Published in final edited form as:
*Alcohol Clin Exp Res*. 2014 March ; 38(3): 871–878. doi:10.1111/acer.12284.

# Inappropriate Feeding Behaviors and Dietary Intakes in Children with FASD or Probable PAE

**Rachel L. Werts, B.S.**[1], **Sandra C. Van Calcar, R.D., Ph.D.**[1,2], **David S. Wargowski, M.D.**[1,2], and **Susan M. Smith, Ph.D.**[1,3]

[1]Waisman Center, University of Wisconsin School of Medicine and Public Health

[2]Department of Pediatrics, University of Wisconsin School of Medicine and Public Health

[3]Department of Nutritional Sciences, University of Wisconsin-Madison, Madison WI 53706

## Abstract

**Background—**Prenatal alcohol exposure (PAE) is a leading cause of significant neurobehavioral and neurocognitive deficits. Its potential consequences for eating behaviors, nutritional status and other nutritional issues in childhood have received little attention.

**Methods—**Nineteen children (11 male, 8 female) of mean age 9.6 years, referred for FASD screening and assessment, were analyzed with physical exams and caregiver questionnaires to identify possible abnormalities in food and eating behaviors. Fourteen children contributed 24-hour diet recalls and were assessed for nutritional status.

**Results—**Seventy-nine percent of participants were diagnosed with FASD and 63.2% had confirmed PAE. Fifty percent of females were overweight or obese, whereas 37% of males had reduced stature, weight, or BMI for their age. Recurring feeding problems included constant snacking (36.8%), lack of satiety (26.3%), and picky eating/poor appetite (31.6%). None had oral feeding problems. Constipation was common (26.3%). Macronutrient intakes were largely normal but sugar consumption was excessive (140%-190% of recommendations) in 57% of subjects. Vitamin A intake exceeded the Upper Limit for 64% of participants, whereas ≥50% had intakes <80% of RDAs for choline, vitamin E, potassium, β-carotene, and essential fatty acids; 100% had vitamin D intakes <80% of the RDA.

**Conclusions—**PAE may be associated with altered acquisition and distribution of body mass with increasing age. Disordered eating was common. The increased feeding behaviors surrounding lack of satiety suggest self-regulation may be altered. Constipation could reflect low dietary fiber or altered gastrointestinal function. These exploratory data suggest that children with PAE may be at risk for nutritional deficiencies, which are influenced by inappropriate food preferences, disordered eating patterns, medication use, and the stressful dynamics surrounding food preparation and mealtime.

### Keywords

Fetal Alcohol Spectrum Disorders; nutrition; abnormal feeding; pediatric obesity; vitamin D; choline

Corresponding Author: Susan Smith, Ph.D., Professor, Department of Nutritional Sciences, University of Wisconsin-Madison, 1415 Linden Drive, Madison, WI 53706, Tel (608) 263-4316, Fax (608) 262-5860, suesmith@nutrisci.wisc.edu.

## Introduction

Prenatal alcohol exposure (PAE) is a leading cause of significant neurodevelopmental disability and affects between 0.5% to 5% of individuals, depending on the population (May et al., 2009). Individuals with Fetal Alcohol Syndrome (FAS) exhibit the diagnostic trio of somatic growth deficiencies, specific craniofacial alterations, and central nervous system deficits. Many more individuals have known PAE and similar levels of neurodevelopmental disability, but do not meet these diagnostic criteria, and these effects comprise a range of outcomes described as Fetal Alcohol Spectrum Disorder (FASD) (Riley and McGee, 2005). PAE adversely affects specific behavioral domains including learning, attention, cognition, memory, and executive function (Mattson et al., 2013). These are paralleled with structural brain changes that include reduced white matter formation and dysmorphologies affecting the cortex, hippocampus, cerebellum, and other regions. fMRI reveals regional differences in brain activity during cognitive task performance in those exposed to alcohol compared with controls (Coles and Li, 2011; Lebel et al. 2011; Norman et al., 2009).

Little attention has been given to whether these neurobehavioral changes also affect the dynamics surrounding nutrition and eating behaviors. Ingestion behaviors are controlled by a complex neurocircuitry between brain, intestinal, and adipocyte compartments that produce endocrine hormones that control appetite and satiety (Yeo and Heisler, 2012). Food texture sensing, mastication, and swallowing are governed by craniofacial nerves, and PAE impairs the development of their neural crest progenitors (Smith and Debelak-Kragtorp, 2006). Epigenetic modifications also affect postnatal nutritional outcomes, for example, growth rates via IGF2 and appetitive behavior in Prader-Willi syndrome (Jirtle and Skinner, 2007), and PAE can alter epigenetic marks (Kaminen-Ahola et al., 2010).

There has been very little investigation into the possibility that PAE may adversely affect food and eating behaviors, nutritional status and other nutritional issues. Although reduced postnatal body growth is part of the FAS diagnostic criteria, one study noted an association of FAS and FASD with increasing adiposity in young adult females, whereas growth deficiency was more common in young adult males (Spohr et al., 2007). There are several reports of infants with FASD also diagnosed with “Failure to Thrive” due to gastrointestinal dysfunctions including pseudoobstruction and gastroesophageal reflux, conditions that reduced feeding behaviors and necessitated specialized nutritional support (Sujay et al. 2012; Uc et al. 1997). PAE can also delay oromotor development, producing feeding dysfunction and dependence upon enteral or even parenteral feeding to correct the growth impairment (Van Dyke et al. 1982).

This exploratory study was designed to broadly address the possibility that eating behaviors, nutritional status and other nutritional issues found in childhood may be affected by PAE. Inquiries were made into the feeding behaviors, eating patterns, food preferences and cravings, and nutrient intake, as well as body mass distribution and weight status of a cohort of children with suspected FASD.

## Materials and Methods

### Study Population

The study population consisted of participants from Wisconsin who were referred by a physician for screening and assessment for FASD based on prior medical history. Nineteen children were assessed at the FASD Diagnostic Clinic at the Waisman Center, University of Wisconsin-Madison, between 2003 and 2006. Prior to participation, written informed parental consent and child assent were obtained via protocols approved by the Institutional Review Board of the University of Wisconsin-Madison. Children were evaluated by a

trained dysmorphologist (D.S.W.). Anthropometric data of children were recorded on-site and plotted on growth charts obtained from the CDC (Kuczmarski et al., 2002). Medical history data were based on medical record review and physical exam. Diet and eating behavior information were collected during a nutrition interview with caregivers and their child by a Registered Dietitian.

### Dietary Behaviors Analysis

A three-page diet questionnaire was mailed to each participant's caregivers and filled out prior to the diagnostic clinic. The questionnaire was designed specifically for the nutrition interview of the FASD clinic visit and made inquiries into nutrition-related medical concerns, current medications, enrollment in social programs, child feeding history, current feeding habits, recurring feeding-related problems, typical food selection, food safety/security, and other general nutrition-related concerns. The dietitian reviewed the anthropometric data and inquired into physical activity, which was classified as sedentary, low activity, active, or very active, to assess estimated nutrition needs. A 24-hour diet recall was provided by the caregivers of 14 out of the 19 children screened. Diet recalls were analyzed for nutrient and micronutrient content using a standardized diet analysis software (The Food Processor, ESHA Research, Salem OR) that combines nutrient data from more than 1700 sources including the USDA, manufacturers and restaurants. Information about some micronutrients is more limited and was analyzed for 76.9% (choline), 82.7% (copper), 93.3%, (β-carotene) and 83.5% (essential fatty acids) of dietary items.

The diet recall data were analyzed to calculate Estimated Energy Requirement (EER) for each participant using the standard equations from the Dietary Reference Intakes (DRI) (Otten et al. 2006). The equation accounts for age, gender, physical activity level and BMI status and an “active” activity level was assumed for all participants. Calculated EER was compared to total calorie intake calculated from the Food Processor Database.

Age- and sex-specific definitions for underweight (<5$^{th}$ percentile), normal (5$^{th}$ to <85% percentile), overweight (85$^{th}$ to <95% percentile) or obese (≥95$^{th}$ percentile) BMI status followed CDC guidelines (Mei et al,. 2002). The most recent Recommended Daily Allowances (RDAs) of the Dietary Reference Intakes (DRI) were used to determine percent over or under of recommended macronutrient intake for age (Food and Nutrition Board, 2012). Micronutrient intakes were compared to the RDA and Upper Tolerable Intake Levels (UL) (Food and Nutrition Board, 2012). Participants with levels >80% RDA but <UL were determined to be of adequate nutrient status. Carotenoids do not have a specified RDA, so intakes were compared to recent recommendations (Otten et al., 2006). Nutritional adequacy was assessed by comparing the average intake of select nutrients of the study population to the same age groups of the general U.S. population (Bailey et al., 2010; Chester et al., 2011; Ervin et al., 2012; Hoy and Goldman, 2012; Wright et al., 2003). There are limited data on average U.S. dietary intakes for young children, therefore, not all intakes could be compared with the general population.

## Results

### Population Characteristics

The population characteristics of the study group are presented in Table 1. Of 19 participants, 11 were male with an average age of 10.6 years (range 3.9-15.1 years), and 8 were female, average age 8.2 years (range 4.3-13.8 years). Average age was 9.6 years for the entire cohort. Prenatal alcohol exposure was confirmed in 63.2% of participants and suspected in the remainder. A total of 79.0% of study participants were diagnosed as having FAS (10.5%), partial FAS (21.0%) or Alcohol-Related Neurodevelopmental Disorders

(ARND, 47.4%). One individual was diagnosed as possible ARND and three received "no diagnosis". Seven participants (36.8%) exhibited facial dysmorphology, one had low birth weight (5.3%), and all had behavioral disorders or neurodevelopmental delays that prompted screening.

With respect to morphometric characteristics, one individual, a 4-year old male, had a BMI that placed him in the <5$^{th}$ percentile for BMI/age, indicating underweight. Two additional males were at the 5$^{th}$ percentile with respect to height/age and <10$^{th}$ percentile with respect to weight for age. However, these two and the remaining males had normal BMI for age, except for one who was obese (BMI ≥95$^{th}$ percentile) and one who was overweight (85$^{th}$ percentile). In contrast, none of the females were below their age-specific height or weight norms. Moreover, half (4/8) of the females were either obese (3/8) or overweight (1/8), and they had a higher reported percentile weight-for-height and weight-for-age, lower incidence of healthy weight, and higher incidence of overweight and obesity (Table 2). All children with ≥85% BMI were 7.75 yrs or older. Three of the six overweight/obese children were taking anti-depressive medication, and four of six children with ≤30% BMI were taking stimulant/ADHD medication. None were diabetic. Those providing an activity level all reported an "active" lifestyle; therefore, for the purpose of calculating energy requirements, we assumed this level for all participants.

Medical problems reported in ≥25% of respondents were developmental delay (42.1%), behavioral disorders / ADHD (36.8%), psychiatric problems (26.3%), and neuromotor disorders (26.3%). The latter included impaired/delayed motor skills (3), impaired sensory processing (1), and poor visual-motor coordination (1). Constipation was reported in 26.3% of respondents. Two children had a history of anemia. There were no reports of conditions affecting ingestive or digestive behaviors including colostomy, dental problems, gastrointestinal disorders, metabolic disorders, or thyroid disorders. One child had a former cleft lip/palate. There were no reports of food allergy. Medications of appreciable use were stimulants/behavioral disorder treatment drugs (36.8%) and depression/psychiatric treatment drugs (26.2%). Medication classes used at a lower incidence included hypertension treatments (10.5%) and anticonvulsants (5.3%). None reported laxative use.

## Dietary Behaviors

Caregivers were asked to report perceived "recurring problems," defined as unwanted behaviors related to feeding and eating habits occurring at least three times per month. Recurring problems reported by at least 25% of the study population were "picky eater/poor appetite" (31.6%), "constant snacking" (36.8%), and "never seems full or satisfied" (26.3%). Two individuals had a recurring incidence of refusing certain textures and one had chewing difficulties. No incidence of gagging/choking, swallowing disorders and problems with self-feeding were reported, suggesting no severe oral/motor dysfunction.

We performed dietary analysis for the 14 participants who completed the 24-hour diet recall. We calculated individual Estimated Energy Requirement (EER), adjusted for age, gender, and BMI status. Three individuals consumed >100% than their estimated needs, based on their intake from 24-hour recalls, and two of these three were overweight (Table 3). One individual's EER was substantially below the 80% cut-off for energy adequacy; that individual was obese.

Macronutrient intake analysis was based on the Acceptable Macronutrient Distribution Ranges (AMDR) set by the most recent DRI (Otten et al., 2006). Consumption of carbohydrate, protein, and fat, as a percentage of total calories, was at target levels for most individuals (Table 4). However, 64% did not consume the recommended intake of dietary fiber for their age. Conversely, over half the participants obtained more than 25% of their

total calories as simple sugars (mono- and disaccharides) and exceeded target recommendations for their respective age, gender, and BMI (Ervin et al., 2012). With respect to essential fatty acids, half of the cohort did not consume sufficient omega-6 fatty acid (linoleic acid) and 71% did not consume sufficient omega-3 fatty acid (α-linolenic acid; Otten et al., 2006).

Intakes for most micronutrients were adequate, including vitamins B6, B12 and C, thiamin, riboflavin, niacin, and folate, and the minerals calcium, copper, iron, and zinc (Table 5). Vitamin A intake, including pro-Vitamin A forms, was excessive and 64.3% had intakes above the UL. Similarly, excessive sodium intake was found in 85.7% of participants. There were several micronutrient inadequacies, defined as intake below 80% of the RDA. The most notable included low intake of vitamin D (100% of participants), Vitamin E (85.7%), Vitamin K (57.1%) and potassium (85.7%). A substantial number of participants did not consume sufficient choline (71.4%) or beta-carotene (64.3%). No participant reported use of multivitamins or mineral supplements, home remedies, or herbal/alternative medicines.

Select macronutrients and micronutrients intakes for several age ranges of the study population were compared to that nutrient's intake for the same age group in the general U.S. population (Table 6). Macronutrient intakes were similar to age-specific norms for U.S. children as a whole, as were intakes for iron, sodium, choline and potassium. However, percent of total kilocalories from sugar was substantially higher (150% to 190%) in this cohort as compared with age-matched average U.S. intakes (Ervin et al. 2012). While dietary calcium was consistent with typical intakes, Vitamin D intake was substantially below average for this age range (Bailey et al., 2010).

## Discussion

This exploratory study is the first investigation into the possible feeding and nutritional consequences of prenatal alcohol exposure. The most notable results suggest that PAE may be associated with female obesity, constant snacking, lack of satiety, constipation, and low vitamin D status. Several outcomes were more common than in the age-matched, U.S. population, suggesting that PAE may have influenced these digestive and eating behaviors. Because adequate nutrition is essential for healthy child growth and development, nutritional disorders could negatively impact the developmental potential of children who experienced PAE. Disordered feeding behaviors can also stress the family dynamic, particularly around mealtime. These problems may be an unappreciated consequence of PAE.

The obesity/overweight incidence for these female subjects was 50%, a rate substantially greater than the U.S. average of 31.3% for girls of all races ages 2-19 (Ogden et al., 2010). In contrast, the obesity/overweight incidence for these males (18.2%) was well below the U.S. average (32.1%).The sample size was too small to determine whether obesity rates significantly differed between the sexes. However, these data are consistent with a longitudinal study of FASD patients, which found that underweight and shorter stature persisted in males into adulthood, while body weight and adiposity increased in post-pubertal females (Spohr et al., 2007). Klug et al. (2003) similarly found that partial FAS was associated with an increased BMI vs. FAS and no-FAS. Although that study found no gender influence, male status did increase the risk for alcohol-associated height and weight reductions. Supporting these clinical observations is the recent demonstration of increased adiposity and pancreatic dysmorphology in adult guinea pigs that experienced PAE (Dobson et al., 2012). In the general population, obesity risk is the sum of complex interactions between genes and environment. PAE can affect physiological processes that influence BMI including epigenetic marks and metabolic dysregulation (Kaminen-Ahola et al. 2010; Glavas et al., 2007). BMI is also heavily influenced by behavioral practices that can include food

availability, food reward use, exercise opportunity / ability, and the use of psychotherapeutic medications that affect appetite and energy expenditure such as stimulants and antidepressants (Luke et al., 1996). We note that stimulant/ADHD medications, which can suppress appetite, were used by almost half of the males and only two females studied here. Studies into this question must consider these complex physiological, pharmacological, and behavioral influences upon BMI.

Constant snacking is common during childhood and adolescence. However, in this population, snacking behavior was unusually associated with a more marked complaint of lack of satiety. Dietary self-regulation is a learned skill shaped by environmental factors such as early experiences with food and eating and energy expenditure, as well as inherent factors such as genetics and neurodevelopment (Birch and Fischer, 1998). Our findings of increased female obesity, constant snacking in both sexes, and parental complaints of lack of satiety suggest a potential association between FASD and impaired dietary self-regulation. Ethanol's adverse effects on brain development could extend to the neuroendocrine signals that control appetite and reward in regions such as the hypothalamus and ventral tegmental area. Poor dietary self-regulation could also be linked to the low impulse control common in those diagnosed with FASD.

The most common medical complications reported here, behavioral disorders, developmental delay, and psychiatric problems, are commonly reported in FASD (Mattson et al., 2012) and likely contributed to the clinic referral. In contrast, constipation is an uncommon childhood complaint. An estimated 3% of child visits to U.S. pediatric clinics present with some degree of constipation (van den Berg et al., 2006), a value that contrasts sharply with the 26.3% incidence reported here. Constipation is not a common sign or symptom of FASD. The higher incidence noted here could, in part, reflect the participants' inadequate fiber intake. These symptoms also could be caused by abnormal enteric nerve structure or function. The enteric nervous system is derived from the neural crest, which is a known target of ethanol's neurotoxicity (Smith and Kragtorp-Debelak, 2006). Enteric disruptions including retrograde and stationary contractions were documented in several instances of FASD (Uc et al., 1997). More research is needed to investigate the potential extent of constipation issues in the FAS/FASD population.

Micronutrient intakes in this population were largely adequate and evaluation of diet records revealed a high consumption of fortified foods such as cereals and breads. However, intakes of Vitamins D, E, K, potassium, and choline were consistently low and posed a risk for deficiencies. The choline inadequacy is noteworthy given that choline has been suggested to improve neurobehavioral outcomes in rats affected by PAE (Ryan et al., 2008). Most of the cohort consumed inadequate potassium and excessive sodium, due to high consumption of processed foods and cheeses rather than whole vegetables and grains. This potassium-sodium imbalance is disconcerting given that 2 of 14 children were hypertensive, a condition not normally associated with PAE. One affected child was overweight and thus at higher risk for hypertension (Tu et al., 2011). The incidence of pediatric hypertension in the U.S. is 3.7% (Din-Dzietham et al., 2007) compared with the 10.8% reported here. More study is needed to determine whether hypertension is more common in those with PAE. Overall these dietary patterns indicated that children with PAE could be at risk for several nutritional deficiencies.

Additionally, 100% of this cohort was at risk for Vitamin D deficiency, with intakes well below that of U.S. children (Bailey et al., 2010). Although their dairy consumption and calcium consumption were adequate, their poor vitamin D intake was explained by their high consumption of cheese products, which have a low vitamin D content. When describing their child's favorite foods, 31.6% of caregivers specifically cited cheese or cheese-

containing foods (i.e. pizza, cheeseburger, macaroni and cheese), whereas only 25% favored a high vitamin D food (milk or yogurt). The low intake is troubling because children in northern regions such as Wisconsin synthesize sufficient vitamin D only seasonally, due to the sun's low winter angle. Vitamin D is essential for calcium absorption, bone health, immune function, and other roles. This population's residency and dietary practices substantially heightened their risk for vitamin D deficiency. These exploratory data suggest that children with PAE may be at risk for several nutritional deficiencies that are influenced by their food preferences, disordered patterns of eating, and perhaps the stressful dynamics surrounding food preparation and mealtime.

This study had several limitations. There was no control group, so responses were compared with age- and sex-matched U.S. norms. The study population was limited to 19 participants, 14 of which contributed a 24-hour diet recall. Although 79% of children had confirmed PAE or were diagnosed with FAS or FASD, the observed associations may not represent t children with FASD as a whole. The 24-hour dietary recall provides a single dietary snapshot and its accuracy relies upon memory, even with trained questioners. Diet histories of children tend to overreport caloric intake, whereas adolescents and adults generally underreport especially as BMI increases (Jonnalagadda et al., 2000; Livingstone et al., 1992). Thus the reported dietary patterns could have been skewed by inaccurate memory of caregivers. This is supported by a majority of participants reporting a total caloric intake below 100% of the calculated EER. However, this difference could also be explained by an over-estimation of energy needs because we assumed “active” fitness for those not providing an activity level, and because self-reports tend to overestimate child physical activity (Slootmaker et al. 2009).

In conclusion, female obesity, high sugar intake, inadequate micronutrient intake, constipation, and patterns of disordered eating were common in a population with confirmed PAE and/or FAS/FASD. Because the diagnosis was not always confirmed, these observations may not be representative of the FAS/FASD population. However, a similarly high incidence of nutrient inadequacies was recently documented in young children (3-5 yr old) diagnosed with FASD (Fugelstad et al., accepted). Given the importance of adequate nutrition to healthy child development, greater attention to nutritional issues in FASD has the potential to improve these individuals' outcomes.

## Acknowledgments

Funding Support: The FASD clinic was supported in part by NIH Core Grant P30 HD03352, by Northrop Grumman Information Technology through the FASD Center for Excellence, and Wisconsin Department of Health Services through the Wisconsin FASD Treatment Outreach Project.

## References

Bailey RL, Dodd KW, Goldman JA, Gahche JJ, Dwyer JT, Moshfegh AJ, Sempos CT, Picciano MF. Estimation of total usual calcium and vitamin D intakes in the United States. J Nutr. 2010; 140:817–822. [PubMed: 20181782]

Birch LL, Fischer JO. Development of eating behaviors among children and adolescents. J Am Acad Pediatr. 1998; 101:539–549.

Chester DN, Goldman JD, Ahuja JK, Moshfegh AJ. Dietary Intakes of Choline: What We Eat in America, NHANES 2007-2008. Food Surveys Research Group Dietary Data Brief No 9. 2011

Coles CD, Li Z. Functional neuroimaging in the examination of effects of prenatal alcohol exposure. Neuropsychol Rev. 2011; 21:119–132. [PubMed: 21484401]

Dobson CC, Mongillo DL, Brien DC, Stepita R, Poklewska-Koziell M, Winterborn A, Holloway AC, Brien JF, Reynolds JN. Chronic prenatal ethanol exposure increases adiposity and disrupts

pancreatic morphology in adult guinea pig offspring. Nutr Diabetes. 2012; 2:e57. [PubMed: 23247731]

Ervin RB, Kit BK, Carroll MD, Ogden CL. Consumption of added sugar among U.S. children and adolescents, 2005-2008. NCHS Data Brief. 2012; 87:1–8. [PubMed: 22617043]

Food and Nutrition Board. Dietary Reference Intakes: Recommend Intakes for Individuals. National Academy of Sciences, Institute of Medicine; 2011.

Fugelstad AJ, Fink BA, Eckerle JK, Boys CJ, Hoecker HL, Kroupina MG, Zeisel SH, Georgieff MK, Wozniak JR. Inadequate intake of nutrients essential for neurodevelopment in children with fetal alcohol spectrum disorders. Neurotox Teratol(accepted).

Glavas MM, Ellis L, Yu WK, Weinberg J. Effects of prenatal ethanol exposure on basal limbic-hypothalamic-pituitary-adrenal regulation: role of corticosterone. Alcohol Clin Exp Res. 2007; 31:1598–1610. [PubMed: 17760789]

Hoy MK, Goldman JD. Potassium intake of the U.S. population: What We Eat in America, NHANES 2009-2010. Food Surveys Research Group Dietary Data Brief No 10. 2012

Jonnalagadda SS, Mitchell DC, Smiciklas-Wright H, Meaker KB, Van Heel N, Karmally W, Ershow AG, Kris-Etherton PM. Accuracy of energy intake data estimated by a multiple-pass, 24-hour dietary recall technique. J Am Dietetic Assn. 2000; 100:303–308.

Kaminen-Ahola N, Ahola A, Flatscher-Bader T, Wilkins SJ, Anderson GJ, Whitelaw E, Chong S. Postnatal growth restriction and gene expression changes in a mouse model of fetal alcohol syndrome. Birth Defects Res A. 2010; 88:818–826.

Klug MG, Burd L, Martsolf JT, Ebertowski M. Body mass index in fetal alcohol syndrome. Neurotoxicol Teratol. 2003; 25:689–696. [PubMed: 14624968]

Kuczmarski RJ, Ogden CL, Guo SS, Grummer-Strawn LM, Flegal KM, Mei Z, Wei R, Curtin LR, Roche AF, Johnson CL. 2000 CDC growth charts for the United States: methods and development. Vital Health Stat 11. 2002; 246:1–190. [PubMed: 12043359]

Lebel C, Roussotte F, Sowell ER. Imaging the impact of prenatal alcohol exposure on the structure of the developing human brain. Neuropsychol Rev. 2011; 21:102–118. [PubMed: 21369875]

Livingstone MB, Prentice AM, Coward WA, Strain JJ, Black AE, Davies PS, Stewart CM, McKenna PG, Whitehead RG. Validation of estimates of energy intake by weighed dietary record and diet history in children and adolescents. Am J Clin Nutr. 1992; 56:29–35. [PubMed: 1609757]

Luke A, Sutton M, Schoeller DA, Roizen NJ. Nutrient intake and obesity in prepubescent children with Down syndrome. J Am Diet Assoc. 1996; 96:1262–1267. [PubMed: 8948387]

Mattson SN, Roesch SC, Glass L, Deweese BN, Coles CD, Kable JA, May PA, Kalberg WO, Sowell ER, Adnams CM, Jones KL, Riley EP, CIFASD. Further development of a neurobehavioral profile of fetal alcohol spectrum disorders. Alcohol Clin Exp Res. 2013; 37:517–528. [PubMed: 22974253]

May PA, Gossage JP, Kalberg WO, Robinson LK, Buckley D, Manning M, Hoyme HE. Prevalence and epidemiologic characteristics of FASD from various research methods with an emphasis on recent in-school studies. Dev Disabil Res Rev. 2009; 15:176–192. [PubMed: 19731384]

Mei Z, Grummer-Strawn LM, Pietrobelli A, Goulding A, Goran MI, Dietz WH. Validity of body mass index compared with other body-composition screening indexes for the assessment of body fatness in children and adolescents. Am J Clin Nutr. 2002; 75:978–85. [PubMed: 12036802]

Norman AL, Crocker N, Mattson SN, Riley EP. Neuroimaging and fetal alcohol spectrum disorders. Dev Disabil Res Rev. 2009; 15:209–217. [PubMed: 19731391]

Ogden CL, Carroll MD, Curtin LR, Lamb MM, Flegal KM. Prevalence of high body mass index in US children and adolescents, 2007-2008. JAMA. 2010; 303:242–249. [PubMed: 20071470]

Otten, JJ.; Hellwig, JP.; Meyers, LD., editors. Dietary Reference Intakes: The Essential Guide to Nutrient Requirements. National Academies Press; Washington, D.C: 2006.

Riley EP, McGee CL. Fetal alcohol spectrum disorders: an overview with emphasis on changes in brain and behavior. Exp Biol Med. 2005; 230:357–365.

Ryan SH, Williams JK, Thomas JD. Choline supplementation attenuates learning deficits associated with neonatal alcohol exposure in the rat: effects of varying the timing of choline administration. Brain Res. 2008; 1237:91–100. [PubMed: 18786517]

Slootmaker SM, Schuit AJ, Chinapaw MJ, Seidell JC, van Mechelen W. Disagreement in physical activity assessed by accelerometer and self-report in subgroups of age, gender, education and weight status. Int J Behav Nutr Phys Act. 2009; 6:17. [PubMed: 19320985]

Smith, SM.; Debelak-Kragtorp, KA. Neural crest and alcohol exposure. In: Miller, MW., editor. Brain Development: Normal Processes and the Effects of Alcohol and Nicotine. Oxford University Press; Oxford UK: 2006. p. 279-294.

Spohr HL, Willms J, Steinhausen HC. Fetal Alcohol Spectrum Disorders in Young Adulthood. J Pediat. 2007; 150:175–179. [PubMed: 17236896]

Sujay NK, Jones M, Whittle E, Murphy H, Auth MKH. Severe gastrooesophageal reflux disease associated with Foetal Alcohol Syndrome. Case Report Pediat. 2012; 2012:509253.

Tu W, Eckert GJ, DiMeglio LA, Yu Z, Jung J, Pratt JH. Intensified Effect of Adiposity on Blood Pressure in Overweight and Obese Children. Hypertension. 2011; 58:818–824. [PubMed: 21968752]

Uc A, Vasiliauskas E, Piccoli DA, Flores AF, Di Lorenzo C, Hyman PE. Chronic intestinal pseudoobstruction associated with Fetal Alcohol Syndrome. Digest Dis Sci. 1997; 42:1163–1167. [PubMed: 9201078]

van den Berg MM, Benninga MA, Di Lorenzo C. Epidemiology of childhood constipation: a systematic review. Am J Gastroenterol. 2006; 101:2401–2409. [PubMed: 17032205]

Van Dyke DC, Mackay L, Ziaylek E. Management of severe deeding dysfunction in children with Fetal Alcohol Syndrome. 1982; 21:336–339.

Wright JD, Wang CY, Kennedy-Stephenson J, Ervin RB. Dietary intake of ten key nutrients for public health, United States: 1999-2000. Adv Data. 2003; 334:1–4. [PubMed: 12743879]

## Table 1

### Population Characteristics

| | | |
|---|---|---|
| Sex | Male | 57.9% (11)* |
| | Female | 42.1% (8) |
| Mean Age | All participants | 9.6 ± 3.6 yrs |
| | Males | 10.6 ± 3.7 yrs |
| | Females | 8.2 ± 3.2 yrs |
| Prenatal Alcohol Exposure | Confirmed | 63.2% (12) |
| | Suspected | 36.8% (7) |
| Diagnosis | FAS | 10.5% (2) |
| | PFAS | 21.0% (4) |
| | ARND | 47.4% (9) |
| | Partial ARND | 5.3% (1) |
| | No diagnosis | 15.8% (3) |
| Mean Birth Weight | All participants | 3302g ± 556g (13) |
| | > 2500 g | 63.2% (12) |
| | ≤ 2500 g | 5.3% (1) |
| | Unknown | 31.6% (6) |
| Dysmorphic Face | | 36.8% (7) |

*Value in parentheses indicates number of participants out of 19 total.

Table 2

**Body Growth Characteristics**

| Number of Participants | Mean %ile Height/Age (m/yr) | Mean %ile Weight/Age (kg/yr) | % Normal Weight (5-84.9%ile) | % OverWeight (85-94.9%ile) | % UnderWeight (<5%ile) | %Obese (≥95 %ile) | % Overweight or Obese |
|---|---|---|---|---|---|---|---|
| Total | 57% | 48% | 63.2% | 10.5% | 5.3% | 21% | 31.6% |
| Males | 51% | 38% | 72.7% | 9.1% | 9.1% | 9.1% | 18.2% |
| Females | 71% | 68% | 50% | 12.5% | 0% | 37.5% | 50.0% |

%ile, percentile.

**Table 3**

**Estimated Energy Requirement (EER) of Subjects**

| Gender | Age, yr | BMI Status | EER (kcal/d) | Reported Intake (kcal/d) | % Intake vs. EER |
|---|---|---|---|---|---|
| M | 5.7 | Normal | 1532 | 1208 | 79.8%† |
| M | 13.5 | Normal | 2207 | 2147 | 97.3% |
| M | 13.2 | Obese | 4045 | 2716 | 67.1%† |
| M | 9.0 | Normal | 1967 | 1677 | 86.3% |
| M | 14.0 | Overweight | 3530 | 4590 | 130.0%* |
| M | 6.8 | Normal | 1808 | 1425 | 78.8%† |
| M | 11.2 | Normal | 2126 | 1936 | 91.0% |
| M | 12.0 | Normal | 2068 | 2133 | 103.2%* |
| F | 7.0 | Normal | 1775 | 1650 | 92.9% |
| F | 8.7 | Overweight | 2263 | 2841 | 125.5%* |
| F | 4.3 | Normal | 1530 | 1430 | 93.5% |
| F | 8.8 | Obese | 2183 | 1917 | 87.8% |
| F | 4.3 | Normal | 1693 | 1366 | 80.7% |
| F | 11.0 | Normal | 1998 | 1595 | 79.8%† |

*Denotes a positive energy balance.

†Denotes a negative energy balance. An intake <80% of EER was considered inadequate. Calculations were made for the 14 of 19 participants who completed the 24hr diet recall. BMI, Body Mass Index.

## Table 4

### Macronutrient Intake of Subjects

| Macronutrient | % Below Recommendation | % Adequate | % Above Recommendation |
|---|---|---|---|
| Total Carbohydrate | | | |
| Total | 0.0% | 92.9% | 7.1% |
| Males | 0.0% | 87.5% | 12.5% |
| Females | 0.0% | 100% | 0.0% |
| Protein | | | |
| Total | 7.1% | 92.9% | 0.0% |
| Males | 12.5% | 87.5% | 0.0% |
| Females | 0.0% | 100% | 0.0% |
| Fat | | | |
| Total | 14.3% | 71.4% | 14.3% |
| Males | 12.5% | 75.0% | 12.5% |
| Females | 16.7% | 66.7% | 16.7% |
| Dietary Fiber | | | |
| Total | 64.3% | 35.7% | 0.0% |
| Males | 50.0% | 50.0% | 0.0% |
| Females | 83.3% | 16.7% | 0.0% |
| Sugar | | | |
| Total | 0.0% | 42.9% | 57.1% |
| Males | 0.0% | 62.5% | 37.5% |
| Females | 0.0% | 16.7% | 83.3% |
| Linoleic Acid | | | |
| Total | 50.0% | 50.0% | 0.0% |
| Males | 62.5% | 37.5% | 0.0% |
| Females | 50.0% | 50.0% | 0.0% |
| Alpha-Linolenic Acid | | | |
| Total | 71.4% | 28.6% | 0.0% |
| Males | 62.5% | 37.5% | 0.0% |
| Females | 83.3% | 16.7% | 0.0% |

All percentages are corrected for age-specific Acceptable Macronutrient Distribution Ranges from the NRC. Intakes that were >80% RDA but <UL were defined as adequate. N=14 participants, 8 males and 6 females.

## Table 5

### Micronutrient Intake of Subjects

| Micronutrient | | <80% RDA | Adequate | > UL |
|---|---|---|---|---|
| Vitamin A | Total | 7.1% | 28.6% | 64.3% |
| | Males | 0.0% | 37.5% | 62.5% |
| | Females | 16.7% | 16.7% | 66.7% |
| Vitamin C | Total | 14.3% | 85.7% | 0.0% |
| | Males | 25.0% | 75.0% | 0.0% |
| | Females | 0.0% | 100% | 0.0% |
| Vitamin D | Total | 100% | 0.0% | 0.0% |
| | Males | 100% | 0.0% | 0.0% |
| | Females | 100% | 0.0% | 0.0% |
| Vitamin E | Total | 85.7% | 14.3% | 0.0% |
| | Males | 100% | 0.0% | 0.0% |
| | Females | 66.7% | 33.3% | 0.0% |
| Vitamin K | Total | 57.1% | 42.9% | 0.0% |
| | Males | 62.5% | 37.5% | 0.0% |
| | Females | 50.0% | 50.0% | 0.0% |
| Thiamin | Total | 7.1% | 92.9% | 0.0% |
| | Males | 12.5% | 87.5% | 0.0% |
| | Females | 0.0% | 100% | 0.0% |
| Riboflavin | Total | 0.0% | 100% | 0.0% |
| | Males | 0.0% | 100% | 0.0% |
| | Females | 0.0% | 100% | 0.0% |
| Niacin | Total | 7.1% | 35.7% | 57.1% |
| | Males | 12.5% | 25.0% | 62.5% |
| | Females | 0.0% | 50.0% | 50.0% |
| Vitamin B6 | Total | 0.0% | 100% | 0.0% |
| | Males | 0.0% | 100% | 0.0% |
| | Females | 0.0% | 100% | 0.0% |
| Vitamin B12 | Total | 0.0% | 100% | 0.0% |
| | Males | 0.0% | 100% | 0.0% |
| | Females | 0.0% | 100% | 0.0% |
| Folate | Total | 7.1% | 57.1% | 35.7% |
| | Males | 12.5% | 50.0% | 35.7% |
| | Females | 0.0% | 67.3% | 33.3% |
| Calcium | Total | 28.6% | 71.4% | 0.0% |
| | Males | 25.0% | 75.0% | 0.0% |
| | Females | 33.3% | 66.7% | 0.0% |
| Copper | Total | 0.0% | 100% | 0.0% |
| | Males | 0.0% | 100% | 0.0% |
| | Females | 0.0% | 100% | 0.0% |

| Micronutrient | | <80% RDA | Adequate | > UL |
|---|---|---|---|---|
| Iron | Total | 7.1% | 92.9% | 0.0% |
| | Males | 0.0% | 100% | 0.0% |
| | Females | 16.7% | 83.3% | 0.0% |
| Potassium | Total | 85.7% | 14.3% | 0.0% |
| | Males | 75.0% | 25.0% | 0.0% |
| | Females | 100% | 0.0% | 0.0% |
| Sodium | Total | 0.0% | 14.3% | 85.7% |
| | Males | 0.0% | 25.0% | 75.0% |
| | Females | 0.0% | 0.0% | 100% |
| Zinc | Total | 0.0% | 92.9% | 7.1% |
| | Males | 0.0% | 100% | 0.0% |
| | Females | 0.0% | 83.3% | 16.7% |
| Choline | Total | 71.4% | 28.6% | 0.0% |
| | Males | 62.5% | 37.5% | 0.0% |
| | Females | 83.3% | 16.7% | 0.0% |
| β-Carotene | Total | 64.3% | 14.3% | 21.4% |
| | Males | 75.0% | 12.5% | 12.5% |
| | Females | 50.0% | 16.7% | 33.3% |

* Vitamin A content includes retinol and carotenoids (alpha-carotene, beta-carotene, and cryptoxanthins); Beta-carotene does not have an RDI, but a recommended range of 3-6 mg/day. RDA, Recommended Daily Allowance; UL, Tolerable Upper Intake Level. N=14, 8 males and 6 females.

## Table 6
## Comparison of Study Population to U.S. Population Norms

| Nutrient | Average Intake Study Population (mean age 9.3yr) | Average Intake of U.S. Children | | |
|---|---|---|---|---|
| | | **Ages < 6yr** | **Ages 6 – 11 yrs** | **Ages 12-19 yr** |
| Carbohydrate [1,2] | 57.6% | 55.4% | 55.2% | 54.8% |
| Protein [1,2] | 15.0% | 13.2% | 13.2% | 13.7% |
| Fats & Lipids [1,2] | 29.0% | 32.9% | 32.9% | 32.0% |
| Sugar [3] | 25.3% | | | |
| | 23.3% (M) | 13.5% (M) | 16.6% (M) | 17.5% (M) |
| | 27.9% (F) | 13.2% (F) | 15.7% (F) | 16.6% (F) |
| Folate, μg [2] | 431 μg | 255 μg | 339 μg | 372 μg |
| Iron, mg [2] | 15.1 mg | 12.9 mg | 14.4 mg | 15.9 mg |
| Sodium, mg [2] | 3049 mg | 2114 mg | 3255 mg | 3586 mg |
| Choline, mg [4] | 227.6 mg | | | |
| | 243 mg (M) | 200 mg (M) | 250 mg (M) | 330 mg (M) |
| | 207 mg (F) | 205 mg (F) | 210 mg (F) | 215 mg (F) |
| Potassium, mg[5] | 2632 mg | | | |
| | 2805 mg (M) | 2092 mg (M) | 2248 (M) | 2750 mg (M) |
| | 2401 mg (F) | 2046 mg (F) | 2092 mg (F) | 2008 mg (F) |
| | | **Ages 4 – 8 yrs** | **Ages 9 – 13 yrs** | **Ages 14-18 yrs** |
| Calcium, mg [6] | 1202 mg | | | |
| | 1344 mg (M) | 1058 mg (M) | 1074 mg (M) | 1266 mg (M) |
| | 1013 mg (F) | 869 mg (F) | 1039 mg (F) | 1249 mg (F) |
| Vitamin D, IU [6] | 121 IU | | | |
| | 128 IU (M) | 264 IU (M) | 336 IU (M) | 228 IU (M) |
| | 112 IU (F) | 316 IU (F) | 320 IU (F) | 244 IU (F) |

[1] Expressed as % of total calories.

References for U.S. Intakes:

[2] Wright et al., 2003;

[3] Ervin et al., 2012;

[4] Chester et al., 2011;

[5] Hoy and Goldman, 2012;

[6] Bailey et al., 2010.

(M), value for 8 males; (F), value for 6 females; IU, international units. Sodium value does not include discretionary use.