Reviewer’s report

Title: Prevalence of Attention Deficit/Hyperactivity Disorder Among Adults in Obesity Treatment

Authors:

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Reviewer: Dr Lisa J. Rapport

Level of interest: A paper of considerable general medical or scientific interest

Advice on publication: Accept after discretionary revisions

In general, the majority of my comments were adequately addressed in the revision. I believe that the author’s theoretical rationale for a neurobiologic link between ADHD and obesity is sound. However, the current data do contain some limitations that should not be downplayed, especially because they may drive the direction of future research in this area.

The diagnostic procedure has been adequately clarified in the revision. Given that this was a purely clinical setting, it is understandable that the procedure could not take on the rigorous quality observed in research settings. Ideally, diagnosis of ADHD would include measures in addition to semi-structured interview. The limitations of the diagnostic procedure may explain some of the inconsistencies in the results.

(1) The revised manuscript indicates that none of the patients met the criteria for hyperactive/impulsive subtype ADHD and that few impulsive symptoms were endorsed by this group. The diagnosis of all ADHD+OB patients as inattentive subtype seems at odds with the relative rates of inattentive, hyperactive/impulsive, and combined types in the ADHD literature. Additionally, the observation that inattention predominated the symptom profile among these ADHD patients seems contrary to the author's proposed neurobiologic link between ADHD and obesity (i.e., a common neurobiologic substrate underlies impulsive/disinhibited behavior observed in both disorders). The author's theory would be supported by a high prevalence of impulsive symptoms in the ADHD+OB group and not by a high prevalence of inattentive symptoms in that group.

The author should consider commenting on the findings regarding subtype and the relative absence of impulsive symptoms, as well as offer some explanation in the context of the proposed theory. Perhaps the diagnostic procedure was less sensitive to impulsive symptoms. If the author is correct that these patients minimize symptoms and are unlikely to endorse even "symptomatic behavior obvious to others," it may be that endorsement of inattentive symptoms associated with ADHD held less personal stigma for these patients. In recent years, ADHD appears to have transcended the cultural stigma associated with most mental disorders; for many individuals, the diagnosis takes on an explanatory quality that relieves the holder from some personal responsibility. ADHD symptoms may be more easily endorsed compared to negative personality characteristics as the source of dyscontrol. Per my initial review, endorsement bias of this nature may have artificially inflated the number of ADHD and other diagnoses in the sample and/or distorted the profile of ADHD symptoms observed.
One manner to investigate this issue further would be to examine whether obese persons with ADHD also endorse higher rates of depression, anxiety, and other disorders than do obese persons without ADHD. The author is correct that depression and anxiety are common comorbid conditions of both obesity and ADHD. However, we would not expect higher rates of these comorbidities among obese persons with versus without ADHD, unless something is driving the difference, which could be a central neurobiologic dysfunction or a tendency toward over-endorsement/positive response bias. If some general measure of self-reported distress or endorsement style (e.g., negative affectivity) leaves little or no unique variance accounted for by ADHD symptoms, then over-endorsement underlie the result.

(2) In my opinion, the two tables added to the revision overwork the data and are not necessary. The author could simply report the percentages and corresponding confidence intervals of ADHD within the obesity groups in the Results text. As an aside, it may be clearer for the readership if the percentages were reported in a common direction (e.g., diagnosis of ADHD was made for 42.6% of Obesity-III, 22.8% of Obesity I & II, and 18.9% of Overweight persons in the sample). At present, the text reports the percentages of ADHD in some groups and the percentages of persons without ADHD in other groups. The initial version of the manuscript seemed clearer in this regard.

2a) The data in Table 3 currently are described inaccurately (or it is worded in a manner that could easily be misinterpreted): “There were 26 patients with both ADHD and Obesity III, which is 42.6% (CI: 36.3% to 48.9%) of all the ADHD patients in the sample.” 26 patients is 44.1% of all 59 ADHD patients in the sample; however, it is 42.6% of the Obesity III group.

2b) "Chi-square test of patient groups vs. obesity classes showed their values were not independent... that is, differences in the distribution of patients among categories was significant." The wording of the result is very vague. The appropriate 2 x 2 post hoc tests would clarify the result, and these should be incorporated into the description (e.g., "the proportion of persons with ADHD in the Obesity-III class was significantly higher than the proportions of persons with ADHD in the Obesity-I/II and Overweight classes").

(3) Table 2 is inaccurate. The n for Cell 1 (ADHD in Obesity III) should = 26, not 61.

(4) The Discussion states, "In other words, in OB+ADHD, it is the symptoms of ADHD rather than level of obesity that *produces* the greater impediment to treatment success." Per my initial review, it is probably best to avoid causal statements in correlational research. Perhaps the author would consider an alternative such as, "In other words, in OB+ADHD, impediments related to symptoms of ADHD rather than level of obesity are more strongly associated with treatment outcome." Or simply, "In other words, in OB+ADHD, symptoms of ADHD are more strongly associated with treatment outcome than is level of obesity."

**Competing interests:**

None declared.