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Overweight and Obesity Among Children and Adolescents with Fetal Alcohol Spectrum Disorders

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Abstract

Background—Because prenatal alcohol exposure is associated with growth deficiency, little attention has been paid to the potential for overweight and obesity in children with fetal alcohol spectrum disorders (FASD). This study examined the prevalence of overweight/obesity (body mass index [BMI]) in a large clinical sample of children with FASD.

Methods—Children, aged 2 to 19 years, who were evaluated for FASD at University Clinics, included 445 with an FASD diagnosis and 171 with No-FASD diagnosis. Prevalence of overweight/obesity (BMI ≥85 percentile) was compared to national and state prevalence. BMI was examined in relation to FASD diagnosis, gender, and age. Dietary intake data were examined for a young subsample (n = 42).

Results—Thirty-four percent with any FASD diagnosis were overweight or obese, which did not differ from the No-FASD group or U.S. prevalence. Underweight was prevalent in those with fetal alcohol syndrome (FAS) (17%). However, increased rates of overweight/obesity were seen in those with partial FAS (40%). Among adolescents, those with any FASD diagnosis had increased overweight/obesity (42%), particularly among females (50%). The rate in adolescent females with FASD (50%) was nearly 3 times higher than state prevalence for adolescent females (17 to 18%), $p < 0.001$. In the young subsample, those who were overweight/obese consumed more calories, protein, and total fat per day than those who were not overweight or obese.

Conclusions—Rates of overweight/obesity are increased in children with partial FAS. In adolescents, rates are increased for any FASD diagnosis (particularly in females). Results are suggestive of possible metabolic/endocrine disruption in FASD—a hypothesis for which there is evidence from animal models. These data suggest that clinicians may consider prenatal alcohol exposure as a risk factor for metabolic/endocrine disruption, should evaluate diet as a risk in this

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population, and may need to target interventions to females prior to puberty to effect changes in overweight-related outcomes.

Keywords

Body Mass Index; Nutrition; Obesity; Fetal Alcohol Spectrum Disorders

Growth deficiency is a defining feature of fetal alcohol syndrome (FAS) along with dysmorphic facial features and central nervous system dysfunction (Hoyme et al., 2005; Jones and Smith, 1973). Many prenatally exposed individuals do not display physical characteristics of FAS, but have neurocognitive impairment and/or structural brain anomalies (Bookstein et al., 2001; Burden et al., 2005; Mattson et al., 1998; Sowell et al., 2001). Fetal alcohol spectrum disorders (FASD) cover a range of disability and include individuals who fit all or part of the defining profile (e.g., neurocognitive impairments without growth deficiency) (Riley and McGee, 2005; Warren et al., 2004). There is wide phenotypic variation in early growth in FASD, ranging from deficient to normal growth. Despite studies demonstrating associations between overweight and prenatal exposure to other drugs including cocaine (LaGasse et al., 2011) and tobacco (Hill et al., 2005), very little is known about the prevalence of overweight and obesity in FASD over the long term.

In the United States, nearly one-third of children between the ages 2 and 19 are overweight or obese (Ogden et al., 2012). Although growth deficiency is a long-term, persistent characteristic in FAS (the most severe form of FASD), those with less-severe FASD diagnoses (e.g., partial FAS, alcohol-related neurodevelopmental disorder [ARND]) may be at increased risk of overweight compared to the population in the long term (Werts et al., 2014). Individuals with partial FAS have been observed to have higher body mass indexes (BMIs) than those with FAS (Klug et al., 2003). Additionally, growth deficiency normalizes over time for some individuals with FASD, and weight percentiles increase throughout childhood and adolescence, particularly for those with partial FAS (Klug et al., 2003) and for females (Spohr et al., 2007). In those studies, the observed weight increases could have reflected a normalization of growth deficiency, with BMIs ultimately falling in the healthy weight range, or the weight gain may have been excessive, with individuals developing overweight or obesity. The goal of the current study was to specifically determine the prevalence of overweight and obesity in a large sample of children and adolescents with FASD. Identifying this prevalence has important clinical implications because this population has generally been monitored and treated early on for growth deficiency (underweight and short stature), but not considered at risk for long-term overweigh/obesity.

A secondary aim was to identify characteristics of individuals with FASD who are overweight or obese. Understanding these factors will assist clinicians in effectively monitoring and promoting healthy growth in this generally high-risk population. We examined age, gender, and FASD diagnosis. These characteristics could potentially help identify individuals who might benefit the most from weight-management interventions, for example. These data might also identify specific developmental periods when risk is highest and/or when specific interventions may have the most benefit. Last, we examined dietary intake in a subsample of young children to evaluate whether caloric intake was associated

with overweight and obesity to provide data about potential early signs of metabolic, endocrine, and/or eating disturbance in this population.

MATERIALS AND METHODS

Participants

Participants included children, aged 2 to 19 years, who were clinically evaluated for FASD at the University of Minnesota FASD Clinic or International Adoption Clinic between April 2005 and April 2013 (Table 1). All participants provided written consent for the use of their deidentified data when registering for clinic. Children were seen by a psychologist for a neuropsychological evaluation and by a pediatrician or geneticist, both with formal training in the University of Washington diagnostic system for FASD (Astley, 2004). Only children with complete height and weight data were included. Diagnoses were made according to modified Institute of Medicine (IOM) criteria (Hoyme et al., 2005). Because IOM criteria do not quantitate cognitive/behavioral deficits for diagnosis, our clinics define deficits as more than 1.5 standard deviations below the mean for age in 3 or more domains on standardized tests (e.g., intellectual, language, motor, visual-perceptual, adaptive, and behavioral). Although IOM criteria do not require confirmed alcohol exposure for a partial FAS diagnosis, our clinics use a conservative criterion and require confirmed exposure for partial FAS.

The final sample included 445 participants with an FASD diagnosis: FAS (n = 64), partial FAS (n = 165), ARND (n = 216). We also included a group who were evaluated in the clinics, but did not receive an FASD diagnosis (a No-FASD group; n = 171) as a contrast to the FASD group. Rather than including a traditional “healthy” control group, the No-FASD group was included to better control for potential confounding factors (e.g., the clinically referred No-FASD group shares many characteristics with the FASD group, including comorbid psychiatric conditions, race, socioeconomic status (SES), and family characteristics, among others). Participants with partial FAS were, on average, slightly older than those with FAS (p = 0.008). Those with partial FAS or ARND were slightly older than the No-FASD group (p < 0.001, p = 0.040, respectively; Table 1).

Dietary intake data were available for a subsample of the FASD participants between the ages 2 and 5 who were enrolled in a 9-month study of choline supplementation (n = 42) (Wozniak et al., 2013). All procedures were approved by a University IRB and the parents of participants in the substudy underwent an informed consent procedure.

BMI

Height and weight measurements, collected by a pediatrician in clinic, were used to calculate BMI. For the subsample, the measurements collected by a pediatrician at study enrollment were used. BMI-for-age percentiles, specific for each gender, were calculated using the Centers for Disease Control and Prevention (CDC) 2000 growth charts (Kuczmarski et al., 2000). BMI categories were assigned as follows: underweight (<5th percentile), healthy weight (5th to <85th percentile), overweight (85th to <95th percentile), and obese (≥95th percentile) (Barlow and the Expert Committee, 2007). The overweight/

obese category included those in either the overweight or obese category (BMI ≥ 85th percentile).

Dietary Intake

Dietary intake data for the subsample were collected 3 times: at study enrollment, 6 months, and 9 months, through parent interviews using the Automated Self-Administered 24-hour Recall (ASA24) (National Cancer Institute, 2009). The ASA 24 interviews were carried out in the laboratory and were administered by a research assistant. Height and weight measurements were collected on the same day as the baseline dietary recall. A portion of the macronutrient data has been previously published (Fuglestad et al., 2013). In the current study, additional participants' data are included and the data are analyzed in the context of BMI rather than age. Details on the dietary recall methods are described elsewhere (Fuglestad et al., 2013). Primary measures included dietary intake of energy (kilocalories) and macronutrients. Mean values for energy intake and each macronutrient were generated from all available interview points to create an observed daily nutrient intake. Most participants (n = 25) had 3 data points. For others, means were from 2 dietary recalls (n = 10) or a single dietary recall (n = 7).

Independent t-tests were used to determine whether macronutrient intake and/or total energy intake was greater among those who were overweight/obese and those who were not overweight. Observed nutrient intakes were compared using 1-sample t-tests to national dietary intake data from 24-hour dietary recalls of children aged 2 to 5 years (What We Eat in America, National Health and Nutritional Examination Survey [NHANES] 2007–2008 [U.S. Department of Agriculture—Agricultural Research Service, 2010]) to determine whether intake in children with FASD differs from same-age children in the United States.

RESULTS

BMI

Thirty-four percent of those with FASD were either overweight (18%) or obese (16%) and 7% were underweight (Table 2). There was more underweight and less overweight/obesity in those with FAS compared to those with either partial FAS, $\chi^2(3) = 16.91$, $p = 0.001$, or those with ARND, $\chi^2(3) = 18.94$, $p < 0.001$. A statistical trend, $\chi^2(3) = 7.51$, $p = 0.057$, suggested a difference in BMI categories between the younger age group (ages 2 to <5 years) and the older age group (ages 13 to 19 years), with more overweight and obesity in the older group (Table 2). When the overweight and obese categories were combined, this difference by age group was significant. The rate of overweight/obesity was significantly higher in the older group (42%) than the younger group (28%), $\chi^2(1) = 4.40$, $p = 0.036$. The rate of underweight was not significantly different between the 2 age groups, $\chi^2(1) = 2.48$, $p = 0.115$.

There were no differences in BMI between the FASD group and the No-FASD group. However, those with FAS had a higher rate of underweight (17%) compared to the No-FASD group (5%), $\chi^2(1) = 9.81$, $p = 0.002$, and a lower rate of overweight/obesity (14%) than the No-FASD group (30%), $\chi^2(1) = 6.34$, $p = 0.012$. There was a statistical trend

suggesting more overweight/obesity in those with partial FAS (40%) compared to the No-FASD group (30%), $\chi^2(1) = 3.62$, $p = 0.057$.

The rate of overweight/obesity in the FASD group was compared both to the overall prevalence in the United States (Ogden et al., 2012) and to the prevalence in Minnesota (Division of Nutrition Physical Activity and Obesity, 2012), the home-state of the FASD sample. State prevalence rates were used in addition to national data because they reflect regional trends and provide a more accurate comparison. State prevalence rates were also used for sex and age comparisons for this same reason.

There was no difference in the rate of overweight/obesity in the whole FASD group (34%) compared to national data for those between the ages of 2 and 19 years (31.7%), $\chi^2(1) = 1.71$, $p = 0.191$. However, compared to the rate of overweight/obesity in the national data (31.7%), the rate was lower in those with FAS (14%), $\chi^2(1) = 9.02$, $p = 0.003$, but higher in partial FAS (40%), $\chi^2(1) = 5.53$, $p = 0.019$. There was no difference between those with ARND (36%) and the national data, $\chi^2(1) = 2.13$, $p = 0.145$.

The base rates of overweight/obesity in Minnesota children are available for 2 age ranges: preschoolers (2- to <5-year-olds) and adolescents (9th graders and 12th graders) (Division of Nutrition Physical Activity and Obesity, 2012). For the preschool group, there was no difference in the rate of overweight/obesity between the FASD group (28%) and Minnesota children (29%), $\chi^2(1) = 0.03$, $p = 0.862$. For the Minnesota adolescent group, the base rates were very similar for 9th and 12th graders (only 1 percentage point difference), so the rates were averaged. In Minnesota, the rates of overweight/obesity differ between males (26 to 27%) and females (17 to 18%), and therefore, data were analyzed separately. The rate of overweight/obesity in adolescent males with any FASD (37%) was higher than the rate for adolescent males in Minnesota (26 to 27%), $\chi^2(1) = 7.00$, $p = 0.008$. The rate of overweight/obesity in adolescent females with any FASD (50%) was also higher than the rate for adolescent females in Minnesota (17 to 18%), $\chi^2(1) = 48.29$, $p < 0.001$.

Dietary Intake

Of the 42 participants in the subsample with dietary intake data, 26%were either overweight (14%) or obese (12%), and 2% were underweight (Table 3), similar to the overall clinic sample. BMI was not associated with gender or age. There was a trend for different rates of overweight/obesity among the diagnostic categories, $\chi^2(2) = 5.63$, $p = 0.060$. Seventy-three percent of those in the subsample who were overweight/obese were in the partial FAS category, and no child with full FAS in the subsample had overweight/obesity. In the subsample, the overweight/obese children had higher intakes of most macronutrients than the nonoverweight children. The macronutrient intakes of the overweight/obese children were more similar than the nonoverweight children to the NHANES data (U.S. Department of Agriculture—Agricultural Research Service, 2010). Although the total dietary intakes differed between the nonoverweight and the overweight/obese children, the distribution of macronutrients (% of energy in the diet from each macronutrient) did not differ between the groups and was within the acceptable macronutrient distribution range (AMDR). The AMDR is the range of intake of an energy source that provides intakes of essential nutrients

and is associated with a reduced risk of chronic disease (Food and Nutrition Board, Institute of Medicine, 2005).

DISCUSSION

The primary goal of this study was to report on the rates of overweight/obesity in a large sample of children and adolescents diagnosed with FASD, because there are almost no available data in the literature other than a few small studies which did, in fact, show increased overweight in the population. A secondary goal was to better characterize individuals with FASD who are overweight or obese to provide a context for clinicians that may ultimately help with the promotion of healthy growth in these children and adolescents.

The data indicate that there were differences in weight status based on FASD diagnosis. The highest prevalence of overweight and obesity was in those with partial FAS, with 40% either overweight or obese. The lowest prevalence of overweight was in those with FAS, with only 14% overweight or obese. Underweight was most prevalent among those with an FAS diagnosis, with at least 1 in 6 being underweight. These results are consistent with a prior study showing that, among individuals with FASD, those with partial FAS have the highest BMI and those with FAS have the lowest (Klug et al., 2003). The data also agree with a recent study showing increased BMIs in children with prenatal alcohol exposure, especially in females (Werts et al., 2014).

A partial explanation for the observed diagnostic group differences in BMI is that growth deficiency is one of the criteria used to determine the FASD diagnosis. The fact that the lowest rate of overweight/obesity occurred in the FAS group is partly because growth deficiency is a required feature for an FAS diagnosis. However, it is important to note that underweight itself is not required for growth deficiency; rather, the criterion of growth deficiency can be met by either height or weight that is at or below the tenth percentile for age.

In this sample, rates of overweight/obesity in FASD were most increased in adolescents compared to younger children. Those who are aged between 13 and 19 years were 1.5 times more likely to be overweight or obese than those under the age of 5. The disproportionate rate of overweight/obesity rate in adolescents with FASD compared to preschoolers was expected given that the same trend is found in normal populations (Ogden et al., 2012). However, the magnitude of the difference in rates between adolescents and preschoolers with FASD was twice that seen in the U.S. population. In the FASD sample, the rate of overweight/obesity was 28% for those ages 2 to <5 years and 42% for adolescents (14% higher in adolescence). In contrast, in the U.S. population, there is only a 7% discrepancy between preschoolers and adolescents (Ogden et al., 2012). These findings were reinforced when examining Minnesota data, with the FASD group showing normal rates of overweight/obesity for preschoolers and increased rates of overweight/obesity for adolescents.

Although the prevalence of overweight/obesity was higher for both adolescent males and females with FASD compared to Minnesota as a whole, the rate was particularly high for adolescent females. Thirty-seven percent of adolescent males with FASD were overweight/

obese, and these adolescent boys were 1.4 times more likely to be overweight/obese than their peers in Minnesota. More strikingly, 50%of adolescent females with FASD were overweight/obese, and these adolescent females were nearly 3 times more likely to be overweight/obese than peers in Minnesota. The magnitude of these increased rates is particularly striking because a history of growth deficiency (height, weight, or both) is a core feature of FASD. These age and gender effects are consistent with previous studies in individuals with prenatal alcohol exposure showing that weight percentiles increase throughout childhood and adolescence (Klug et al., 2003; Spohr et al., 2007) and that males have more persistent growth deficiency whereas females have greater postpubertal weight gains (Spohr et al., 2007; Werts et al., 2014). Greater adiposity in females compared to males has also been observed in animal models of prenatal ethanol exposure (Dobson et al., 2012).

In young children with FASD, there were dietary intake differences between those who were overweight or obese and those who were not. Specifically, those who were overweight were consuming on average more calories, protein, and total fat per day. However, the diets of those who were overweight were very similar to the typical diets of preschool-age children in the United States (NHANES [U.S. Department of Agriculture—Agricultural Research Service, 2010]). We did not collect data on activity level or medication use and therefore could not determine whether children with FASD were in energy balance. In future studies, a more comprehensive evaluation of energy balance would be informative (such as the estimated energy requirements that are based on age, gender, physical activity level, and BMI status; Food and Nutrition Board, Institute of Medicine, 2005).

Obesity is the result of a complex interaction between genetic, environmental, social, economic, endocrine, nutritional, and behavioral factors (Must et al., 2009). FASD could be associated with obesity via any of these factors or interactions. Animal models suggest possible mechanisms, including prenatal alcohol-related alterations in metabolic and endocrine function that lead to increased adiposity in the adult offspring (Chen and Nyomba, 2003; Dobson et al., 2012). Alternatively, Chen and Nyomba (2003) demonstrated that prenatal alcohol exposure in rats impaired glucose homeostasis and insulin resistance, which was exacerbated by a high-fat diet, and that animals exposed prenatally to alcohol and postnatally to a high-fat diet also tended to store more energy, independently of appetite. Similarly, increased adiposity was observed in a guinea pig model of prenatal alcohol exposure (Dobson et al., 2012) in a study that noted that a pattern of intrauterine growth restriction followed by “catch-up” growth has been associated with metabolic syndrome (Bertram and Hanson, 2001), a process that may play an important role in overweight and obesity in humans with FASD. Clearly, a number of large comprehensive studies will be needed to fully understand the range of individual and interactive effects.

There are several limitations to acknowledge with regard to this study. Weight status in the United States is associated with race (Ogden et al., 2012); however, we were not able to examine the association between race and overweight/obesity due to lack of individual racial data because these data are not collected in the clinic. In the U.S. population, obesity rates are lowest among non-Hispanic Caucasian children compared to other groups. Although approximately two-thirds of the patients seen at the University FASD clinic are non-

Hispanic and Caucasian, a high percentage (29%) are Native American because of established referral patterns. In the United States, American Indians and Alaska Natives make up <2% of the population (U.S. Census Bureau: State and County QuickFacts, 2013). Estimates for overweight/obesity in American Indian or Alaskan Native children are as high as 46 to 48% (Zephier et al., 2006). Thus, the probable over representation of Native Americans in our sample may have increased the overall rate of overweight and obesity. Although there is no published evidence to suggest a specific relationship between Native American heritage and specific FASD diagnosis among those referred to diagnostic clinics, it is theoretically possible that a higher proportion of Native American individuals specifically in the partial FAS group could have inflated the observed rates of overweight and obesity. A second related limitation to the current study is that psychotropic drugs can have effects on weight status (Dent et al., 2012), and we were not able to examine the association between psychotropic medications and weight status in this study.

Another potential caveat relates to the possible influence of nonalcohol factors that are known to be related to obesity, such as SES. Although full SES data were not available for the clinical sample, the majority of patients evaluated for FASD in the University Clinics are living in adopted homes or in foster care (only 14% of children and adolescents with FASD live with a biological parent). The average SES of the families therefore is higher than the SES of the biological families and is similar to the SES of the general Minnesota population. The clinically referred No-FASD group was included as a contrast to address these potential confounding factors. It is also worth noting that the primary goal of this study was not to establish a causal link between prenatal alcohol exposure and obesity, but rather to determine whether overweight and obesity are prevalent and need to be monitored clinically in children and adolescents with FASD, a population for whom growth deficiency (e.g., underweight) has historically been the primary clinical concern growth.

An additional limitation to acknowledge relates to the fact that the sample was a clinically referred one. As such, the sample is not entirely representative of the whole population of individuals with FASD. Clinical samples, in general, are more affected than nonreferred individuals living in the community. In this case, we expect that our clinically derived participants likely have more behavioral, learning, and cognitive difficulties than the larger population affected by FASD. Although there is no specific evidence to suggest that overweight and obesity are necessarily over represented in clinically derived samples of those with FASD, it is a possibility.

Despite the limitations discussed, these data have important clinical implications. In a population in which growth deficiency is typically the main concern, we observed a high rate of overweight and obesity by the time these individuals reach adolescence. The prevalence of overweight was increased among adolescents with FASD compared to the general population, and there appear to be particularly high rates in female adolescents with FASD as well as individuals with partial FAS. These data, perhaps, suggest that preadolescence may represent a window of opportunity for interventions designed to prevent obesity. The data also suggest that females, particularly those with partial FAS, may warrant specific attention from prevention efforts. Although we did not observe increased rates of overweight/obesity in children under the age of 5 with FASD, those who were overweight or

obese were consuming more calories, protein, and total fat per day than their nonoverweight peers, suggesting another possible target for early identification and intervention. Because as many as 80% of obese children become obese adults (Wyllie, 2005), identification of individuals at increased risk of overweight or obesity is essential for the implementation of effective prevention efforts aimed at promoting healthy weight.

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Table 1

Sample Characteristics

%or mean (SD)	FAS *n* = 64	Partial FAS *n* = 165	ARND *n* = 216	No-FASD diagnosis *n* = 171	Statistical test
Full sample					
Age (years)	9.5 (4.9)	11.5 (4.8)	10.5 (5.0)	9.5 (5.0)	$F(3, 612) = 5.07$, $p = 0.002$
IQ	81 (16)	81 (12)	85 (15)	88 (15)	$F(3, 439) = 5.93$, $p = 0.001$
Gender					
Male	64%	57%	61%	55%	$\chi^2(3) = 2.26$, $p = 0.520$
Female	36%	43%	39%	45%	
Subsample	*n* = 8	*n* = 19	*n* = 15	–	
Age (years)	3.7 (0.9)	4.3 (0.6)	3.9 (0.6)	–	$F(2, 39) = 1.88$, $p = 0.166$
IQ	81 (19)	78 (17)	93 (19)		$F(2, 38) = 2.73$, $p = 0.078$
Gender					
Male	38%	26%	27%	–	$\chi^2(2) = 0.39$, $p = 0.824$
Female	63%	74%	73%	–	
Racial category					
American Indian/Alaska Native	12.5%	11%	33%	–	$\chi^2(8) = 8.16$, $p = 0.418$
Asian	0%	5%	0%	–	
Black or African American	12.5%	21%	27%	–	
White	75%	47%	27%	–	
More than 1 race	0%	16%	13%	–	
Ethnic category					
Hispanic or Latino	25%	0%	0%	–	$\chi^2(2) = 8.67$, $p = 0.013$
Non-Hispanic or Latino	75%	100%	100%	–	

ARND, alcohol-related neurodevelopmental disorder; FAS, fetal alcohol syndrome; FASD, fetal alcohol spectrum disorder.

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Table 2

BMI[a] by FASD Diagnosis, Gender, and Age

	BMI category						
	Underweight	**Healthy weight**	**Overweight**	**Obese**	**BMI category statistical test**	**BMI percentile *M* (SD)**	**BMI percentile statistical test**
FASD diagnostic category[b]							
FAS (*n* = 64)	17%	69%	6%	8%	$\chi^2(6) = 22.59$, $p = 0.001$	37.8 (32.1)	$F(2, 442) = 24.48$, $p < 0.001$
Partial FAS (*n* = 165)	7%	53%	22%	18%		66.0 (30.7)	
ARND (*n* = 216)	5%	59%	19%	17%		66.7 (29.5)	
Gender[b]							
Male (*n* = 266)	7%	58%	18%	17%	$\chi^2(3) = 0.60$, $p = 0.895$	62.2 (32.2)	$t(443) = -0.12$, $p = 0.901$
Female (*n* = 179)	8%	59%	19%	15%		62.5 (31.5)	
Age group[b]							
2 to <5 years (*n* = 78)	10%	62%	10%	18%	$\chi^2(3) = 7.51$, $p = 0.057$	57.8 (33.1)	$t(257) = -1.95$, $p = 0.052$
13 to 19 years (*n* = 181)	5%	53%	23%	19%		66.2 (31.1)	
Gender and age[b]							
Age group: 2 to <5 years							
Male (*n* = 44)	13%	57%	7%	23%	$\chi^2(3) = 3.94$, $p = 0.268$	58.9 (34.1)	$t(76) = 0.34$, $p = 0.736$
Female (*n* = 34)	6%	67%	15%	12%		56.3 (32.1)	
Age group: 13 to 19 years							
Male (*n* = 115)	6%	57%	20%	17%	$\chi^2(3) = 3.11$, $p = 0.376$	62.9 (32.2)	$t(179) = -1.88$, $p = 0.062$
Female (*n* = 66)	3%	47%	27%	23%		71.8 (28.5)	
FASD							
Any FASD diagnosis (*n* = 445)	7%	58%	18%	16%	$\chi^2(3) = 3.10$, $p = 0.377$	62.3 (31.9)	$t(614) = 0.03$, $p = 0.974$
No-FASD diagnosis (*n* = 171)	5%	66%	15%	15%		62.4 (29.1)	

ARND, alcohol-related neurodevelopmental disorder; BMI, body mass index; FAS, fetal alcohol syndrome; FASD, fetal alcohol spectrum disorder.

[a] BMI percentile for age, CDC 2000 growth charts (Kuczmarski et al., 2000).

[b] Only includes those with an FASD diagnosis.

Table 3

Macronutrient Intake from Food in Children with FASD by weight Category

	Dietary reference intakes[a] 1 to 3 years; 4 to 8 years	NHANES[b] (2 to 5 years) mean	Observed nutrient intake		*t*-Tests (*p*-value)		
			Nonoverweight *n* = 31 mean (SD)	Overweight/ obese *n* = 11 mean (SD)	Nonoverweight vs. overweight/obese	Nonoverweight vs. NHANES	Overweight/obese vs. NHANES
Total Energy (kcal/d)	–	1,523	1,344 (347)	1,694 (508)	0.015	0.007	0.290
Protein (g/d)	13; 19	54	49 (15)	65 (18)	0.006	0.066	0.068
Carbohydrate (g/d)	130; 130	207	186 (55)	228 (79)	0.063	0.046	0.401
Total fat (g/d)	–	56	47 (15)	61 (18.3)	0.016	0.004	0.327
Protein (%energy)	5 to 20; 10 to 30[c]	–	14.5 (3.0)	15.7 (2.5)	0.272	–	–
Carbohydrate (%energy)	45 to 65; 45 to 65[c]	–	55.7 (7.7)	53.1 (5.9)	0.309	–	–
Total fat (%energy)	30 to 40; 25 to 35[c]	–	31.6 (6.4)	33.2 (4.9)	0.457	–	–

[a] Food and Nutrition Board, Institute of Medicine (2005).

[b] Data from *What We Eat in America*, NHANES 2007–2008 (U.S. Department of Agriculture --- Agricultural Research Service, 2010).

[c] Acceptable Macronutrient Distribution Range (AMDR).