Association Between Paternal Alcoholism and Adverse Outcomes Observed in Individuals with Prenatal Alcohol Exposure Receiving a Fetal Alcohol Spectrum Disorder Diagnostic Evaluation at the Washington State Fetal Alcohol Syndrome Diagnostic & Prevention Network Clinic

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Abstract

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Background: Prenatal alcohol exposure (PAE) can cause a spectrum of adverse outcomes called fetal alcohol spectrum disorders (FASD). This spectrum includes a multitude of life-long adverse outcomes such as behavioral issues, learning challenges, brain structural changes, and physical abnormalities. Since the 1990s, the Fetal Alcohol Syndrome Diagnostic & Prevention Network Clinic has used a multitude of neuropsychological tests conducted by an interdisciplinary team to understand, diagnose, and provide services to individuals living with PAE. At the FASDPN, a 4-Digit Diagnostic Code is used when evaluating and diagnosing patients. Throughout the decades, it has been evident that maternal alcohol consumption is a risk factor for developing FASD, however less is known about the role of paternal alcoholism. This study examined whether reported paternal alcoholism was associated with an increased severity of a FASD diagnosis and adverse outcomes among patients with PAE.

Methods: A retrospective study using data abstracted from FASDPN patient records was conducted with patient consent and human subjects approval to assess the association between paternal alcoholism and severity of FASD diagnosis as well as adverse fetal outcomes. 468 out of 871 patients were selected by a multitude of inclusion and exclusion criteria. We sampled all different diagnoses, races, ethnicities, sexes at birth, all ages, and patients whose biological father was known. Patients were excluded if no information was given on paternal alcoholism and/or any biological father's health history. Paternal alcoholism was recorded if the section for paternal alcoholism was check marked by individuals completing the New Patient Information form. Growth, facial and structural, neurological, and functional brain outcomes were compared between the two groups. Maternal alcohol exposure was also compared between the two groups. Chi-squared tests, independent t-tests, and Oneway ANOVA with linear-by-linear association when appropriate, were used to compare outcomes between the two groups. Statistical significance set at $p < 0.05$.

Results: Out of the 468 patients, 184 (39%) did not have reported paternal alcoholism and 284 (61%) did have reported paternal alcoholism. Paternal alcoholism was significantly associated with increased severity of FASD diagnosis, increased neuropsychological impairment, and more severe maternal alcohol consumption during pregnancy (all p-values < 0.05). Paternal alcoholism was not observed to be significantly associated with growth deficiency or the FAS facial features (all p-values > 0.05).

Conclusion and Discussion: Our findings suggest paternal alcoholism may be an important risk factor contributing to adverse neurodevelopmental outcomes of patients with PAE. These findings are consistent with both laboratory and clinical studies published by other investigators in the last 5 years. These observations warrant further epidemiological studies to examine

paternal alcohol consumption effects on the longitudinal brain, development, and growth outcomes of individuals with PAE.

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Introduction

Over the last couple of decades in the United States, awareness of the negative effects of alcohol consumption during pregnancy has increased through a multitude of legislative and public health efforts. However, there is still an estimated one infant with fetal alcohol syndrome (FAS) born for every 1,000 live births in the United States (Mayo Clinic, 2024). The majority of individuals exposed to and damaged by prenatal alcohol exposure (PAE) do not have FAS. Rather, they present across a spectrum of adverse outcomes called Fetal Alcohol Spectrum Disorders (FASD). FASD is the umbrella term used to describe the spectrum of adverse outcomes that individuals with PAE present with. According to the FASD 4-Digit Diagnostic Code used at the University of Washington's Fetal Alcohol Syndrome Diagnostic & Prevention Network (FASDPN), three distinct diagnoses fall under the umbrella of FASD: Fetal Alcohol Syndrome (FAS), Static Encephalopathy/ Alcohol Exposed (SE/AE), and Neurodevelopmental Disorder/ Alcohol Exposed (ND/AE). FAS is at the severe end of the spectrum and is characterized by growth deficiency, a unique cluster of three minor facial anomalies, and structural, neurological, and/or functional brain abnormalities (Astley Hemingway, 2024). SE/AE is characterized by structural, neurological, and/or severe functional brain abnormalities without the physical features that would otherwise meet criteria for FAS. ND/AE is at the moderate end of the spectrum and is characterized by moderate brain dysfunction with or without the growth deficiency or FAS facial phenotype. Individuals with PAE frequently present with other prenatal and postnatal risks that, like PAE, can adversely impact growth and cognitive/behavioral development (Hemingway, 2024).

An important first step in the prevention of FASD is the prevention of PAE. To confirm prevention efforts are working, one must track the prevalence of PAE over time. The CDC

created two surveillance programs for tracking PAE: the Pregnancy Risk Assessment Monitory System (PRAMS) and the Behavioral Risk (BRFSS) back in the 1990s (Terry et al., 2024). States individually elect to participate in these surveillance programs. WA has participated since 1993 (Astley Hemingway et al., 2024). The CDC reports that up to 1 in 20 U.S. school-aged children might have FASD (CDC, 2025). Currently, an estimated 14% of pregnant women in the United States report drinking alcohol during pregnancy, and about 5% reported binge drinking (Gosdin et al., 2022).

In 1993, the CDC sponsored the creation of the University of Washington FASD clinic as a primary prevention study for FASD (Astley et al., 2000, 200a). In 1995, the WA State legislature expanded the single clinic into a network of statewide clinics through Senate Bill 5688 and House Bill 1168. Over 3000 patients have received FASD diagnostic evaluations to date. All FASDPN clinics use an interdisciplinary approach to diagnosis using the FASD 4-Digit Diagnostic Code created by (Astley) Hemingway (2024). The mission of the FASDPN is the prevention of FASD through diagnosis, training, screening, intervention, education, and research (www.fasdpn.org) (Astley, 2013).

A question that has until recently remained unanswered in the field of FASD is whether paternal alcohol consumption poses a risk for the developing fetus. Studies in recent years have provided compelling evidence of paternal effects of alcohol consumption on the developing fetus. Using mouse models, a study from the American Society for Clinical Investigation found a plausible connection between male drinking and the development of alcohol related growth deficiencies and craniofacial abnormalities (Thomas et al., 2023). In addition, a population-based study conducted by May (et al., 2025) and colleagues found that after controlling for maternal drinking during pregnancy, paternal alcohol consumption was associated with significant adverse

effects on the child's height, verbal intelligence quotient centile, and head circumference (OFC). These studies inspired another researcher, Michael Golding PhD, to assess paternal drinking and the epigenetic influences on mitochondrial function, child's health, and FASD (Golding, 2025). Golding has found evidence that the epigenetic influences carried in sperm can alter placental development, which increases the likelihood of impacts on the child's health. This is evident from his research, which explains that male alcohol use transmits a memory that alters the mitochondrial stress in the sperm, which impacts placental health and craniofacial shape (2025). Further epidemiological/ epigenetic studies focused on paternal alcohol consumption and its effects on the developing fetus are needed to corroborate these findings.

The objective of this study was to determine if there is an association between paternal alcoholism and adverse fetal outcomes among individuals with PAE who received an FASD diagnostic evaluation at the FASDPN clinic.

Methods

Study Design and Study Setting

A retrospective observational study was conducted using existing patient records collected by the University of Washington FASDPN clinic with patient consent and University of Washington Human Subjects approval. The objective of this study was to determine if associations exist between reported paternal alcoholism and the prevalence and severity of 1) FASD diagnoses (FAS, SE/AE, ND/AE) and 2) the prevalence and/or severity of the individual adverse outcomes that define FASD (growth deficiency, minor facial anomalies and structural, neurological and/or functional brain abnormalities). Over the decades, patients' records were collected in preparation for their FASD diagnostic evaluation. These patient records included the

New Patient Information Form (NPIF). This form collects pertinent information on growth and brain development over the lifetime along with school and placement records. It also provides an overview of medical and mental health conditions that run through the family including the biological mother and father. The list of conditions included paternal alcoholism.

Study Participants

The study population with prenatal alcohol exposure was pulled from the records of ~3,000 patients who had received an FASD evaluation from the FASDPN since 1993. Starting with the patients most recently evaluated at the FASD clinic in 2024, patient records were reviewed to identify the subset that met the following inclusion/exclusion criteria.

Inclusion criteria: All patients who received a diagnosis of FAS, SE/AE, ND/AE, or not FAS/AE, all races, all ethnicities, either sex assigned at birth, all ages at the time of diagnosis, and biological father was known.

Exclusion criterion: If the list of conditions on the NPIF for the biological father had no conditions marked, suggesting the biological father's medical/mental health conditions, including alcoholism, were unknown.

Typically, the prevalence of FASD diagnoses rendered is not evenly distributed across the spectrum. On average, 10% receive a diagnosis of FAS, 25% SE/AE, 50% ND/AE, and 8% are diagnosed not FASD/AE (not FASD despite the presence of prenatal alcohol exposure). Records were reviewed until a comparable number of records fell into each FASD diagnostic classification. A total of 871 patient records were reviewed. A total of 452 patients with PAE met the inclusion/exclusion criteria. The records of an additional 16 control subjects were included that met the above criteria but had a confirmed absence of maternal alcohol use during

pregnancy, resulting in a total study population of 468 subjects. The FASDPN has patient consent and UW Human Subjects approval for the use of this data for research purposes.

Data Collection

The following sociodemographic, exposure, and outcome variables were collected from the patients' records. Sociodemographic variables included sex at birth, age in years at diagnosis, race/ethnicity (reported on the NPIF), and reported paternal alcoholism (yes, no). Maternal prenatal alcohol exposure variables included prenatal alcohol exposure, average number of drinks, maximum number of drinks, average number of days/week of drinking before and during pregnancy, trimesters of drinking, maternal alcohol treatment, and maternal alcoholism. FASD diagnostic outcomes were identified as FAS, SE/AE, ND/AE, not FASD/AE, not FASD/no AE. Growth measures included height and weight percentiles, ABC-scores **(Figure 2)** and 4-Digit Ranks **(Figure 1).**

FAS facial features include 1) Palpebral fissure length (PFL) zscore, ABC-score; 2) Philtrum smoothness 5-point Rank, ABC-score; 3) Lip thinness: circularity, 5-point lip rank, ABC-score; Face ABC-score and 4-Digit Rank. Brain abnormalities using the 4-Digit Diagnostic code **(Figure 1)** included structural abnormalities (microcephaly ($\leq 3^{rd}$ percentile), lowest OFC percentile), neurological abnormalities (seizure disorders), and functional abnormalities (3-Digit CNS (Central Nervous System) Rank (1. Unlikely, 2. Possible, 3. Probable, 4. Definite structural damage)). Neuropsychological measures include standardized test scores from the following: neuropsychological tests: WISC FSIQ (Full Scale Intelligence Quotient) , PIQ (Performance Intelligence Quotient), VIQ (Verbal Intelligence Quotient), WISC (Wechsler Intelligence Scale for Children) processing speed, WISC vocabulary, Keymath, RCFT (Rey Complex Figure Test

of memory and executive function), DKEF (Delis-Kaplan Executive Function System) Trails, DKEF Tower, DKEF Color Word, CVLT (California Verbal Learning Test for memory), QNST (Quick Neurological Screening Test for motor and neurologic function), CCC (Child Communication Checklist for language), CELF4 (Clinical Evaluation of Language Fundamentals), Bayley3 (Bayley Scales of Infant and Toddler Development) Cognition. Three-point Likert Ranks for the following brain domains: intellect, achievement, adaptation, executive function/memory, motor/sensory, psychiatric diagnoses, ADHD, behavior, and infant development.

Paternal alcoholism is documented on the NPIF (**Figure 3**.) If a condition is present, the caregiver places a check mark on the line. If the condition does not have a checkmark, it either means the biological father was not diagnosed with the condition or the person completing the NPIF did not know if the father was diagnosed with the condition. Paternal alcoholism was considered a no if other conditions for the father were checked, indicating that the father's history was known and did not include alcoholism. If none of the conditions for the father were checked, it was assumed the father's history was unknown. Only patients with Yes or No for alcoholism as defined above were included in the study.

FASD Diagnosis

FASD is diagnosed at the WA State FASDPN by an interdisciplinary team using the FASD 4-Digit Diagnostic Code ((Astley) Hemingway, 2024). This diagnostic system is data-driven using information abstracted from the NPIF, past medical/psychological/educational assessments, psychological assessments, and caregiver interviews conducted on the day of the diagnostic evaluation. The four digits of the FASD 4-Digit Code reflect the magnitude of

expression of the four key diagnostic features of FASD in the following order: (1) growth deficiency, (2) the FAS facial phenotype, (3) brain abnormalities, and (4) prenatal alcohol exposure (Figure 1). Each is ranked on a 4-point Likert scale (1 normal, 2 mild, 3 moderate, 4 severe). A 4-digit Code of 4444 reflects the most severe presentation of FAS. A code of 1111 reflects an individual with normal growth and development and confirmed absence of PAE. There are multiple combinations of 4-Digit Codes that fall under the umbrella of FASD. These codes are collapsed into 3 clinically meaningful FASD diagnoses (FAS, SE/AE, and ND/AE) (**Figure 1**). For example, there are 40 different 4-Digit Codes that meet the criteria for FAS. Individuals with prenatal alcohol exposure often present with a myriad of other prenatal and postnatal risks that could also adversely impact growth and development. These, too, are documented and ranked.

Growth and facial measures are collapsed into ABC-scores (A = normal, B = moderate, C = severe) and further collapsed into 4-Digit Ranks in accordance with the Tables presented in **Figure 2**.

Data Analysis

Descriptive statistics were used to summarize characteristics of the study population. Means and standard deviations (SD) were used for our continuous variables while frequencies/ percentages were used for our categorical variables.

Chi-square tests, including linear-by-linear analyses, were used to compare the categorical and ordinal outcomes between fathers with and without alcoholism. These outcomes include the FASD diagnostic category, growth rank measures, CNS structural and function ranks, Likert rankings, and maternal prenatal alcohol exposure measures.

Independent T-tests and Oneway ANOVAs were used to compare continuous outcomes such as birth length centile, birth weight centile, height percentile, weight percentile, OFC percentile, palpebral fissure length percentile, lip circularity percentile, and standardized neuropsychological test scores between reported paternal alcoholism and no report of paternal alcoholism.

All analyses were performed using RStudio Statistical Software version 4.4.3. Statistical significance was set at $p < 0.05$. Due to the observational exploratory nature of this study, p-values were not adjusted for multiple comparisons.

Results

Sociodemographic characteristics of the study population (n = 468) are shown in **Table 1.** Two hundred and eighty-four (61%) patients reported paternal alcoholism, while 184 (39%) did not. Overall, the demographic characteristics were comparable between the groups with and without reported paternal alcoholism. For sex assigned at birth, 151 (53.2%) of males had reported paternal alcoholism, while 91 (49.5%) did not. One hundred and thirty-three (46.8%) of females had reported paternal alcoholism, and 93 (50.5%) did not. Race/ ethnicity and age distributions were also comparable between those with and without reported paternal alcoholism.

Patients with PAE and paternal alcoholism had a higher prevalence of FASD diagnoses at the more severe end of the fetal alcohol spectrum than patients with PAE and no reported paternal alcoholism (**Table 2**).

When evaluating the individual features (growth, face, brain, and PAE) that define FASD, no significant differences in patient growth or FAS facial features were observed between patients with and without paternal alcoholism (**Tables 2, 3**). Among the structural and

neurological measures of brain abnormalities (**Table 4**), patients with paternal alcoholism had smaller mean OFC percentiles (39.2 percentile) than patients without paternal alcoholism (45.2 percentile). This contrast was near significant ($p = 0.06$).

A number of significant differences were observed across numerous domains of brain function as measured using standardized neuropsychological tests administered by psychologists (**Tables 5-6**). Patients with reported paternal alcoholism had significantly higher impairment in intellect, processing speed, math proficiency, adaptive function, executive function, memory, visual/motor integration and language.

Maternal alcohol use was positively correlated with paternal alcoholism (**Table 7**). All measures of maternal alcohol use were significantly more severe among patients with paternal alcoholism than among patients without paternal alcoholism (average number of drinks per drinking occasion, maximum number of drinks per drinking occasion and average number of days/week of drinking before and during pregnancy; number of trimesters of drinking, maternal alcohol treatment, maternal problems with alcohol abuse, and maternal alcoholism)).

Discussion

In our study, patients with PAE and paternal alcoholism had a higher prevalence of FASD diagnoses at the more severe end of the fetal alcohol spectrum than patients with PAE and no reported paternal alcoholism. Patients with paternal alcoholism also had significantly smaller head circumferences and greater impairment across the following domains of brain function (intellect, processing speed, math proficiency, adaptive function, executive function, memory, visual/motor integration and language). Our study did not find significant contrasts in growth or FAS facial features between patients with and without paternal alcoholism. Since paternal

alcoholism in our study was significantly correlated with maternal use of alcohol during pregnancy, our study was unable to separate the adverse impacts of paternal alcohol use from maternal alcohol use. Our findings are broadly consistent with a recent, population-based study (May et al., 2025) despite the considerable contrasts in study populations, FASD diagnostic systems used and number of metrics available to define paternal alcoholism between our two studies.

May et al., (2025) used data from five population-based, cross-sectional studies assessing the prevalence of FASD among first-grade children (7 years of age) in Western Cape Province of South Africa. They found that paternal alcohol consumption appeared to have some limited, independent negative influence on specific child outcomes and traits at 7 years of age. After controlling maternal alcohol consumption in each trimester and maternal tobacco use during pregnancy, there were significant associations found between reported paternal drinking that were independent of maternal drinking on child height, head circumference, and verbal IQ. May et al., (2025) reported their population-based epidemiologic findings indicated that maternal drinking, when combined with heavy male drinking, was associated with the most severe FASD diagnostic outcomes. Three of their four findings (small head circumference, verbal IQ and more severe FASD diagnoses) matched our findings. It is important to note that May's study design and study population enabled them to adjust for confounding by maternal alcohol use during pregnancy. Our study was unable to adjust for maternal alcohol use during pregnancy since the FASDPN clinic does not evaluate patients with no prenatal alcohol exposure.

In contrast to the findings of May et al., (2025), our study observed multiple domains of significantly impaired brain function, not just a reduction in verbal IQ. This is likely due to the fact that May et al., study population was only 7 years of age; too young (e.g., brains not fully

developed yet) to engage in the more sophisticated assessments of executive function, memory, language etc. Roughly 60% of our study samples were over the age of 7 years permitting assessment of more domains of brain function.

We did not find significant differences in growth or FAS facial features between patients with or without paternal alcoholism recorded. Our results are similar to those of May et al., (2025) who found no clear increase in adverse dysmorphology when paternal alcohol consumption was involved. These findings suggest that paternal alcoholism may not independently influence the physical presentation of FASD. Interestingly however, a laboratory-based study conducted by Thomas et al., (2023) did observe physical contrasts between mice offspring who had both maternal and paternal alcohol exposure and mice who had only maternal alcohol exposure. They found that paternal alcohol exposure led to increased shifts in craniofacial patterning. These included significant changes in the offspring's lower jaw, upper jaw, head size (microcephaly), ocular size, midfacial depth, and snout-occipital distance (2023).

Laboratory-based studies using mice (Golding, 2025; Luan et al., 2022; Thomas et al., 2023) have helped shed light on potential underlying mechanisms for how paternal alcoholism may adversely impact fetal development. Golding (2025) found that chronic use of alcohol in male mice causes oxidative stress on mitochondria when metabolizing alcohol in the liver. Golding et al., (2025) hypothesized that this stress induced on the mitochondria by the liver passes into the male's sperm which transmits a memory onto the offspring. This eventually increases the stress of the mitochondria in the developing fetus which directly affects placental growth, which can alter craniofacial growth and future health outcomes of offspring. Luan et al. (2022) looked at the effects that preconception paternal alcohol consumption had on the neurodevelopment of mice offspring through its impact on sperm. Their study found that

preconception paternal alcohol exposure increased the risk of epigenetic modifications of sperm, which include chromosomal abnormalities and gene mutations. This directly influences adverse behavioral development (anxiety, depression, aggressive behavior, and sleep problems) in mice offspring and an increased risk of developing alcohol dependency behaviors. Thomas et al., (2023) observed significant craniofacial and CNS growth deficiencies. They found that paternal prenatal alcohol exposure is an unexamined factor in the development of offspring. These laboratory studies conducted by Golding, (2025) Thomas et, al. (2023) and Luan et, al. (2022), have hypothesized the underlying mechanisms (epigenetic, genetic, and environmental pathways) by which paternal alcohol consumption may adversely impact fetal development. Even though FASDPN does not have epigenetic measures to compare and contrast between patients with or without paternal alcoholism, it demonstrates similar results showcased in the above referenced studies. The participants in our study population experienced a multitude of adverse prenatal and postnatal adversities (e.g., prenatal tobacco exposure, prenatal illicit drug exposure, no prenatal care, neglect, sexual/physical abuse, out-of-home placements, socioeconomic instability) which paternal alcoholism may co-occur ((Astley) Hemingway et al., 2020). All of these risks can contribute to negative adverse outcomes in individuals with prenatal alcohol exposure.

The following limitations should be considered when interpreting the outcomes observed in this study. Paternal alcohol consumption was based on the reported history of alcoholism of the father by the patient's current caregiver. Most patients evaluated in the FASDPN clinic no longer have contact with their birth father. This could lead to recall bias and misclassification of paternal alcoholism. In addition, unlike other published studies (May et al., 2025), our dataset had only one measure of paternal alcohol use conveniently available (alcoholism), limiting the

power to detect associations that might have been revealed if measures of quantity, frequency and timing had been available. Finally, the subset of the FASDPN clinical population selected for this study presented with a significant correlation between maternal and paternal alcohol use limiting the ability to effectively control for maternal alcohol use. Despite these limitations, the significant correlations between paternal alcoholism and adverse brain outcomes observed in this study overlapped considerably with those reported in other clinical and laboratory populations (May et al., 2025; Thomas et al., 2023; Golding, 2025; Luan et al., 2022).

This study contributes to the growing literature reporting significant associations between paternal alcohol consumption and its adverse effects on fetal development. The associations observed between paternal alcoholism and structural, neurological and neuropsychological outcomes highlight the need for further research on the impact of paternal alcohol use on fetal outcomes. Our results highlight the need for increased comprehensive family alcohol consumption histories, including paternal alcohol consumption during maternal health visits and FASD assessments. Future research needs to incorporate more detailed measures of paternal alcohol consumption, such as frequency, duration, and history of alcohol dependency before and after each pregnancy. As is evident in recent clinical studies, research needs to also target the biological mechanisms (genetic and epigenetic) in adjusting for maternal alcohol consumption, family environment, and socioeconomic status. This will help identify if paternal alcohol consumption is independently affecting adverse prenatal outcomes.

In conclusion, patients with prenatal alcohol exposure with reported paternal alcoholism had increased severity of neurodevelopmental and neuropsychological measures than patients without paternal alcoholism. Even though growth measures and facial characteristics were very similar between groups, they were not statistically significant, showcasing a need for more

detailed measures in future studies. Our findings support the increased research into the role paternal alcohol consumption has in the development and severity of FASD related outcomes.

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spectrum disorder is explained by their prenatal alcohol exposure and their other prenatal and postnatal risks? Advances in Pediatric Research 7:41. doi:10.35248/2385-4529.20.7.41.

Figures

Figure 1. Graphic portrayal of the FASD 4-Digit Diagnostic Code

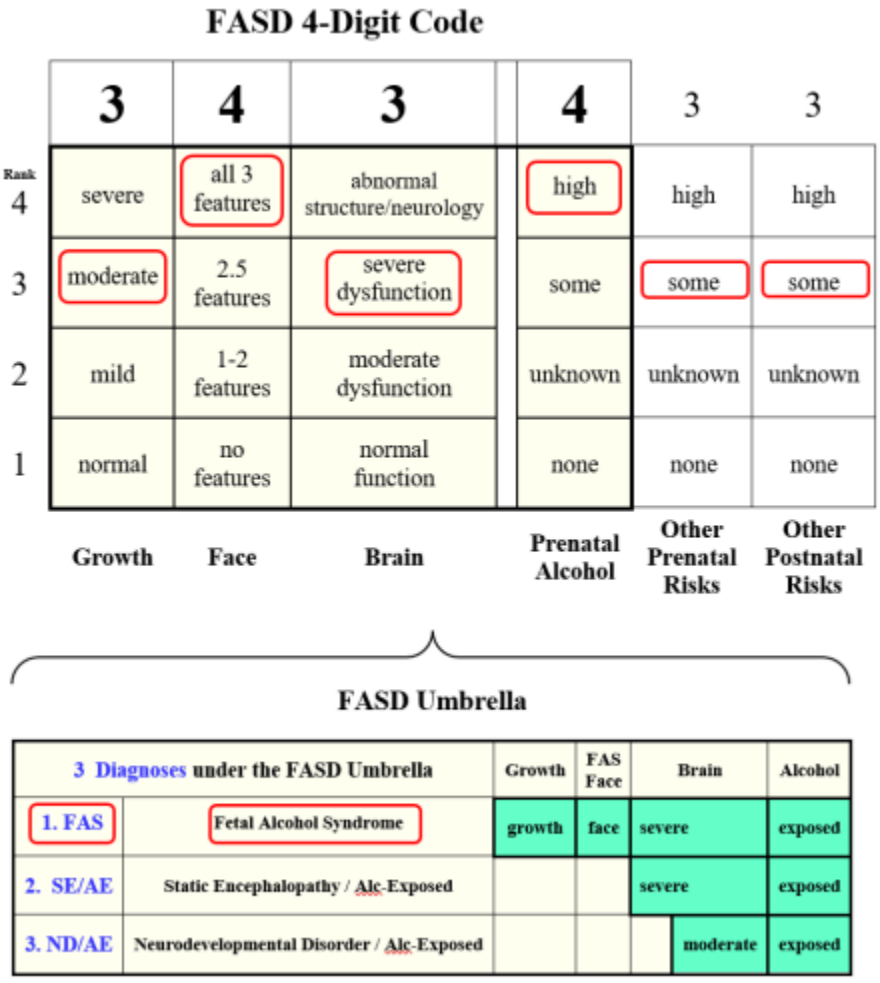
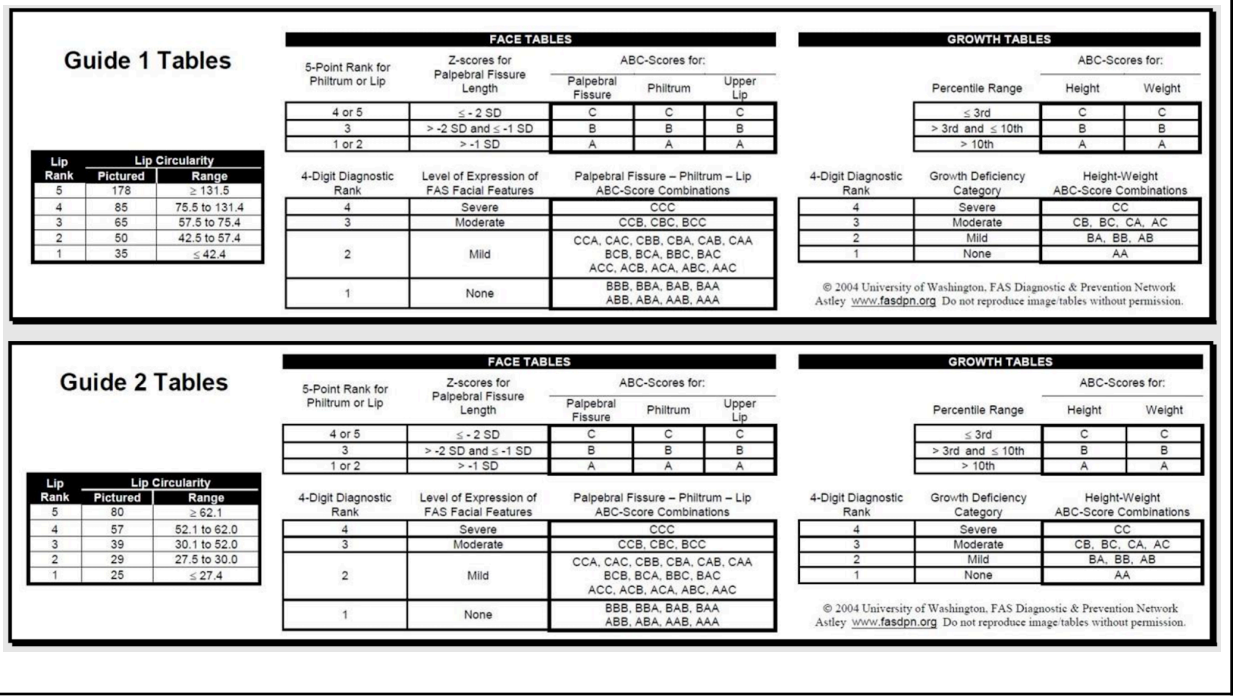


Figure 2. Growth and facial tables for conversion to ABC-Scores and 4-Digit Codes.



Guide 1 Tables

Lip Rank	Lip Circularity: Pictured	Lip Circularity: Range
5	178	≥ 131.5
4	85	75.5 to 131.4
3	65	57.5 to 75.4
2	50	42.5 to 57.4
1	35	≤ 42.4

FACE TABLES

5-Point Rank for Philtrum or Lip	Z-scores for Palpebral Fissure Length	ABC-Scores for: Palpebral Fissure	Philtrum	Upper Lip
4 or 5	≤ - 2 SD	C	C	C
3	> -2 SD and ≤ -1 SD	B	B	B
1 or 2	> -1 SD	A	A	A

4-Digit Diagnostic Rank	Level of Expression of FAS Facial Features	Palpebral Fissure – Philtrum – Lip ABC-Score Combinations
4	Severe	CCC
3	Moderate	CCB, CBC, BCC
2	Mild	CCA, CAC, CBB, CBA, CAB, CAA BCB, BCA, BBC, BAC ACC, ACB, ACA, ABC, AAC
1	None	BBB, BBA, BAB, BAA ABB, ABA, AAB, AAA

GROWTH TABLES

Percentile Range	ABC-Scores for: Height	Weight
≤ 3rd	C	C
> 3rd and ≤ 10th	B	B
> 10th	A	A

4-Digit Diagnostic Rank	Growth Deficiency Category	Height-Weight ABC-Score Combinations
4	Severe	CC
3	Moderate	CB, BC, CA, AC
2	Mild	BA, BB, AB
1	None	AA

Guide 2 Tables

Lip Rank	Lip Circularity: Pictured	Lip Circularity: Range
5	80	≥ 62.1
4	57	52.1 to 62.0
3	39	30.1 to 52.0
2	29	27.5 to 30.0
1	25	≤ 27.4

FACE TABLES

5-Point Rank for Philtrum or Lip	Z-scores for Palpebral Fissure Length	ABC-Scores for: Palpebral Fissure	Philtrum	Upper Lip
4 or 5	≤ - 2 SD	C	C	C
3	> -2 SD and ≤ -1 SD	B	B	B
1 or 2	> -1 SD	A	A	A

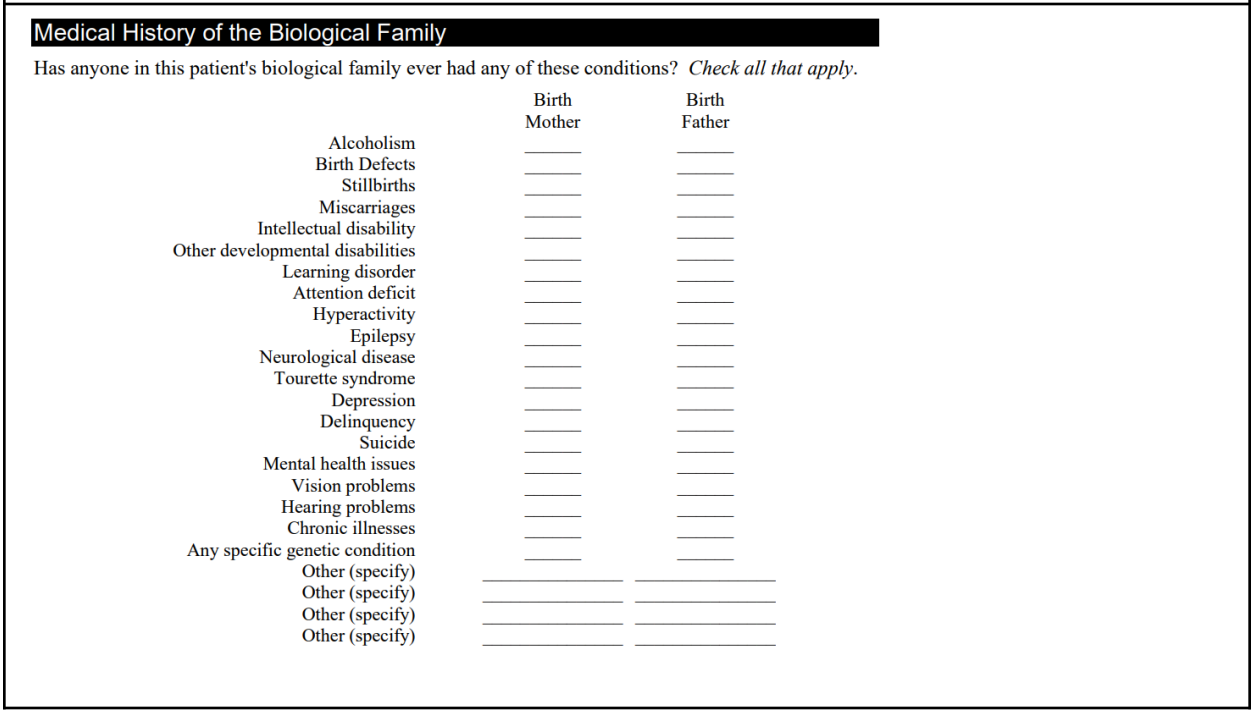
4-Digit Diagnostic Rank	Level of Expression of FAS Facial Features	Palpebral Fissure – Philtrum – Lip ABC-Score Combinations
4	Severe	CCC
3	Moderate	CCB, CBC, BCC
2	Mild	CCA, CAC, CBB, CBA, CAB, CAA BCB, BCA, BBC, BAC ACC, ACB, ACA, ABC, AAC
1	None	BBB, BBA, BAB, BAA ABB, ABA, AAB, AAA

GROWTH TABLES

Percentile Range	ABC-Scores for: Height	Weight
≤ 3rd	C	C
> 3rd and ≤ 10th	B	B
> 10th	A	A

4-Digit Diagnostic Rank	Growth Deficiency Category	Height-Weight ABC-Score Combinations
4	Severe	CC
3	Moderate	CB, BC, CA, AC
2	Mild	BA, BB, AB
1	None	AA

Figure 3. Medical History of the Biological Family documented on the NPIF.



Medical History of the Biological Family

Has anyone in this patient's biological family ever had any of these conditions? *Check all that apply.*

	Birth Mother	Birth Father
Alcoholism	_____	_____
Birth Defects	_____	_____
Stillbirths	_____	_____
Miscarriages	_____	_____
Intellectual disability	_____	_____
Other developmental disabilities	_____	_____
Learning disorder	_____	_____
Attention deficit	_____	_____
Hyperactivity	_____	_____
Epilepsy	_____	_____
Neurological disease	_____	_____
Tourette syndrome	_____	_____
Depression	_____	_____
Delinquency	_____	_____
Suicide	_____	_____
Mental health issues	_____	_____
Vision problems	_____	_____
Hearing problems	_____	_____
Chronic illnesses	_____	_____
Any specific genetic condition	_____	_____
Other (specify)	_____	_____
Other (specify)	_____	_____
Other (specify)	_____	_____
Other (specify)	_____	_____

Tables

Table 1. Selected sociodemographic characteristics of patients receiving a diagnosis at the FASDPN				
Characteristics	All Participants	Paternal Alcoholism		P-value
	N = 468	No, n = 184	Yes, n = 284	
Age at Diagnosis in Years: mean(SD)	8.42 (6.29)	8.17 (5.49)	8.57 (6.76)	0.51*
Age Category at Diagnosis: n (valid %)				P= 0.98**
0 - 2.9 Years	97 (20.7%)	37 (20.1%)	60 (21.1%)	
3 - 5.9 Years	91 (19.4%)	38 (20.7%)	53 (18.7%)	
6 - 12.9 Years	188 (40.2%)	72 (39.1%)	116 (40.8%)	
13 - 17.9 Years	65 (13.9%)	27 (14.7%)	38 (13.4%)	
18+ Years	27 (5.8%)	10 (5.4%)	17 (6.0%)	
Sex at Birth: n (valid %)				P= 0.45**
Male	242 (51.7%)	91 (49.5%)	151 (53.2%)	
Female	226 (48.3%)	93 (50.5%)	133 (46.8%)	
Race/Ethnicity: n (valid %)				P= 0.73**
White	247 (52.8%)	102 (55.4%)	145 (51.1%)	
Black	29 (6.2%)	10 (5.4%)	19 (6.7%)	
Indian	33 (7.1%)	9 (4.9%)	24 (8.5%)	
Asian	1 (0.2%)	1 (0.5%)	0 (0.0%)	
Hispanic	10 (2.1%)	3 (1.6%)	7 (2.5%)	
All others	147 (31.4%)	59 (32.1%)	88 (31.0%)	
Unknown	1 (0.2%)	0 (0.0%)	1 (0.4%)	

*T test

** Age chi-square = 0, Sex chi-square = 0.62, Race/Ethnicity chi-square = 0.12

Table 2. Growth measures for patients with and without paternal alcoholism				
Characteristics	All Participants	Paternal Alcoholism		P-value
	N = 468	No: n = 184	Yes; n = 284	
Diagnostic Category: n (valid %)				P < 0.001*
FAS	107 (22.9%)	39 (21.2%)	68 (23.9%)	
SE/AE	126 (26.9%)	45 (24.5%)	80 (28.2%)	
ND/AE	158 (33.8%)	60 (32.6%)	98 (34.5%)	
Not FASD/AE	61 (13.0%)	40 (21.7%)	38 (13.4%)	
Not FASD/noAE	16 (3.4%)	16 (8.7%)	0 (0.0%)	
Height Measures: n (valid %)				P= 0.81*
1. Rank A, Normal	263 (64.5%)	98 (64.5%)	165 (64.5%)	
2. Rank B, Moderate	54 (13.2%)	22 (14.5%)	32 (12.5%)	
3. Rank C, Severe	91 (22.3%)	32 (21.1%)	59 (23.0%)	
Weight Measures: n (valid %)				P= 0.96*
1. Rank A, Normal	299 (73.5%)	111 (73.0%)	188 (73.7%)	
2. Rank B, Moderate	49 (12.0%)	20 (13.2%)	29 (11.4%)	
3. Rank C, Severe	59 (14.5%)	21 (13.8%)	38 (14.9%)	
Birth Length Centile: n (mean) SD)	341	126 (37.8) 37.8	215 (41.7) 31.3	P= 0.26**
Birth Weight Centile: n (mean) SD)	387	147 (39.8) 26.9	240 (40.9) 28.7	P= 0.69**
Height Centile at Diagnosis: n (mean) SD)	454	177 (40.7) 31.3	277 (40.2) 31.9	P= 0.87**
Weight Centile at Diagnosis: n (mean) SD)	455	179 (49.9) 32.8	276 (50.9) 33.8	P= 0.76**

*Diagnostic Category chi-square = 17.4, Height chi-square = 0.05, Weight chi-square = 0.003
**Birth Length T- = -1.13, Birth Weight T = -0.39, Height Centile T = 0.17, Weight Centile T = -0.30

Table 3. Face measures in patients with and without paternal alcoholism				
Characteristics	All Participants	Paternal Alcoholism		P-value
	N = 468	No; n = 184	Yes; n = 284	
Face Rank: n (valid %)				P= 0.06*
1. Rank 1, No Features	231 (49.4%	99 (53.8%)	132 (46.5%)	
2. Rank 2, 1-2 Features	108 (23.1%)	36 (19.6%)	72 (25.4%)	
3. Rank 3, 2.5 Features	62 (13.2%)	27 (14.7%)	35 (12.3%)	
4. Rank 4, All 3 Features	67 (14.3%)	22 (12.0%)	45 (15.8%)	
Palpebral Fissure ABC-Score: n (valid %)				P= 0.57*
A: > 1 SD (Normal)	210 (44.9%)	78 (42.4%)	132 (46.5%)	
B: > -2SD and <= -1 SD (moderate)	97 (20.7%)	44 (23.9%)	53 (18.7%)	
C: ≤ -2 SDs (severe)	159 (34.0%)	61 (33.2%)	98 (34.5%)	
Philtrum ABC-Score: n (valid %)				P = 0.89*
A. Rank 1 or 2	148 (31.6%)	57 (31.0%)	91 (32.0%)	
B. Rank 3	177 (37.8%)	73 (39.7%)	104 (36.6%)	
C. Rank 4 or 5	141 (30.1%)	53 (28.8%)	88 (31.0%)	
Philtrum Ranks: n (valid %)				P = 0.79*
1: Very Deep	21 (4.6%)	7 (3.9%)	14 (5.0%)	
2: Somewhat Deep	121 (26.2%)	48 (26.7%)	73 (26.0%)	
3: Normal	177 (38.4%)	72 (40.0%)	105 (37.4%)	
4: Somewhat Smooth	114 (24.7%)	44 (24.4%)	70 (24.9%)	
5: Very Smooth	28 (6.1%)	9 (5.0%)	19 (6.8%)	
Lip ABC-Score: n (valid %)				P = 0.65*
A: Rank 1 or 2	167 (35.7%)	70 (38.0%)	97 (34.2%)	
B: Rank 3	141 (30.1%)	57 (31.0%)	84 (29.6%)	
C: Rank 4 or 5	158 (33.8%)	56 (30.4%)	102 (35.9%)	
Lip Ranks: n (valid %)				P = 0.26*
1: Very Thick	23 (5.0%)	13 (7.1%)	10 (3.6%)	
2: Somewhat Thick	143 (30.9%)	56 (30.8%)	87 (31.0%)	
3: Normal Thickness	145 (31.3%)	58 (31.9%)	87 (31.0%)	
4: Somewhat Thin	120 (25.9%)	42 (23.1%)	78 (27.8%)	
5: Very Thin	32 (6.9%)	13 (7.1%)	19 (6.8%)	
Palpebral Fissure Length Zscore n,(mean) SD	457	179 (-1.26) 1.49	278 (-1.32) 1.58	P = 0.67**
Lip Circulatory: n,(mean) S)	443	177 (66.47) 31.4	266 (69.0) 28.9	P = 0.38**

*Face Rank chi-square = 1.5, Palpebral Fissure ABC chi-square = 2.04, Philtrum ABC chi-square = 0.587, Philtrum Ranks chi-square = 0.07, Lip ABC chi-square = 1.64, Lip Ranks chi-square = 1.25

** Palpebral Fissure Zscore T = 0.42, Lip Circulatory T = -0.87

Table 4. Brain structure and neurological measures of patients with and without paternal alcoholism				
Characteristics	All Participants	Paternal Alcoholism		P-value
	N = 468	No; n = 184	Yes; n = 284	
CNS Rank: likelihood of structural damage: n (valid %)				P = 0.09*
1: Unlikely	77 (16.5%)	39 (21.2%)	38 (13.4%)	
2: Possible	158 (33.8%)	60 (32.6%)	98 (34.5%)	
3: Probable	139 (29.7%)	50 (27.2%)	89 (31.3%)	
4: Definite	94 (20.1%)	35 (19.0%)	59 (20.8%)	
Occipitofrontal Circumference: n (valid %)				P = 0.71*
>10th percentile	347 (75.4%)	137 (76.1%)	210 (75.0%)	
4-10th percentile	45 (9.8%)	18 (10.0%)	27 (9.6%)	
≤ 3rd percentile (microcephaly)	68 (14.8%)	25 (13.9%)	43 (15.4%)	
Occipitofrontal Circumference Percentiles: n (mean) SD**	457	178 (45.2) 32.4	279 (39.6) 30.2	P = 0.06**
Seizure Disorders: n (valid %)				P = 0.91*
1: Normal	419 (90.3%)	166 (90.7%)	253 (90.0%)	
2: Moderate	28 (6.0%)	10 (5.5%)	18 (6.4%)	
3: Severe	17 (3.7%)	7 (3.8%)	10 (3.6%)	

* CNS Rank chi-square = 2.73, OFC chi-square = 0.136, Seizure Disorders chi-square = 0.19

** OFC Percentiles T = 1.85

Table 5. Brain function of patients with and without paternal alcoholism				
Characteristics	All Participants	Paternal Alcoholism		P-value
	N = 468	No; n = 184	Yes; n = 284	
CNS Function Rank: n (valid %)				P = 0.29*
Rank 1: No Dysfunction	93 (19.9%)	42 (22.8%)	51 (18.0%)	
Rank 2: Moderate Dysfunction	192 (41.0%)	73 (39.7%)	119 41.9%)	
Rank 3: Severe dysfunction	183 (39.1%)	69 (37.5%)	114 (40.1%)	
Academic Achievement Rank: n (valid %)				P = 0.17*
Rank 1: Normal	56 (26.8%)	30 (38.0%)	26 (20.0%)	
Rank 2: Mildly Abnormal	63 (30.1%)	14 (17.7%)	49 (37.7%)	
Rank3: Severely Abnormal	90 (43.1%)	35 (44.3%)	55 (42.3%)	
Intelligence Rank: n (valid %)				P = 0.63*
Rank 1: Normal, FSIQ $\geq$ 86	156 (44.8%)	64 (47.1%)	92 (43.4%)	
Rank 2: Mildly Abnormal, FSIQ > 70 - $\leq$ 85	122 (35.1%)	45 (33.1%)	77 (36.3%)	
Rank 3: Severely Abnormal, FSIQ $\leq$ 70	70 (20.1%)	27 (19.9%)	43 (20.3%)	
Adaptation Rank: n (valid %)				P = 0.0088
Rank 1: Normal	40 (18.3%)	25 (27.8%)	15 (11.7%)	
Rank 2: Mildly Abnormal	48 (22.0%)	18 (20.0%)	30 (23.4%)	
Rank 3: Severely Abnormal	130 (59.6%)	47 (52.2%)	83 (64.8%)	
Executive Function/ Memory Rank: n (valid %)				P = 0.001*
Rank 1: Normal	49 (19.1%)	29 (28.7%)	20 (12.8%)	
Rank 2: Mildly Abnormal	55 (21.4%)	22 (21.8%)	33 (21.2%)	
Rank 3: Severely Abnormal	153 (59.5%)	50 (49.5%)	103 (66.0%)	
Motor / Sensory Rank: n (valid %)				P = 0.15*
Rank 1: Normal	91 (21.2%)	42 (24.6%)	49 (18.9%)	

Rank	170 (39.5%)	67 (39.2%)	103 (39.8%)
Rank 3: Severely Abnormal	169 (39.3%)	62 (36.2%)	107 (41.3%)
Language Rank: n (valid %)			P = 0.96*
Rank 1: Normal	177 (43.0%)	75 (45.2%)	102 (41.5%)
Rank 2: Mildly Abnormal	119 (28.9%)	41 (24.7%)	78 (31.7%)
Rank 3: Severely Abnormal	116 (28.2%)	50 (30.1%)	66 (26.8%)
Psychiatric Diagnoses Rank: n (valid %)			P = 0.003*
Rank 1: Normal	97 (25.5%)	58 (37.7%)	39 (17.2%)
Rank 2: Mildly Abnormal	98 (25.7%)	26 (16.9%)	72 (31.7%)
Rank 3: Severely Abnormal	186 (48.8%)	70 (45.5%)	116 (51.1%)
Behavioral and Social Rank: n (valid %)			P = 0.013*
Rank 1: Normal	66 (17.1%)	35 (22.6%)	31 (13.4%)
Rank 2: Mildly Abnormal	81 (21.0%)	34 (21.9%)	47 (20.3%)
Rank 3: Severely Abnormal	239 (61.9%)	86 (55.5%)	153 (66.2%)
Development Rank: n (valid %)			P = 0.09*
Rank 1: Normal	83 (31.1%)	43 (36.1%)	40 (27.0%)
Rank 2: Mildly Abnormal	106 (39.7%)	46 (38.7%)	60 (40.5%)
Rank 3: Severely Abnormal	78 (29.2%)	30 (25.2%)	48 (32.4%)
Rey-Osterrieth Complex Figure Test (RCFT): n (valid %)			P = 0.02*
1: > 16%	40 (25.5%)	21 (33.9%)	19 (20.0%)
2: 11-16%	10 (6.4%)	4 (6.5%)	6 (6.3%)
3: 6-10%	2 (1.3%)	1 (1.6%)	1 (1.1%)
4: 2-5%	10 (6.4%)	7 (11.3%)	3 (3.2%)
5: ≤1%	95 (60.5%)	29 (46.8%)	66 (69.5%)

* CNS Function chi-square = 1.14, Achievement Rank chi-square = 1.86, Intelligence Rank chi-square = 0.24, Adaptation Rank chi-square = 7.10, Executive Function/ Memory Rank chi-square = 10.31, Motor/Sensory Rank chi-square = 2.06, Language Rank chi-square = 0.003, Psychiatric Diagnoses Rank chi-square = 9.08, Behavioral and Social Rank chi-square = 6.22, Developmental Rank chi-square = 2.91, RCFT chi-square = 5.14

Table 6. Contrasts in subjects' standardized scores on neuropsychological tests between biological fathers with and without reported alcoholism

Characteristic	All participants	Reported Paternal Alcoholism N (mean) SD		Test statistic, P-value
	N = 468	No; n = 184	Yes;n = 284	
FSIQ *	270	103 (95.3) 20.1	167 (89.9) 16.5	T= 2.3, P = 0.026
PIQ*	67	36 (104.6) 22.9	31 (82.1) 13.5	T = 4.8, P < 0.001
VIQ*	70	36 (103.1) 20.3	34 (83.7) 17.5	T = 4.3, P < 0.001
WISC Process Speed*	95	43 (99.3) 20.0	52 (87.5) 12.9	T = 3.4, P = 0.001
WISC Vocab**	100	44 (10.1) 4.4	56 (7.9) 3.6	T = 2.5, P = 0.01
KeyMath-3 Test*,	34	20 (111.8) 19.5	14 (81.3) 18.7	T = 4.6, P < 0.001
VABS Adaptive Score***	40	24 (88.2) 16.3	16 (63) 23.3	T = 4, P < 0.001
Rey Immediate Recall****	146	57 (38.7) 13.9	89 (31.4) 11.1	T = 3.5, P = 0.001
Rey Delayed Recall****	140	55 (39.5) 14.3	85 (31.2) 10.9	T = 3.9, P < 0.001
DKEF Trails/switching**	49	22 (10.6) 4.3	27 (5.7) 4.3	T = 3.9, P < 0.001
DKEF Tower/Achievement**	69	29 (9.9) 2.4	40 (8.6) 2.6	T = 2.3, P = 0.02
DKEF Tower/Rule Violation^	42	22 (68.2) 36.5	20 (19.5) 29.8	T = 4.7, P < 0.001
DKEF ColorWord/Inhibition**	66	28 (10.1) 2.7	38 (7.8) 3.9	T = 2.7, P = 0.01
DKEF Verbal Fluency**	34	20 (10.5) 2.4	14 (9.4) 3.2	T = 1.2, P = 0.24
CVLT****	51	26 (53.9) 13.4	25 (44.5) 10.2	T = 2.8, P = 0.007
QNST3r*****	133	46 (34.6) 19.2	87 (30.4) 15.9	T = 1.3, P = 0.2
VMI*	202	86 (86.7) 16.4	116 (81.7) 15.3	T = 2.2, P = 0.03
CCC*	183	72 (80.6) 14.2	111 (80.2) 17.8	T = 0.1, P = 0.19
CELF4 Recall Sentences**	88	29 (7.7) 3.5	59 (8.2) 3.1	T = -0.7, P = 0.45
CELF4 Formulated Sentences**	92	34 (9.3) 4.1	58 (10.2) 3.7	T = -1.0, P = 0.29
Bayley3 Developmental*	34	16 (82.1) 24.2	18 (79.9) 21.4	T = 0.3, P = 0.78

* = Standard Score, ** = Scaled Score, *** Composite Score, **** = T-score, ***** = Raw Score, ^ = Cumulative Score
FISQ: Full Scale Intelligence Quotient, PIQ: Performance Intelligence Quotient, VIQ: Verbal Intelligence Quotient, WISC: Wechsler Intelligence Scale for Children, VABS: Vineland Adaptive Behavior Scale, DKEF: Delis-Kaplan Executive Function System, CVLT: California Verbal Learning Test for memory, QNST-3: Quick Neurological Screening Test, VMI: Developmental Test of Visual- Motor Integration, CCC: Child Communication Checklist for language, CELF4: Clinical Evaluation of Language Fundamentals, Bayley3: Bayley Scales of Infant and Toddler Development

Table 7. Maternal Prenatal Alcohol Exposure Measures between patients with and without paternal alcoholism				
Mothers Characteristics	All Participants	Paternal Alcoholism N(%)		P-value
	N = 468	No = 184	Yes = 284	
Average number of days/week of drinking before pregnancy: n (valid %)				P = < 0.001*
1	25 (13.7%)	17 (23.6%)	8 (7.3%)	
2	16 (8.8%)	9 (12.5%)	7 (6.4%)	
3	20 (11.0%)	10 (13.9%)	10 (9.1%)	
4	17 (9.3%)	6 (8.3%)	11 (10.0%)	
5	21 (9.3%)	4 (5.6%)	17 (15.5%)	
6	14 (7.7%)	4 (5.6%)	10 (9.1%)	
7	69 (37.9%)	22 (30.6%)	47 (42.7%)	
Average number of drinks per drinking occasion: n (valid %)				P < 0.001*
No drinks	16 (9.3%)	16 (21.1%)	0 (0.0%)	
One Drink	15 (8.7%)	7 (9.2%)	8 (8.3%)	
2 Drinks	28 (16.3%)	13 (17.1%)	15 (15.6%)	
3 Drinks	20 (11.6%)	10 (13.2%)	10 (10.4%)	
4 Drinks	16 (9.3%)	6 (7.9%)	10 (10.4%)	
5+ Drinks	77 (44.8%)	24 (31.6%)	53 (55.2%)	
Average number of days/week of drinking during pregnancy: n (valid %)				P = 0.005*
0-4 days a week	103 (49.3%)	53 (60.9%)	50 (41.0%)	
5-7 days a week	106 (50.7%)	34 (39.1%)	72 (59.0%)	
Alcohol consumed during trimesters: n (valid %)				P < 0.001*
None	36 (10.7%)	31 (22.6%)	5 (2.5%)	
First Trimester Only	49 (14.5%)	20 (14.6%)	29 (14.4%)	
First and Second Trimesters Only	56 (16.6%)	22 (16.1%)	34 (16.9%)	
All 3 Trimesters	117 (52.4%)	59 (43.1%)	118 (58.7%)	
Mother had a documented problem with alcohol use: n (valid %)				P < 0.001*
No	48 (12.2%)	34 (22.7%)	14 (5.8%)	
Suspected	7 (1.8%)	3 (2.0%)	4 (1.7%)	
Yes	337 (86.0%)	113 (75.3%)	224 (92.6%)	
Mother diagnosed with alcoholism: n (valid %)				P < 0.001*
No	73 (22.1%)	46 (36.8%)	27 (13.2%)	
Suspected	18 (5.5%)	5 (4.0%)	13 (6.3%)	
Yes	239 (72.4%)	74 (59.2%)	165 (80.5%)	

Mother Ever Received Alcohol Treatment: n (valid %)			P < 0.001*
No	104 (31.8%)	60 (45.5%)	44 (22.6%)
Suspected	6 (1.8%)	2 (1.5%)	4 (2.1%)
Yes	217 (66.4%)	70 (53.0%)	147 (75.4%)

*Average number of days/week before pregnancy chi-square = 12.3, Average number per drinking occasion chi-square = 18.4, Average number of days/week during pregnancy chi-square = 8.04, Alcohol consumed chi-square = 24.4, Documented problem with alcohol chi-square = 24.5, Diagnosed with alcoholism chi-square = 22.6, Ever received alcohol treatment chi-square = 18.6