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ORIGINAL PAPER

Overweight and Obesity: Prevalence and Correlates in a Large Clinical Sample of Children with Autism Spectrum Disorder

Katharine E. Zuckerman · Alison P. Hill · Kimberly Guion · Lisa Voltolina · Eric Fombonne

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Abstract Autism Spectrum Disorders (ASDs) and childhood obesity (OBY) are rising public health concerns. This study aimed to evaluate the prevalence of overweight (OWT) and OBY in a sample of 376 Oregon children with ASD, and to assess correlates of OWT and OBY in this sample. We used descriptive statistics, bivariate, and focused multivariate analyses to determine whether socio-demographic characteristics, ASD symptoms, ASD cognitive and adaptive functioning, behavioral problems, and treatments for ASD were associated with OWT and OBY in ASD. Overall 18.1 % of children met criteria for OWT and 17.0 % met criteria for OBY. OBY was associated with sleep difficulties, melatonin use, and affective problems. Interventions that consider unique needs of children with ASD may hold promise for improving weight status among children with ASD.

Keywords Autism spectrum disorder · Obesity · Overweight · Children

K. E. Zuckerman (✉)
Division of General Pediatrics and Child and Adolescent Health Measurement Initiative, Department of Pediatrics, Oregon Health & Science University, 707 SW Gaines Rd., Mail Code CDRC-P, Portland, OR 97239-3098, USA
e-mail: zuckerma@ohsu.edu

A. P. Hill
Center for Spoken Language Understanding, Oregon Health & Science University, 840 SW Gaines Rd., Mail Code GH40, Portland, OR 97239, USA

A. P. Hill · K. Guion · L. Voltolina · E. Fombonne
Institute of Development and Disability, Oregon Health & Science University, 707 SW Gaines Rd., Mail Code CDRC-P, Portland, OR 97239, USA

K. Guion
Division of Child Psychology, Oregon Health & Science University, 707 SW Gaines Rd., Mail Code CDRC-P, Portland, OR 97239, USA

L. Voltolina
Department of Behavioral Neuroscience, Oregon Health & Science University, Portland, OR, USA

E. Fombonne
Department of Psychiatry, Oregon Health & Science University, Portland, OR, USA

Introduction

Childhood overweight (OWT) and obesity (OBY) affect nearly 1/3 of U.S. children, and the prevalence of these conditions has increased at least four-fold since the 1970s (Kipping et al. 2008; Ogden et al. 2012). OWT, defined as child body mass index [BMI]/age ≥85 percentile but <95 % percentile, and OBY, defined as child BMI/age ≥95 percentile (Krebs et al. 2007), are both associated with substantial health risks for children (Dietz and Robinson 2005; Unger et al. 1990), including risk of type 2 diabetes (Pinhas-Hamiel et al. 1996), hypertension (Friedemann et al. 2012; Williams et al. 1992), dyslipidemia (Williams et al. 1992), orthopedic problems (Taylor et al. 2006), and sleep apnea (Verhulst et al. 2007). In addition, childhood OBY places a child at increased risk of OWT or OBY in adulthood, and has been associated with premature death, regardless of adult weight status (Bjorge et al. 2008; Must et al. 2012; van Dam et al. 2006).

Little is known about the prevalence and correlates of OWT and OBY among children with Autism Spectrum Disorder (ASD). However, research on this group of children is taking on increased public health importance, since ASD is also quite prevalent (Autism and Developmental

Disabilities Monitoring Network Surveillance Year 2008 Principal Investigators 2012; Maenner and Durkin 2010; Windham et al. 2011) and is increasing in prevalence (Blumberg et al. 2013).

It is unclear whether risk factors for obesity in ASD are the same or different from risk factors for children generally. However, obesity in ASD may be particularly problematic for a variety of reasons. First, core symptoms of ASD may relate to weight problems: for instance, children with ASD may lack social motivation to participate in family meals or in structured physical activities with other children (Lee et al. 2008), which might promote healthy weight. Likewise, families and therapists may be more likely to use food as a reward in children with ASD due to lack of social motivation. Many children with ASD also demonstrate selective eating as a restrictive/repetitive behavior pattern (Zimmer et al. 2012), and have been shown to have higher intake of low-nutrition, energy-dense foods (Evans et al. 2012). The severity or type of a child's symptoms may also affect his or her ability to participate in physical activities that might mitigate weight gain. For instance, children with ASD who have a more depressive or withdrawn subtype may be less likely to participate in healthy physical activities or social eating patterns that protect children from unhealthy weight.

ASD comorbidities may also impact weight trajectories. For instance, poor sleep quality, which is common in ASD (Richdale 1999), may be both a cause (Dev et al. 2013) and a consequence (Bixler et al. 2009) of unhealthy weight. Gastrointestinal (GI) disturbances, such as constipation, are also frequent in ASD and have been associated with poor dietary quality and obesity (Fishman et al. 2004). Likewise, gross motor deficits, which are common in ASD (Ghaziuddin and Butler 1998; Ming et al. 2007), may prevent children from participating in age-appropriate physical activities that could mitigate weight gain (Minihan et al. 2007). Safe and appropriate physical activity opportunities geared towards inclusion of children with ASD are often limited as well.

ASD treatments may also alter risk for OWT and OBY. The associations of common dietary treatments for children with ASD, such as gluten-free/casein-free diet, with weight status are unknown. Additionally, little is known about the effects of medication use on weight in children with ASD. However, many common medications for treating ASD and its comorbidities have alterations in weight as side effects: for instance, children taking atypical neuroleptics, a common ASD behavioral treatment, are known to have problems with elevated BMI (Hellings et al. 2001). Conversely, children taking stimulants to treat attentional problems, which are also prevalent in ASD, may be relatively protected from excess weight gain (Faraone et al. 2008).

Finally, some children with ASD might have specific genetic vulnerabilities that affect weight control. A known example of this finding is a group of children with microdeletions at chromosome 16p11, who have both ASD and increased risk of unhealthy weight (Miller et al. 2009).

Only a few prior studies have investigated rates of OWT and OBY among children with ASD, and they have shown quite variable results. Studies report OWT prevalence ranging from 8 to 35 % (de Vinck-Baroody et al. 2013; Egan et al. 2013; Evans et al. 2012; Hyman et al. 2012; Rimmer et al. 2010) and OBY prevalence ranging from 8 to 43 % (Chen et al. 2010; Curtin et al. 2005, 2010; de Vinck-Baroody et al. 2013; Egan et al. 2013; Evans et al. 2012; Ho et al. 1997; Hyman et al. 2012). Overall, however, reported rates are relatively high compared to U.S. population samples: for instance in the 2009–2010 National Health and Nutrition Examination Survey (NHANES), 14.5 % of children age 2–19 met criteria for OWT and 16.9 % met criteria for OBY (Ogden et al. 2012). Only two studies compared children with ASD to typical peers in the same sample. Curtin et al. (2010) found higher rate of OBY in ASD that bordered on significance (30.4 vs 23.6 %; $p = .075$) but did not address rates of OWT; Evans et al. (2012) also found a near-significant difference in rates of OBY (17 vs 9 %; $p = .09$) but no significant differences in OWT; however, Evans' sample size was considerably smaller than Curtin's sample. All studies of OWT and OBY in ASD have been limited by small sample sizes (Curtin et al. 2005; Egan et al. 2013; Evans et al. 2012; Ho et al. 1997), use of parent-reported weight and height parameters (Chen et al. 2010; Curtin et al. 2005), non-standard definitions of obesity (Ho et al. 1997), or convenience sampling (Rimmer et al. 2010).

In addition, few prior studies have directly addressed which factors are associated with OBY in ASD. In a chart review, Curtin et al. (2005) found a non-significant trend toward increased BMI among older children with ASD. Several studies have suggested that children with more severe ASD symptoms are at higher risk for OWT or OBY. For instance, Ho et al. (1997) found that OBY had a positive correlation with autism severity as measured by the Childhood Autism Rating Scale (CARS). Similarly, Egan et al. (2013) reported that children with autistic disorder are more likely to have OBY than children with Asperger Disorder or Pervasive Developmental Disorder – Not Otherwise Specified. Several studies addressed medications use: Dreyer found that children with ASD taking atypical antipsychotic medications were more likely to have OBY, and that those taking stimulant medications were less likely to have OBY; however, it was unclear if any statistical testing was performed (Dreyer et al. 2008). Egan et al. (2013) found no associations between any psychotropic medication use and OWT/OBY, but sample size of those on medication ($n = 13$) was too small to examine medication classes separately.

In this study we sought to add to existing literature by investigating the prevalence of OWT and OBY in a large clinical sample of children (n = 388) who were diagnosed with ASD based on DSM-IV-TR criteria, using the Autism Treatment Network (ATN) dataset. This dataset has comprehensive, validated data about the characteristics of each child's ASD as well as his or her cognitive, behavioral and adaptive functioning. In addition, this dataset had detailed information on demographic factors, treatments for ASD, and ASD comorbidities. Other studies using ATN participants have focused on nutrient intake (Hyman et al. 2012) and demographic associations with weight status (de Vinck-Baroody et al. 2013). Our study aimed to take a broader view of ASD-specific and general childhood risk factors for OWT and OBY in this sample. The study's primary research questions were: (1) What is the prevalence of OWT and OBY in this sample? (2) What child and family demographic characteristics, ASD characteristics (including cognitive and adaptive functioning), ASD comorbidities (GI problems and sleep problems), and ASD treatments were associated with increased risk of OWT and OBY?

Methods

Participants

The Autism Speaks Autism Treatment Network (ATN), a collaboration of 17 academic health centers in the United States and Canada, has developed a common registry protocol for children enrolled at each network site. Site-wide registry inclusion criteria are age 2–18 years and confirmation of an ASD diagnosis, supported by DSM-IV-TR criteria and administration of the Autism Diagnostic Observation Schedule (ADOS) (Lord et al. 1999). Eligible families are invited to participate in the registry, which involves written consent and the collection of clinical data that are regarded as routine standard of care of ASD, such medication usage, health, and behavior assessments. Data are entered into the ATN Registry by trained study coordinators at each site. The current study included 376 children enrolled in the ATN at Oregon Health and Science University (OHSU). Protocols were deemed exempt from review by the OHSU Institutional Review Board. The study sample included all children enrolled in the OHSU ATN Registry (based on the date of consent) from May 2008 to September 2012.

Measures

Body Mass Index (BMI)

Weight and height data for each child were obtained as part of a physical exam, by trained clinical staff, using a metric scale and wall-mounted stadiometer. Age- and sex-specific BMI percentiles were calculated for each participant based on CDC population norms (Kuczmarski et al. 2000). Children were classified as either healthy weight (HWT $n = 244$; 5 $\leq$BMI/age $<$85 percentile), overweight (OWT $n = 68$; 85 $\leq$BMI/age $<$95 percentile), or obese (OBY $n = 64$; BMI/age $\geq$95 percentile) for group comparisons. Underweight children (BMI $<$5 percentile; $n = 12$) were excluded from the analysis.

Socio-Demographic Characteristics

Socio-demographic characteristics included child gender, age, race/ethnicity, and caregiver(s) education levels, based on caregiver report. Race was categorized as white, black/African American, Asian, or other (Native American or Alaskan Native, Native Hawaiian or Pacific Islander, mixed race). Because of the small numbers in some categories, these were collapsed to white/Caucasian and non-white/non-Caucasian for analyses. Ethnicity was categorized as either Hispanic/Latino origin or non-Hispanic/non-Latino. Caregiver education level was classified as the maximum educational level of the child's primary or secondary caregiver. For analyses, these were grouped as follows: high school graduate or less, some college, or college graduate or higher.

ASD Characteristics and Cognitive/Adaptive Functioning

Autism Severity The ADOS is a standardized observational assessment that is organized into four modules, based on the child's expressive language level (Lord et al. 1999). The ADOS Calibrated Severity Score (CSS) was used as an indicator of ASD severity for children (Gotham et al. 2009). Standardized domain scores were also calculated (Hus et al. 2012), as indicators of symptom severity in two domains: social affect and Restricted and Repetitive Behaviors (RRB). Children who received Module 4 ($n = 10$) were excluded from these analyses as CSSs are defined only for ADOS Modules 1–3.

Adaptive Functioning We used the Vineland Adaptive Behavior Scale- Second Edition (VABS-II) to assess functional skills used in everyday life (Sparrow et al. 2005). The domains assessed in the VABS-II include Communication, Socialization, Daily Living Skills, and Motor Skills. The VABS-II also provides an Adaptive Behavior Composite Score, which is an estimate of overall adaptive functioning. Standardized scores of the VABS-II have a mean of 100 and a SD of 15. Test–retest reliability for the VABS-II has been established: subdomain reliability coefficients are excellent with most values exceeding 0.85.

Cognitive Abilities Cognitive abilities were assessed using a range of measures for $n = 306$ participants. The majority of children (n = 205) were administered the Mullen Scales of Early Learning (MSEL; [Mullen 1995]). The MSEL Early Learning Composite (ELC) Standard Score was used as an estimate of Full Scale IQ (FSIQ). Remaining participants were administered either the full Stanford Binet Scales of Intelligence-5th Edition (SB5, $n = 98$) (Roid 2003), the abbreviated SB5 battery (ABIQ, $n = 2$), or the Wechsler Preschool and Primary Scales of Intelligence-Third Edition ($n = 1$; [Wechsler 2002]). Preliminary analysis revealed that SB5 FSIQs were normally distributed, whereas MSEL ELCs significantly deviated from normal. Inspection of the data revealed that about half of the children who received the MSEL ($n = 105$) were assigned the lowest ELC score possible (49), creating scores that were significantly positively skewed. To address this issue, we attempted to impute FSIQs for these children using the regression relationship of ELC to VABS-II Adaptive Behavior Composite scores for children with ELCs greater than 49. However, FSIQ distributions (range, skewness, kurtosis, etc.) continued to differ between the MSEL and SB5. As a result, in final analyses, we did not analyze FSIQ as a continuous variable across measures, and instead created FSIQ categories for group comparisons as follows: average to above average range ($\geq$85), below average range (70–84), and intellectual disability range (<70).

Behavioral Problems Behavioral problems were measured using the *Child Behavior Checklist (CBCL)*. The CBCL is a norm-referenced parent-report questionnaire that assesses specific behavioral and emotional problems in children and adolescents (Achenbach and Rescorla 2000, 2001). Parents completed either the Preschool form of the CBCL (1.5–5 years, 100 items) or the School Age form (6–18 years, 113 items). Parents rated each behavior on a 3-point Likert-scale (0 = not true, 1 = somewhat/sometimes true, 2 = very often true). Mean test–retest reliabilities of $r = .85$ and $r = .88$ across an eight day period have been reported for the Preschool and School-Age forms, respectively. We analyzed the T-scores ($M = 50$, $SD = 10$) of the seven subscales with clear counterparts in both versions (Syndrome scales: Internalizing Problems, Externalizing Problems, Total Problems; DSM-IV-oriented scales: Affective Problems, Anxiety Problems, Attention Deficit/Hyperactivity Problems, Oppositional Defiant Problems).

ASD Comorbidities

Sleep Problems Problematic sleep behaviors were derived from an abbreviated version of the Children's Sleep Habits Questionnaire (CSHQ), a validated parental questionnaire describing sleep behaviors in children ages 2–10 (Owens et al. 2000). The CSHQ includes 39 items and is rated over the previous week by parents to screen for the most common sleep problems. The majority of sleep questions are answered on a 3-point scale (1 = rarely, 2 = sometimes, 3 = usually). The CSHQ contains items related to eight sleep domains: (1) bedtime resistance (2) sleep onset latency, (3) sleep duration, (4) anxiety around sleep, (5) night awakenings, (6) sleep disordered breathing, (7) parasomnias and (8) morning waking/daytime sleepiness. The Total Sleep Disturbance Score is the sum of scores across 33 items; scores at or above 41 have been reported as a sensitive cutoff for clinically significant sleep problems in typical populations (Owens et al. 2000), and a cutoff of 47 has been proposed specific to ASD populations (Katz et al. 2013). Because the CSHQ has been studied well only to age 10 years, we limited analyses of sleep associations with BMI to children younger than 11 years of age.

GI Disturbance As part of the ATN Parent Survey, parents indicated whether they currently had concerns about their child's GI problems (i.e., "gastrointestinal [belly] problems [diarrhea, constipation, pain]"). "Yes"/"No" responses to this question were used as a measure of GI disturbance.

ASD Treatments

Use of Complementary and Alternative Medicine (CAM) The medical history questionnaire completed by the parent at entry to the registry includes questions about CAM treatments. Parents endorse whether their child receives any of the following: chiropractic care, dietary supplements (amino acids, high dosing vitamin B6 and magnesium, essential fatty acids, probiotics, digestive enzymes, glutathione), or dietary interventions (gluten-free, casein-free, no processed sugars). Because of the infrequent rate of CAM treatments, the variables were collapsed into a primary outcome measure of any CAM use. In a separate exploratory analysis, we also examined dietary interventions versus no dietary intervention.

Use of Psychotropic Medications and Melatonin Medications were those received at time of enrollment and confirmed by ATN clinicians. We categorized medications as stimulants, selective serotonin reuptake inhibitors (SSRIs), α-adrenergic agents, anticonvulsants, antihistamines, and atypical neuroleptics. Use of melatonin was also recorded. Due to low frequencies for medications within each category, variables were collapsed for two analyses: (1) any psychotropic drug use versus none; and (2) melatonin use versus no melatonin use.

Statistical Analyses

The goal of all analyses was to test the associations between BMI status (HWT, OWT, OBY) with socio-demographic characteristics, ASD symptoms, comorbidities, and treatments in this sample of children with ASD. Because our focus was on exploring possible associations with BMI, we used univariate methods to identify variables that differed significantly among the three BMI groups. For categorical variables, group comparisons were conducted using Chi square tests. Significant results were subsequently assessed with post hoc tests on variables with p values <.10 to identify significant pairwise differences. For continuous variables, Levene's tests indicated that the assumption of homogeneity of variables was met for each variable. Prior to analysis, we screened for skewness, kurtosis, and outliers in the data. All continuous variables (VABS-II, ADOS, CBCL, CSHQ scores) were not normally distributed and transformations were unsuccessful, so group comparisons on continuous variables were conducted using one-way Kruskal–Wallis tests, followed by pairwise post hoc tests where the omnibus test had $p < .10$. Permutation tests were also performed and in all cases were consistent with non-parametric tests, so results of non-parametric tests are reported. Statistical significance was set at the $p < .05$ level, but post hoc testing was performed on any variable with a p value of <.10 in order to explore trends. Finally, due to possible confounding between melatonin use and sleep problems, we used stepwise logistic regression to examine the joint associations between melatonin use and sleep difficulties with BMI status.

Missing Data

Missing data were an issue for several measures used in the current study. Missing data for several variables (GI disturbance, use of CAM, use of psychotropic drugs, use of melatonin) were replaced with zeros in analyses as a conservative approach to avoid overestimation of proportions. Participants with missing data were excluded from analyses for measures of ASD severity (ADOS CSS, $n = 49$), adaptive functioning (VABS-II, $n = 38$), and cognitive abilities ($n = 71$). Participants with 50 % or more missing CSHQ items ($n = 40$ for total sleep disturbance scores) were excluded from analyses. For participants with less than 50 % of CSHQ items missing ($n = 97$), missing data were imputed using mean responses for non-missing items.

Results

Prevalence of OWT and OBY

Sample characteristics are reported in Table 1. The prevalence of HWT (BMI/age ≥5 percentile but <85 percentile) was 64.9 % ($n = 244$), OWT (BMI/age ≥85 percentile but <95 percentile) was 18.1 % ($n = 68$), and OBY (BMI ≥ 95th percentile) was 17.0 % ($n = 64$). Prevalence of BMI ≥85 percentile was 34.6 % in males and 33.3 % in females, with estimates varying non-significantly across age ranges (Fig. 1).

Table 1 Sample ($N = 376$)

	M (SD) or n (%)
Sociodemographic characteristics	
Age	5.5 (3.2)
Male	311 (82.7)
Race	
White	288 (76.6)
Black/African American	15 (4.0)
Asian	25 (6.7)
Other	11 (2.9)
Missing	37 (9.8)
Ethnicity	
Hispanic or Latino origin	44 (11.7)
Not of Hispanic or Latino origin	297 (78.9)
Missing	35 (9.3)
Caregiver(s)' highest level of education	
High school or less	86 (22.9)
Some college	152 (40.4)
College graduate or more	68 (18.1)
Missing	70 (18.6)
Autism characteristics and functioning	
ADOS module	
Module 1	199 (52.9)
Module 2	94 (25.0)
Module 3	46 (12.2)
Module 4	10 (2.7)
Missing	27 (7.2)
VABS-II composite score	67.5 (13.5)
FSIQ	
Average to high (≥85)	60 (16.0)
Borderline Intelligence (70–84)	40 (10.6)
<70	206 (54.8)
Missing	70 (18.6)

ADOS Autism Diagnostic Observation Schedule, *FSIQ* Full Scale IQ; *VABS-II* Vineland Adaptive Behavior Scales-II

Associations with OWT and OBY

Socio-Demographic Associations

Bivariate analyses showed that age, gender, caregiver education level, and race/ethnicity, and autism severity were unrelated to BMI status (*p*s >.10; Table 2). Age was non-normally distributed with significant positive skew

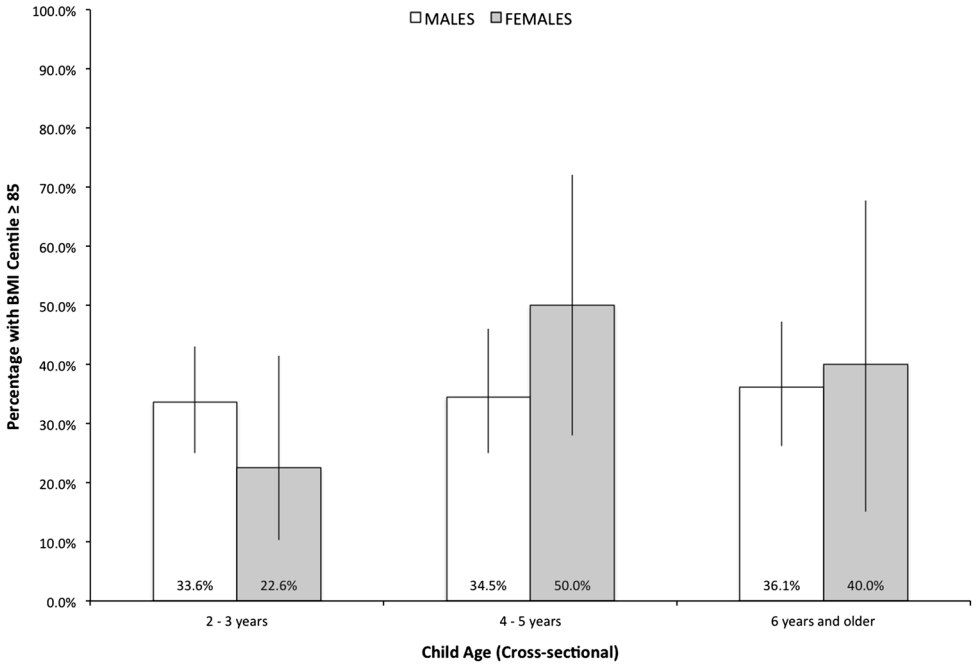

Fig. 1 Percentages of males and females with BMI centile ≥85 by age (with 95 % confidence intervals)

Table 2 Sociodemographic characteristics by BMI group

	M (SD) or percent			Test statistic	*p* value
	HWT $n = 244$	OWT $n = 68$	OBY $n = 64$		
% Male	81.9	82.4	85.9	$\chi^2 = 0.57$	.753
Age, *M (SD)*	5.3 (2.9)	5.8 (3.3)	5.9 (3.9)	$H = 2.39$	.303
Caregiver education (%)					
High school or less	27.4	21.7	37.5	$\chi^2 = 7.61$	.107
Some college	50.5	46.7	50.0		
College graduate or more	22.1	31.7	12.5		
% non-white	16.0	17.7	8.6	$\chi^2 = 2.38$	.305
% Hispanic/ Latino	11.6	12.7	17.7	$\chi^2 = 1.63$	.441

Post-hoc testing was not conducted for any of the above variables due to no significant findings on omnibus test; percentages are within BMI group (columns)

(Mean age = 5.5; Median age = 4.3) and was thus analyzed using non-parametric Kruskal–Wallis tests.

ASD Severity, Adaptive and Cognitive Functioning, and Behavioral Problems

ASD severity, as calculated by the ADOS total CSS, had no significant associations with BMI status. In analyses of ADOS subdomain severity scores, OWT children had significantly lower RRB CSSs than the other two groups (higher scores indicate more severe RRBs) (Table 3). To further examine this finding, we analyzed ADOS RRB CSSs separately for Modules 1 ($n = 199$) and 2 ($n = 94$) as these were the most frequent modules administered in the sample. Although the effect of weight status was not significant within each module, the OWT group tended to have lower RRB CSSs than HWT and OBY in both Module 1 ($H = 5.73$, $p = .06$) and Module 2 ($H = 4.35$, $p = .11$), which is consistent with the pattern observed with RRB CSSs in the full sample (Table 3).

Adaptive functioning, as measured VABS-II standardized scores, was not related to BMI (Table 3). Cognitive functioning, measured as the association between BMI status and FSIQ was also not significant (χ^2 $p = .18$). There was a trend for a higher proportion of FSIQs <70 and a lower proportion of children with FSIQs ≥85 in the OBY group; however, the corresponding effect size (Cramer's $V = 0.10$) indicates a weak association between BMI status and FSIQ in the current sample.

The analysis of behavioral problems showed a trend for total CBCL scores to be elevated in the OBY group compared to the other two groups (Table 3). Although non-significant, this also seemed to be true for both Internalizing and Externalizing Problems. When DSM-IV CBCL scales were examined, the OBY group showed a significant elevation in Affective Problems, and to a lesser extent Anxiety Problems (*ns*), whereas the HWT and OWT groups were closely comparable. Because items on the Affective Problems scale differs between the two CBCL forms, we separately examined Affective T-scores for

Table 3 ASD severity, adaptive and cognitive functioning, and behavioral problems by BMI groups

	M (SD) or percent			Test statistic	*p* value	Post-hoc testing *p* values		
	HWT	OWT	OBY			HWT OWT	HWT OBY	OWT OBY
ASD severity (ADOS calibrated severity scores[a])	$n = 213$	$n = 58$	$n = 56$					
Total CSS	7.2 (2.0)	7.0 (1.8)	7.3 (1.8)	$H = 0.99$	.609			
Social affect CSS	6.8 (2.2)	7.1 (1.8)	6.8 (2.0)	$H = 0.81$	.667			
Restricted and repetitive behaviors CSS	7.9 (1.7)	7.0 (2.0)	7.9 (1.6)	$H = 9.06$	.011	.003	.953	.018
Adaptive functioning (VABS-II[b])	$n = 222$	$n = 58$	$n = 59$					
Adaptive behavior composite	68.2 (13.3)	66.0 (14.6)	66.1 (12.9)	$H = 3.00$	.223			
Communication	70.0 (15.4)	69.5 (15.8)	68.0 (13.7)	$H = 1.03$	.597			
Socialization	68.2 (10.3)	67.5 (8.6)	66.9 (7.9)	$H = 1.10$	.577			
Daily living skills	73.7 (14.3)	73.8 (12.6)	72.6 (13.5)	$H = 0.51$	.777			
Cognitive functioning (FSIQ)	$n = 202$	$n = 51$	$n = 53$					
Average to above average range (% ≥85)	21.8	17.0	13.7	$\chi^2 = 6.31$	.177			
Below average range (% 70–84)	12.4	20.7	7.8					
Intellectual disability range (% <70)	65.8	62.3	78.4					
Behavioral problems (CBCL T-scores[c])	$n = 239$	$n = 67$	$n = 63$					
Total problems	66.0 (9.3)	65.8 (10.9)	69.0 (10.6)	$H = 5.01$	.082	.811	.026	.107
Internalizing problems	64.4 (8.3)	64.0 (10.4)	65.8 (9.1)	$H = 1.09$	.578			
Externalizing problems	61.9 (11.2)	62.3 (12.7)	63.6 (12.9)	$H = 1.57$	.456			
Affective problems	62.7 (8.5)	63.5 (9.5)	66.9 (9.7)	$H = 8.30$	.016	.614	.004	.052
Anxiety problems	60.7 (9.5)	60.7 (9.6)	63.5 (11.1)	$H = 2.69$	.259			
Attention deficit/hyperactivity problems	62.8 (8.3)	62.4 (9.0)	63.7 (8.2)	$H = 1.09$	.578			
Oppositional defiant problems	61.2 (9.5)	62.2(10.0)	61.6 (10.1)	$H = 0.29$	.864			

Percentages are within BMI group (columns)

ADOS Autism Diagnostic Observation Schedule, *CBCL* Child Behavior Checklist, *CSS* Calculated Severity Score, *FSIQ* Full Scale IQ, *VABS-II* Vineland Adaptive Behavior Scales-II

[a] ADOS CSSs range from 1 to 10

[b] VABS-II standard scores are $M = 100$, $SD = 15$

[c] CBCL T-scores are $M = 50$, $SD = 10$

those who received the Preschool (<6 years, $n = 270$) or the School Age form (≥6 years, $n = 99$). On the Preschool CBCL, the effect of BMI status was significant ($H = 11.57$, $p = .003$), with affective problems in OBY significantly higher than HWT ($p < .001$) and OWT ($p = .02$). However, on the School Age CBCL, Affective Problems did not differ by BMI status ($H = 0.24$, $p = .89$). There was no difference across the three groups for DSM-IV scales related to disruptive symptoms.

ASD Comorbidities

In the subsample of children age 11 years of age and younger with CSHQ data ($n = 288$), clinically significant sleep problems (using the cutoff score of 41) were present in 86 % of OBY, 84 % of OWT, and 76 % of HWT children ($\chi^2 = 3.08$, $p = .22$). Using the proposed cutoff of 47 for ASD populations (Katz et al. 2013), sleep problems were present in 64 % of OBY, 55 % of OWT, and 49 % of HWT children ($\chi^2 = 3.87$, $p = .14$). Total sleep disturbance scores were significantly elevated in OBY relative to HWT children. This pattern was also true for the CSHQ subscales describing sleep-disordered breathing (OBY > HWT & OWT) and daytime sleepiness (OBY > HWT; Table 4). For a subset of participants for whom duration of sleep (in hours) was reported ($n = 192$), no group differences were found. No differences were found between OWT and HWT for total sleep disturbance or any subscale scores. GI problems were quite prevalent in all groups (32.8 % of OBY, 23.5 % of OWT, and 26.2 % of HWT children), but did not differ according to BMI category ($\chi^2 = 1.59$, $p = .45$).

ASD Treatments

No associations were found with use of any CAM or psychotropic medications (Table 4) or with specific classes of medications (SSRIs and atypical neuroleptics; other

Table 4 Autism comorbidities and treatments by BMI group

	M (SD) or percent			Test statistic	*p* value	Post-hoc testing *p* values		
	HWT	OWT	OBY			HWT OWT	HWT OBY	OWT OBY
Comorbidities								
Sleep problems (CSHQ scores[a])	$n = 187$	$n = 51$	$n = 50$					
Bedtime resistance	9.5 (3.6)	9.0 (3.4)	10.3 (3.5)	$H = 3.57$	.168			
Sleep onset delay	1.8 (0.8)	1.9 (0.9)	1.9 (0.8)	$H = 1.18$	.555			
Sleep duration	4.3 (1.7)	4.2 (1.6)	4.6 (1.8)	$H = 1.66$	.437			
Sleep anxiety	6.2 (2.0)	6.1 (2.0)	6.8 (2.0)	$H = 4.54$	.104			
Night wakings	4.7 (1.8)	4.4 (1.5)	4.7 (2.0)	$H = 0.54$	.763			
Parasomnias	9.7 (2.2)	9.7 (2.5)	10.5 (2.7)	$H = 3.93$	.140			
Sleep disordered breathing	3.3 (0.7)	3.5 (1.0)	3.7 (1.2)	$H = 15.45$	<.001	.159	<.001	.038
Daytime sleepiness	11.7 (2.8)	12.3 (2.8)	12.5 (2.9)	$H = 4.94$	.085	.149	.049	.669
Total sleep disturbance	48.1 (9.3)	48.3 (8.8)	51.6 (9.0)	$H = 7.04$	.030	.765	.008	.059
Gastrointestinal problems[b]	26.2	23.5	32.8	$\chi^2 = 1.59$	.452			
Treatments	$n = 244$	$n = 68$	$n = 64$					
% Using any CAM	28.0	22.7	24.6	$\chi^2 = 0.86$	.652			
% Using any psychotropic medication[c]	32.4	36.8	35.9	$\chi^2 = 0.62$	.734			
% Taking melatonin	20.9	16.2	32.8	$\chi^2 = 5.86$	.054	.489	.066	.043

Percentages are within BMI group (columns)

CAM Complementary or Alternative Medicine

[a] Children 11 years of age and older were excluded from CSHQ analyses

[b] For GI problems: HWT $n = 244$; OWT $n = 68$; OBY $n = 64$

[c] Separate analyses of SSRIs and atypical neuroleptics were also not significant

medication classes had too small sample size to analyze). Only 10.6 % of our sample received dietary interventions (gluten-free, casein-free, no processed sugars), and these were not associated with BMI ($\chi^2 = 1.71$, Fisher's exact test $p = .65$).

Melatonin use was higher in OBY children (32.8 %) compared to HWT (20.9 %, $p = .07$) and OWT (16.2 %, $p = .04$) children. Melatonin use was also significantly associated with the presence of sleep problems using either the general cut-off of 41 ($\chi^2 = 3.88$, $p = .05$) or the ASD-specific cut-off of 47 ($\chi^2 = 7.97$, $p < .01$). Melatonin use was reported for 29 % of children with clinically significant sleep problems and 14.6 % of those without sleep problems, according to the higher cut-off score. To evaluate whether the elevated levels of sleep problems in the OBY group could account for the previously noted association between BMI status and melatonin use, a stepwise logistic regression was performed to predict BMI status (OBY vs HWT + OWT) for children 11 and younger. Melatonin use was entered in step one and had only a weak association with OBY status when HWT and OWT groups were combined ($\beta = 0.50$, $SE = .35$, $p = .15$, $OR = 1.65$ [CI 0.82–3.23]) in the sample of children for whom sleep scores were available. When total sleep disturbance scores were added in step two, sleep score was significantly associated with OBY status ($\beta = 0.33$, $SE = .16$, $p = .03$, $OR = 1.39$ [CI 1.03–1.89]); and the p value for melatonin increased to 0.39 ($\beta = 0.31$, $SE = .36$, $p = .39$, $OR = 1.36$ [CI 0.65–2.72]), suggesting that melatonin was likely a confounder for sleep problems as measured by the CSHQ.

Discussion

In this study, we found that 18.1 % of children with ASD were overweight and 17 % were obese. Overall, 35.1 % of children in this sample were at an unhealthy weight, which falls in the middle range of previous recent reports of BMI in ASD (Curtin et al. 2010; Egan et al. 2013; Hyman et al. 2012). The overall rate of elevated BMI that we found is similar to population-based measures of OWT and OBY children overall in the U.S. The 2009–2010 NHANES, where 31.8 % of U.S. children age 2–19 were at or above the 85th percentile of BMI for age, and 16.9 % were at or above the 95 percentile for age (Ogden et al. 2012). However, given that the mean age of children in our study was significantly lower than NHANES, the similar rate we found is concerning. Since obesity is more prevalent in older children, the relatively high rate we found suggests

that obesity may be affecting children with ASD particularly severely. Our findings suggest that many current efforts to curb the prevalence of OWT/OBY in pediatric populations, may need to be adapted to the specific needs of children on the autism spectrum.

In contrast to national data (Bethell et al. 2010; Ogden et al. 2012), but consistent with prior studies of obesity in ASD (Egan et al. 2013), sociodemographic characteristics were not strongly associated with weight status in this sample. In terms of ASD symptoms, overall severity, cognitive, and adaptive measures lacked significant associations with ASD. However, we did find significantly lower levels of RRB among OWT (but not obese) children with ASD. This pattern of findings is somewhat difficult to interpret. It is possible RRB may manifest as restrictive eating patterns that limit weight, and that when these problems are less severe, weight gain is accelerated. It is also possible that some RRBs themselves burn calories. Finally, there may be an association between RRB severity and use of medications that increase appetite, such as stimulants; these factors could be explored in future research.

Behavioral problems also had notable associations with BMI in this sample. In particular we found a consistent (though non-significant) trend for children with OBY to score higher than normal weight and OWT children on all domains of the CBCL. These higher numbers only reached significance for the DSM-IV oriented affective problems scale, and seemed to be concentrated in those children under age 6 who received the preschool version. The clinical significance of these findings should be interpreted cautiously: in particular, it is possible that specific items in the CBCL battery may have altered results. For example, "overeating" is an item in the Preschool form, but is not included in the affective scale on the School Age form, which may explain some of the age differences we found. In addition, several CBCL items in both forms assess sleep quality, which we found to also be associated with OBY in ASD. However, there is a strong literature trend suggesting that depressive symptoms may be both a cause and consequence of childhood obesity (Goodman and Whitaker 2002; Sjoberg et al. 2005; Wardle and Cooke 2005); therefore, it is possible these results represent a true pattern. Likewise, though non-significant, the consistent trend in higher CBCL scores in the majority of domains (with the exception of Oppositional Defiant Problems) among children with OBY is worthy of note. If they are found to persist in future research, they would suggest that OBY is associated with more global behavioral impairment among children with ASD.

This study also found that ASD comorbidities had significant associations with OWT/OBY among children with ASD. There were abundant associations between poor sleep quality and weight status among children <11 years with ASD: 86 % of OBY, 84 % of OWT, and 76 % of HWT children had clinically significant sleep problems, and obese children with ASD were more likely to have sleep-disordered breathing, daytime sleepiness, and total sleep disturbances than HWT children. These findings are consistent with the general literature about sleep problems in children with obesity (Bixler et al. 2009; Dev et al. 2013). However, given that sleep problems overall are highly prevalent among children with ASD, we view having obesity as an additional risk factor that makes a child's risk for sleep disturbance extremely high. The impact of combined obesity and sleep problems on daytime learning for children with ASD (who already face educational challenges) may be of clinical relevance (Reynolds and Malow 2011; Schreck et al. 2004); this combination of problems should be seen by parents and health care providers as a 'red flag.'

Consistent with prior studies (Curtin et al. 2005; Egan et al. 2013), we found few treatment associations with ASD. The one significant treatment association we found, melatonin use, was likely related to increased sleep problems among children with elevated BMI.

This study had a number of limitations. First, all data were cross-sectional in nature. Given that obesity can be both a cause and a consequence of so many additional problems, the associations we found should be interpreted with caution, and a causal direction should not be inferred. Since this was a secondary data analysis, some factors that might be important in the epidemiology for obesity for children with ASD were not available. Direct measures of physical activity, dietary intake, and markers for poor nutrition associated with obesity (e.g. anemia, vitamin D deficiency), for instance, were lacking. Due to small sample size, we did not investigate the correlates of underweight children with ASD. The population that we studied was primarily young (mean age <6 years), and predominantly white non-Latino race/ethnicity. Given the known associations of older age and minority race/ethnicity with OWT and OBY (Bethell et al. 2010), our findings may therefore underestimate true OWT and OBY rates. Likewise, since the sample was obtained from autism referral centers, the children included may be of higher socioeconomic status than most U.S. children with autism, which may also have depressed the rate of OWT/OBY we found.

Missing data is a significant limitation of the study. We took a conservative approach to missingness by coding missing variables as null where we felt positive responses would have been noted. We imputed CSHQ scores due to large amounts of missing data for this scale. Other variables also were problematic for other reasons: for instance, the ADOS RRB CSS may be of limited value in higher-functioning children, who may not display their RRBs in a

clinical setting. Some of the parent-report measures, such as the CSHQ, may have been completed prior to the child's clinic visit and therefore may not be contemporaneous with BMI measurement. Our measure of GI disturbance was a single item that has not been previously validated; more robust measures of GI disturbance may be necessary to truly understand the relationship between GI problems and weight status in children with ASD.

Due to the exploratory nature of this study, as well as missing values for many variables in our dataset, we focused on exploring possible associations with BMI using bivariate analyses. Longitudinal studies are needed to better understand the trends we observed in this cross-sectional sample, and multivariate analyses would clarify several of the associations we report. Finally, several of the correlates identified in the present study were only marginally associated with BMI status. As the number of group comparisons conducted may have increased the probability of Type I error, replication of these results is needed before strong conclusions can be drawn.

Nonetheless, the study has important practice and policy implications. Given that OWT and OBY may affect as many as 1 in 3 children with ASD, focusing on the unique barriers to healthy weight in this population may be important to their long-term health outcomes, development, and functioning. In particular, our study suggests that more attention to sleep and mood disorders among OWT and obese children with ASD could important; longitudinal studies are need to assess whether adequate treatment of these factors will lead to improved weight status in ASD. The findings also support the concept that improved treatment of weight in children with ASD may reduce certain problematic ASD features, such as sleep and affective functioning. Activities that support healthy weight in all children, such as physical activity, reduced screen time, and family meals, are also likely very important for children with ASD, but parents of children with ASD may need more help in finding ways to implement these activities into their child's daily life.

Overall, there is a great need for more focused research in this area. Future studies addressing obesity in ASD should particularly consider employing more direct measures of dietary intake and physical activity, collecting BMI data in a clinical setting, and including a group of non-autistic children for comparison. Such a measurement strategy would allow clinicians and policy makers to better understand which obesity-related risk and protective factors play the most important roles in ASD and ultimately allow for the development of tailored lifestyle interventions.

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