Letters to the Editor

What About Fetal Alcohol Exposure?

TO THE EDITOR: When we saw the title of the Review and Overview article by O’Donnell and Meaney (1), “Fetal Origins of Mental Health: The Developmental Origins of Health and Disease Hypothesis,” published in the April 2017 issue of the Journal, we were overjoyed because we thought this was indication that our field was finally getting a leg up on prevention. Although the thesis of the review was directionally correct (i.e., the quality of fetal growth and development predicting the risk for a range of noncommunicable, chronic illnesses), the review missed one of the most obvious causes of these problems: fetal alcohol exposure. We appreciated the discussion that fetal growth predicts risk for later psychopathology, such as attention deficit hyperactivity disorder (ADHD), but missing was one of the most common causes of low birth weight and prematurity: fetal alcohol exposure.

Fetal alcohol exposure is one of the leading causes of intellectual disability and is associated with impairment in executive functioning, learning, memory, mood or behavioral regulation, attention, and impulse control. DSM-5 lists the prevalence of the proposed diagnosis of neurobehavioral disorder associated with prenatal alcohol exposure (ND-PAE) as between 2% and 5% in the United States. The problem has been found at even higher rates in other countries. For example, May et al. (2) found rates of ND-PAE of 18.2%–25.9% in four rural communities in South Africa, and Fitzpatrick et al. (3) found rates of 12% in a rural Australian community. More disturbingly, we have found rates of 39% in a family medicine center on Chicago’s South Side (4), and this research revealed that patients—whose mental health problems had fetal origins—were misdiagnosed as having bipolar disorder, depression, schizophrenia, and ADHD.

To the authors’ credit, they did briefly mention that children born small were likely to be born to mothers with high-risk lifestyles that include increased alcohol consumption. However, our study revealed that the vast majority of mothers who were caring for adult children with ND-PAE engaged only in social drinking before they realized they were pregnant (4). We hope this letter to the editor will place more emphasis on more common problems in life, such as ND-PAE.

REFERENCES

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Broader Focus Required to Understand the Effects of the Perinatal Environment on Child Neurodevelopment: Response to Bell and Chimata

TO THE EDITOR: We thank Drs. Bell and Chimata for their comments on our article (1). They raise a number of points that warrant further discussion. First, we completely agree that prenatal alcohol exposure is a modifiable risk factor that influences fetal growth and neurodevelopment, as evidenced by fetal alcohol spectrum disorders and neurobehavioral disorder associated with prenatal alcohol exposure (ND-PAE). Drs. Bell and Chimata raise an important point: maternal alcohol consumption, and thus the salience of this risk factor, varies across cultures and socioeconomic gradients. However, we (2) previously found no significant association between maternal prenatal alcohol exposure and child emotional or behavioral problems at 48 months of age within the Avon Longitudinal Study of Parents and Children cohort (data available from first author). Zuccolo and colleagues (3) reported a positive association between maternal prenatal alcohol consumption and child academic achievement within the same cohort. The authors show that this paradoxical finding reflects confounding by maternal socioeconomic status, which is positively associated with moderate drinking in this relatively low-risk community sample (3). However, the authors do observe the expected inverse association between maternal prenatal alcohol consumption and child...
academic success in this cohort when genetic factors are considered (3). Similarly, Muggli et al. (4) found that the effects of light to moderate maternal alcohol consumption on child craniofacial shape are moderated by maternal ratings of the perceived effects of drinking. Collectively, these findings highlight the importance of considering individual-level as well as contextual factors in studies of prenatal alcohol exposure, which was a major theme in our article.

Second, Drs. Bell and Chimata call for an emphasis on prenatal alcohol exposure because of its known association with low birth weight and preterm birth. Obstetric outcomes are clearly important for a broad range of developmental outcomes, but there is good evidence that the negative effects of prenatal adversity on neurodevelopment are not solely mediated by increased obstetric risk (1). Therefore, we suggest that prenatal risk factors should not necessarily be prioritized based on associations with obstetric outcomes alone.

Third, Drs. Bell and Chimata expressed the hope that their letter will “place more emphasis on more common problems in life,” such as ND-PAE. And with reason. However, maternal perinatal depression represents the most common complication of pregnancy. As many as one in five women in developed countries experience perinatal depression, a number that is significantly higher in low- and middle-income countries (5), while elevated maternal anxiety is associated with an approximate doubling of risk for mental disorders in childhood (2). We certainly do not discount the importance of prenatal alcohol exposure or ND-PAE; rather, as we emphasize in our article, a broader focus is required to better understand the lasting influence of the in utero environment on child neurodevelopment. An emphasis on any one risk factor in isolation from an individual’s genomic risk and the wider psychosocial context is likely to be uninformative. The work of the PhenX Pregnancy Working Group is of interest in this context. This initiative seeks to standardize data collection in perinatal cohorts and capture a constellation of risk factors (6). Such efforts may, in time, help us understand the sources of individual variation in developmental outcomes and advance prevention efforts in perinatal psychiatry.

REFERENCES

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Are Personality Disorders Assessed in Young People?

TO THE EDITOR: Personality disorders are highly prevalent, disabling, and costly. Decades of research suggest that they commonly emerge in childhood and adolescence, demonstrate early stability, and, critically, respond well to early treatment and prevention efforts (1). It is vital that early-onset personality disorders are properly identified, as accurate diagnosis is essential for implementation of effective interventions.

Despite consistent empirical support for the validity of pediatric personality disorders, there are indications that practitioners resist personality disorder assessment in young people. Yet aside from several practitioner surveys (e.g., reference 2), large-scale data are lacking on the extent of this underdiagnosis. We therefore analyzed responses from a large national survey of university students who reported whether they had been diagnosed previously with a mental illness by a health professional. We compared those reports with the prevalence of personality disorder diagnoses ascertained with structured interviews in a university student subsample of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) (3).

The Healthy Minds Study (4) included 113,515 students from 105 U.S. universities who provided complete histories of psychiatric diagnoses. As shown in Table 1, about one in 200 Healthy Minds Study students was diagnosed with any personality disorder, and rates of individual personality disorders were as low as one in 10,000. By comparison, more than five in 100 respondents had been diagnosed with major depressive disorder. The discrepancy in prevalence between personality disorder and depression was even more pronounced in the Healthy Minds Study treatment-seeking subsample.

The comparison of the “true” disorder rates from the NESARC with the “diagnosed” rates from the Healthy Minds Study illustrates that the vast majority of young people who have a personality disorder are undiagnosed. The true versus
**TABLE 1. University Student Histories of Personality Disorder Diagnosis**

<table>
<thead>
<tr>
<th>Personality Disorder</th>
<th>All Healthy Minds Study Participants (N=113,515)</th>
<th>Healthy Minds Study Treatment-Seeking Participants (N=29,974)</th>
<th>NESARC Subsample (N=2,188)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major depression</td>
<td>6,108</td>
<td>4,974</td>
<td>154</td>
</tr>
<tr>
<td>Paranoid personality disorder</td>
<td>53</td>
<td>40</td>
<td>106</td>
</tr>
<tr>
<td>Schizoid personality disorder</td>
<td>35</td>
<td>28</td>
<td>72</td>
</tr>
<tr>
<td>Schizotypal personality disorder</td>
<td>19</td>
<td>15</td>
<td>—</td>
</tr>
<tr>
<td>Antisocial personality disorder</td>
<td>126</td>
<td>95</td>
<td>103</td>
</tr>
<tr>
<td>Borderline personality disorder</td>
<td>289</td>
<td>240</td>
<td>76</td>
</tr>
<tr>
<td>Histrionic personality disorder</td>
<td>15</td>
<td>11</td>
<td>—</td>
</tr>
<tr>
<td>Narcissistic personality disorder</td>
<td>36</td>
<td>29</td>
<td>76</td>
</tr>
<tr>
<td>Avoidant personality disorder</td>
<td>67</td>
<td>47</td>
<td>50</td>
</tr>
<tr>
<td>Dependent personality disorder</td>
<td>41</td>
<td>28</td>
<td>11</td>
</tr>
<tr>
<td>Obsessive-compulsive personality disorder</td>
<td>82</td>
<td>69</td>
<td>180</td>
</tr>
<tr>
<td>Any personality disorder</td>
<td>529</td>
<td>409</td>
<td>387</td>
</tr>
</tbody>
</table>

*a Respondents were, on average, 22.91 years old (SD = 5.49); 64% were female; and 73% identified as white.

*b Healthy Minds Study participants who sought mental health treatment in the past 12 months.

*c University student subsample of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC).

*d Schizotypal, borderline, and narcissistic personality disorders were not assessed in this NESARC study sample.

diagnosed prevalence rates differ by a factor of approximately 40 for personality disorders, compared with a factor of 1.3 for major depression.

We caution that our contrasts rely on patients’ reports of diagnoses, which may be imperfect proxies of true assessment results. Also, the lion’s share of research on pediatric personality disorders has targeted borderline personality disorder, but we could not evaluate the underdiagnosis of borderline personality disorder because it was not surveyed in the NESARC university subsample. With those caveats in mind, we conclude that practitioners are not assessing or treating personality disorders prior to adulthood, despite a clear need for early intervention. Given the data supporting the concurrent and prognostic importance of personality disorder diagnoses in youths, clinicians arguably should assess them.

REFERENCES


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current episode, 5 years prior due to clonazepam discontinuation. Given the patient's history of recurrent catatonia after benzodiazepine discontinuation, the patient was advised to continue 0.5 mg of clonazepam daily.

Benzodiazepine withdrawal catatonia typically occurs after long-term benzodiazepine use, with onset of symptoms 3–7 days after discontinuation (2). Older adults may be more susceptible to benzodiazepine withdrawal catatonia than the general population (3). A recent review identified 16 cases of catatonia due to benzodiazepine withdrawal, eight of which involved older adults. All patients were initially treated with benzodiazepines. Long-term management strategies varied and included continuation of benzodiazepine (six cases), taper of benzodiazepine over 2 weeks (one case), and administration of ECT (one case) (2). To date, this is the first case describing recurrent benzodiazepine withdrawal catatonia.

Benzodiazepine withdrawal catatonia may become more common as the prevalence of older adults on long-term benzodiazepines increases and clinicians begin tapering benzodiazepines often leads to immediate improvement in benzodiazepine withdrawal catatonia. However, uncertainty remains about optimal long-term management. Potential treatment options include prolonged benzodiazepine taper or long-term maintenance at the lowest effective dose. Alternative treatments for older adults with catatonia include memantine, topirimate, amantadine, or ECT in severe cases (4). However, these treatments have not yet been evaluated in the management of benzodiazepine withdrawal catatonia.

REFERENCES

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