Letters

COMMENT & RESPONSE

In Reply We thank Solanto for her interest in our article[1] and for providing some thoughtful ideas on how to understand these intriguing similar findings from 3 population samples that challenge the notion of attention-deficit/hyperactivity disorder (ADHD) as only a child-onset neurodevelopmental disorder.[2,3] Solanto suggests that our study[1] might have missed a substantial proportion of cases of ADHD predominantly inattentive type (ADHD-PI) in childhood, consequently decreasing the rate of adult ADHD with roots in childhood. She based this hypothesis on the idea that we had collected data on ADHD symptoms in childhood before *DSM-IV* criteria.

In fact, the 1993 Pelotas Birth Cohort collected data on childhood ADHD in 2004, a decade after *DSM-IV* was launched. However, as mentioned in the article,[2] we assessed childhood ADHD with a screening instrument that emphasizes hyperactive-impulsive symptoms. Considering the potential lower performance of the instrument for ADHD-PI subtype, Solanto's hypothesis makes sense and warrants proper testing.

In 2004, a subsample of 288 participants at age 11 years was also assessed with the Development and Well-Being Assessment, a semistructured interview that generates *DSM-IV* diagnoses, including ADHD-PI. In this subsample, 24% of the ADHD cases were of ADHD-PI, 40% of which were not detected by the screening instrument. Therefore, we can conclude that 10% (40% of 24%) of the childhood ADHD cases were not included in the childhood ADHD group. Extrapolating for the entire sample at 11 years of age, the scenario suggests that the 393 cases in childhood represent 90% of the real ADHD cases and the ADHD-PI cases that were not diagnosed by the instrument would add 44 new cases. It is important to note that this mathematical reasoning addresses only the issue related to ADHD-PI. It does not take into account the performance of the screening for other subtypes of ADHD.

Although the ADHD combined type seems to be the most persistent subtype,[4] let us assume conservatively that all these ADHD-PI cases in childhood would continue to present ADHD in young adulthood. This assumption would increase the proportion of young adults with ADHD with childhood history of ADHD symptoms from 13% (reported in the article) to 21%. Thus, we cannot throw the baby out with the bath water. We were also surprised with our findings and explored our data from diverse angles to assess potential flaws. Others did the same.[5] The main message continues to be that most young adults with the ADHD phenotype do not have a childhood history of significant ADHD symptoms.

Finally, we are in complete agreement with Solanto's proposal that the translation of data from population-based to clinical studies is challenging and that more studies are needed to understand the reasons for this surprising rate of adult-onset ADHD cases in population samples. Indeed, a group of researchers interested in this controversial issue has begun to work on numerous data-driven hypotheses.[6]

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