Abnormal Eating Behaviors Are Common in Children with Fetal Alcohol Spectrum Disorder

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Objective To compare the eating behaviors and nutrition-related concerns in children with fetal alcohol spectrum disorder (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 ($P < .001$) and solid food introduction ($P < .001$). 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 $P \leq .002$). They consumed the equivalent of an additional meal/snack daily ($P < .01$). Children with FASD were more likely to have a past diagnosis of underweight ($P < .001$). Mean body mass index was significantly reduced for males ($P = .009$) but not females ($P = .775$) with FASD, and only 2 children with FASD were currently underweight. Children with FASD were more physically active ($P < .01$).

Conclusions 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 children with hyperphagia may warrant referral for FASD screening. *(J Pediatr 2016;169:194-200)*.

Prenatal alcohol exposure is a leading cause of neurodevelopmental disability. The most severe manifestations of prenatal alcohol exposure, diagnoses within the fetal alcohol spectrum disorder (FASD), affect 2.4%-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.[2,3] These differences lessen in later childhood,[3,4] and by periadolescence, both reduced[5-7] and increased weight gain[8-10] is reported compared with unexposed controls. These discrepancies may reflect social and environmental factors.[5,11] 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 (BMIs) ≥85th percentile, especially for females and for cohorts consuming western-style diets.[10,12-15]

Infants with FASD have reduced suckle that delays transition to solid foods.[16] Older children exhibit dysfunctional feeding behaviors including constant snacking, poor satiety, and picky eating/poor appetite.[15] 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.[15,17] This suggests a pattern of abnormal feeding behaviors and food consumption that is specific to children with FASD.

Our previous report of eating behaviors[15] 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 2 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

ADHD	Attention deficit hyperactivity disorder
ASD	Autism spectrum disorder
BMI	Body mass index
FAS	Fetal alcohol syndrome
FASD	Fetal alcohol spectrum disorder

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Supported by the National Institutes of Health (R37AA11085, R01AA22999 [both to S.S.], P30 HD03352 [to Waisman Center], R21AA19580, R33AA19580 [both to J.W.], and T32 DK007665 [to R.A.-K.]). The authors declare no conflicts of interest.

http://dx.doi.org/10.1016/j.jpeds.2015.10.049

were recruited through the University of Minnesota's 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 criteria,[18] which considered their growth, facial dysmorphology, and alcohol exposure. Because Institute of Medicine criteria do not specifically characterize cognitive functioning, we further applied Centers for Disease Control and Prevention central nervous system criteria for FASD[19] (see Wozniak et al[20] for details). Specifically, in order to meet our criteria for alcohol-related neurodevelopmental disorder, participants had to meet the Hoyme criteria for alcohol-related neurodevelopmental disorder, plus have deficits of at least 1 SD in 3 domains of cognitive function. Exclusion criteria were the presence of a non-FASD developmental disorder (ie, autism, Down syndrome), neurologic disorder, brain injury, or other medical condition affecting the brain, or very low birthweight (<1500 g). Hoyme criteria allow for diagnosis of both fetal alcohol syndrome (FAS) and partial FAS 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 partial FAS. For an additional 6 individuals, FASD was strongly suspected but a diagnosis could not be made: 2 had confirmed heavy prenatal alcohol exposure and at least 1 facial feature, 3 had suspected prenatal alcohol exposure and 1 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]

Table I. 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 ± SEM, y		
All participants	9.43 ± 0.45	8.08 ± 0.46*
Male	9.67 ± 0.64	8.82 ± 0.69
Female	9.21 ± 0.65	7.34 ± 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)

ARND, alcohol-related neurodevelopmental disorder.
*$P = .04$.

The 81 control participants included 53 typically developing children who were recruited through the University of Minnesota's 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 institutional review boards at both institutions, and written informed parent consent was obtained prior to study participation.

All the University of Minnesota-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 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.[21] 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.[10]

A 6-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.V.) specializing in developmental disabilities and was modified from Werts et al[15] 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, California) and SAS statistical software (v 9.4; SAS Institute Inc, Cary, North Carolina) 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 1-way ANOVA to examine confounding influences of race, sex, choline intervention, eating behavior, and BMI, upon outcomes. For responses involving a range (ie, 1-2 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) because of the inclusion of participants from the choline