Published in final edited form as:

J Pediatr. 2016 February ; 169: 194–200.e1. doi:10.1016/j.jpeds.2015.10.049.

Abnormal Eating Behaviors are Common in Children with Fetal Alcohol Spectrum Disorders

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Abstract

Objective—To compare the eating behaviors and nutrition-related concerns in children with fetal alcohol spectrum disorders (FASD) with those in typically-developing children.

Study design—A survey that assessed eating behaviors was completed between October 2013 and May 2014 by the caregivers of children screened for FASD at the University of Minnesota's Fetal Alcohol Spectrum Disorders Program, and typically-developing children recruited from that clinic or from the Research Participation Core of the Waisman Center, University of Wisconsin.

Results—Compared with controls (N=81), children with FASD (N=74) had delayed acquisition of self-feeding behavior ($P<0.001$) and solid food introduction ($P<0.001$). Impaired satiety was common and independent of medication use: 23.0% were never full/satisfied, 31.1% snacked constantly, and 27.0% concealed food (all $P=0.002$). They consumed the equivalent of an additional meal/snack daily ($P<0.01$). Children with FASD were more likely to have a past diagnosis of underweight ($P<0.001$). Mean body mass index was significantly reduced for males ($P=0.009$) but not females ($P=0.775$) with FASD, and only two children with FASD were currently underweight. Children with FASD were more physically active ($P<0.01$).

Conclusion—Abnormal eating patterns are common in children with FASD and may contribute to their delayed growth and nutritional inadequacies. Their poor satiety may reflect poor impulse control. Children with FASD may benefit from diet counseling. Conversely, some hyperphagic children may warrant referral for FASD screening.

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The authors declare no conflicts of interest.

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Keywords

Fetal alcohol spectrum disorder; abnormal eating; ingestive behaviors; pediatric obesity; nutrition

Prenatal alcohol exposure is a leading cause of neurodevelopmental disability. The most severe manifestations of prenatal alcohol exposure, diagnoses within the fetal alcohol spectrum disorders (FASD), affect 2.4% to 4.6% of school-age children (1). Children with FASD share some growth characteristics with those exposed prenatally to other drugs and tobacco. In infancy and early childhood FASD strongly associates with below-normal weight, height, and head circumference (4, 5). These differences lessen in later childhood (5, 6), and by periadolescence both reduced (7-9) and increased weight gain (10-12) is reported compared with unexposed controls. These discrepancies may reflect social and environmental factors (7, 13). When growth quality is evaluated, periadolescents with FASD may accrue adiposity rather than lean mass, and may have a greater likelihood of having body mass indexes (BMI) 85th percentile, especially for females and for cohorts consuming western-style diets (12, 14-17).

Infants with FASD have reduced suckle that delays transition to solid foods (18). Older children exhibit dysfunctional feeding behaviors including constant snacking, poor satiety, and picky eating/poor appetite (19). In otherwise well-nourished populations, nutrient intakes were below recommendations for several vitamins, minerals, and essential fatty acids, and sugar, fat, and caloric consumption was excessive (17, 19). This suggests a pattern of abnormal feeding behaviors and food consumption that is specific to children with FASD.

Our previous report of eating behaviors (17) only evaluated children with prenatal alcohol exposure or suspected prenatal alcohol exposure. Here we extend that work and compare those behaviors in children with FASD against those of typically-developing children.

Methods

The study population consisted of two groups children with FASD (n=74) and a control group not exposed to alcohol (n=81). None of the children were studied previously with respect to their eating behaviors. Participants in the FASD group were recruited through the University of Minnesota's Fetal Alcohol Spectrum Disorders (UMN FASD) Program. The diagnostic categorizations of those with FASD are described in Table I. Participants were enrolled after an initial telephone screening to determine eligibility. The FASD group was diagnosed according to modified Institute of Medicine (IOM) criteria (21), which considered their growth, facial dysmorphology, and alcohol exposure. Because IOM criteria do not specifically characterize cognitive functioning, we further applied Centers for Disease Control and Prevention (CDC) central nervous system (CNS) criteria for FASD (22) (see 20 for details). Specifically, in order to meet our criteria for Alcohol Related Neurodevelopmental Disorder (ARND), participants had to meet the Hoyme criteria for ARND, plus have deficits of at least one standard deviation in three domains of cognitive function. Exclusion criteria were the presence of a non-FASD developmental disorder (i.e. autism, Down syndrome), neurological disorder, brain injury, or other medical condition

affecting the brain, or very low birthweight (<1500 grams). Hoyme criteria allow for diagnosis of both fetal alcohol syndrome (FAS) and partial fetal alcohol syndrome (pFAS) without confirmed alcohol exposure. Sixty-one children with FASD had confirmed prenatal alcohol exposure and 7 had unconfirmed prenatal alcohol exposure but were included because they met the Hoyme criteria for FAS or pFAS. For an additional six individuals, FASD was strongly suspected but a diagnosis could not be made: two had confirmed heavy prenatal alcohol exposure and at least one facial feature, three had suspected prenatal alcohol exposure and one facial feature with cognitive impairment, and one had suspected prenatal alcohol exposure and some cognitive impairment. Of those in the FASD group, 24 had participated in a 9-month study of choline supplementation (20).

The 81 control participants included 53 typically developing children who were recruited through the UMN FASD Program and 28 typically-developing siblings of children with developmental disabilities who were recruited from a registry of the Research Participation Core of the Waisman Center, University of Wisconsin-Madison. None of the 81 control participants had prenatal alcohol exposure or an FASD diagnosis. Protocols were reviewed and approved by the IRBs at both institutions, and written informed parent consent was obtained prior to study participation.

All the UMN-recruited FASD and control children received a physical examination by a physician at an in-person visit at the University of Minnesota. Height and weight of each participant was measured and recorded by a physician on equipment maintained by the University's Center for Neurobehavioral Development or Clinical and Translational Science Institute. Age- and sex-adjusted Body Mass Index (BMI) percentile was calculated for 48 children in the control groups and 72 alcohol-exposed children from growth charts provided by the Centers for Disease Control and Prevention (23). Following their guidelines, overweight was defined as BMI 85th percentile, obesity as 95th percentile, and underweight as 5th percentile. The BMIs for 24 children with FASD enrolled in the choline trial (20) were reported previously (12).

A six-page questionnaire with a mix of dichotomous questions, open-ended questions, and structured multiple choice questions was mailed to and completed by the child's primary caregiver. The questionnaire was developed by a pediatric Registered Dietitian (S.C.V.) specializing in developmental disabilities and was modified from (17) to further interrogate areas of concern identified therein. Questions addressed the child's current eating behaviors, eating frequency, eating environment, and physical activity, and past or present medical history and nutrition-related concerns.

Data Analyses

Data were analyzed either using Sigmaplot 12.3 (Systat Software, San Jose, CA) and SAS statistical software (v.9.4; SAS Institute Inc, Cary, NC, USA) with an alpha level of 0.05 for significance. Group comparisons were made with the χ^2 test, independent *t*-test, or Mann-Whitney *U*-test where appropriate. We also performed Kruskal-Wallis one-way analysis of variance to examine confounding influences of race, sex, choline intervention, eating behavior, and BMI, upon outcomes. For responses involving a range (i.e. 1 to 2 snacks per day), the mean of the range was calculated and analyzed using ANOVAs. All statistical

analysis related to racial background only included children whose background was provided.

Results

The children with FASD were younger (mean age = 8.08 years) than control children (mean age = 9.43 years) due to the inclusion of participants from the choline study of 2-5 year olds (Table I). For children whose racial background was known, the majority in both groups was white (FASD: 33.8% white, Controls: 48.8% white). African American backgrounds were under-represented in the control population relative to the population with FASD, and there were no Native Americans among the controls as contrasted with 16% in the alcohol-exposed group. For over half (52.7%) of the children with FASD, maternal and perinatal history was unavailable or incomplete (many of the children were adopted) compared with 2.5% of controls ($\chi^2(1, 155)=47.6, P<0.001$). For those with a known history, alcohol exposure was associated with significantly decreased prenatal vitamin use (FASD 51.4%, Controls 98.8%; $\chi^2(1, 155)=38.2, P<0.001$) and increased prevalence of premature delivery (25.7% vs. 3.7%, $\chi^2(1, 155)=10.5, P<0.001$). Alcohol-exposed pregnancies were more likely to be associated with exposure to additional drugs of abuse (unspecified) ($\chi^2(1, 155)=62.3, P<0.001$), cigarettes (FASD 34.3%, Controls 0%; $\chi^2(1, 155)=27.4, P<0.001$) and increased prevalence of gestational depression (50.0% vs. 3.7%, $\chi^2(1, 158)=33.2, P<0.001$).

Children with FASD had higher prevalence of prescribed psychiatric medications (43.2% vs. 0%; $\chi^2(1, 155)=38.3, P<0.001$) including stimulants (33.7%), other ADHD-affiliated medications (16.2%), antidepressants (12.2%), and mood modulators (6.8%). They were significantly more likely to be diagnosed with a mental health disorder (16.2% vs. 1.2%, $\chi^2(1, 155)=9.4, P<0.001$). They had greater use of sleep-aids (10.8% vs. 0%, $\chi^2(1, 155)=7.2, P<0.01$), the most common of these being melatonin. There were no differences in the use of other medications (i.e. anticonvulsants, antihypertensives, anti-diabetic).

We surveyed the prevalence of conditions that could adversely impact nutritional outcome (Table II). Children with FASD did not have a higher prevalence of gastrointestinal disorders including celiac disease, irritable bowel syndrome, or food allergies such as gluten intolerance, apart from a non-significant trend for increased constipation (23.0% vs. 11.1%, $\chi^2(1, 155)=3.1, P=0.08$). They had a higher prevalence of eating difficulties including increased oral aversion (6.8% vs. 0%, $\chi^2(1, 155)=3.7, P<0.05$), increased frequent food refusals on the basis of texture (17.6% vs. 3.7%, $\chi^2(1, 155)=6.1, P<0.01$), and increased difficulty in handling silverware (12.1% vs. 1.2% of controls, $\chi^2(1, 155)=6.0, P=0.02$). There was a trend to lower multivitamin use (41.9% vs. 58.0%, $\chi^2(1, 155)=3.4, P<0.07$). The children with FASD did not require special diets and did not have a higher prevalence of food allergies.

Growth impairment is a hallmark of FAS (16) and, expectedly, children with FASD were more likely to have been diagnosed in infancy as small-for-gestational-age (16.2% vs. 2.5%, $\chi^2(1, 155)=7.3, P=0.007$) and/or failure to thrive (12.2% vs. 1.2%, $\chi^2(1, 155)=6.0, P=0.015$) (Table II). There was a trend for lower median BMI in those with FASD (FASD: 16.7, Controls: 16.9, $U=1366, P=0.053$) and this represented an effect of sex, as males with FASD

had a significantly lower mean BMI than male controls (16.3 vs. 18.2, $U=256.5$, $P=0.009$) whereas mean BMI for females did not differ ($P=0.775$). Mean BMI was unaffected by race or gestational size. Caregivers of children with FASD were more likely to perceive their child as underweight (23.0% vs. 1.2%, $\chi^2(1, 155)=15.8$, $P<0.001$) and less likely as overweight ($P=0.64$), perceptions at odds with actual BMI. When analyzed by age (Figure 1), median BMI was lower in the prepubertal (2-10 years) children with FASD (16.5 vs. 19.0, $U=528.5$, $P<0.05$) and was similar in postpuberty (11-19 years). Linear regression revealed a significant difference between the best-fit lines for children with FASD and controls in the post-pubertal group ($t=2.5$, $P=0.02$), suggesting that BMI may increase with age more in children with FASD. There was no association between BMI and satiety, meal/snack consumption, stimulant use, or physical activity.

The acquisition of feeding skills is an important landmark for child development (24). Children with FASD were significantly more likely to experience delayed acquisition of age-appropriate eating skills, compared with controls (18.9% vs. 1.2%; $\chi^2(1, 155)=11.9$, $P<0.001$). Specifically, the median age for solid foods introduction was significantly older for children with FASD (0.75 year vs. 0.50 year, $U=1397.5$, $P<0.001$), as was their age at self-feeding (1.08 year vs. 1.00 year, $U=1111$, $P<0.001$) (Figure 2).

We previously reported a high frequency of abnormal eating behaviors in children with FASD (17), and we replicated those observations in this larger population. Children with FASD had significant impairments in satiety control (Table II). Their caregivers were more likely to report that the child was “never full or satisfied” (23.0% vs. 4.9%, $\chi^2(1, 155)=9.3$, $P=0.002$), snacked constantly (31.1% vs. 6.2%, $\chi^2(1, 155)=14.6$, $P<0.001$), made repeated attempts to sneak food (27.0% vs. 1.2%, $\chi^2(1, 155)=19.8$, $P<0.001$), and exhibited pica (10.8% vs. 0%, $\chi^2(1, 155)=7.2$, $P=0.007$). They were also more likely to report that their child ate too quickly (24.3% vs. 3.7%, $\chi^2(1, 155)=12.36$, $P<0.001$). In response to an open-ended question about eating behavior, multiple caregivers volunteered that the child “lacked an off-switch” with respect to appetite. Other abnormal eating behaviors included poor appetite (14.9% vs. 2.5%, $\chi^2(1, 155)=6.0$, $P=0.01$) and mealtime disinterest (20.3% vs. 0%, $\chi^2(1, 155)=15.9$, $P<0.001$). Although children with FASD were more likely to be picky eaters (32.4% vs. 17.3%, $\chi^2(1, 155)=4.0$, $P=0.05$), there was not a consistent pattern of foods that initiated refusal.

Consistent with their high prevalence of poor satiety, children with FASD consumed one additional meal or snack per day as compared with control children (4.92 ± 0.16 vs. 4.3 ± 0.21 , $F(1,112)=3.0$, $P<0.01$), and this increase was in the form of daily snacks (2.44 ± 0.14 vs. 1.67 ± 0.21 , $F(1,116)=10.6$, $P<0.001$) (Figure 3; available at www.jpeds.com). Children with FASD were more likely to consume greater than six meals or snacks daily (17.6% vs. 6.2%, $\chi^2(1, 155)=3.8$, $P=0.03$), and this was not related to stimulant use. Participation rates in subsidized school food programs did not differ between groups.

Children with FASD had poor self-regulation at mealtime and these behaviors altered the family eating environment. They were significantly more likely to leave the table before meal completion (35.1% vs. 8.6%, $\chi^2(1, 155)=14.8$, $P<0.001$). Their problematic mealtime behaviors included inability to sit through a meal (24.1%), inability to follow or remember

table manners (13.5%), and inappropriate communication skills ('talking loudly', 'cries'; 17.7%). Nearly one-half of their caregivers (48%) reported that these behaviors made it difficult to dine out, compared with 6.1% of controls ($\chi^2(1, 155)=33.0, P<0.001$). We predicted this behavior might impede home meal preparation, but the frequency of meal preparation within the home did not differ across groups. Caregivers of children with FASD maintained a structured eating environment. Both groups of children were equally likely to eat with the family, eat at the dining table, and eat what the family ate. Fewer children with FASD ate meals in front of a computer or television (12.2% vs. 26.0%, $\chi^2(1, 155)=3.9, P<0.05$), suggesting that the parents of those with FASD impose additional levels of structure to try to work around the difficulties of mealtime.

Physical activity is an important modifier of body composition, appetite, and obesity and type-2 diabetes risk. Children with FASD had higher prevalence of weekend physical activity (3.1 ± 0.13 hr/day vs. 2.6 ± 0.15 hr/day, $F(1,92)=7.50, P<0.01$) and trended to higher weekday activity (2.39 ± 0.12 hour/day vs. 2.08 ± 0.14 hour/day, $F(1,108)=2.80, P<0.10$). Despite their greater physical activity, caregivers reported that children with FASD experienced greater difficulty with participating in group physical activities or organized games (46.5% vs. 7.5%, $\chi^2(1, 155)=27.7, P<0.001$). Challenges included difficulty in following rules (21.5%) and in handling social interactions (14.9%). For children with FASD, those who took stimulant medications had decreased levels of overall activity (2.0 hr/day vs. 4.0 hr/day, $U=231.5, P<0.001$) and >3hr of television/computer use per day (20.4% vs. 0%, $P<0.01$) compared with those who did not.

Discussion

Abnormal eating behavior is common in several developmental disabilities including Prader-Willi Syndrome, autism spectrum disorders (ASD), and attention-deficit hyperactivity disorder (ADHD), but has not been well documented in FASD (25-28). In the early 1990s, Demb et al (29) and Ayoob et al (30) separately identified a subset of children in foster care who exhibited hyperphagia without weight gain. Both noted that most of those children experienced prenatal alcohol exposure and/or drug abuse, and speculated that prenatal alcohol exposure might play a causative role. Our data independently endorse this hypothesis and extend it to encompass a broader pattern of disordered eating. The eating behaviors described here for children with FASD appear to be distinct from those described for other developmental disabilities. Whereas ADHD is also associated with disordered eating, this typically presents as bulimia nervosa and can be accompanied by purge behaviors (25), characteristics not observed in this FASD cohort. Children with ASD also display aberrant eating behaviors that include rigid food choice, rigid mealtime routines, and over-sensitivity to food taste, texture or smell (26, 27). These behaviors were not more common in children with FASD except for texture refusal. Also unlike children with ASD (27), those with FASD did not have an elevated incidence of gastrointestinal disorders. Thus the poor satiety, constant snacking, and sneaking of food were behaviors distinct from ADHD and ASD and, if anything, superficially resemble the eating behaviors of Prader-Willi syndrome.

The basis for the poor satiety control in children with FASD is unknown. These children had high use of psychotherapeutic medications, which can influence eating behaviors. For example, some SSRIs stimulate appetite, whereas stimulants impair appetite and users become hyperphagic as the medication levels decline. However, the medication/appetite interaction is complex; stimulants can reduce appetite in children with ADHD, perhaps by reducing impulsive eating (25, 31). Our analysis failed to identify a consistent association between satiety and medication use or class, and additional work on this question is needed. Satiety is also controlled physiologically, by hormonal signals that communicate between the small intestine, adipose tissue, and brain regions such as the ventral tegmental area (32). Prenatal alcohol exposure is known to alter the number and activity of pro-opiomelanocortin cells (33), which can influence appetite, but the potential consequence of these changes upon appetite has not been explored. Self-regulation is also an important determinant of feeding behavior, and the accompanying prefrontal cortex deficits and reduced executive functions that are well-documented in individuals with FASD (2) could make it difficult to acquire the appropriate social and behavioral responses to orexigenic signals. Because eating and satiety is the product of complex interactions between physiological signals and learned behaviors (32), it is reasonable to suggest that the disordered eating in these children results from multiple influences that shape this behavior including deficits in self-regulation, medication use, and altered neuroendocrine signals.

The children with FASD were more likely to have been small for gestational age or underweight during early childhood, which is expected because growth impairment is part of the diagnostic criteria for FAS (16). Perinatal growth impairment increases the risk for metabolic syndrome in later life (34). In that context, several studies report that prenatal alcohol exposure may increase risk for adiposity in periadolescence and particularly for girls (10-12, 14, 15, 17), although we did not find a difference in BMI here. An additional contributor to these children's growth characteristics may be their disordered eating, which can override the satiety cues that regulate caloric intake (38). Multiple caregivers reported the need to monitor and restrict portion size due to the child's overeating, practices that may have limited the accrual of adiposity.

Physical activity also shapes BMI and appetite. The higher rates of physical activity in children with FASD may reflect their increased risk for hyperactivity. Although these children were eager to participate in physical activity, their cognitive, social and emotional deficits made it difficult for them to participate in organized activities and sports. Physical activity should be encouraged for those with FASD as it improves glucose tolerance, muscle development, motor coordination, and may stimulate hippocampal neurogenesis and synaptogenesis. Our findings suggest that their exercise goals may be best achieved through individual activities or participation in sports that deemphasize rules, taking into account physical and cognitive limitations.

This study has several limitations. It relied on caregiver self-report, which is known to underestimate eating behavior. Its cross-sectional design precludes a determination of causation. Some of these children with FASD were enrolled in a choline supplement trial (20) and this may have enhanced caregiver awareness of appropriate dietary behaviors, although reanalysis of data using choline as a cofactor found no significant associations

between choline participation and major study outcomes. The choline subset was younger and thus the FASD cohort was somewhat younger than the control group. Information was unavailable on additional modifiers of BMI including socioeconomic status, parental education level, and prenatal smoking exposure. Although the FASD cohort had significantly more children of African-American and Native American ethnicity, populations with higher obesity risk (41), we found no association between race, BMI, and prenatal alcohol exposure. Although BMI has good validity for assessing body fatness in children and adolescents (40), it does not measure body composition directly.

Based upon our studies of two independent cohorts, we recommend that children who experienced prenatal alcohol exposure, regardless of BMI, should be evaluated for disordered eating. These behaviors may contribute to the growth reductions (4-13) and nutritional inadequacies seen in children with FASD (17, 19). Consultation with an eating therapist may be helpful to develop eating self-regulation and reduce the risk for obesity and/or disordered eating.

Acknowledgments

Supported by the National Institutes of Health (R37AA11085, R01AA22999 [both to S.S.], P30 HD033352 [to Waisman Center], R21AA19580, R33AA19580 [both to J.W.], and T32 DK007665 [to R.K.]).

We thank Carrie Arneson, Elizabeth Emmerich, and Katarina Spelter (Waisman Center, University of Wisconsin) for managing subject recruitment and mailings.

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Figure 1.

Age- and sex-corrected BMI were plotted against age for typically-developing individuals and those with FASD for children ages 2.5 to 10 years and ages 10 to 17 years.

Figure 2.

Age of acquisition for ingestive behaviors in typically-developing and alcohol-exposed children. Left, Age at which child started to consume solid foods. Right, Age at which child was self-feeding. Box plots depict median age, range, and quartiles.

Figure 3.

online. Meal and snack frequency for controls and children with FASD. Top, Number of total meals plus snacks consumed daily as a percentage of total study participants. Bottom, Percentage of study participants who reported they typically consume a given meal or snack.

Table 1
Population Characteristics

		Controls (N=81)	Alcohol-Exposed (N=74)
Sex	Male	51.9% (42)	44.6% (33)
	Female	48.1% (39)	55.4% (44)
Mean Age $\pm$ SEM, yrs			
	All participants	9.43 $\pm$ 0.45	8.08 $\pm$ 0.46 [†]
	Male	9.67 $\pm$ 0.64	8.82 $\pm$ 0.69
	Female	9.21 $\pm$ 0.65	7.34 $\pm$ 0.62
Demographics			
	Native American	0% (0)	16.2% (12)
	Asian	5.0% (4)	8.1% (6)
	African American	5.0% (4)	20.3% (15)
	Pacific Islander	0% (0)	0% (0)
	White	48.8% (40)	33.8% (25)
	Multiracial	8.8% (7)	18.9% (14)
	Unknown	32.5% (26)	2.7% (2)
	Hispanic/Latino	0% (0)	4.0% (3)
	Non-Hispanic/Latino	67.9% (55)	90.5% (67)
	Unknown	31.0% (26)	4.0% (3)
Diagnosis			
	FAS	0% (0)	28.4% (21)
	Partial FAS	0% (0)	28.4% (21)
	ARND	0% (0)	35.1% (26)
	Possible FASD	0% (0)	8.1% (6)

[†] $P=0.04$

Table 2
Nutritional and Eating Behaviors in Children with FASD

Behavior	Controls (n)	Alcohol-Exposed (n)	P
Nutrition-Related Medical Concerns			
Past Concerns			
Failure to Thrive	1.2 (1)	12.2 (9)	0.015
Small for Gestational Age	2.5 (2)	16.2 (12)	0.007
Impaired Rooting Reflex	0 (0)	2.7 (2)	0.44
Current Concerns			
Oral Aversion	0 (0)	6.8 (5)	0.05
Inability to Self-Feed	0 (0)	4.1 (3)	0.21
Gastroesophageal Reflux	4.9 (4)	13.5 (10)	0.11
Tube/Non-oral Feeding	0 (0)	0 (0)	
Food Allergies	3.7 (3)	9.5 (7)	0.26
Irritable Bowel Syndrome	0 (0)	1.4 (1)	0.96
Celiac Disease	0 (0)	0 (0)	
Crohn's Disease	0 (0)	0 (0)	
Diarrhea	2.5 (2)	0 (0)	0.49
Colic/Gas Pains	1.2 (1)	5.4 (4)	0.31
Constipation	11.1 (9)	23.0 (17)	0.08
Current BMI			
Underweight (caregiver perception)	1.2 (1)	23.0 (17)	<0.001
Obese/Overweight (caregiver perception)	5.1 (4)	8.1 (6)	0.64
Median BMI	16.9 (48)	16.7 (72)	0.053
Mean BMI ($\pm$ SEM) - Males	18.2 $\pm$ 0.8	16.3 $\pm$ 0.7	0.009
- Females	19.0 $\pm$ 0.9	18.9 $\pm$ 6.8	0.775
5 th percentile	0% (0)	2.8% (2)	0.662
>5% - < 85 percentile	81.2% (39)	68.1% (49)	0.164
85 th percentile	18.7% (9)	29.2% (21)	0.282
85 th - <95 percentile	8.3% (4)	16.7% (12)	0.298
95 th percentile	10.4% (5)	12.5% (9)	0.954
Of all females, % 85 percentile	22.3% (5/22)	33.3% (13/39)	0.637
Of those 85 percentile, % females	55.6% (5/9)	61.9% (13/21)	1.000
Current Recurring Eating Problems			
Vomiting	1.2 (1)	4.1 (3)	0.55
Lactose Intolerance	1.2 (1)	5.4 (4)	0.31
Gluten Intolerant	0 (0)	1.4 (1)	0.96
Food Intolerance	2.5 (2)	0 (0)	0.49
Bad Teeth/Sore Mouth	2.5 (2)	4.1 (3)	0.92
Trouble Breathing when Eating	0 (0)	1.4 (1)	0.96
Trouble Chewing Food	0 (0)	6.8 (5)	0.05
Frequently Spits Out Food	1.2 (1)	6.8 (5)	0.17

Behavior	Controls (n)	Alcohol-Exposed (n)	P
Oral Sensitivity	0 (0)	4.1 (3)	0.21
Refuses to Eat Certain Textures	3.7 (3)	17.6 (13)	0.01
Swallow/Gag/Choke	3.7 (3)	5.4 (4)	0.90
Head Control/Sitting up	0 (0)	0 (0)	
Negative Response to Facial Contact	0 (0)	8.1 (6)	0.03
Difficulty with Silverware	1.2 (1)	12.1 (9)	0.02
Eats Too Quickly	3.7 (3)	24.3 (18)	<0.001
Picky Eater	17.3 (14)	32.4 (24)	0.05
Never Seems Full/Satisfied	4.9 (4)	23.0 (17)	0.002
Constant Snacking	6.2 (5)	31.1 (23)	<0.001
Attempts to Sneak Food	1.2 (1)	27.0 (20)	<0.001
Pica	0 (0)	10.8 (8)	0.007
Tires or Sleeps when Eating	0 (0)	2.7 (2)	0.44
Disinterest in Mealtimes	0 (0)	20.3 (15)	<0.001
Leaves Table before Finishing	8.6 (7)	35.1 (26)	<0.001
Poor Appetite	2.5 (2)	14.9 (11)	0.01
Upset at Meals	2.5 (2)	9.5 (7)	0.13
Anorexia	0 (0)	0 (0)	
Bulimia Nervosa	0 (0)	0 (0)	