood onset of bipolar disorder is possible. Such evidence, however, does not alter my overall conclusions that such an onset appears to be very rare, that the diagnosis is difficult to establish due to the poor reliability and validity of criteria in this age group, and that current diagnostic practices appear to be associated with significant harms.

Historical context

Malhi's final criticism is that my article did not discuss the historical reasons for the introduction of DMDD in detail. Such a focus was outside the scope of my article, which examined the construct validity of paediatric bipolar disorder using clearly pre-specified criteria, and has been discussed in detail elsewhere (Lochman *et al*., 2015; Carlson, 2016; Parry, 2021). I also note that Malhi and colleagues seemed to overlook this issue in their own previous papers on paediatric bipolar disorder (Malhi *et al*., 2020; Malhi and Bell, 2021), although they cover some aspects in a separate opinion paper on DMDD (Malhi and Bell, 2019).

While endorsing my points about DMDD's limited reliability and validity, Malhi et al. identify a further problem with DMDD: its failure to prevent polypharmacy and hospitalisation in those receiving a diagnosis. Another limitation not discussed is DMDD's restricted influence on some proponents of paediatric bipolar disorder who continue to use the latter term (Parry, 2021). More significantly, however, the historical context of DMDD that Malhi et al. seek to highlight would seem to have implications for their proposal for altered terminology. In particular, given DMDD's failure as an alternative diagnosis to paediatric bipolar disorder, it is unclear whether it would be helpful to introduce other diagnostic terms with the apparent same goal and vulnerability to co-option.

Malhi et al.'s proposed nomenclature

Malhi et al. argue that 'paediatric bipolar disorder' should be replaced with two alternative 'developmentally informed categories': 'childhood bipolar disorder' for pre-pubescents, which they suggest should be used 'largely for research', and 'adolescent bipolar disorder' for post-pubescents, which they do not offer analogous restrictions. They also suggest that the term 'paediatric bipolar disorder' could be redefined to refer to adult patients' recall of symptoms occurring prior to adulthood. Malhi et al.'s proposal would have the advantage of clarifying a distinction sometimes obfuscated by proponents of the disorder whereby adolescents are included within the diagnosis to give greater legitimacy to prepubescent forms (Goodwin and Jamison, 2007; Parry *et al*., 2018). Their proposal, however, does not address many other problems associated with the diagnosis. Indeed, given the construct's poor reliability and validity in pre-pubertal children, it is unclear why Malhi et al. seek to preserve the construct with a new designation, rather than abandon it altogether. Providing such designations could further reify and legitimise the condition, while leaving their preferred terms open to unintended misuse.

A related issue with Malhi et al.'s proposal is that the terms they seek to introduce have been used previously. 'Childhood bipolar disorder' has long been used interchangeably with 'paediatric bipolar disorder' to refer to supposed pre-pubertal forms (Geller and Luby, 1997; Biederman *et al*., 2003). Likewise, 'adolescent bipolar disorder' has been used to introduce dubious diagnostic features for bipolar disorder within this age group (e.g., defying authority figures, partying, aspiring to become a rock star, and developing romantic fantasies about teachers; Geller and Luby, 1997, see Parry, 2021). Malhi et al. do not discuss this issue, so it is unclear whether they view this as problematic. Such previous uses, however, would seem to indicate that the terms offer little to curtail current overdiagnosis. Their recommendation that the childhood term be reserved 'largely for research' appears to offer little protection, especially as paediatric bipolar disorder itself was first proposed as a research hypothesis. To the contrary, Malhi et al.'s proposal could potentially exacerbate overdiagnosis by rebranding and more explicitly targeting adolescents.

More details would be needed to evaluate Malhi et al.'s proposal further. It would be helpful to know, for example, how their classification scheme based on puberty would demarcate childhood, adolescent, and adult forms of bipolar disorder, an issue that is not necessarily straightforward (e.g., chronological age, hormones, secondary sex characteristics), as well as the specific clinical features that they suggest might differ across

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categories. It would also be useful to know their reasons for distinguishing bipolar disorder in late adolescence, a relatively common presentation, from that occurring in adulthood to the extent that they consider it to warrant a separate diagnosis. Other details to clarify involve the practicalities of redefining 'paediatric bipolar disorder', a now widely used term, and how they plan to mitigate the confusion that would arise. A further issue is the research that Malhi et al. recommend for pre-pubertal forms of bipolar disorder given that they propose a term for this purpose despite accepting the construct's problematic validity. Regardless of these details, however, Malhi et al.'s proposal does not address the role of cross-sectional assessments, checklist diagnostic criteria, proposed alternative phenotypes, and other clinician and system-level factors that contribute to the construct's ongoing popularity and influence.

Conclusion

Malhi et al., despite agreeing with the bulk of my article and my conclusions about the problematic nature of paediatric bipolar disorder as a construct, differ in their views on the scope of the problem and what might constitute an effective response. The ongoing popularity of the diagnosis reflects, in my view, a wide array of clinician, health system, and other social factors, so is unlikely to be solved easily, not least by switching labels. Instead, I would suggest that attempts to reduce overdiagnosis involve careful, critical engagement with the primary research, a focus on the tangible harms to patients, and efforts to identify and, where possible, address the human- and system-level factors that perpetuate current practice.

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