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Time to transition from paediatric to adolescent bipolar disorder

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Letter to the Editor

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A recent perspective piece systematically examined the diagnosis of paediatric bipolar disorder (PBD) and critically considered the shortcomings of our current nosology for capturing this construct (Connors, 2023). We agree with the critique and, in particular, the statements in the concluding paragraph that "*. . . the phenomenon reflects limitations in current clinical nosology*" and further "*. . . combined with 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*".

However, we were somewhat puzzled by the customary suggestion in the concluding sentence that "*. . . 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*" as it seemed to undermine the position that the author had secured through the robust argument for eschewing current diagnostic practice. In other words, having raised concerns about the diagnosis of PBD and outlined reasons to doubt its usefulness in its current form, the recommendation for current practice, namely, to undertake greater care when assessing children, does not accord with the problems and limitations identified. Specifically, there is a modest but important inconsistency, and it is this that we wish to examine.

Part of the reason perhaps why Dr Connors, like others, defers dramatic nosological change is that there is still a widely held latent belief that the aetiopathogenesis of bipolar disorder is partly biologically mediated and that elements of this can serve as a marker of the illness and can be reliably identified in childhood. This is implied by the statement that *"the possibility of bipolar disorder having its first onset in childhood is generally not disputed"*. We disagree. This belief is an assumption and nothing more because, as yet, we have no clear proof of *"biological mechanisms"* that meaningfully underpin the clinical manifestations of what we term bipolar disorder. That is not to say we do not have associations and changes that seem to correlate with illness parameters; indeed, in this regard, we are overrun with possibilities. However, we do not have a definitive biological marker that tracks the illness in terms of course or outcome or is able to anticipate onset or predict treatment response.

Another critique of the account given by Connors is that even though the various failed attempts to alter our diagnostic taxonomy to capture PBD are nicely laid out in his article, the reasoning as to *why* these changes were introduced in the first place is not made explicit. To appreciate this fully, the chronology of how paediatric bipolar disorder ran into problems and disruptive mood dysregulation disorder (DMDD) came into being needs to be briefly revisited.

Connors' overview of the increase in the diagnosis of PBD in the early 2000s is accurate, as is the attribution of cause to overdiagnosis in several major centres in the USA. However, even once the reasons for the emerging 'epidemic' of PBD became known, it proved difficult to curb over-diagnosis, particularly because there was significant reluctance to change views and clinical practice, especially on the part of those *"promulgating the construct"*. Arguably, the diagnosis of PBD had gained so much momentum that rather than alter its criteria and modify clinical practice, it was perhaps seen to be easier to 'camouflage' PBD cases and maybe this was part of the reason why DMDD was created and introduced rather hurriedly in DSM-5. This crude attempt at disguising the problem of over-diagnosis and obfuscating its prevalence seems to have worked, as noted by Findling and colleagues (2022), who report that "*. . . DMDD was developed, in part, in hopes of addressing concerns that many youths were being erroneously prescribed antipsychotics owing to a misdiagnosis of BPD* [denotes Paediatric Bipolar Disorder in this article]. *The results of the present study suggest that the introduction of the DMDD diagnosis has been associated with the expected reductions in new treatment episode rates and treated prevalence of BPD*". However, despite DMDD distracting from PBD, it seems to generate problems of its own. The same study also revealed that youth diagnosed with DMDD are more likely to be treated with polypharmacy and have higher rates of comorbidity and hospitalisation than those with PBD (Findling *et al.*, 2022).

Put simply, DMDD appeared in DSM-5 in 2013 in order to capture children with chronic irritability that were otherwise being given a diagnosis of paediatric bipolar disorder. However, as noted by Connors and illustrated pointedly by ourselves, this totally new diagnosis lacked validity and reliability and is difficult to apply in clinical practice (Malhi and Bell, 2019). Thus, by crudely shoe-horning this new diagnostic category into DSM-5, it only added to the number of potential misdiagnoses that could arise rather than addressing the core problem, namely, the difficulty of applying current bipolar disorder diagnostic criteria to children and adolescents.

In addition to the failings of DMDD, Connors also states that the problems plaguing PBD also stem from geographical issues such as the US healthcare system, the influence of specific opinion leaders, and pharmaceutical companies. And whilst again we agree that these factors no doubt contribute to some extent, we are less sanguine about biological markers being able to provide answers to the deep-seated problems within our diagnostic nosology, especially in the near future.

This is why we feel that current efforts should be focused at least equally, if not more so, within the descriptive plane and on refining our current diagnostic taxonomy, rather than pursuing a causal ontology, especially in post-pubertal children (adolescents). This is because, whilst we agree with Connors that theoretically, mania may in some instances, start in childhood, as it stands, we lack the ability to detect and diagnose PBD with sufficient accuracy and reliability in pre-pubertal children. Indeed, the divergence in the reliability of bipolar disorder diagnosis between pre-pubertal children and adolescents is stark (Parry *et al*., 2021), to the extent that we feel the term 'paediatric bipolar disorder' should perhaps be abandoned altogether and replaced with developmentally informed categories.

The fundamental problem is that PBD is a somewhat vague descriptor that is applied to a developmentally broad age range, namely, anyone that is not yet an adult. In other words, the term captures children of all ages and, importantly, two very contrasting groups that extend on either side of puberty. By attempting to capture bipolar disorder across such a developmentally heterogeneous group, populations with starkly different reliabilities in diagnosis are lumped together, and this significantly limits our ability to identify a valid and clinically meaningful phenotype.

This key problem could be addressed by using developmentally informed categories, which may include 'adolescent bipolar disorder' (ABD), which would refer to bipolar disorder that occurs post-puberty. The diagnostic counterpart of ABD, namely, 'childhood bipolar disorder', should perhaps be used largely for research purposes and withheld clinically until such a time it can be firmly established phenomenologically in pre-pubescent children. To aid the transition to these new terms, the term 'paediatric bipolar disorder' may still have some limited utility in that it may be used to describe symptoms that an individual with established bipolar disorder recalls upon inquiry as having occurred any time before adulthood. These early symptoms may have reflected the emergence of bipolar disorder and may provide a useful timeline of the illness for both the patient and clinician in clinical practice – underscoring its chronic nature and the need for a longitudinal management perspective.

In essence, we feel that the flaws in our nosology need to be rectified; otherwise, any findings that rest on the diagnosis of PBD will be non-specific at best and incorrect at worst. Thus, we suggest that the category of paediatric bipolar disorder, as it currently stands, should be supplanted by developmentally informed diagnoses such as adolescent bipolar disorder, which specifically captures bipolar disorder that occurs post-puberty. By addressing the critical problems within our nosology first and in a systematic manner, we will lay down a reliable and meaningful foundation for research aiming to identify potential early markers of the disorder.

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