Stark Discrepancy in Pediatric Bipolar Diagnoses Between the US and UK/Australia

To the Editor:

James *et al.*[1] reported stark differences in inpatient diagnostic rates of pediatric bipolar disorder (PBD) between the United Kingdom and the United States in the June 2014 issue of the *Journal*. The scale of the discrepancy is huge: by 5 years of age, the rate of PBD discharge diagnoses in US inpatient units exceeded the rate for BD diagnoses by 19 years of age in the United Kingdom!

Australia and New Zealand are closer to the British rates than to those of the United States.[2] In the childhood-early adolescent inpatient unit at the Royal Children's Hospital, Brisbane, Australia, there were 505 patients (3–15 years old, mean 9.8 years) admitted over 5 years, from July 1, 2009 to July 1, 2014. Only 2 had *International Classification of Diseases, Tenth Revision (ICD-10)* code F31 bipolar spectrum diagnoses: a 14-year-old boy with code F31.3 (bipolar disorder: mild-moderate depression) and a 14-year-old girl with code F31.6 (bipolar disorder: mixed). In addition, there was a 14-year-old girl with code F25.2 (schizoaffective disorder: mixed type), a 13-year-old girl with code F25.9 (schizoaffective disorder: unspecified), and 15 youth (12–14 years old) with code F20 (other psychotic disorders). The unit's catchment is most of Queensland, whose population is 4.67 million people. Thus, prepubertal cases of BD in Australia's third largest state are almost nonexistent.

Stringaris and Youngstrom[3] explored the US-UK discrepancy in their editorial on the article by James *et al.* ("Unpacking the Differences in US/UK Rates of Clinical Diagnoses of Early-Onset Bipolar Disorder," June 2014), referencing a meta-analysis[4] positing the "true prevalence of [pediatric] BD does not vary between countries" and is 1.8%, so the problem must be in the "administrative prevalence" (p. 609). In fact, the meta-analysis they cited has significant methodologic problems and does not address prepubertal childhood rates.[5]

As Stringaris and Youngstrom speculated, differences in discharge rates more likely reflect differences in diagnosing PBD. US psychiatrists use a "wider construct of BD" than their British counterparts. Evidence for this includes US-UK divergence on clinical vignettes,[6] where the US *DSM* focus on checking operationalized criteria contrasts with the *ICD* focus on pattern recognition. Pattern recognition requires experience seeing patients longitudinally. US insurance companies tend to only cover short lengths of stay, necessitating brief assessments. Thus, US clinicians are deprived of vital experience in such longitudinal phenomenology. Note the huge differences in length of stay between the 2 countries.

In addition, researchers use and interpret standardized assessments differently based on a "liberal" (more the US view) or "conservative" (more the non-US view) orientation.[5] Thus, the use of existing research measurements will not clarify the differences or bring them more in line.

In addition to other explanations offered by Stringaris and Youngstrom, diagnostic up-coding for reimbursement purposes is not necessary in most health care systems outside US-managed care. Pharmaceutical company influence on parents through direct-to-consumer advertising and support of PBD parent advocacy groups has not occurred outside the United States. The significant financial support of PBD researchers by the pharmaceutical industry has been minimal outside the United States. The capacity to focus on a biopsychosocial case formulation and multimodal management of emotional and behavioral problems is a common feature of clinical practice in non-US health care, leading to less emphasis on an Axis I diagnosis.[7]

Stringaris and Youngstrom expressed the opinion that a minority of UK child psychiatrists doubt the existence of PBD. However, a debate on PBD at the 2010 Royal College of Psychiatrists' Faculty of Child and Adolescent Psychiatry conference indicated most UK child psychiatrists dispute the validity of US PBD phenotypes. This skepticism concurs with the 2006 British National Institute for Health and Clinical Excellence guidelines that PBD phenotypes were research hypotheses only and that there were 0 inpatient BD diagnoses in preadolescent British children from 2000 through 2010.

Peter I. Parry, MBBS, FRANZCP, RANZCP
School of Medicine
Royal Brisbane Clinical School
University of Queensland
Brisbane, QLD, Australia
p.parry1@uq.edu.au

Louise Marie-Elaine Richards, MBChB, MRCPsych, DCH
Watford Child and Family Clinic
Peace Children's Centre
Hertfordshire, UK

Ethics approval for the release of anonymized patient data from the Child and Family Therapy Unit (psychiatric inpatient unit) at the Royal Children's Hospital, Herston, Brisbane, Queensland, Australia was obtained. The reference number for this ethics approval is HREC/14/QRCH/118. The contact person for data analysis is Ms. Rebekah Stewart (Rebekah.Stewart2@health.qld.gov.au).

Drs. Parry and Richards report no biomedical financial interests or potential conflicts of interest.

http://dx.doi.org/10.1016/j.jaac.2014.08.012

REFERENCES

1. James A, Hoang U, Seagroatt V, Clacey J, Goldacre M, Leibenluft E. A comparison of American and English hospital discharge rates for pediatric bipolar disorder, 2000 to 2010. J Am Acad Child Adolesc Psychiatry. 2014;53:614-624.
2. Parry P, Furber G, Allison S. The paediatric bipolar hypothesis: the view from Australia and New Zealand. Child Adolesc Mental Health. 2010;14:140-147.
3. Stringaris A, Youngstrom E. Unpacking the differences in US/UK rates of clinical diagnoses of early-onset bipolar disorder. J Am Acad Child Adolesc Psychiatry. 2014;53:609-611.
4. Van Meter AR, Moreira AL, Youngstrom EA. Meta-analysis of epidemiologic studies of pediatric bipolar disorder. J Clin Psychiatry. 2011;72:1250-1256.
5. Carlson GA, Klein DN. How to understand divergent views on bipolar disorder in youth. Annu Rev Clin Psychol. 2014;10:529-551.
6. Dubicka B, Carlson GA, Vail A, Harrington R. Prepubertal mania: diagnostic differences between US and UK clinicians. Eur Child Adolesc Psychiatry. 2008;17:153-161.
7. Parry PI, Levin EC. Pediatric bipolar disorder in an era of "mindless psychiatry". J Trauma Dissociation. 2012;13:51-68.

Drs. Stringaris and Youngstrom reply:

We thank Drs. Parry and Richards for their response to our editorial related to the article by James *et al.* in the June 2014 issue of the *Journal*. The authors raise a number of important points that we would like to take up for further discussion.

First, the authors refer to "US pediatric bipolar (PBD) phenotypes" throughout the letter. We would caution against such generalizations. There are important differences in approaches to PBD within the United States. Indeed, most of the scientific debate about PBD happened between US groups.

Second, the authors use as an argument against "US PBD phenotypes" the fact that other countries do not recognize them. This is a rather weak argument, because it implies that for some reason, the United Kingdom, Australia, or New Zealand are somehow intrinsically psychiatrically superior. Such a statement can easily be interpreted as snobbery.

Third, the authors suggest that long hospital stays are better because they allow clinicians to take a "longitudinal" view of patients. This is a problematic argument. Children's hospital admissions should not be for the benefit of clinicians' observations; they should be planned strictly to serve young people and families. Good psychiatrists are perfectly capable of observing their patients in their natural milieu, namely the community.

Fourth, the authors suggest that a vote and a committee's decision should swing us all to becoming BD deniers. This should, of course, be rejected outright, because scientific matters ought to be decided by science rather than by majority decision or decree.

Fifth, the authors seem to suggest that an increase in the rates of PBD is a bad thing in its own right. This is hard to defend: depression was not formerly a diagnosis for young people, yet it thankfully is now, with characteristics similar to those of adult depression.[1] Similarly, the rates of recognition of epilepsy increased because people have been less inclined to ascribe it to metaphysical causes.

Sixth, the assertion that psychiatric diagnoses must be corroborated by multiple informants to be confirmed flies in the face of clinical reality. Reporter agreement in child psychiatry is reassuring but is typically modest across diagnoses.[2] Although overt mania will rarely go unnoticed by a young person's relatives, hypomania and impairing manic symptoms are devastatingly under-recognized, even in adults.[3]

As clearly stated in our letter, none of us takes the position that all candidate BD phenotypes in the United States (or elsewhere) correspond to true BD. In fact, we have devoted part of our scientific careers to testing (and often rejecting) such phenotypes.[4] We also noted in our editorial that there are some plausible reasons why such rates may have been inflated. Yet a rapidly growing body of solid empirical research clearly shows that BD does occur in youth, and that it does merit more attention than it has received so far. The authors say that where they themselves can afford to place "less emphasis on an Axis-I diagnosis." Maybe so, but the question is whether avoiding a diagnosis is good for patients. A good biopsychosocial formulation does not take away the need for careful diagnosis—we actually believe that it makes it imperative. BD is among the top 10 causes of the global burden of disease,[5] with an annual cost of £2 billion ($3.4 billion) in the United