### Title

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### Journal

Alcohol Clinical and Experimental Research, 40(5)

### ISSN

0145-6008



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### Publication Date

2016-05-01

### DOI

10.1111/acer.13035

Peer reviewed

HHS Public Access

Author manuscript

*Alcohol Clin Exp Res*. Author manuscript; available in PMC 2017 May 01.

Published in final edited form as:

*Alcohol Clin Exp Res*. 2016 May ; 40(5): 1075–1082. doi:10.1111/acer.13035.

# Dietary Nutrient Intake in School-Aged Children with Heavy Prenatal Alcohol Exposure

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## Abstract

**Background—**Nutrition is an important factor that affects brain development. Nutritional deficiencies can exacerbate alcohol's damaging effects. Conversely, nutritional supplementation can serve a protective role against alcohol damage and may prove to be a worthwhile intervention strategy. The present study investigated dietary intake in school-aged children with heavy prenatal alcohol exposure to understand their nutritional status, compared to a national sample of typically developing children and Dietary Reference Intakes.

**Methods—**Dietary intake data were collected from children with confirmed histories of heavy prenatal alcohol exposure (5–10 years, *N*=55) using the Automated Self-administered 24-hour Dietary Recall (ASA24). Observed nutrient levels were compared to the Dietary Reference Intakes to evaluate adequacy of nutrient intake as well as to national averages for same-aged children (*What We Eat in America*, NHANES 2007–2008).

**Results—**Alcohol-exposed children exhibited poorer nutritional status compared to the typically developing NHANES sample, consuming lower levels of protein, omega-3 fatty acids, magnesium, potassium, zinc, vitamins C and K, niacin, and choline. Moreover, their diets did not meet Recommended Dietary Allowance or Adequate Intake for dietary fiber, potassium, vitamins E and K, omega-3 fatty acids, and choline.

**Conclusions—**The present findings are consistent with prior studies investigating nutritional intake in preschoolers with FASD, indicating that these children are vulnerable to nutritional inadequacies. Moreover, data suggest a specific profile of dietary intake in this population. As

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several nutrients are important for cognitive development, targeted interventions in clinical populations might be effective in boosting outcomes. Thus, further clinical investigation into the role of nutrition in improving cognitive outcomes is warranted.

**Keywords**

fetal alcohol spectrum disorders (FASD); fetal alcohol syndrome (FAS); prenatal alcohol exposure; nutrition; dietary intake

## INTRODUCTION

Fetal alcohol spectrum disorders (FASD) represent a range of outcomes resulting from alcohol exposure during pregnancy that disrupts fetal development (Bertrand et al., 2004). FASD is a global health concern (Warren et al., 2001), estimated to affect as high as 2–5% of young school children in the United States and Western Europe (May et al., 2009, May et al., 2011) and 7–9% in South Africa (May et al., 2007, May et al., 2009). Prenatal alcohol exposure disrupts brain development, leading to a broad range of neurobehavioral impairments. It is one of the leading known preventable causes of birth defects, intellectual disability, and neurodevelopmental disorders around the world (Pulsifer, 1996, Abel and Sokol, 1987).

Understanding the risk and protective factors that may influence the development of children with FASD is critical for the development of effective prevention and intervention strategies. Nutrition is an important risk factor for FASD. Nutrition interacts with alcohol in various ways that may potentially exacerbate or protect against alcohol's teratogenicity. Poor maternal nutrition and less body mass is a significant problem for mothers who drink alcohol during pregnancy, particularly in high-risk populations (May et al., 2004). For example, studies have shown that women in the Western Cape Province of South Africa, regardless of drinking status, have major nutritional deficiencies; however, mothers of children with FASD were reported to consume significantly lower intake of vitamin $B_2$, calcium, docosapentaenoic acid, and choline compared to non-drinking mothers (May et al., 2014). In a follow-up study, with a different sample of women in the same South African community, these investigators found that mothers of children with FASD consumed more nutrients than non-drinking mothers but alcohol diminished any potential beneficial effects of additional nutrients (May et al., 2015). Furthermore, an investigation of the nutritional status of pregnant women in Russia and the Ukraine revealed that alcohol-consuming mothers have significantly lower levels of plasma zinc and copper when compared to nondrinking mothers attending the same prenatal clinics (Keen et al., 2010). Such nutritional deficiencies pose a severe threat to healthy fetal development, and evidence from animal models clearly demonstrates that undernutrition increases alcohol-related fetal toxicity (Weinberg et al., 1990, Wiener et al., 1981, Shankar et al., 2006, Shankar et al., 2007, Keppen et al., 1985, Huebner et al., 2015).

Although nutritional deficiencies may convey increased risk for FASD, findings in animal studies suggest that nutrition may serve as a protective factor against alcohol-related cognitive impairments. Preclinical studies have shown that dietary supplementation with

specific nutrients—either prenatally or postnatally—can mitigate alcohol-related neurodevelopmental damage and improve cognitive outcomes (Patten et al., 2013a, Patten et al., 2013b, Idrus et al., 2013, Thomas et al., 2007, Thomas et al., 2004, Thomas et al., 2000, Ryan et al., 2008, Summers et al., 2008). However, less is known about how prenatal alcohol exposure or maternal nutrition may affect the nutritional status of offspring. Few studies have investigated the eating behaviors and nutrition of children with FASD (Werts et al., 2014, Fuglestad et al., 2013, Amos-Kroohs et al., 2015). Werts et al. (2014) and Amos-Kroohs et al. (2015) found that children with prenatal alcohol exposure demonstrate abnormal eating behaviors, including delayed acquisition of self-feeding skills, poor appetite, constant snacking, and impaired satiety control. Additionally, these studies revealed an elevated prevalence of gastrointestinal disorders and incidence of obesity among children with FASD. Furthermore, Fuglestad and colleagues (2013) reported that preschoolers with FASD (ages 2.5–4.9 years) consumed lower dietary intakes of saturated fat, vitamin D, and calcium compared to their typically developing peers in the U.S., and that the majority of children did not meet the Dietary Reference Intakes (Institute of Medicine, 2006) for fiber, omega 3 fatty acids, vitamin D, vitamin E, vitamin K, choline, and calcium.

The goal of the current study was to investigate the dietary intake among school-aged children with FASD. An increased understanding of children's nutritional status may highlight aspects of dietary intake and specific nutrients that would be most beneficial to address in dietary supplementation studies and nutritional intervention programs. Thus, this study is an important step in translating preclinical nutritional findings to a human clinical population and understanding whether nutrition can improve cognitive outcomes for children with FASD.

## MATERIALS AND METHODS

### Participants

Participants included 55 children with confirmed histories of heavy prenatal alcohol exposure between the ages of 5 and 10 years ($M$ = 8.28, SD = 1.75). Individuals were recruited through and assessed at two primary sites: the Center for Behavioral Teratology (CBT) at San Diego State University and Double ARC, a nonprofit organization providing services to families of children with FASD in Toledo, Ohio. Children were also recruited through the Genetics and Dysmorphology Clinic at Rady Children's Hospital–San Diego, postings on websites and listservs for families of children with FASD, and ClinicalTrials.gov (Identifier: NCT01911299).

Eligible participants were required to meet criteria for heavy prenatal alcohol exposure and be primary English speakers. Heavy exposure was defined as at least 4 drinks per occasion at least once per week or at least 14 drinks per week during pregnancy. History of prenatal alcohol exposure was determined retrospectively through a review of available medical, social service, or adoption records as well as maternal and/or family/friend report, when available. In many cases when detailed information about timing, duration, and quantity of alcohol consumption was unavailable, mothers were reported to be "alcoholic," alcohol abusing, or alcohol dependent during pregnancy. Thirty-one participants received a dysmorphology examination conducted by Dr. Kenneth Lyons Jones to determine FAS

diagnosis based on craniofacial and growth anomalies; FAS was defined by the presence of two or more key facial features (short palpebral fissures, smooth philtrum, thin vermillion), growth deficiency (≤10th percentile for height or weight), and head circumference ≤10th percentile (for more details, see Jones et al., 2006). Additionally, 6 participants had received a formal diagnosis of an FASD (i.e., including a dysmorphological and physical exam) from other sources (e.g., by dysmorphologists other than Dr. Jones; use of different diagnostic systems, such as the 4-Digit-Code; Astley and Clarren, 2000). The remaining 18 children all met eligibility criteria of having confirmed histories of heavy prenatal alcohol exposure, as defined above, but were not evaluated for a diagnosis.

Exclusion criteria included history of significant head injury with loss of consciousness greater than 30 minutes, significant physical (e.g., uncorrected visual impairment, hemiparesis), neurologic (e.g., seizure disorder), or psychiatric (e.g., active psychosis) disability that precluded involvement in the study, or evidence of any other known causes of mental deficiency (e.g., congenital hypothyroidism, neurofibromatosis, chromosomal abnormalities).

Caregivers and participants provided informed consent and assent to participate in the study, and all procedures were approved by the Institutional Review Boards at San Diego State University and the University of California, San Diego.

## Measures & Statistical Analyses

**Demographics—**Information about subject age, gender, race, ethnicity, and home placement was collected through caregiver report. Body weight percentile was calculated based on the Centers for Disease Control and Prevention (CDC) growth charts, comparing participants' weight to that of other children in the same age group and gender (Kuczmarski et al., 2000). Socioeconomic status (SES) was assessed with the clinician-rated, Hollingshead 4-Factor Index of Social Position. The index assesses caregivers' gender, marital status, highest level of formal education, and current occupation. These factors are weighted, summed, and combined into a continuous measure of social index. Scores range from 8 to 66, with higher scores indicative of higher SES (Hollingshead, 1975). Differences in demographic data across study sites were analyzed using Chi-square statistics (gender, race, ethnicity, handedness, FAS diagnosis, and home placement) and independent-samples *t*-tests (age, body weight, and SES).

**Dietary Intake—**Levels of various nutrients consumed in children's diets were collected twice, 6 weeks apart, using the Automated Self-administered 24-hour Dietary Recall (ASA24; Subar et al., 2012). The ASA24 is a web-based application that guides respondents through the completion of a 24-hour dietary recall for the previous day. Caregivers were asked to report all foods, drinks, and dietary supplements consumed by their children in the previous day from a list of food and drink terms derived from the National Health and Nutrition Examination Survey (NHANES). The ASA24 asks detailed questions about food preparation, portion size, and additions so that food codes from the U.S. Department of Agriculture (USDA) Food and Nutrient Database for Dietary Surveys (FNDDS) can be assigned. Detailed information and analysis is provided about individual-level nutrients and

food group estimates in an individual's diet based on the USDA Food and Nutrient Database for Dietary Surveys, the corresponding USDA MyPyramid Equivalents Database, and the NHANES Dietary Supplement Database. Data on 10 macronutrients, 12 vitamins/micronutrients, and 8 minerals were obtained and included for analyses. Concomitant supplement use was also recorded with each dietary recall.

Because participants in this study were also enrolled in an ongoing clinical trial of choline supplementation, preliminary analyses were conducted to determine whether dietary intake data across the two assessment points could be combined. No significant differences were observed for dietary choline or any other nutrient intake over time, for either treatment groups (i.e., group×time interaction, $p$s > .05). Additionally, there were no known conditions of the intervention study that would have resulted in alterations in children's diet. Caregivers were blind to group randomization, were encouraged not to change their children's diets during the duration of the study, and denied any changes to their children's nutritional supplement regimen on follow-up. Therefore, mean values for each nutrient were created, averaged across the two assessment points, to obtain observed daily nutrient intake levels.

One-sample $t$-tests were used to compare observed nutrient intakes to national data for children ages 5–10 years (*What We Eat in America*, NHANES 2007–2008; Agricultural Research Service - Food Surveys Research Group, 2010). Data from the NHANES 2007–2008 were also collected using 24-hour dietary recalls and with similar collection and processing methods as the ASA24; both the NHANES and ASA24 employed the USDA's Automated Multiple-Pass Method during interview-administered 24-hour recalls, and both datasets utilized the USDA's Food and Nutrient Database for Dietary Surveys, version 4.1, making them comparable.

Additionally, to evaluate whether the observed nutrient intakes were adequate based on recommended values, observed nutrient intakes were compared to the Dietary Reference Intakes (Institute of Medicine, 2006). Dietary Reference Intakes (DRIs) are nutrient-based reference values established for a given age range to meet or exceed the requirements of the majority of healthy individuals within that age range. For the current sample, participants' intakes were compared to DRIs for 5–8 years olds or 9–10 year olds, based on their age. The DRIs include three reference values, the Estimated Average Requirement (EAR), Recommended Dietary Allowance (RDA), and the Adequate Intake (AI). The EAR is the average daily intake level that is estimated to meet the nutrient needs of 50% of healthy individuals; the RDA is the estimated level sufficient to meet the nutrient requirements of 97–98% of healthy individuals; and the AI is the recommended average daily intake level when neither the EAR nor RDA have been established due to insufficient research. Due to the large number of comparisons, the Holm-Bonferroni sequential procedure was used to correct for multiple tests within each category of nutrients (i.e., macronutrients, vitamins, and minerals).

Statistical analyses were conducted using SPSS Statistics version 20.0 (IBM Corporation, 2011). An alpha level of $p < .05$ (two-tailed) was used to determine statistical significance.

# RESULTS

## Demographic Data and Sample Characteristics

Demographic information and sample characteristics are presented in Table 1. Of the 55 participants, 6 children met formal diagnostic criteria for FAS, 31 children did not meet criteria for full FAS based on dysmorphological exam but received a diagnosis on the spectrum (e.g., partial FAS; alcohol-related neurodevelopmental disorder; static encephalopathy, alcohol exposed), and 18 children had confirmed histories of heavy prenatal alcohol exposure but were not evaluated for a diagnosis. Mean SES for the population was 47.5 (median = 50, mode = 50), with a range of 9 through 66. Thus, participants were largely from middle class families (e.g., medium business owners, minor professionals, technicians; Hollingshead, 1975), although the sample was representative of the entire socioeconomic spectrum. Seven children (12.7%) were living with biological parents, whereas 48 (87.3%) were with adoptive families. Of those in adoptive families, 12 were with living with biological relatives (e.g., grandparents, great-grandparents, uncles, aunts) and 3 were adopted internationally.

Additionally, as this was a multi-site study, site differences on demographic and dietary variables were assessed. Sites differed on age, $t(53) = -2.33$, $p = .02$, body weight percentile, $t(53) = -2.92$, $p = .01$, and SES, $t(53) = 2.33$, $p = .02$. Participants at the Ohio site were older (Ohio $M = 8.7$ years, San Diego $M = 7.6$ years), were at a higher body weight percentile (Ohio $M = 56.1$ percentile, San Diego $M = 30.0$ percentile), and were from lower SES (Ohio $M = 44.6$, San Diego $M = 53.0$) compared to participants at the San Diego site. No differences were observed between study locations on gender, $\chi^2(1) = 1.74$, $p = .19$, race, $\chi^2(1) = 3.89$, $p = .14$, ethnicity, $\chi^2(2) = 1.47$, $p = .48$, handedness, $\chi^2(2) = 5.25$, $p = .07$, FAS diagnosis, $\chi^2(2) = 3.17$, $p = .21$, or home placement, $\chi^2(1) = 1.81$, $p = .18$. Site differences on dietary intake were observed for vitamin $B_2$, $t(53) = -2.45$, $p = .01$), vitamin $B_{12}$, $t(53) = -2.59$, $p = .01$, vitamin D, $t(53) = -2.03$, $p = .05$, and selenium, $t(53) = -2.45$, $p = .02$, but no other nutrients or total caloric intake. Participants at the San Diego site consumed lower levels of these nutrients, compared to Ohio site (vitamin $B_2$: Ohio $M = 2.13$ mg, San Diego $M = 1.60$ mg; vitamin $B_{12}$: Ohio $M = 4.87$ μg, San Diego $M = 3.35$ μg; vitamin D: Ohio $M = 4.99$ μg, San Diego $M = 3.68$ μg; selenium: Ohio $M = 95.2$ μg, San Diego $M = 71.3$ μg). Finally, there were no gender differences on any dietary intake variable.

## Dietary Intake

Nutrient intake data for the sample, compared to estimates from the NHANES and DRIs, are reported in Table 2. NHANES estimates were based on a sample size of $N = 1047$ children ages 5–10 years. Participants did not significantly differ on body weight (sample $M = 61.8$, NHANES $M = 66.9$; $p = .07$) or total caloric intake (adjusted $p > .05$) from the NHANES sample. Compared to the national sample, children with prenatal alcohol exposure consumed significantly lower levels of protein, omega-3 fatty acids, magnesium, potassium, zinc, vitamin $B_6$, vitamin C, vitamin K, niacin, and choline in their diets (adjusted $p$s $< .05$).

Compared to the Dietary Reference Intakes, alcohol-exposed children's diets were inadequate for several important macronutrients, vitamins, and minerals. Participants'

nutrient intake levels were significantly lower than the AI for dietary fiber, potassium, omega-3 fatty acids, and choline (adjusted *p*s < .05). Additionally, vitamin K levels were significantly lower than the AI for 5–8 year-olds (adjusted $p = .01$) but not 9–10 year-olds (adjusted $p > .99$); conversely, calcium intake was significantly lower in 9–10 year-olds (adjusted $p = .02$) but not 5–8 year-olds (adjusted $p = .96$). Observed levels of vitamin E for the sample were significantly lower than the RDA (adjusted *p*s < .03) but not the EAR (*p*s > .12), suggesting that participants' diets were not quite inadequate but likely suboptimal for this vitamin. Over 50% of the sample failed to meet the RDA/EAR or AI for all reported nutrients, as well as for vitamin D.

Although nutritional supplements were not included in these analyses, 36% of children in the sample ($n = 20$) were reported by caregivers to take at least one daily supplement. Supplements included multivitamins (33%; $n = 18$), vitamin D/calcium (6%; $n = 3$), omega-3 fatty acids (9%; $n = 5$), probiotic or digestive supplement (7%; $n = 4$), zinc/magnesium/iron (2%; $n = 1$), and lithium orotate (2%; $n = 1$).

To assess for the representativeness of each recall to children's usual diets, caregivers were asked whether food amounts reported in each recall were typical. At the first assessment point, a majority of participants' diets were reported to be typical (78.2%; $n = 43$), 16.4% ($n = 9$) were reported to be less than usual, and 3.6% ($n = 2$) were more than usual. Similar rates were observed after 6 weeks; 75.0% ($n = 39$) of diets were reported to be typical, 17.3% ($n = 9$) were less than usual, and 7.7% ($n = 4$) were more than usual.

## DISCUSSION

This study is one of the few to have examined dietary intake in children who have been exposed to heavy prenatal alcohol exposure. Examination of participants' dietary nutrient intakes revealed that children with prenatal alcohol exposure have poorer nutritional intake compared to similar-age, typically developing children in the U.S. Despite having similar caloric intake and comparable body weights to children in the NHANES sample, children with FASD consumed significantly lower levels of protein, omega-3 fatty acids, magnesium, potassium, zinc, vitamin $B_6$, vitamin C, vitamin K, niacin, and choline in their diets. Moreover, compared to the DRIs established by the Institute of Medicine, children with FASD did not meet recommended dietary intake levels for several important nutrients, including dietary fiber, omega-3 fatty acids, calcium, potassium, vitamin E, vitamin K, and choline. Over 50% of the sample did not meet the RDA or AI for these nutrients, as well as vitamin D. Interestingly, when similar analyses were applied to the NHANES data (i.e., calculated percentage of children who did not meet DRIs for the same group of nutrients), over 50% of children in the NHANES sample did not meet DRIs for the exact same nutrients as children with FASD: dietary fiber, calcium, potassium, vitamin D, vitamin E, vitamin K, choline, and omega-3 fatty acids. Nevertheless, of these nutrients, children with FASD were further below the DRIs for potassium, choline, and omega-3 fatty acids than the NHANES sample based on our nutrient specific comparisons.

Together, these findings suggest that children with FASD may consume less nutrient dense foods than their typically developing counterparts and that their dietary inadequacies may be

related to poor eating habits and food choices. Previous studies report that children with FASD have recurrent behavioral problems related to eating habits, including being picky eaters or having poor appetites, constant snacking, and never seeming full or satisfied (Werts et al., 2014, Amos-Kroohs et al., 2015). These irregularities likely impact adequate consumption of nutritious foods.

The present study further contributes to a growing and much-needed area of research regarding nutrition and its potential role in affecting outcomes for children with FASD. Specifically, these findings extend results from a previous study, which indicated that preschoolers with FASD did not meet recommended intake levels for several nutrients and had poorer nutritional status compared to similar-aged, typically developing children (Fuglestad et al., 2013), and we demonstrate that nutritional inadequacy continues among older, school-aged children. In fact, with regard to nutritional adequacy based on DRIs, our findings were analogous with that of Fuglestad and colleagues, who reported that greater than 50% of their sample did not meet DRIs for fiber, calcium, vitamins D, E, and K, choline, and omega-3 fatty acid. These same nutrients were found to be inadequate in a majority of our sample as well (with the addition of potassium and vitamin $B_6$, which were not observed in the Fuglestad, et al. study). Similarly, Werts and colleagues (2014) found that over 50% of their sample did not meet DRIs for vitamins D and vitamin K, potassium, choline, beta-carotene, and essential fatty acids. Despite some variability, which might be expected across multiple studies and participant samples, striking similarities were found among the three studies that have reported on the nutritional status of children with FASD (Fuglestad et al., 2013, Werts et al., 2014). Across these studies, nutrients consistently found to be inadequate included calcium, potassium, vitamins D, E, and K, choline, and essential fatty acids. Together, these studies suggest that children with FASD may be particularly vulnerable to specific dietary inadequacies and that perhaps there is a nutritional profile characteristic of children with FASD.

An improved understanding of inadequate dietary patterns observed in children with FASD provides opportunities for targeted interventions, and these data suggest nutritional supplementation as a potential treatment for children with FASD. Of the nutrients for which dietary intake was inadequate, several have particular importance in brain development and function as well as cognitive health. Notably, compelling evidence supports the beneficial role of vitamin D (Buell and Dawson-Hughes, 2008, Eyles et al., 2009), choline (Zeisel and Niculescu, 2006, Blusztajn, 1998), and omega-3 fatty acid (Innis, 2007) in the developing brain as well as clinical associations between nutrient status and both global and specific areas of cognitive function. These same nutrients have also demonstrated relevance in FASD, as preclinical studies have demonstrated improved cognition/behavior in alcohol-exposed subjects following supplementation. Choline supplementation has been most extensively studied in preclinical models and has been found to ameliorate impairments related to the hippocampus and prefrontal cortex, including spatial learning, object recognition, working memory, and hyperactivity (Ryan et al., 2008, Thomas et al., 2004, Thomas et al., 2000, Thomas et al., 2007). A recently published randomized clinical trial of choline supplementation in children with FASD revealed improved memory function in young children between ages 2.5 to 4 years (Wozniak et al., 2015). Additionally, when administered with routinely recommended multivitamin/mineral prenatal supplements

during pregnancy, choline has been found to impact neurophysiological encoding and memory in alcohol-exposed infants (Kable et al., 2015). Cognitive findings have been supported by evidence that choline prevents abnormal hippocampal cholinergic development caused prenatal alcohol exposure (Monk et al., 2012) and reduces alcohol-related alterations in DNA methylation in the hippocampus and prefrontal cortex (Otero et al., 2012). Additionally, animal studies have found that vitamin D can improve behavioral flexibility by reducing perseverative behaviors induced by developmental alcohol exposure (Idrus et al., 2013), and omega-3 fatty acids prevent oxidative stress and enhances antioxidant protection in the hippocampus, prefrontal cortex, and cerebellum and improves hippocampal synaptic plasticity (Patten et al., 2013a, Patten et al., 2013b).

## Strengths and Limitations

A few limitations to the present study should be acknowledged. This study did not include a control group, which limited the ability to make direct comparisons to a typically developing sample and, as such, comparisons were made to age- and sex-matched U.S. norms. Another important limitation of this study is that blood nutrient levels were not assessed; instead, nutritional status was determined using a 24-hour dietary recall. Relatedly, a potential limitation of 24-hour dietary recalls (as opposed to a multiple-day or -month food frequency questionnaire or dietary record) is whether they are representative of children's typical diet. However, extensive research has demonstrated that 24-hour dietary recalls can provide high-quality dietary intake data with minimal bias, and this method has been preferred in epidemiological studies, including the NHANES data (Moshfegh et al., 2008). By using the same Automated Multiple-Pass Method for collecting 24-hour dietary recalls as in the NHANES, direct comparisons from our sample to that of the NHANES data could be made. Nevertheless, in order to increase reliability, dietary data were collected at multiple time points and averaged to create more stable values. Additionally, caregivers were asked to rate whether food amounts reported in each recall were typical, and a majority of participants' intakes were described as typical (≥75%). Finally, although participants in this study did not significantly differ from than the NHANES cohort in weight, there was a trend towards lower body weight in children with FASD compared to the typically developing sample. Growth deficiency was one of the initial defining features of FAS (Jones et al., 1973), and alcohol-exposed children tend to be smaller in weight and stature than their non-alcohol-exposed peers, which may impact their overall nutritional requirements. However, the importance of growth in the overall presentation of prenatal alcohol exposure has diminished (O'Leary et al., 2009), and more contemporary diagnostic approaches have relaxed or completely removed the criterion (Cook et al., 2015). Furthermore, in the current sample, there was no difference in overall caloric intake between our sample and that of the NHANES, indicating that differences in nutrient intake were not merely due to lower overall food intake.

Despite these limitations, the current study has notable strengths and adds to the available literature in several respects. This study was able to find the similar dietary deficiencies as seen in a previous nutritional study of preschoolers with FASD and extend those findings in an older age group of children. Additionally, the sample of subjects in this study, collected from two centers across the United States, is larger than those of previous studies,

encompasses a wide range of SES, and is more representative of the general FASD population.

### Summary and Future Directions

In conclusion, we have found that school-aged children with FASD have poorer nutritional intake compared to typically developing children in the U.S., and they did not meet dietary standards outlined by the Institute of Medicine. These results, together with previous studies, suggest a specific profile of dietary intake in children with FASD. As adequate nutrition is critical for healthy brain and cognitive development (Nyaradi et al., 2013), nutritional insufficiency may contribute to or exacerbate risk of impaired cognition and behavior in children with FASD, and nutritional supplementation may be an important treatment or intervention to ameliorate cognitive difficulties in this population. Future studies should extend these findings by validating this nutritional profile using blood nutrient levels. Moreover, additional studies are needed to further investigate the relationship between nutritional status and neurocognition in children with FASD as well as the effectiveness of nutritional supplementation in clinical samples.

## Acknowledgments

Research described in this manuscript was supported by NIAAA grant numbers R01 AA012446 (Thomas) and F31 AA021630 (Nguyen).

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## Table 1

Demographic and sample characteristics.

| Variable | Study Sample (*N* = 55) |
|---|---|
| Gender [*n* (%)] | |
| Males | 27 (49.1) |
| Females | 28 (50.9) |
| Age [*M* (*SD*)] | 8.3 (1.8) |
| Age Strata [*n* (%)] | |
| 5–8 years | 34 (61.8) |
| 9–10 years | 21 (38.2) |
| Body Weight [*M* (*SD*)] | 61.8 (20.9) |
| Body Weight Percentile [*M* (*SD*)] | 47.1 (33.7) |
| Race [*n* (%)] | |
| Caucasian | 37 (67.3) |
| African American | 10 (18.2) |
| Multiracial | 8 (14.5) |
| Ethnicity [*n* (%)] | |
| Hispanic | 5 (9.1) |
| Not Hispanic | 43 (78.2) |
| Not reported | 7 (12.7) |
| SES[†] [*M* (*SD*)] | 47.5 (13.2) |
| FAS Diagnosis[*] [*n* (%)] | |
| FAS | 6 (10.9) |
| Prenatally exposed, non-FAS | 31 (56.4) |
| Not diagnosed | 18 (32.7) |
| Home Placement [*n* (%)] | |
| Biological | 7 (12.7) |
| Adopted | 48 (87.3) |
| Site [*n* (%)] | |
| San Diego | 19 (34.5) |
| Ohio | 36 (65.5) |

[†]Hollingshead 4-Factor Index

[*]FAS, children met three diagnostic criteria based for FAS on dysmorphological exam; Prenatally exposed non-FAS, children did not meet criteria for full FAS based on dysmorphological exam but received a diagnosis on the spectrum; Not diagnosed, children were not evaluated for a diagnosis.

FAS, fetal alcohol syndrome; SES, socioeconomic status

**Table 2**

Observed dietary nutrient intakes compared to NHANES and Dietary Reference Intakes.

| | | | Observed Mean Nutrient Intake | | |
|---|---|---|---|---|---|
| | **RDA/AI 5–8y; 9–10y** | **NHANES 5–10 Mean** | **5–8 years (*n* = 34)** | **9–10 years (*n* = 21)** | **Total sample (*N* = 55)** |
| ***Macronutrients/Energy*** | | | | | |
| Energy (kcal) | – | 2009 | 1674 (425) | 2043 (663) | 1815 (553) |
| Protein (g) | 19; 19 | 74.3 | 59.1 (21.2) | 71.0 (16.8) | **63.5 (20.2)** † |
| Carbohydrate (g) | 130; 130 | 252 | 221 (68.3) | 260 (95.4) | 236 (81.2) |
| Sugars (g) | – | 120 | 101 (38.1) | 114 (59.5) | 106 (47.4) |
| Dietary fiber (g) | 25; 31m, 26f | 14.1 | **13.6 (5.5)** * | **13.9 (4.6)** * | 13.7 (5.1) |
| Total fat (g) | – | 75.5 | 64.0 (17.6) | 82.1 (30.7) | 70.9 (24.8) |
| Saturated fat (g) | – | 25.0 | 21.9 (5.9) | 29.6 (10.4) | 24.9 (8.7) |
| Monounsaturated fat (g) | – | 27.8 | 24.3 (7.7) | 29.8 (12.4) | 26.4 (10.0) |
| Polyunsaturated fat (g) | – | 16.2 | 12.5 (4.6) | 15.9 (8.5) | 13.8 (6.5) |
| Omega-3 fatty acids (g) | .9; 1.2m, 1.0f | .12 | **.017 (.016)** * | **.055 (.10)** * | **.031 (.065)** † |
| ***Vitamins*** | | | | | |
| Vitamin A (μg) | 400; 600 | 577 | 480 (233) | 646 (275) | 544 (260) |
| Vitamin $B_1$ (mg) | .6; .9 | 1.54 | 1.48 (.72) | 1.75 (.61) | 1.58 (.68) |
| Vitamin $B_2$ (mg) | .6; .9 | 2.10 | 1.74 (.72) | 2.27 (.68) | 1.94 (.75) |
| Vitamin $B_6$ (mg) | .6; 1 | 1.83 | 1.45 (.61) | 1.77 (.68) | **1.57 (.65)** † |
| Vitamin $B_{12}$ (μg) | 1.2; 1.8 | 5.15 | 3.79 (1.98) | 5.25 (2.21) | 4.34 (2.17) |

*Alcohol Clin Exp Res*. Author manuscript; available in PMC 2017 May 01.

| | | | Observed Mean Nutrient Intake | | |
|---|---|---|---|---|---|
| | **RDA/AI 5–8y; 9–10y** | **NHANES 5–10 Mean** | **5–8 years (*n* = 34)** | **9–10 years (*n* = 21)** | **Total sample (*N* = 55)** |
| Vitamin C (mg) | 25, 45 | 90.7 | 66.7 (61.2) | 66.2 (63.0) | **66.5 (61.4)**† |
| Vitamin D (μg) | 5; 5 | 5.09 | 4.27 (2.49) | 4.96 (2.08) | 4.53 (2.34) |
| Vitamin E (mg) | 7; 11 | 6.78 | **5.57 (2.78)*** | **7.31 (5.50)*** | 6.23 (4.08) |
| Vitamin K (μg) | 55; 60 | 79.2 | **40.7 (24.2)*** | 60.0 (48.0) | **48.1 (36.1)**† |
| Folate (DFE) (μg) | 200; 300 | 511 | 324 (158) | 555 (270) | 499 (260) |
| Choline (mg) | 250; 375 | 295 | **207 (71.2)*** | **241 (70.7)*** | **220 (72.3)**† |
| Niacin (mg) | 8; 12 | 22.7 | 18.2 (7.0) | 22.6 (7.2) | **19.9 (7.3)**† |
| ***Minerals*** | | | | | |
| Calcium (mg) | 800; 1300 | 931 | 803 (301) | **1058 (347)*** | 900 (340) |
| Copper (mg) | .44; .7 | 1.20 | .978 (.34) | 1.10 (.41) | **1.03 (.37)**† |
| Iron (mg) | 10; 8 | 14.5 | 12.8 (5.7) | 15.6 (6.1) | 13.9 (6.0) |
| Magnesium (mg) | 130; 240 | 259 | 212 (70.7) | 230 (82.8) | **219 (75.3)**† |
| Phosphorus (mg) | 500; 1250 | 1243 | 1038 (346) | 1287 (306) | 1133 (350) |
| Potassium (mg) | 3800; 4500 | 2417 | **1871 (677)*** | **1996 (669)*** | **1917 (670)**† |
| Selenium (μg) | 30; 40 | 99.5 | 79.5 (37.1) | 99.0 (31.1) | 87.0 (36.0) |
| Zinc (mg) | 5; 8 | 11.5 | 9.05 (3.79) | 11.2 (4.29) | **9.88 (4.09)**† |

Data are presented as *M* (SD). Comparisons between sample means and reference values were conducted using one-sample *t*-tests. Age group estimates (5–8 and 9–10 years) were compared to RDA/AI reference values; the total sample estimate was compared to the NHANES mean. The Holm-Bonferroni sequential procedure was used to correct for multiple tests within each category of nutrients.

*Observed dietary intake significantly lower than Institute of Medicine RDA/AI levels, adjusted $p < .05$.

†Observed dietary intake significantly lower than *NHANES* national sample, adjusted $p < .05$.

AI, Adequate Intake; DFE, dietary folate equivalent; f, female; m, male; NHANES, National Health and Nutrition Examination Survey; RDA, Recommended Dietary Allowance; y, years