

# Symptoms of attention deficit hyperactivity disorder in severely obese women

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**ABSTRACT. Objective:** Past and current symptoms of Attention Deficit Hyperactivity Disorder (ADHD) were assessed in a clinical sample of severely obese females. **Method:** Core symptoms of ADHD were examined in 75 consecutive, severely obese (BMI  $\geq 35$ ) women referred to a medical specialist for the non-surgical treatment of obesity. Subjects completed both a retrospective report of childhood symptoms of ADHD (Wender Utah Scale) and two standardized adult ADHD symptom scales. **Results:** The frequency of clinically suggestive elevations in ADHD scores was substantially and significantly higher than the normative samples in 9 out of 11 symptom subscales. Inattentive symptoms, but not hyperactive symptoms of ADHD, were frequently reported. Overall, 26.7% of the sample reported significant symptoms of ADHD in both childhood and adulthood. **Conclusions:** This preliminary study suggests that severely obese women report significant symptomatology related to both childhood and adult ADHD.

(*Eating Weight Disord.* 10: e10-e13 2005). ©2005, Editrice Kurtis

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## INTRODUCTION

Clinically we have noted that a substantial number of obese clients exhibited consistent difficulty keeping an accurate diet record, planning and preparing meals, eating regularly, and maintaining an exercise schedule. While there are numerous explanations for such behavior we began to consider the possibility that perhaps, for some, it might be related to an underlying neurological condition; attention deficit hyperactivity disorder (ADHD).

The scientific basis for understanding ADHD has evolved very considerably over the past decades. Once considered a disorder of childhood that expressed itself predominantly through hyperactivity in males, it is now recognized to occur throughout the lifespan, across genders, and is best characterized by deficiencies in executive functioning or self-regulation (1-3). While authors differ in their precise definition of the deficiencies which best characterize ADHD, current diagnostic criteria (4) include problems with distractibility, organization, follow-through, forgetfulness, maintaining effort, attention (symptoms of inattention), as well as problems with physical restlessness, excessive talking, and impulse control.

There is an emerging literature that does suggest a possible contribution of ADHD symptoms to the development of disordered eating. Two published case studies (5, 6) have described the treatment of individuals with bulimia nervosa and comorbid ADHD. Both studies report a substantial reduction in both ADHD and eating pathology when the subjects were treated with methylphenidate. Altfas (7) conducted a retrospective chart review of 215 obese patients being treated for obesity to determine the prevalence of ADHD and its relation to successful weight loss. He found that 27% of the sample had been given a clear diagnosis of ADHD and that they had significantly less success in weight loss than those patients without signs of the disorder. Another recent study (8) investigating the prevalence of obesity in a group of male children with ADHD found rates of obesity higher than population norms. Finally, Levitan et al. (9) have recently reported a moderately strong correlation between childhood symptoms of inattention and adult maximal body mass index (BMI) in overeating women with seasonal affective disorder.

We report here our assessment of ADHD symptomatology in a sample of women referred to a medical specialist for the non-surgical treatment of obesity.

### Key words:

Attention, deficit, hyperactivity disorder, adult obesity, ADHD.

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Received: February 2, 2004

Accepted: June 15, 2004

## METHODS

### *Subjects*

The sample consisted of consecutive women having a BMI  $\geq 35$  who were referred by their family doctors to a medical (non-surgical) obesity clinic over a 14-month period. The clinic is located in a major urban center and is culturally diverse. Patients are generally treated with behavioral and/or medical interventions. Thirty-three percent of the initial referrals did not complete the questionnaires, largely because they did not follow through in pursuing treatment beyond the initial interview. The final sample consisted of 75 women with a mean age of 40.4 years (SD=10.8) and a mean BMI of 43.1 (SD=7.25). All subjects completed a consent form at the initial interview before being given the questionnaires.

### *Instruments*

#### The Wender Utah Rating Scale (WURS)

Because ADHD is considered to be a lifelong neurological condition, the diagnosis in adults requires the existence of significant symptoms during childhood. The Wender Utah Rating Scale (WURS) is a 61-item retrospective survey of behaviors characteristic of childhood ADHD, including problems with concentration, disorganization, impulsiveness, emotionality, school, and social problems. Ward et al. (10) found that a cutoff score of 36 on a subset of 25 core items from the WURS (WURS-25) could correctly classify 96% of individuals identified as meeting Utah criteria (11) for ADHD, as well as 96% of a normal sample. This 25-item subscale also differentiates core ADHD symptoms from those of major depression (10).

#### Conners' Adult ADHD Rating Scale (CAARS)

To measure the presence of current symptoms of ADHD, all subjects completed the long self-report form of the CAARS (12). This 66-item instrument has multiple scales measuring each primary symptom domain (inattentiveness and hyperactivity/impulsivity), as well as a self-esteem scale that is non-specific to ADHD. A major factor in selecting the CAARS was its provision of separate norms for males and females broken down by four age groups. This is important since symptom levels have been shown to vary both by age as well as gender (12, 13). For this reason, all scores were converted to T-scores using the appropriate age and gender norms before analysis.

#### Brown ADD Scale for Adults

The last 41 of the 75 consecutively referred subjects also completed the Brown Adult ADD Scale (2) in addition to the CAARS and WURS. The instrument was added because of our

desire to further investigate the high levels of symptoms of inattention we had observed in the first stage of the study. The Brown scales differ from the CAARS in that they do not assess hyperactivity and impulsivity, but examine five different domains of executive function.

### *Statistics*

Because the study design did not involve a control group, statistical analyses employ the standardization sample for the CAARS and Brown. The object of interest in these analyses concerned the relative frequency of clinically significant symptomatology. For both standardized tests, clinically significant symptomatology was defined by a subject receiving a T-score of 65 or above, which would place their level of reported symptoms at or above the 93<sup>rd</sup> percentile (2, 12). The normal subjects for the Conners scale were collected from "diverse regions of the US and Canada" and were not prescreened in any way (12). For the Brown, the normal comparison sample was collected "from work settings and from a civic organization" (2). For the analysis of the Wender Utah scale, the previously established cutoff score of 36 on the core WURS-25 (10) was employed, which corresponds to the 96<sup>th</sup> percentile in their study of normal subjects. The normal subjects for the Wender WURS were parents of elementary school children who were judged to be "well-adjusted" by their teachers.

Goodness-of-fit chi-square tests were used to test the hypothesis that the observed frequency of elevated symptoms was greater than expected in the normative samples. Since the experiment involved a large number of planned comparisons, the alpha level was set at  $p=0.01$  for individual tests in order to control for experiment-wise Type I error.

## RESULTS

### *Symptoms of childhood ADHD based on the WURS*

Based on the previously established cutoff score of 36 on the WURS-25, 38.6% of the subjects met the criteria predictive of ADHD. This was significantly higher than the 4% prevalence previously reported in a normal sample (10) ( $\chi^2$  (df=1) = 234,  $p<0.001$ ). This difference corresponds to a relative risk of 9.67 compared to the normative sample.

### *Adult ADHD measures*

Table 1 indicates the percentages of obese subjects who had clinically relevant scale ele-

**TABLE 1**

Percentage of sample (N=75) with clinical elevations on the CAARS

Scale	Percentage T≥65	χ <sup>2</sup> ,df=1	Relative risk
Inattention/Memory	34.7	72.5, p<0.001	4.3
Hyperactive/Restless	9.3	0.18, NS	1.2
Impulsivity/Emotionality	24.0	26.1, p<0.001	3.0
DSM-IV Inattentive	41.3	113.2, p<0.001	5.2
DSM-IV Hyper-impulse	16.0	6.5, p=0.011, NS	2.0
ADHD Index	30.7	52.4, p<0.001	3.8

Goodness-of-fit chi square calculated against an expected frequency of seven percent.

vations (T≥65) based on the CAARS. Two of the three scales assessing the hyperactive/impulsive dimension of ADHD did not show high levels of clinically significant elevation in the obese sample. All other scales showed significantly higher levels of symptomatology.

Table 2 summarizes similar data based on the Brown Scale (n=41). As shown, a very substantial percentage of this obese sample had scores at or above the 93<sup>rd</sup> percentile (T≥65). Strikingly, a full 61% of this sub sample had total symptom scores in the range suggestive of probable ADHD (2).

A more stringent test of the presence of ADHD symptoms in the severely obese would be to require both a childhood history as well as significant adult symptomatology. To examine rates of probable ADHD based on this conservative methodology, subjects were next classified as likely cases if they had a childhood history indicated by an elevated Wender subscore (≥36) as well as two elevated CAARS symptom scales (T≥65). Using these criteria, 20 subjects or 26.6% of the sample were classified as probable ADHD cases. This is well above DSM-IV adult preva-

**TABLE 2**

Percentage of subsample (n=41) with Clinical Elevations on the Brown.

Scale	Percentage T≥65	χ <sup>2</sup> ,df=1	Relative risk
Activation	43.9	143.9	7.7
Attention	58.5	158.6	8.0
Effort	43.9	143.9	7.7
Affect	53.7	129.8	7.3
Memory	48.8	103.9	6.7
Total	61.0	174.1	8.3

Goodness-of-fit chi square calculated against an expected frequency of seven percent. All χ<sup>2</sup> tests p<0.001.

lence estimates of 3-5% (4) and far in excess of the frequency of clinical elevations expected on any one of the three measures.

To address the question of whether there was a simple causal relationship between level of obesity and level of ADHD symptoms, correlation coefficients were calculated between all ADHD symptom measures and BMI. All Pearson coefficients were low and non-significant (p>0.05).

## DISCUSSION

While the current study does not allow us to ascertain the cause of the deficit, it is striking that a very high percentage of this sample of severely obese women report very substantial problems with the set of symptoms that we characterize as reflecting ADHD. Furthermore, many of these women report significant symptoms dating back to their childhood.

How might the cognitive and behavioral characteristics that describe ADHD contribute to the development of obesity? Several authors (1, 3, 14) have emphasized that ADHD is characterized by problems with self-regulation; these include problems with working memory, attention, primary arousal, affect regulation, organization, and inhibition. It is well established that both adults and children with ADHD suffer from very high rates of comorbid disorders including depression, anxiety, sleep disorders, and substance abuse (14). Dietary regulation is a complex process (15-17) and would appear to be quite vulnerable to disruption in humans. It is possible that in the same way that ADHD undermines the regulation of emotions, sleep and moderate alcohol use, it may also disrupt dietary regulation.

Given that this was a preliminary descriptive study based solely on self-report questionnaires, these findings must be considered tentative and in need of clarification and expansion in other obese samples. The absence of a comparable medically referred control group is a major limitation, as is the lack of corroborating information from family members and school reports.

The study does not allow us to rule out the possibility that the high rates of reported symptoms might be due to another disorder such as depression or an untreated sleep disorder. This explanation would not readily account for the high levels of reported childhood symptoms, but we are currently conducting a follow-up study in which we assess these dimensions independently.

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