Author's response to reviews

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

Authors:

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Changes in this revision:

1. Omitted superfluous tables (2 and 3), but preserved discussion of the data in the Results section.

2. Results section revised to provide clearer presentation of ADHD category vs. obesity class numerical results, statistical analyses, and proportions of sample population in relevant subgroups.

3. In Results section, revised numerical entries that were incorrect.

4. In the Discussion section, added comments about DSM-IV ADHD Inattentive subtype relating to its diagnostic criteria and symptomatic patterns, and implications for connections to neurophysiological mechanisms.

5. Revised the portion of the Discussion section pertaining to biases that might have affected prevalence results.

6. Various spelling, grammatical and phrasing errors in the manuscript were corrected.

Reply to review: Dr. Rapport

1. Two major points are: *A) the Inattentive subtype of ADHD does not exclude hyperactive or impulsive behaviors, and *B) impulsivity is not necessarily the only behavioral factor binding ADHD to obesity.

The manuscript states that patients didn't meet *DSM-IV criteria* for the Hyperactive/Impulsive or Combined subtype of ADHD, which does not mean impulsive or hyperactive *symptoms* were absent. It has been widely reported that by early adulthood, there is marked attenuation of DSM-defined hyperactive and impulsive behaviors. Therefore, adults seldom present as having H/I or Combined ADHD, though frequently do continue to manifest such symptoms, if less obviously, meaning there wasn't "a relative absence" of hyperactive/impulsive symptoms among OB+ADHD adults. (That is, a decrease in, or different manifestation of, these symptoms is expected.) Published relative prevalence rates of ADHD subtypes apply only to children, because prevalence of ADHD (and its subtypes) in adult populations is not yet known--reseach yields highly divergent results depending on definitions used, age of subjects, etc.

The formal diagnosis of Inattentive subtype doesn't negate potential neurobiological links between ADHD and obesity. As noted above, "Inattentive" patients often have impulsive symptoms, though such behavior tends to differ from DSM descriptions (oriented toward children). The manuscript doesn't actually discuss a "substrate that underlies impulsive/disinhibited behavior observed in both disorders", although impulsivity (or disinhibition) is likely prominent in many instances of ADHD and obesity. Rather, the manuscript looks at a number of potential "substrates" in OB+ADHD, some of

which may be mediated by impulsive or other behaviors (e.g., compulsive spectrum, binge-eating). The paragraph (in the background section) alluding to similarities between impulsive aggression in adolescents and impulsive eating in OB+ADHD adults is only "pointing to a connection" between disorders rather than attempting to define the nature of any connection.

While impulsivity is almost certainly implicated in OB+ADHD, there is no reason to think that attentional factors in ADHD could not be at least as likely to contribute to comorbid obesity. For example, poor planning, poor organization, forgetfulness, poor task initiation, poor task persistence, distractability, inattention to detail, etc., are antithetical to characteristics known to be associated with self-regulation of eating behavior and physical activity.

On these bases, there is no inherent contradiction between diagnosis of Inattentive subtype of ADHD, and ideas about common biological processes in ADHD and obesity. Given the current "state of the art", that far too little is known about either disorder, it isn't possible to offer a coherent model of the neural (and non-neural) substrates underlying a relationship between, or the nature of, comorbid ADHD and obesity. I trust the day will arrive that we really know enough to be able to construct such models.

Accordingly, it is a good idea to give a brief summary of ADHD subtypes as applied to adult subjects (Probably it's best to assume a reader might be unfamiliar with ADHD in adults.) A point to make is that the sex of the subject has a bearing on ADHD subtype. It is known that in girls, the DSM-IV Inattentive subtype is present in the majority who have ADHD, and the disorder is likely to be diagnosed later (if at all), and less likely to be treated in childhood. The predominance of the Inattentive subtype is augmented in the sample insofar as ~90% were female, increasing the odds that even if diagnosed in childhood, most would have been classified as Inattentive, and would continue to have that subtype.

The potential for endorsement bias toward ADHD symptoms is noted by the reviewer who stated, "...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." Responding to this opinion is difficult: it doesn't match experience with OB+ADHD patients, and its basis in scientifically established fact is unclear.

Mitigating against endorsement bias are the details of the procedures used in diagnosing patients' conditions, and characteristics of the patients observed during the course of working with them. In making any diagnosis, it is a principle of optimum clinical practice to avoid biasing or "leading" the patient to any particular conclusion before the facts of the patient's condition are clear. From the first visit, a careful clinician will inquire about a broad range of behavior, history of symptoms, prior treatment, and so on. Medical evaluation, e.g., physical exam, and lab tests are completed. Other procedures, like neuropsychological testing, may be obtained. During this phase, specific diagnoses are not discussed with the patient, precisely to minimize distortions and misdiagnosis. The order is always collect the information first, and later, if, and only if, there is enough information, then make a diagnosis and present it to the patient.

The OB+ADHD patients were coming to the clinic for medical management of obesity. They did not attribute their difficulties to a mental disorder, and in particular, did not claim symptoms of ADHD as a cause of their problems. As behaviors were observed, and information gathered, patients were not told it was about ADHD, or any disorder, until the evidence had accumulated to the point a specific diagnosis could be concluded. Prior to being told about the ADHD diagnosis, patients didn't know they had ADHD, and didn't know they were endorsing symptoms of any particular condition. None ever said anything like, "You know, I have ADHD and it's causing my problems..." After being presented with the diagnosis of ADHD, most came to terms with it, but very few embraced it, in fact, a number rejected the idea and never did accept it.

On the other hand, there are other forms of endorsement bias that are readily observed in this and other clinical populations. For example, patients will commonly attribute distress to a somatic source rather than disturbance of mood, thought or dyscontrol of actions which the clinician views as the true problem. This endorsement bias for somatic symptoms is well-documented. Similarly, in my experience, a minority of ADHD patients prefer endorsing depression or anxiety over ADHD as a cause of their difficulties, though hard data quantifying the extent of this phenomenon is not yet available. However, experience suggests it is equally logical to postulate that there is an endorsement bias toward depression, rather than ADHD, when both are present.

Differential rates of depression and anxiety among obese individuals with and without ADHD would indeed be worthwhile to study. If a difference were found, explanations could include neural mechanisms or endorsement bias, but other causes driving such a difference are also credible. For example, consider the reports that adults with ADHD have higher rates of auto accidents, divorce, job failure, and conduct problems. Interpersonal conflicts and losses, and role deficits and failures are known to be associated with onset of depression, potentially explaining a higher rate of depression among those with ADHD.
Another example takes the point of view that depression has a multifactorial causation, with a variety of risk factors contributing to its development, conceptually similar to the risk-factor approach to cardiovascular disease, or type-II diabetes. Obesity is associated with increased incidence of depression (especially in women), and for this example, consider ADHD as leading to higher rates of depression. Therefore, if ADHD and obesity are independent risk factors for depression (which would be likely) then patients with both ADHD and obesity would have a higher probability of becoming depressed than those having obesity without ADHD, other risks being equal.

Further elaboration on certain points is possible, but nonetheless it could not honestly be asserted that it is impossible for endorsement bias to have affected the reported rate of ADHD in the sample. While it was intended that the prior revision of the manuscript would address this factor, it was revised again to discuss the issue in more detail. Ultimately, like other questions about ADHD in adults, the issue of endorsement bias won't really be answerable until there are reliable diagnostic methods based on functional or biological markers (e.g., neuroimaging techniques), reducing the clinician's dependence on self-reports of symptomatic behavior, which is the present day basis of determining who has ADHD, and who does not.

2. Can't disagree about the tables. The percentages in the text have been revised to be clearer.

2a. Arithmetic errors and misentered numbers have been corrected.

2b. The previous revision was an attempt to keep the results from being too cluttered. The text has been further revised to make it clearer.

3. Table was removed.

4. I concur. Text was revised.

Reply to review: Dr. Mick

Thank you.

Jules R.Altfas