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Obesity in Children with Autism Spectrum Disorders

Carol Curtin, MSW,
University of Massachusetts Medical School – E.K. Shriver Center, 200 Trapelo Road, Waltham, MA 02452

Mirjana Jojic, MD, and
Department of Psychiatry, UMass Medical Center - University Campus, 55 Lake Avenue North, Worcester, MA 01605

Linda G. Bandini, PhD, RD
University of Massachusetts Medical School – E.K. Shriver Center, 200 Trapelo Road, Waltham, MA 02452

Abstract

Research suggests that children with autism spectrum disorders (ASD) have a prevalence of obesity at least as high as that seen in typically developing (TD) children. Many of the risk factors for children with ASD are likely the same as for TD children, especially within the context of today's obesogenic environment. However, the unique needs and challenges that this population faces may also render them more susceptible to the adverse effects of typical risk factors, and they may also be vulnerable to additional risk factors not shared by children in the general population. Psychopharmacological treatment, genetics, disordered sleep, atypical eating patterns, and challenges for engaging in sufficient physical activity may be uniquely associated with the development of obesity in children with ASD. Obesity and its associated sequelae potentially represent a significant threat to independent living, self-care, quality of life, and health for individuals with ASD. This article provides a summary of the literature on the prevalence of obesity in children with ASD and the putative obesity risk factors that this population may experience.

Keywords

Autism Spectrum Disorders; Obesity; Children; Eating Patterns; Genetics; Physical Activity; Psychopharmacology; Sleep

Introduction

Obesity in children has become a significant health concern, and the prevalence of childhood obesity has tripled over the last twenty years. Data from the National Health & Nutrition Examination Survey (NHANES) indicate that nearly a third of children ages 2-19 in the general population are overweight or obese.[1] Evidence from clinic-based studies and nationally representative surveys suggests that children with autism spectrum disorders (ASD) have a prevalence of obesity at least as high as that seen in typically developing (TD) children. While significant efforts are underway to understand and treat obesity in the

general pediatric population, relatively little work has focused on children with ASD. In general, children who are obese are likely to remain so as adults, and excess weight substantially increases risk for chronic diseases such as diabetes, cardiovascular disease, and certain cancers.[2] Given the increasing prevalence of ASD, the prevention of secondary conditions associated with obesity in children in this population is a pressing public health issue, with implications for independent living and quality of life.

Research on the prevalence of obesity and associated risk factors in children with ASD remains limited. Many of the risk factors for children with ASD are likely the same as for TD children, especially within the context of today's obesogenic environment. However, the unique needs and challenges that this population faces may also render them more susceptible to typical risk factors and they may also be vulnerable to additional risk factors not shared by children in the general population. Psychopharmacological treatment, genetics, disordered sleep, atypical eating patterns, and challenges for engaging in sufficient physical activity may be uniquely associated with the development of obesity in children with ASD.

The purpose of this article is to summarize the literature on the prevalence of obesity in ASD and the putative obesity risk factors that this population may experience. A literature search was undertaken using electronic databases of PubMed, Google Scholar, Ovid, and MEDLINE to locate relevant literature published in English in the last 25 years using search term combinations including the population term (e.g., autism, autism spectrum disorder) and key words for each of the sections such as obesity, overweight, obesity prevalence, weight status, genetics, medications, eating patterns, food selectivity, physical activity, etc. Additionally, several bibliographies were inspected manually to identify additional relevant articles.

The prevalence of obesity in children with ASD

Overweight and obesity are generally recognized as the presence of excess body fat or adipose tissue. Obesity is classified by body mass index (BMI) which is calculated as weight in kilograms divided by the square of height in meters (kg/m^2). For children in the United States, sex-specific BMI-for-age percentiles are calculated based on the 2000 US growth reference.[3] Youth who are considered to be overweight have a BMI-for age that is greater than or equal to the 85^{th} percentile, and those who would be classified as obese have a BMI-for-age at or above the 95^{th} percentile.[4] Countries outside of the US have used different criteria and cut-points at different points in time.

Only a few studies have reported data on weight status of children with ASD. An early Japanese study of 140 school children with autism ages 7-18 years found that 25% were obese.[5] In a second large Japanese study of 20,013 children (6-17 years) with intellectual disability attending special schools, 413 of whom had autism, Takeuchi[6] reported an obesity prevalence of 22% in boys and 11% in girls with autism. Mouridsen et al.[7] examined the weight status of 117 young children with autism in Denmark (mean age 5.5 for boys and 5.2 for girls) and found that BMI for males but not for females was significantly lower than in an age-matched reference population. Curtin et al.[8] conducted a chart review of a small

sample of children with ASD from a tertiary care clinic and found that the prevalence of overweight and obesity was 35.7% and 19.0% respectively, and thus as significant a problem in children with ASD as for children in the general population. Xiong et al.[9] found that among 380 boys and 49 girls in a clinical sample with autism ages 2-11 years in China, 33% were overweight and 18% were obese. Egan et al.[10] performed a retrospective chart review of 273 young children (mean age 3.89 years) with ASD seen by developmental specialists and found a prevalence of overweight and obesity of 39.0% and 23.1% among children with autistic disorder and Asperger's disorder/PDD-NOS, respectively. In a study of 111 children with ASD ages 2-9 yrs. seen in a developmental clinic in China, Xia and colleagues[11] found that the prevalence of overweight and obesity was 31.5% based on the WHO 1995 standards. Curtin et al.[12] also examined the prevalence of obesity in children with ASD using data from the National Study of Children's Health and found that children with ASD were 40% more likely to be obese than children in the general population. Finally, in a small study of 53 children with ASD and 58 TD children recruited from the community, Evans et al.[13] found that 17% of the children with ASD met criteria for obesity compared to 9% of TD children. However, this difference only reached borderline significance (p=0.09), likely due to the study's small sample size. Notably, the children with ASD included in the study were not taking medications that would have affected their weight status.

These findings suggest that children with ASD are at risk for obesity at the same or higher rate than children generally, and thus this issue warrants both research and clinical attention because of the adverse health effects known to be associated with obesity. In the next sections we review the current evidence on known and putative risk factors for obesity in children with ASD, and then discuss the implications for future research and clinical management/intervention.

The genetics of obesity and ASD

Numerous twin and family studies indicate that ASD has a biologic basis, with concordance rates higher for monozygotic twins (70-90%) as compared to dizygotic twins (20-30%)[14,15]; unfortunately, the specific genetic determinants have not been fully identified.[16,17] Both inherited and de novo single nucleotide polymorphisms (variations occurring in a single nucleotide, i.e., A, T, C, or G) and copy number variants (CNVs, misplaced or duplicated segments of chromosomes) have been implicated in the causation of ASD.[18–20] Although direct cause-and-effect from specific genetic variants has yet to be elucidated, approximately 7-20% of idiopathic cases of autism result from CNVs, with both deletions and duplications playing a role.[16] Recent studies examining chromosome microarrays have identified approximately ten CNVs associated not only with ASD, but several psychiatric (schizophrenia, bipolar disorder) and prodromal (congenital heart disease, hypotonia, micro/macrocephaly, seizures) disorders. Most notably, genomic imbalances and high rates of recurrent CNVs are emerging as risk factors for obesity,[21,22] suggesting an inherited correlation between obesity and ASD.

Genomic duplications and deletions at 16p11.2 are among the most commonly associated with ASD, and deletions in this region have recently have been shown to play a role in early-onset childhood obesity.[23] Zufferey et al.[24] examined 285 child and adult carriers of a

~600 kb deletion at the 16p11.2 locus. They found that over 80% of the carriers exhibited psychiatric disorders, including attention deficit and disruptive behavior disorders, anxiety disorders, and substance related disorders, while 15% of the pediatric carriers were diagnosed with ASD. More than 50% of pediatric 16p11.2 carriers were obese, with weight gain starting at 3.5 years and progressing rapidly until age 7. This number of obese gene carriers increased to 75% in adulthood. Despite the link between 16p11.2 carriers, ASD, and obesity, the researchers found that obesity occurred independently of ASD.

Smaller studies have examined shorter deletion sequences (5Mb-30.1Mb) on 16p11.2. Yu et al.[23] found that of 28 individuals identified with the deletion, nine were classified as obese and six as overweight, with excessive weight gain occurring between the age of five and six years, consistent with the findings of Zufferey et al. [24] A multiplex family study of three boys, a twin pair and an elder brother, found a similar link between autism, mild intellectual disability, and early-onset severe obesity, although the elder brother had less severe autism than the twins. The fact that the gene can be present in individuals who either do not manifest with overweight or obesity,[23] or have differing degrees of autistic symptomatology, suggests variability in penetrance and gene expression.[25]

Although the 16p11.2 gene aberration is the most commonly associated with ASD, several other loci associated with the disorder have been identified. Included is the deletion at 11p14.1 that has been implicated in both obesity and autism, albeit in a small sample.[26] Duplication at 15q11.2, a gene originally thought to be associated solely with Prader-Willi and Angelman syndrome, is also now believed to play a role in other disorders associated with developmental delay, ASD, and obesity.[27] Maged1, part of the MAGE gene family, also involved in the development of Prader-Willi syndrome, may present as progressive obesity with deficits in social interactions suggestive of ASD symptomatology.[28] More recently, a Prader-Willi phenotype of Fragile X syndrome, also known to be associated with ASD,[29,30] was discovered in 13 patients (aged 5-27); all 13 cases were found to have obesity and hyperphagia, while 10 of 13 were diagnosed with ASD.[31] Overall, 200 ASD susceptibility genes have been identified to date;[17] given the significant gene variability and penetrance emerging in both animal and human models of ASD, more work is needed to draw definitive genetic associations between ASD and obesity, with the hope of understanding and combating both.

Psychopharmacological effects on obesity in ASD

The adverse effects of psychotropic medication on weight status is likely the best understood risk factor for obesity in both children and adults with ASD. Psychotropic medication use is prevalent in individuals with ASD. Data obtained from clinical and nationally representative populations of children, including those psychiatrically hospitalized, report that approximately 30% - 60% of children with ASD are prescribed at least one psychotropic medication, and 10% are prescribed more than three medications; stimulants, antidepressants, and antipsychotics are among the most commonly prescribed.[32]

Antipsychotics

Typical and atypical antipsychotics are used extensively to treat psychotic disorders, bipolar disorder, and as adjuncts to antidepressants. Most recently, in 2006 and 2009, the FDA approved risperidone and aripiprazole, respectively, for treatment of irritability associated with ASD.[33] Subsequently, atypical antipsychotics became widely used in ASD and studies have shown they are twice as likely to be prescribed in children with ASD as any other medications.[34]

Atypical antipsychotics (e.g. aripiprazole, clozapine, olanzapine, quetiapine, risperidone, ziprasidone) provide significant benefit in reducing the frequency of extrapyramidal symptoms. However, these second-generation antipsychotics (SGAs) are considerably more apt to cause weight gain.[35–38] In addition to weight gain, metabolic syndrome (defined by abdominal obesity, atherogenic dyslipidemia, raised blood pressure, insulin resistance ± glucose intolerance, proinflammatory state, and prothrombotic state) is of primary concern with SGAs.[39,40] Children appear to be more susceptible than adults to developing obesity and lipid abnormalities associated with SGAs; these findings are noteworthy given that SGAs are first-line treatments in children with ASD-associated irritability.[41]

Risperidone and aripiprazole have the best evidence for treating ASD-associated irritability, and have thus garnered FDA-approval for this indication. Risperidone is the most widely studied of the antipsychotics prescribed to children with ASD, and was the first to be studied in the Research Units on Psychopharmacology Autism Network.[42] All studies of children with ASD treated with risperidone have found weight gain to be significant in treatment versus placebo groups.[39,43] Risperidone was shown to increase appetite in 33% of children with ASD, likely one contributor to the weight increase. Fortunately, weight gain was shown to decelerate with treatment time.[44] Aripiprazole has been studied in two pharmaceutical-funded randomized, double-blind, placebo-controlled trials.[45] The first trial enrolled 98 children ages 6-17[46], the second included 218 children.[47] Marcus et al.[47] confirmed that all treatment groups prescribed aripiprazole 5-15 mg had significant weight gain over placebo, but no one in the study discontinued the medication as a result. Similar to risperidone, sedation was the most common side effect seen with aripiprazole.

Several other atypical antipsychotics have been studied in the treatment of ASD symptoms. One small randomized controlled trial (RCT) with olanzapine estimated a weight increase of 7.5 ± 4.8 lbs. In fact, the risks of weight gain, metabolic syndrome, hyperlipidemia, and DM Type II associated with even short-term use of olanzapine are considerable, and most physicians do not suggest this as first-line agent for children with ASD.[38,45] Ziprasidone is felt to be less obesogenic than other atypicals in adults.[48] Although there are no published RCTs of ziprasidone in ASD, a small trial of 10 adults suggests that the medication reduced total cholesterol and triglyceride levels in patients, while 8 patients lost approximately 13.1 +/- 7.1 lbs. on average.[49] Another open-label study found the medication to be overall weight neutral with a BMI reduction that was not statistically significant.[50] One small, 8-week, open-label trial of quetiapine in 11 adolescent patients did not find any significant difference in body weight of treated individuals.[51] In general, ziprasidone is considered the SGA with the lowest potential for weight gain, followed by aripiprazole, quetiapine, and

risperidone with intermediate risk, while olanzapine is considered to be associated with high risk of weight gain and metabolic disturbances.[48]

The diverse mechanisms by which antipsychotics cause weight gain are not fully understood. It is postulated that weight gain is tied to increased appetite associated with SGA-interaction with neuronal dopamine, serotonin, and histamine receptors.[52] Atypical antipsychotics target several receptors, including 5HT2cR, 5HT3R, α2R, H1R, and β3R, and scientists have theorized that weight gain is associated with the communal effect of SGAs on these receptors.[53] Serotonin and histamine have been most widely implicated in the appetite and metabolic disturbances, and several studies have confirmed an association between the H1 receptor and antipsychotic-induced weight gain.[54,55] Although SGAs are more commonly associated with metabolic disturbances, pioneering work by Leibowitz[56] revealed that the typical antipsychotics, including haloperidol, chlorpromazine, and fluphenazine, also stunted appetite suppression via endogenous amines in the perifornical lateral hypothalamus. Thus, through interactions with multiple neuronal receptors, both typical and atypical antipsychotics have the propensity to significantly impact patients' weight and metabolic profile.

Combating antipsychotic-induced weight gain

The prevalence of antipsychotic-induced weight gain has spurred investigation into pharmacological and behavioral interventions to reduce the impact of these medications on weight and metabolism.[57] Topiramate has shown efficacy in limiting weight gain associated with initiation of olanzapine in adult males with schizophrenia.[58] One case study further reported efficacy of topiramate in reducing antipsychotic-induced weight gain in a 14-year old female with major depressive disorder with psychotic features.[59] More recently, buproprion has been effective in reducing antipsychotic-induced weight gain in 7 subjects by an average of 3.4 kg.[60]

Metformin has also emerged as a safe and effective option in reducing body weight and improving fasting insulin levels and insulin resistance in adults treated with antipsychotics.[61] A placebo-controlled trial conducted in 39 adolescents, ages 10-17, examined weight, insulin sensitivity, and development of diabetes in children treated with olanzapine, risperidone, or quetiapine. Psychiatric diagnoses varied; bipolar disorder and attentional disorders were most common, while 12% of the children were diagnosed with either autism or Asperger's syndrome. Over the 16-week study, metformin was effective in stabilizing weight and decreasing insulin sensitivity, with overall good tolerability.[62]

Although psychopharmacological treatments have shown efficacy in reducing antipsychotic-induced weight gain, behavioral strategies remain important in weight loss and prevention of obesity-associated complications such as hypertension, hyperlipidemia, and diabetes mellitus. Despite the efficacy of metformin as an adjunct to reduce antipsychotic-induced weight gain, Wu et al.[63] found that metformin alone was inferior to metformin when used with a lifestyle intervention that incorporated dietary education and an exercise regimen. Cognitive behavioral therapy has also demonstrated efficacy in reducing weight and binge-eating behaviors in individuals treated with chronic antipsychotic therapy.[64] No such studies have been carried out in children or adults with ASD, and further investigation into

psychopharmacology combined with lifestyle intervention is needed to determine efficacy and feasibility of such treatments in this population.

In lieu of established treatments and preventative measures for antipsychotic-induced weight gain, the American Academy of Child and Adolescent Psychiatry (AACAP) has developed practice parameters that address some of the associated risk of antipsychotic use in children. These include: (1) obtaining a thorough review of current symptoms, past medical history, past medication trials, and establishing need for antipsychotic medication; (2) dosing atypical antipsychotics by the "start low and go slow" method; (3) obtaining baseline BMI and continuing to monitor at regular intervals; and 4) obtaining baseline and ongoing measurements of heart rate, blood pressure, fasting blood glucose, and fasting lipid profiles.[65] Children whose weight rises over the 90th percentile of BMI while on atypical antipsychotics should be referred for weight management and have more frequent lipid and blood glucose monitoring. The practice parameter includes complete guidelines and monitoring recommendations for specific antpsychotics.[65]

Mood Stabilizers

Mood stabilizers (e.g. divalproex/valproic acid, lithium, lamotrigine, levitracetam) are generally not supported for treatment in ASD, although they have shown efficacy in patients with mania and conduct disorders.[66] Consensus is that most mood stabilizers are associated with weight gain, and studies in bipolar disorder have identified divalproex as the likeliest culprit, with one double-blind, placebo-controlled trial of divalproex in ASD noting increased appetite as a significant side effect.[6768] On the other hand, lamotrigine-associated weight gain has been nominal in studies of children with bipolar disorder[69] and one RCT of 27 children with ASD found no significant differences in outcomes or adverse events between treatment groups.[70] Levitracetam, with one controlled, double-blind study of 20 children with ASD, also did not reveal significant weight gain, weight loss, or changes in appetite.[71] There are currently no open label studies or RCTs of lithium in treatment of ASD.

Serotonin Uptake Inhibitors

Serotonin is postulated to play a key role in aggressive drive; as such, several RCTs have examined the effectiveness of antidepressants on irritability and aggression, as well as repetitive and other maladaptive behaviors in ASD.[66] SRIs have shown variable association with weight disturbances.[72] Fluxoetine has been linked with mild anorexia and decreased appetite in individuals with ASD[73], while neither citalopram and escitalopram were associated with weight gain.[74,75] Fluvoxamine studied in adults with ASD revealed mild sedation, sertraline has not shown significant weight gain according a small study and several case studies, and no significant weight gain has been shown in the few studies conducted with venlafaxine.[76] On the other hand, mirtazapine, a noradrenergic and specific serotonergic antidepressant, contributed to increased appetite and significant weight gain (greater than 7% over baseline) in one study of 26 subjects, ages 3.8 to 23.5 (mean age 10.1 +/- 4.8 years), diagnosed with autism and other pervasive developmental disorders.[77] Clomipramine, a tricyclic antidepressant, was associated with fatigue and lethargy, but also decreased appetite, while one adult study showed weight gain in 13 out of 33 individuals

with ASD.[78] However, the latter was a poor quality study. Overall, there are few studies and limited evidence of weight gain associated with serotonin uptake inhibitors in individuals with ASD.

Other potential risk factors for obesity in children with ASD

Very little research has been done to determine the risk factors associated with obesity in children with ASD, which will require longitudinal prospective studies. Some of the putative risk factors that may be particularly relevant to children with ASD are described below.

Sleep problems

Epidemiologic evidence is mounting to suggest a link between short sleep duration and body weight.[79,80] Cross-sectional studies of children and adults have found a link between short sleep duration and overweight[81–84], as have prospective studies.[85–87] In children, several studies have documented that longer sleep duration is inversely related to overweight in children.[85,88,89] Long-term effects of short sleep duration during childhood on obesity risk in adulthood were also observed in a population-based prospective study in New Zealand.[86] Thus, in relation to obesity, the aspect of sleep that appears to be most important is sleep duration.[80]

Sleep problems are common in children with ASD[90,91] and are present at similar rates in those with and without co-occurring intellectual disability.[91–93] How sleep problems fluctuate with age is variably reported, but most investigators find that in contrast to TD children whose early childhood sleep problems often resolve, sleep problems in children with ASD tend to persist.[90,91] The specific sleep problems consistently identified include difficulty falling asleep and difficulty staying asleep.[94] Children with ASD, ages 5 to 16, studied by polysomnography were found to have shorter sleep times by an average of 43 minutes compared to TD children matched for age and sex.[95,96]

Direct and indirect pathways are proposed to explain the link between short sleep duration and excess weight based on experimental and observational studies. Experimental studies in rodents and humans demonstrate sleep deprivation effects on hyperphagia.[96] In a small study of young men[97], sleep restriction increased reports of hunger and appetite. Endocrine effects associated with sleep deficit, including elevated ghrelin and decreased leptin, which together increase hunger and stimulate appetite, were also observed. In the large Wisconsin Sleep Cohort, short sleep duration was associated with low leptin and high ghrelin levels.[98] Sleep problems could be expected to influence energy balance by decreasing activity or increasing energy intake in the context of daily family life. Children who are tired during the day may be less likely to engage in active play. On the food intake side, nighttime snacking may add to caloric intake.

The relationships among sleep disturbances, engagement in physical activity, eating behaviors, and weight status have not been fully elucidated in children with ASD. Given their notable propensity for sleep disturbances, however, research in this area seems warranted.

Food selectivity

It is frequently reported that children with ASD are highly selective eaters (often referred to as “picky eating”), with aversions to specific textures, colors, smells, temperatures, and brand names of foods. The diets of children with selective eating are characterized by a lack variety and may be associated with inadequate nutrient intake.[98–101] Although picky eating occurs in TD children, it appears to be more prevalent in children with ASD[102] and other developmental disabilities.[99] However, very few studies have examined the relationship between food selectivity and overweight.

Schreck et al.[103] examined food selectivity in 128 children with ASD and TD controls ages 5-12 years. They concluded that children with ASD had a significantly greater degree of food selectivity than TD children. Using the same data set in a subsequent analysis, Schreck et al.[98] reported that children with ASD preferred energy-dense foods within food groups (e.g., chicken nuggets, hot dogs, and peanut butter in the protein group; cake, french fries, macaroni, and pizza in the starch group; and ice cream in the dairy group). However, no measures of height and weight were available to examine the relationship of food selectivity to weight status.

In their cross-sectional study of 3-11 year old children with ASD, Bandini et al.[98] found that children with ASD refused more foods than TD peers and had more limited repertoires of foods. They also found that children with ASD ate significantly fewer fruits and vegetables than TD children and reported more daily servings of sugar-sweetened beverages (SSBs) than did TD children. However, only SSB intake in TD children was associated with BMI z-score. In a multivariate analysis, controlling for age, race, parental weight status, and education, the interaction between ASD and consumption of SSBs as a predictor of BMI z-score was not statistically significant, likely due to limited power.[13] In prospective observational studies of TD children, increased intake of SSBs[104–106] as well as decreased intake of fruits and vegetables[107] have been shown to be associated with obesity. Additional research is needed on whether intake of sugar-sweetened beverages increases risk of obesity in children with ASD.

Delayed/impaired motor development

Data from prospective observational studies suggest that increased participation in physical activity and decreased sedentary behavior are two protective factors in the development of obesity in children and adolescents.[108] Children with ASD may be particularly challenged to engage in physical activity by virtue of motor skill difficulties, which have been documented in toddlers[109] and school-age children[109] with ASD. Motor function impairments include unevenness of developmental milestone acquisition, low muscle tone, and postural instability.[110–113] These impairments may compromise children's endurance, balance, motor planning, and ability to participate successfully in motor-related activities. These difficulties may also result in exclusion from activities by peers and reduced motivation to participate in physical activities. The participation of children with ASD in physical activity may be compromised because of their social skill and communicative difficulties; Dziuk et al.[114] found that motor planning difficulties in children with ASD were strongly correlated with

the social, communicative, and behavioral impairments that define ASD. The need for close supervision may also hamper these children's participation.

The data on the extent to which children with ASD engage in physical activity are mixed. Rosser et al.[115] did not find differences in physical activity between children with ASD and TD children. In a cross-sectional study of 3-11 year old children with and without ASD, Bandini et al.[116] found that parents of children with ASD reported that their children participated in fewer types of activities and for less time than did TD children. However, similar to Rosser's findings, when physical activity levels were measured by accelerometry, there were no differences between the two groups. The authors speculated that questionnaires may not capture behaviors such as roaming and pacing that are frequently observed in children with ASD. Pan et al.[117], Macdonald et al.[118], and Memari et al.[119] have reported declines in physical activity with age in cross-sectional analyses in children and adolescents with ASD. The relationship between weight status and physical activity were not reported in these studies. Additional research is needed to determine whether physical activity levels are related to weight status in children with ASD and whether this relationship is influenced by age.

Increased time spent in sedentary behavior has been shown to increase the risk for obesity.[79] As noted, the social, behavioral, and/or intellectual impairments evidenced by children with ASD may make participation in formal and informal forms of physical activity more difficult, potentially increasing the amount of time spent in sedentary behavior. For example, parents of children with ASD report using television for its calming effect on their children and as a respite from caregiver challenges.[120] A recent study by Mazurek et al.[121] comparing screen time between 202 youth with ASD to 179 TD children found that both males and females with ASD (mean age 12.1) spent more time watching video games than did TD children (mean age 12.5). Television time did not differ between boys with ASD and TD boys, but was significantly higher among girls with ASD than TD boys. Further work is also needed to explore the relationship between screen time and weight status in this population.

Family functioning

Dietary, physical activity, and sleep patterns emerge within the context of the family environment; thus, family dynamics are central to any examination of childhood obesity risk factors. Research has documented that parental practices, mealtime routines, and parental feeding styles influence child feeding patterns.[122] A growing body of literature highlights the role that family stress, maternal depression, and family cohesion play in mediating or moderating the development of obesity in children. For example, obese adolescent girls have reported that their families are less cohesive, less expressive, and more authoritarian than non-obese youth.[123] Zeller et al.[124] reported that mothers of obese children ages 8-16 yrs., were more likely to report high levels of emotional distress, more family conflict, greater mealtime problems, and fewer positive family interactions during mealtimes than mothers of children who were not obese. Few disorders pose a greater threat to the psychosocial well-being of family members than ASD; the behaviors associated with ASD can be taxing for even the strongest of families.[125] Parents often report social isolation and difficulty

managing their children's temper tantrums, obsessions, and self-injurious behaviors. No research to date has examined the relation of family stress and obesity in children with ASD, but given that these factors have been shown to be associated with obesity in TD children, this line of research warrants future attention.

Other issues

There are myriad additional possibilities for unique risk factors for obesity in children with ASD that deserve research attention. For example, many children with ASD receive ABA treatment and food is often used as a primary reinforcer. Research is needed to determine whether using food as a reward, including the types and frequency of foods used, impact children's overall food intake and weight status. Additionally, the extent to which early feeding problems and gastrointestinal problems common to children with ASD are associated with obesity are also areas worthy of future investigation.

While addressing the problem of obesity in children with ASD has implications for their health and well-being, efforts to do so cannot be at the expense of the important social opportunities that eating with others confers. Mealtimes are a vehicle for conveying social values and expectations for children. Eating meals with others is a major way in which many cultures, including American culture, convey social relationships and socialization. In fact, it has been argued that “the act of eating is overtly social.”[126] Likewise, anthropologists have suggested that “food and eating are not just biologically significant for the reproduction of families and social groups, but are saturated with social import.”[127] Thus, efforts to address obesity in this population must ensure that the enjoyable and social dimensions of eating and eating with others are supported.

Discussion and Conclusions

The extant literature suggests that obesity is at least as high or higher in children with ASD than in children generally, and thus the comorbidities associated with obesity are a threat to the health and well-being of this population. The research to date is unequivocal in the risk that atypical antipsychotics present for obesity in both children and adults, but little research exists on how to address this problem. Although there is evidence to support psychopharmacological interventions in atypical antipsychotic-induced weight gain, research in children is sparse. Furthermore, given the increase in use of medications in children and concern for long-term consequences, more research is needed to determine safer alternatives to combat weight gain, such as lifestyle interventions. In the meantime, if the use of atypical antipsychotic medications is indicated, prescribers should provide anticipatory guidance to patients and families around the likelihood of weight gain and discuss strategies for managing hunger, increased appetite, and the child's engagement in physical activity. Families may also benefit from a referral to a registered dietician (RD) who can assist them in identifying preferred foods lower in caloric content.

Increasingly, community organizations are expressing an interest in and willingness to include children with ASD in recreational programming, which families should be supported and encouraged to pursue. Very recently the Office for Civil Rights in the US Department of Education[128] clarified obligations of schools to provide students with disabilities an equal

opportunity to participate alongside their peers in after-school athletics and clubs. This is good news for children with ASD and other disabilities, though additional research and professional training will be needed to understand how children with ASD can be engaged successfully in physical activity and extramural sports. Involvement in physical activity may also improve children's sleep, which may bring about improved behavioral control as well as address one of the risk factors believed to be associated with obesity.

As noted, research suggests that children with ASD have higher levels of food selectivity and that their diets may be characterized by calorically dense foods low in nutrient content. Whether these eating patterns contribute to the development of obesity is a topic worthy of future research. Children with ASD, whose eating patterns are selective for foods such as sugar sweetened beverages, cookies/cakes, and savory snacks, may benefit from the input of an RD, an occupational therapist, and a behavioral psychologist. An RD can assess the child's eating patterns and assist with devising a nutritionally sound eating plan. Because sensory issues are so common in children with ASD and may be an underlying factor in selective eating patterns, an occupational therapist may be able to assess sensory processing difficulties and suggest sensory desensitization strategies to help the child to accept new foods. Likewise, it is likely that the eating patterns seen in children with ASD are associated with core features of autism itself, including the rigidity and insistence on sameness. Behavioral approaches that are considered the gold standard treatments for autism, such as applied behavior analysis (ABA), may prove useful in increasing children's eating repertoires and acceptance of a healthy diet.

In sum, prevalence of obesity in children with ASD is at least as high, if not higher, than in the general population of children. Research has documented that atypical antipsychotic medication is a clear risk for weight gain in this population. Studies on diet, physical activity, and sleep in TD children have shown positive associations with obesity, but this work remains to be done in children with ASD, along with other investigations on the implications of GI disorders and using food as reward for learning and behavior management. Obesity and its associated sequelae represent significant threats to independent living, self-care, quality of life, and long-term health outcomes for individuals with ASD. Information about the unique and specific risk factors for development of obesity in children with ASD is needed so that obesity prevention and intervention strategies may be developed that are both appropriate and effective for this population.

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