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.

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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[a]**

| | All Healthy Minds Study Participants (N=113,515) | | Healthy Minds Study Treatment-Seeking Participants (N=29,974)[b] | | NESARC Subsample (N=2,188)[c] | |
|---|---|---|---|---|---|---|
| Psychiatric Disorder | N | % | N | % | N | % |
| Major depression | 6,108 | 5.38 | 4,974 | 16.59 | 154 | 7.04 |
| Paranoid personality disorder | 53 | 0.05 | 40 | 0.13 | 106 | 4.86 |
| Schizoid personality disorder | 35 | 0.03 | 28 | 0.09 | 72 | 3.31 |
| Schizotypal personality disorder | 19 | 0.02 | 15 | 0.05 | —[d] | — |
| Antisocial personality disorder | 126 | 0.11 | 95 | 0.32 | 103 | 4.70 |
| Borderline personality disorder | 289 | 0.25 | 240 | 0.80 | —[d] | — |
| Histrionic personality disorder | 15 | 0.01 | 11 | 0.04 | 76 | 3.47 |
| Narcissistic personality disorder | 36 | 0.03 | 29 | 0.10 | —[d] | — |
| Avoidant personality disorder | 67 | 0.06 | 47 | 0.17 | 50 | 2.31 |
| Dependent personality disorder | 41 | 0.04 | 28 | 0.09 | 11 | 0.51 |
| Obsessive-compulsive personality disorder | 82 | 0.07 | 69 | 0.23 | 180 | 8.24 |
| Any personality disorder | 529 | 0.47 | 409 | 1.36 | 387 | 17.68 |

[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.

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## Recurrent Benzodiazepine Withdrawal Catatonia in an Older Adult

TO THE EDITOR: The prevalence of long-term benzodiazepine use is elevated in individuals ages 65 years and older (1). Recent guidelines recommend against long-term benzodiazepine use, and many clinicians are reducing prescribed doses or discontinuing benzodiazepines in those ages 65 and older (1). A rare complication of benzodiazepine withdrawal is catatonia. Here we describe a case of recurrent benzodiazepine withdrawal catatonia in an older adult.

A 79-year-old woman presented to the emergency department with 2 days of altered mental status characterized by staring, mutism, and motor resistance to commands. Six days prior to presentation, clonazepam was discontinued (daily dose of 0.5 mg prescribed for 10 years). Her history included mild cognitive impairment, unspecified anxiety, and past major depression without catatonia. Purposeless agitation, including stereotypies and combativeness, occurred when staff obtained intravenous access. After receiving 2 mg of lorazepam intravenously, the patient began speaking and became more cooperative. She received another 1 mg of lorazepam intravenously to facilitate incremental improvement in mental status. Metabolic laboratory and head imaging test results were unremarkable. Her vital signs were unchanged from baseline, and tremor was absent. Catatonia signs and symptoms remained absent 12 hours later during psychiatric evaluation. There was no recent history or examination findings of depression, mania, psychosis, or delirium. The most likely explanation for catatonia was benzodiazepine discontinuation. Chart review revealed the patient developed catatonia, with similar symptoms to the