PROFILE OF THE FIRST 1,400 PATIENTS RECEIVING DIAGNOSTIC EVALUATIONS FOR FETAL ALCOHOL SPECTRUM DISORDER AT THE WASHINGTON STATE FETAL ALCOHOL SYNDROME DIAGNOSTIC & PREVENTION NETWORK

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ABSTRACT

Background
An interdisciplinary approach to fetal alcohol spectrum disorder (FASD) diagnosis using rigorously defined diagnostic guidelines has been adopted as best practice. Diagnostic clinics are being established worldwide. If these clinics are to successfully compete for limited health care dollars, it is essential to document their value.

Objective
The primary objectives were to document the value of the largest and longest standing interdisciplinary FASD diagnostic program; the Washington State Fetal Alcohol Syndrome Diagnostic & Prevention Network (WA FAS DPN). Now in its 17th year of operation, the WA FAS DPN is a statewide network of diagnostic clinics all using the 4-Digit Diagnostic Code and contributing to a centralized electronic database.

Methods
The clinical database was used to generate comprehensive profiles of all patients evaluated for FASD from 1993-2005. These profiles were used to answer a multitude of clinical, research, and public health questions including: What is the demand for FASD diagnostic services, who is referred to the clinics, and what are their FASD diagnostic outcomes? Can FAS/D prevalence estimates from this clinical population be used to estimate FAS/D prevalence estimates in the general population? Do FASD diagnostic outcomes vary by race, age or alcohol exposure? Does the presence of other adverse exposures/events lead to more severe outcomes? Does this approach to diagnosis meet the needs of families?

Results
Demand for diagnosis remains very high. Of 1,400 patients (newborn to adult) with confirmed prenatal alcohol exposure, 11% were diagnosed with FAS/PFAS, 28% with static encephalopathy, 52% with neurobehavioral disorder, and 9% with no evidence of CNS abnormality. FASD outcomes varied significantly by age, race, gender, alcohol exposure, and presence of other risk factors. Families reported high satisfaction with the diagnostic process, and receipt of information/services they were unable to obtain elsewhere.

Conclusions
This report documents the immense contribution of a statewide FASD diagnostic program, and underscores the extraordinary value of a comprehensive FASD clinical dataset.

Key Words: *Fetal Alcohol Spectrum Disorders (FASD), FASD 4-Digit Diagnostic Code*

The Washington State Fetal Alcohol Syndrome Diagnostic & Prevention Network (WA FAS DPN) is a network of statewide, fetal alcohol spectrum disorder (FASD) diagnostic clinics linked by the core clinical/research/training clinic located at the Center on Human Development and Disability at the University of Washington (UW) in Seattle

Washington. The FAS DPN began as a single CDC-sponsored clinic at the University of Washington in 1993 in response to a national request for proposals for FASD prevention. The philosophy behind the UW proposal was…If you build a clinical diagnostic program that meets the needs of the families raising children with FASD, they would seek out the services of the clinic. In so doing, each time you identified (diagnosed) a child with FAS/D, you had an opportunity to identify and potentially intervene with a woman at high risk for bearing subsequent children with FAS/D (the child's birth mother). The results of that FASD primary prevention effort are presented in Astley et al.[1,2] When the UW FAS DPN clinic first opened in 1993, it was the first to introduce an interdisciplinary approach to FASD diagnosis.[3] The interdisciplinary team included a medical doctor, two psychologists, a speech-language pathologist, an occupational therapist, a social worker, and a family advocate. A gestalt[4] approach to FASD diagnosis was initially used, reflecting the most current guidelines available at the time. This gestalt approach was replaced in 1995 by a more rigorous, case-defined FASD diagnostic system (the FASD 4-Digit Diagnostic Code[5-8]) developed by the UW FAS DPN. The 4-Digit Code was formally released to the public in 1997, with updates in 1999 and 2004. During the first two years of operation, the single UW FASDPN clinic was overwhelmed by demand for FASD diagnostic services, far exceeding its capacity. In 1995, the Washington Chapter of the National March of Dimes provided funding to establish two satellite FASD clinics in two large cities just north (Everett) and south (Federal Way) of Seattle. In 1995, the state legislature through Senate Bill SB5688 mandated further expansion of the program to six satellite clinics (located in Everett, Federal Way, Tacoma, Yakima, Pullman, and Spokane) linked by the core UW clinic in Seattle, establishing the WA FAS DPN.[9] The WA FAS DPN is now in its 17th year of funding support from the state.

The mission of the WA FAS DPN is FASD prevention through FASD screening, diagnosis, intervention, research, and training. To this end, the WA FAS DPN has created a myriad of diagnostic tools, training programs, and screening programs (FASD 4-Digit Diagnostic Code and Lip-Philtrum Guides[5-8] (1997,1999,2004), FAS Facial Analysis Software[10-12] (2003); Foster Care FAS Screening Program[13,14](1999); FASD 4-Digit Code Online Training Course[15](2004)), all of which are available to clinical professionals free or at cost to maximize access. Over the decades, this interdisciplinary approach to FASD diagnosis using the FASD 4-Digit Code has been adopted worldwide.

The core mission of the FAS DPN has always been the advancement of the field through translational research (the rapid translation of clinical research into practice). The foundation of translational research is data management. From the FAS DPN's first day of operation in 1993, all data from the diagnostic clinics have been methodically collected and entered into an electronic clinical/research database with patient consent and Human Subjects Review Board approval. Over the years, this dataset has grown to over 8,000 cases, each with up to 2,000 fields of information, providing a comprehensive documentation of statewide demand for FASD evaluations and extensive detail on the antecedents and outcomes of these evaluations. This dataset supported the development of the diagnostic tools, screening programs, and training programs listed above, and serves as one of the largest research registries of individuals with FASD (n = 2,000) for enrollment into research studies that directly benefit individuals with FASD and their families.[16-25]

Over the years, the clinical field of FASD has come to adopt, as best practice, an interdisciplinary approach to FASD diagnosis using more rigorous, case-defined diagnostic guidelines.[6,7,26,27] Interdisciplinary FASD diagnostic clinics are being established worldwide. If these clinics are to successfully compete for limited health care dollars, it is essential to document their value. To demonstrate the extraordinary and unique value of a statewide interdisciplinary FASD diagnostic clinical program (and the essential role of data collection), the outcomes of the first 13 years of operation of the WA FAS DPN are presented below. The primary objectives of this study were to:

1. Construct a comprehensive profile (based on factors A-K below) of all 1,400 Washington State residents who obtained an FASD diagnostic evaluation at one of seven

WA FAS DPN clinics between 1993 and 2005.

2. Divide the clinical population into four FASD diagnostic subgroups (ranging from no adverse outcomes to severe adverse outcomes), construct a comprehensive profile of each subgroup (based on factors A-K below), and identify risk and protective factors that differentiate the four groups.

Factors

A. Sociodemographics
B. Birth mother and birth father characteristics
C. Growth
D. FAS facial features
E. CNS structural, neurological, and functional outcomes
F. Patient's behavioral profile: Summary of Caregiver Interview and Child Behavior Check List
G. Prenatal alcohol exposure
H. Other prenatal and postnatal risk factors
I. Prevalence of other syndromes
J. Prevalence of mental health disorders
K. Patient satisfaction with the FASD diagnostic process and access to intervention services.

Primary objectives 1 and 2 allow a multitude of clinical, research, and public health questions to be addressed. For example, if a statewide FASD diagnostic program is built, what is the demand for services, who is referred to the clinics, and what are their FASD diagnostic outcomes? Are there individuals with prenatal alcohol exposure who present with no evidence of adverse outcome? Can FAS/D prevalence estimates from the clinical population be used to estimate FAS/D prevalence estimates in the general population? Do FASD diagnostic outcomes vary by race, age, or level of prenatal alcohol exposure? What is the prevalence of mental health disorders and other syndromes in this patient population? Does the presence of other adverse exposures/events (e.g., prenatal exposure to illicit drugs, poor prenatal care, multiple home placements, physical/sexual abuse) lead to more severe dysfunction? Growth deficiency has always been a hallmark of FAS/D. How prevalent is growth deficiency in this patient population? The FASD literature suggests that infants and adults are less likely to present with the full FAS facial phenotype than school-aged children? Is this true? Should a diagnosis of FAS be rendered in an infant who presents with structural evidence of CNS abnormality (microcephaly), but is too young to assess and confirm the presence of CNS dysfunction (intelligence, executive function, memory, language)? Does the presence of the full FAS facial phenotype increase the correlation between microcephaly and brain dysfunction? Who are the birth mothers and birth fathers of these children? What proportion of these patients are still in the care of their birth parents? How satisfied are patients with the services provided by the clinics? Are they provided information/services they were unable to obtain elsewhere? These questions and many more are answered in this report.

METHODS

The Washington State FAS DPN electronic clinical/research database was utilized to construct a comprehensive profile of all 1,400 Washington State residents (birth through adult) who received an interdisciplinary FASD diagnostic evaluation using the FASD 4-Digit Diagnostic Code at one of the seven WA FAS DPN clinics in the first 13 years (1993-2005) of operation. The protocol was approved by the University of Washington Human Subjects Review Board.

Interdisciplinary FASD Diagnostic Model.
All WA FAS DPN clinics use the same interdisciplinary approach[3] to FASD diagnosis using the FASD 4-Digit Diagnostic Code.[6,7]

Interdisciplinary Model. The WA FAS DPN interdisciplinary teams include a pediatrician, two psychologists, a speech-language pathologist, an occupational therapist, a social worker and a family advocate. The patient population served by the WA FAS DPN has expressed strong preference for an evaluation that can be completed in one visit. Thus, a diagnostic evaluation is conducted in one 4-hour session.[3] In preparation for the evaluation, the patient's birth, medical, school, psychological, and social service records are collected by the clinic coordinator and pre-reviewed by the lead psychologist. On the day of the evaluation, the lead psychologist presents the patient's case history, including the outcomes of

any prior medical/psychological assessments, to the team in a 30-minute case conference. While the case-conference is being conducted, the patient's growth is measured and facial photograph is taken for computerized analysis.[10] After the case-conference, the pediatrician and lead psychologist conduct an interview with the caregiver(s) while the child is assessed over a 2-hour period by the second psychologist, speech-language pathologist, and occupational therapist. The child receives a brief physical examination by the pediatrician at the end of their 2-hour assessment. The caregiver interview and child assessment sessions focus on gathering information that is needed for diagnosis and not already present in the child's records. The battery of assessments administered to each patient (both historically and on the day of the diagnostic evaluation) vary by patient age and area of developmental concern. The team reconvenes for 1 hour to derive the FASD 4-Digit Code and generate an intervention plan. The diagnosis and intervention plan are shared with the family in the final 30 minutes of the evaluation. A single comprehensive medical summary documenting the diagnostic outcome, all data used to derive the diagnostic outcome, and intervention recommendations are submitted to the patient's medical record.

The FASD 4-Digit Code. The 4-Digit Code was developed by the UW FAS DPN in 1997 with the most recent 3rd edition published in 2004.[5-8,23] Briefly, the 4 digits of the FASD 4-Digit Code reflect the magnitude of expression of the 4 key diagnostic features of FASD, in the following order: 1. Growth deficiency, 2. FAS facial phenotype, 3. CNS structural/functional abnormalities, and 4. Prenatal alcohol exposure (Figure 1). The magnitude of expression of each feature is ranked independently on a 4-point Likert scale, with 1 reflecting complete absence of the FASD feature and 4 reflecting a strong "classic" presence of the FASD feature. Each Likert rank is specifically case defined. There are 256 possible 4-Digit Diagnostic Codes, ranging from 1111 to 4444. Each 4-Digit Diagnostic Code falls into 1 of 22 unique clinical diagnostic categories (labeled A through V). Seven of the 22 diagnostic categories (4-Digit Categories A–C and E–H) fall broadly under the designation of FASD (A. FAS/Alcohol Exposed, B. FAS/Alcohol Exposure Unknown, C. Partial FAS/Alcohol Exposed, E-F. Static Encephalopathy/Alcohol Exposed, and G-H. Neurobehavioral Disorder/Alcohol Exposed).

FIG. 1A) FASD 4-Digit Diagnostic Code grid. FASD is defined by growth deficiency, specific FAS facial features, evidence of CNS damage/dysfunction, and prenatal alcohol exposure. The 4-Digit Code ranks each of these areas on 4-point, case-defined, Likert scales. The 4-Digit Code (3444) inserted in the grid is 1 of 12 codes that meet the diagnostic criteria for FAS. **B)** FASD 4-Digit Code FAS facial phenotype (**view image**). The Rank 4 FAS facial phenotype determined with the 4-Digit Diagnostic Code requires the presence of all 3 of the following anomalies: (1) palpebral fissure length 2 or more standard deviations below the norm; (2) smooth philtrum (Rank 4 or 5 on the Lip-Philtrum Guide), an (3) thin upper lip (Rank 4 or 5 on the Lip-Philtrum Guide). Examples of the full Rank 4 FAS facial phenotype for Caucasian, Native American, African American, and Asian American children are shown.

FIG. 1A FASD 4-Digit Diagnostic Code Grid

				3	**4**	**4**	**4**		
Severe	Severe	Definite	(4)		X	X	X	(4)	High risk
Moderate	Moderate	Probable	(3)	X				(3)	Some risk
Mild	Mild	Possible	(2)					(2)	Unknown
None	None	Unlikely	(1)					(1)	No Risk
				Growth	Face	CNS	Alcohol		
Growth Deficiency	**FAS Facial Features**	**CNS Damage**							**Prenatal Alcohol**

Patient Referral Criteria and Diagnostic Capacity

The only criteria required for a patient to be seen in a WA FAS DPN clinic is a confirmed prenatal alcohol exposure history, at any level. The presence of the full FAS facial phenotype (4-Digit Face Rank 4) can be used in lieu of a confirmed alcohol history, since the Rank 4 facial phenotype, as defined by the 4-Digit Code is so specific to prenatal alcohol exposure.[11,12,14] The UW FAS DPN clinic provides evaluations to patients of all ages (newborn to adult). The other statewide FAS DPN clinics focus their services on pediatric populations. The diagnostic capacity of the WA FAS DPN has fluctuated over the years. Current funding levels support 130 evaluations per year: 80 at the UW FAS DPN and 50 at the four statewide FAS DPN clinics.

WA FAS DPN Electronic Clinical/Research Database

All data collected by the WA FAS DPN clinics since 1993 has been entered into an electronic clinical/research database with patient consent and Human Subjects Review Board approval. The majority of the data entered into the database come from two standardized data collection forms: 1) the New Patient Information Form, and 2) the FASD Diagnostic Form. These forms are provided in the Diagnostic Guide for FASD[6] and are posted on the FAS DPN website (**www.fasdpn.org**). The New Patient Information Form is completed by all families requesting an FASD diagnostic evaluation in a WA FAS DPN clinic. The form provides the clinic with key information regarding the patient's sociodemographics, growth, and development, lifetime prenatal and postnatal adverse exposures and events, including prenatal alcohol exposure, and social, educational, medical, psychological, psychiatric, and family history. The FASD Diagnostic Form is designed to capture all information required to derive and support the FASD 4-Digit Diagnostic Code (growth, facial features, CNS structural, neurological, functional measures, prenatal alcohol exposure, all other adverse prenatal and postnatal exposures, events, and conditions including all other physical anomalies and/or syndromes). The FASD Diagnostic Form is completed by the interdisciplinary team at the time of the FASD diagnostic evaluation. Data entered into the FASD Diagnostic Form include all data collected at the time of the FASD diagnostic evaluation as well as all information collected from previous records in preparation for the diagnostic evaluation (birth, medical, school, psychological, psychiatric, social service, placement, and legal records). All data collection forms are reviewed and prepared for data entry into an ACCESS[28] electronic database by SJA. Data is exported from ACCESS to SPSS[29] for statistical analysis. All 4-Digit Codes were upgraded to the most current 2004 version of the FASD 4-Digit Code.[6]

Study Population.

The following inclusion/exclusion criteria were applied to the WA FAS DPN database to establish the study population for this report.

Inclusion Criteria:

1. Received an FASD diagnostic evaluation at one of the seven WA FAS DPN clinics between 1993 and 2005.
2. Was a resident of Washington State at the time of their FASD diagnostic evaluation.
3. Had confirmed prenatal alcohol exposure, at any level. May have an unknown prenatal alcohol exposure history only if their FASD 4-Digit Code diagnostic outcome was full FAS (the Rank 4 FAS facial phenotype is so specific to prenatal alcohol exposure, it can be used in lieu of a prenatal alcohol exposure.[11,12,14,23])
4. Male or female, all ages, all races/ethnicities.

Exclusion Criteria:

1. None.

A total of 1,400 patients met the inclusion/exclusion criteria for this study. Patients evaluated in the WA FAS DPN after 2005 were not included in this study because their data are still in various phases of data entry, monitoring, and cleaning.

Study Groups

The study population was divided into four FASD diagnostic subgroups defined below. A recently completed FASD magnetic resonance study, conducted on a subset of this clinical population, confirmed these first three groups reflect three clinically meaningful and statistically distinct

FASD diagnostic subgroups.[16,23-25] Using the FASD terminology introduced by the Institute of Medicine[30], the SE/AE group most closely reflects 'severe Alcohol-Related Neurodevelopmental Disorder (ARND)' and the ND/AE group most closely reflects 'mild ARND'. The 4th group (Normal CNS/AE) by definition does not fall fully under the umbrella of FASD. This group represents individuals who have a confirmed prenatal alcohol exposure, but present with no evidence of adverse CNS outcomes. Some, but not all, present with growth deficiency and/or FAS facial features. The very existence of this group confirms that not all individual exposed to prenatal alcohol present with evidence of adverse outcomes. Inclusion of this group in this study presents an opportunity to identify potential 'protective' factors against prenatal alcohol exposure. The diagnostic features specific to each group were as follows:

1. *Patients in Group 1* had a 4-Digit diagnosis of **FAS or Partial FAS (FAS/PFAS)** (e.g., 4-Digit Diagnostic Categories A,B,C: with Growth Ranks 1-4, Face Ranks 3-4, CNS Ranks 3 and/or 4, Alcohol Ranks 2-4) (Figure 1). Alcohol Rank 2 (unknown exposure) could only be present if the patient had a diagnosis of full FAS because the Rank 4 FAS facial features are so specific to prenatal alcohol exposure.[11,12,14] In summary, patients in Group 1 had severe cognitive/behavioral dysfunction and the FAS facial phenotype.
2. *Patients in Group 2* had a 4-Digit diagnosis of **Static Encephalopathy / Alcohol Exposed (SE/AE)** (e.g., 4-Digit Diagnostic Categories E,F: with Growth Ranks 1-4, Face Ranks 1-2, CNS Ranks 3 and/or 4, Alcohol Ranks 3-4). In summary, patients in Group 2 had severe cognitive/behavioral dysfunction, comparable to Group 1, but did not have the FAS facial phenotype.
3. *Patients in Group 3* had a 4-Digit diagnosis of **Neurobehavioral Disorder / Alcohol Exposed (ND/AE)** (e.g. 4-Digit Diagnostic Categories G, H: with Growth Ranks 1-4, Face Ranks 1-2, CNS Rank 2, Alcohol Ranks 3-4). In summary, patients in Group 3 had prenatal alcohol exposure comparable to Groups 1 and 2, but in comparison to Groups 1 and 2 had only mild to moderate cognitive/behavioral dysfunction, and did not have the FAS facial phenotype.
4. *Patients in Group 4* had a 4-Digit diagnosis of **Sentinel Physical Findings/Alcohol Exposed** or **No Physical Findings or CNS Abnormalities Detected / Alcohol Exposed (Normal CNS/AE**) (e.g., 4-Digit Diagnostic Categories I and J: with Growth Ranks 1-4, Face Ranks 1-4, CNS Rank 1, and Alcohol Ranks 3-4. In summary, patients in Group 4 had prenatal alcohol exposure, no CNS abnormalities, and may or may not have had growth deficiency and/or FAS facial features.

Data Analysis

Objective 1: Descriptive statistics (means, SDs, proportions) were used to summarize the sociodemographic and clinical profiles of the clinical population as a whole, and each of the four diagnostic subgroups (1. FAS/PFAS; 2. SE/AE; 3. ND/AE; and 4. Normal CNS/AE). Proportions are expressed as valid column percents in all tables unless otherwise specified.

Objective 2: Empirical analyses were conducted to identify risk and protective factors that differentiated the four diagnostic subgroups. Chi-square tests (or Fishers Exact where appropriate) were used to compare proportions between 2 or more subgroups. T-tests were used to compare means between two groups. ANOVA was used to compare means between 3 or more groups. When ANOVA was employed, the overall f-statistic was used to test if differences existed among the group means. When the overall f-statistic was statistically significant, the Duncan post hoc range test was used to identify which group means differed. The Duncan test makes pairwise comparisons using a stepwise procedure. Means are ordered from highest to lowest, and extreme differences are tested first. The Duncan test sets a protection level for the error rate for the collection of tests. The Duncan test identifies homogeneous subsets of means that are not different from one another. For example, if the outcome of a Duncan test is presented as 1,23,4, this means the mean for groups 2 and 3 were comparable to one another, but significantly higher and lower than the means for groups 1 and 4 respectively. Two-tailed p-values of 0.05 were used throughout the analyses. Due to multiple comparisons, resulting p-values should be interpreted accordingly.[31,32] As a

general point of reference (since sample size varied with each analysis), this study had 80% power or greater to detect the following effect sizes (at a two-tailed alpha level of 0.05) when a study group had 65 or more subjects: 1) A difference in means one-half the standard deviation of the mean difference; 2) A 24-point or greater difference in proportions between two groups.

RESULTS

Demand for FASD Diagnostic Services and Ability to Meet the Demand

Although the WA FAS DPN provides FASD diagnostic evaluations to patients from all over the U.S., the vast majority (95%) reside in WA State. Demand for FASD diagnostic services has always exceeded the FAS DPN's capacity, but expansion from the single clinic to a statewide network of clinics doubled its capacity and increased access to FASD diagnostic services. The WA FAS DPN's current capacity is 130 diagnostic evaluations per year. A total of 6,586 families from WA State requested an FASD diagnostic evaluation between 1993 and 2005; on average 506 per year. Patients request an appointment by sending their name and address to the clinic via voicemail or email. All patients requesting an appointment are sent an information packet that includes a description of the clinical services and a New Patient Information Form (NPIF). Patients are requested to complete the NPIF and submit it to the clinic for review. The NPIF documents why a diagnostic evaluation is being requested, what the developmental concerns are, if any, and whether the patient has a confirmed prenatal alcohol exposure. Of the 6,586 requests, 3,004 (47%) completed and submitted the NPIF. In a survey conducted in the mid 1990's, the primary reason stated for not submitting the NPIF was lack of a confirmed prenatal alcohol exposure. Oftentimes, families are requesting evaluations because they are concerned about their child's development, have confirmation of maternal illicit drug use during pregnancy and therefore suspect prenatal alcohol exposure. It has become clear after 17 years of clinical record review that when women use illicit drugs and alcohol during pregnancy, their illicit drug use is far more likely to be documented in medical or social service records than their alcohol use. Of the 3,004 NPIFs submitted, 2,462 (82%) appeared to have a confirmed prenatal alcohol exposure and were thus eligible to be evaluated in clinic. The 18% without a confirmed prenatal alcohol exposure were referred to other appropriate clinics (typically neurodevelopmental clinics). Of the 2,462 patients deemed eligible to be evaluated in the FAS DPN clinics, 1,668 (68%) received a diagnostic evaluation between 1993 and 2005. The average wait to be seen in a clinic, from the time the NPIF was submitted, was 6.7 months. Of the 1,668 patients evaluated in the clinics, 268 were deemed to have an unknown prenatal alcohol exposure, despite what appeared to be a confirmed prenatal alcohol exposure at the time the evaluation was requested. Exclusion of the 268 patients with unknown alcohol exposure produced the sample of 1,400 patients summarized in this report.

FASD Diagnostic Outcomes (Table 1)

Of the 1,400 residents of WA state evaluated in the WA FAS DPN in the first 13 years of operation, 4% were diagnosed with FAS, 7% had PFAS, 28% had Static Encephalopathy (without the FAS facial phenotype), 52% had Neurobehavioral Disorder, 2% presented with growth deficiency and/or FAS facial features, but no evidence of CNS abnormalities, and 7% presented with no growth deficiency, no FAS facial features, and no evidence of CNS structural, neurological, or functional abnormalities, despite their prenatal alcohol exposure. The core clinic at the University of Washington provided diagnostic evaluations for 930 (66%) of the 1,400 patients. The remaining 470 were evaluated at one of the six other FAS DPN statewide clinics. The distribution of FASD diagnoses rendered by the core UW FAS DPN clinic was comparable to the distribution of FASD diagnoses rendered by the six other statewide FAS DPN clinics.

Sociodemographic Profile (Table 2)

Although patients of all races and ethnicities were evaluated in the FAS DPN clinics, the racial distribution of the clinical population was significantly different from the racial distribution of the state ($chi^2 = 100$, $p < 0.000$). The WA State 2000 census reported the following distribution of single races: White 82%, American Indian/Native

Alaskan 2%, Black 3%, Asian 6%).[33] By comparison, Caucasians (48.9%) and Asians (0%) were under-represented in the clinical population and Black (6.6%) and American Indian/Native Alaskan (8.2%) groups were over-represented. Males were significantly more prevalent (58%) than females (42%) (chi^2 18, $p< 0.000$). The vast majority of the population (90%) was under 16 years of age with a mean age of 9.9 years (6.2 SD) and an age range of 7 days old to 50.8 years old. Only 22% percent of the patients were accompanied to clinic by their birth mother. The vast majority (70.5%) were not residing with their birth mother or birth father at the time of their diagnostic evaluation.

Contrasts between FASD Diagnostic Subgroups

Factors A-K below are compared and contrasted between the four clinical subgroups (1. FAS/PFAS, 2. SE/AE, 3. ND/AE, and 4. Normal CNS/AE).

Factors

- **A.** Sociodemographics (Table 2)
- **B.** Birth mother and birth father characteristics (Table 3)
- **C.** Growth (Table 4)
- **D.** FAS facial features (Table 4)
- **E.** CNS structural, neurological, and functional outcomes (Tables 5 and 6)
- **F.** Patient's behavioral profile: Summary of Caregiver Interview and Child Behavior Check List[34] (Tables 7 and 8, Figures 2 and 3)
- **G.** Prenatal alcohol exposure (Table 9)
- **H.** Other prenatal and postnatal risk factors (Table 10)
- **I.** Prevalence of other syndromes (Table 10)
- **J.** Prevalence of mental health disorders (Table 11)
- **K.** Patient satisfaction with the FASD diagnostic process and access to intervention services. (Table 12)

Use of the FASD 4-Digit Code by seven statewide, interdisciplinary teams, over a period of 13 years, produced three clinically and statistically distinct FASD clinical subgroups. The three subgroups (ND/AE, SE/AE and FAS/PFAS) reflected a linear continuum of increasing neuropsychological impairment and physical abnormality, representing the full continuum of FASD.

DISCUSSION

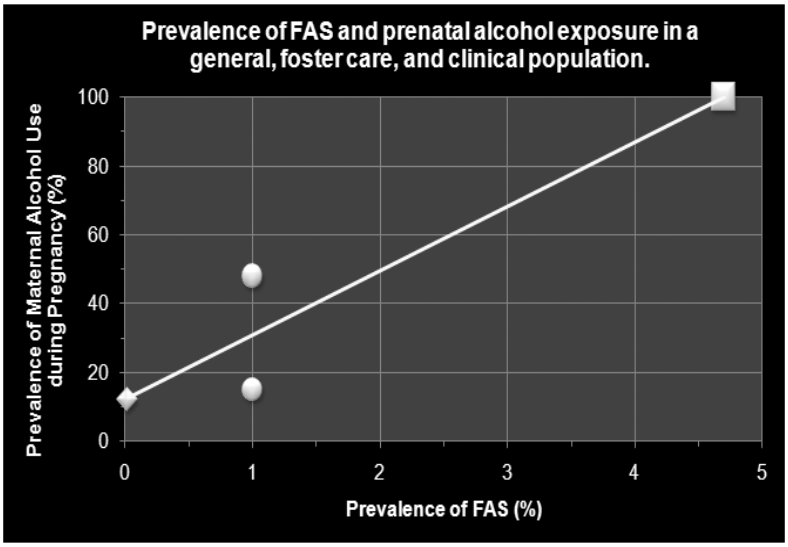
An infinite array of clinical, research, and public health questions can be addressed using the WA FAS DPN clinical dataset. The answers to a selection of questions are presented and discussed below to document the immense value and contribution of a statewide FASD diagnostic program, and underscore the extraordinary value of a comprehensive FASD clinical dataset.

1. Does the prevalence and distribution of the FASD diagnostic outcomes observed in this statewide *clinical* population reflect the prevalence and distribution one would expect to observe in the statewide *general* population?

No. The prevalence of FASD will be higher in this clinical population than in the general population for two reasons: 1) all individuals in this clinical population have a prenatal alcohol exposure, and 2) individuals experiencing difficulties are more likely to be referred to a clinic than those not experiencing difficulties. How much higher will the prevalence be? Below are some FASD prevalence estimates from other population samples (and their corresponding alcohol-exposure estimates) to compare to our clinical sample. The prevalence of FAS in our statewide clinical population was 4.2%. One hundred percent had a confirmed prenatal alcohol exposure. The prevalence of FAS in the King County subset of our statewide clinical population (were Seattle and the University of Washington are located) was 4.7%. Again, 100% had a confirmed prenatal alcohol exposure. In comparison, the prevalence of FAS in a foster care population residing in King County (as documented by a 10-year, active case-ascertainment FAS screening program) was 1.5%.[14] Fifteen percent of this foster care population had a documented prenatal alcohol exposure in their foster records. Forty-eight percent had a confirmed or suspected prenatal alcohol exposure in their foster records. Thus the true prevalence of prenatal alcohol exposure in this foster population was likely somewhere between 15% and 48%. The FAS prevalence estimates from these clinical and high-risk foster populations are 15 to 47 times greater than the FAS prevalence estimate often cited for the general U.S. population (0.1 – 0.3%).[35] National surveys of the general population

estimate 12% of women report drinking during pregnancy.[36] If one plots the prevalence of FAS to the prevalence of alcohol exposure across these three populations (clinical, foster care, and general), an interesting trend appears (Figure 4).

FIG. 4 Prevalence of FAS and prevalence of maternal alcohol use during pregnancy in three populations: ♦General U.S. population (FAS = 0.2%[35], alcohol use = 12.2%[36]). ●King County WA foster care population (FAS = 1%, alcohol use = 15% to 48%).[14] ■King County WA FAS Diagnostic & Prevention Network (FAS DPN) clinical population (FAS = 4.7%, alcohol use = 100%). Best fit linear trend line: y = 18.989x + 12.352; R-squared = 0.89. FAS: fetal alcohol syndrome.



Another related question that is often raised is: How much more prevalent is "ARND" than FAS? The prevalence of SE/AE and ND/AE combined (what other diagnostic systems refer to as ARND[27,30]) was 7.2-fold greater than the prevalence of FAS/PFAS in our clinical population. Does this mean there are 7 times more individuals with ARND than FAS in the *general* population? The true ratio is likely higher for the following reason. Since individuals with severe outcomes are more likely to be referred to a clinic than individuals with less severe outcomes, diagnostic subgroups with the most severe outcomes will likely be disproportionately over-represented in a clinical population. Thus, if FAS is more severe than SE/AE, the prevalence of SE/AE to FAS would likely be higher in the general population than was observed in this clinical population. The published literature suggests ARND is as least three times more prevalent than FAS.[37] Unfortunately, the published literature does not specifically case-define ARND or FAS, so it is difficult to know which of our clinical subgroups to compare them to. The ratio of ARND to FAS, generated from our clinical population, ranges from a low of 2.6-fold (if ARND is defined as SE/AE+ND/AE and FAS is defined as FAS+PFAS) to a high of 18.9-fold (if ARND is defined as SE/AE + ND/AE and FAS is defined as FAS). No matter how one chooses to define ARND and FAS, our clinical data strongly suggest "ARND" is at least 3-fold greater than FAS, but likely much higher. In summary, prevalence estimates derived from clinical populations will exceed those of the general population, but clinical estimates can play an important role in formulating estimates for the general population. An FASD diagnostic clinic is a form of passive population-based FASD screening. The individuals referred are the subset of the general population who were identified by community professionals as at-risk and in need of diagnostic and intervention services.

2. Did the prevalence estimates for FAS/PFAS, SE/AE, and ND/AE vary by race? Yes (Table 13). And these variations were correlated with racial variations in drinking patterns during pregnancy. The prevalence of FAS/PFAS was significantly higher among Caucasians (12.7%) and Blacks (18.5%) than among American/Alaskan Natives (5.2%) (Caucasian versus Native: Chi=5.4, p=0.02; Black versus Native: Chi=9.1, p=0.003). Caucasians and Blacks also reportedly drank significantly more days per week during pregnancy (on average 4.6 and 5.7, respectively) than American/Alaskan Natives (on average 3.6). Interestingly, the only measure of prenatal alcohol exposure that significantly differentiated FAS/PFAS from all other FASD diagnoses, across the entire study population of 1,400, was a higher mean number of days per week of drinking during pregnancy. This same finding was observed in the recently completed FASD magnetic resonance study.[24] Since the window of vulnerability for producing the FAS facial features appears to be very short in duration (a few hours in the mouse[38], a few days in the nonhuman primate[39]), perhaps the more days per week of drinking, the more likely drinking will occur during this narrow window of vulnerability. In contrast to FAS/PFAS, the prevalence of SE/AE "severe ARND without the FAS facial features" was significantly higher in American/Alaskan Natives (41.7%) than in Caucasians (26.6%) or Blacks (20.7%). American/Alaskan Natives reportedly drank a significantly higher number of drinks per drinking occasion during pregnancy than Caucasian or Blacks. Perhaps binge drinking places a fetus at greater risk for CNS structural/functional abnormalities, whereas more frequent drinking increases the odds of also having the FAS facial features.

3. Did the prevalence estimates for FAS/PFAS, SE/AE, and ND/AE vary by age? The prevalence of FAS/PFAS did not vary significantly by age at diagnosis: 0-3.9 yrs (15%), 4-5.9 yrs (9%), 6-10.9 yrs (11%), 11-15.9 yrs (9%), 16+ yrs (10%); Chi=6.3 (p=0.18). An infant was as likely to receive a diagnosis of FAS/PFAS as an adult. As a point of reference, the prevalence of FAS/PFAS across the entire study sample of 1,400 was 11 %. The prevalence of ND/AE varied from 45.3% to 58.4% across the age categories, but these variations were not statistically significant. Again, for reference, the prevalence of ND/AE across all 1,400 subjects was 51.6%. The prevalence of SE/AE did vary significantly by age. Children under the age of 6 years were significantly less likely to receive a diagnosis of SE/AE than older individuals. This may be explained, in part, by the fact that a key clinical feature of SE/AE is significant dysfunction across three or more domains of cognitive/behavioral function. A child typically is not old enough to engage in an assessment of higher level functioning (executive function, memory, language, etc) until they are 7 to 8 years of age. But an individual does not have to have significant dysfunction to meet the CNS criteria for SE/AE. They could meet the criteria with microcephaly. In fact, the CNS criteria for FAS/PFAS and SE/AE are identical (presence of a CNS structural/neurological abnormality and/or significant dysfunction across 3 or more domains of brain function).[6] So why are individuals with SE/AE significantly older (mean = 10.1 years) than individuals with FAS/PFAS (mean = 8.9 years) if the CNS criteria to achieve these two diagnoses are identical? Remember, the only feature that distinguishes FAS/PFAS from SE/AE is the FAS facial phenotype. As it turns out, those with the FAS facial phenotype are significantly more likely to have microcephaly (the prevalence of microcephaly among FAS/PFAS was 45%) than those with comparable brain dysfunction, but no FAS facial phenotype (the prevalence of microcephaly among SE/AE was 25%). This same finding was observed in the recently completed FASD MRI study.[16] More specifically, individuals with FAS/PFAS had significantly and disproportionately smaller frontal lobes than individuals with SE/AE. Since head circumference can be accurately assessed in children less than 8 years of age, but a comprehensive assessment of brain dysfunction cannot, the higher prevalence of microcephaly among the FAS/PFAS group produces a diagnostic subgroup that is significantly younger than the SE/AE subgroup. This observation leads to the next question.

4. Is it clinically cogent to render a diagnosis of FAS in an infant who presents with structural evidence of CNS abnormality (microcephaly), but is too young to assess and confirm the

presence of brain dysfunction (intelligence, executive function, memory, language, etc)? Is the presence of microcephaly in an infant with the FAS facial phenotype predictive of brain dysfunction that will not be revealed until an infant is old enough to participate in higher level functional assessments? The answers to both questions are yes. Among the 154 patients with FAS/PFAS, 69 (44.8%) had microcephaly (Table 5). Of the 69 with microcephaly, 36 (52%) had no evidence of brain dysfunction (Rank 1), 14 (20%) had moderate (Rank 2) brain dysfunction, and 19 (28%) had severe (Rank 3) brain dysfunction. Did the 52% with no evidence of brain dysfunction, truly have normal function, or were they too young to accurately/comprehensively assess function? The data would suggest they were too young to assess. The subset with no evidence of brain dysfunction (Rank 1) had a mean age of 4.7 (6.0 SD) years. The subset with Rank 2 moderate dysfunction had a mean age of 7.5 (5.9 SD) years. And the subset with Rank 3 severe dysfunction had a mean age of 10.3 (5.9 SD) years. The older the patient, the more likely they revealed evidence of moderate to severe dysfunction (ANOVA F=5.8 (df 2), p=.005). This data suggests rendering a diagnosis of FAS/PFAS in a newborn/infant that presents with microcephaly, but is too young to assess/confirm brain dysfunction, is clinically sound. The combined presence of the FAS facial phenotype, microcephaly, and prenatal alcohol exposure serves as a strong risk factor for (predictor of) brain dysfunction. The correlations between increasing magnitude of expression of the 4-Digit FAS facial phenotype and 1) increasing CNS dysfunction ,and 2) decreasing head circumference are quite high (Figures 5A,B).[11,16] Early diagnosis affords early intervention. Postponing a FAS/PFAS diagnosis in children with microcephaly, who were not old enough to participate in higher-level functional assessments to confirm brain dysfunction, could lead to missed opportunities for early intervention.

FIG. 5A) The mean Performance Intelligence Quotient (PIQ) standard score (WISC III [40]) decreased significantly as the FAS facial phenotype increased in magnitude from 4-Digit Face Rank 1 to 4 (ANOVA: F 2.7(3df), p =.046).

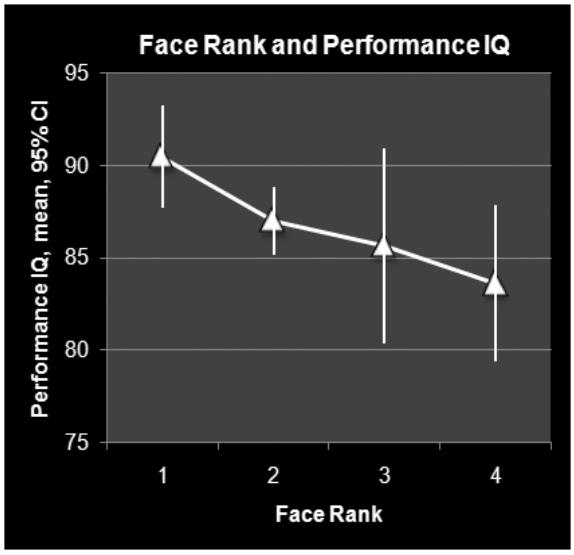
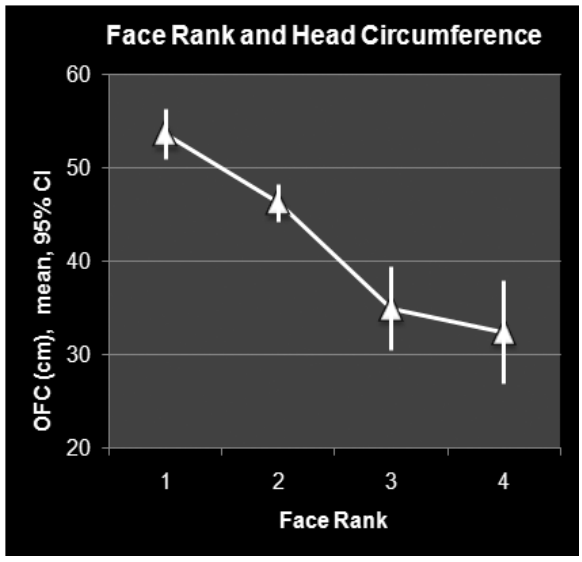


FIG. 5B) The mean occipital frontal head circumference (OFC) in centimeters (cm) decreased significantly as the FAS facial phenotype increased in magnitude from 4-Digit Face Rank 1 to 4 (ANOVA: F 26 (3df), p < .001).



5. Do the CNS functional profiles of the FAS/PFAS, SE/AE and ND/AE groups differ when generated by a single, age-appropriate, comprehensive neuropsychological battery administered to all patients, as compared to when generated by variable neuropsychological batteries that may focus more on deficits than strengths (as is often the case in clinical settings)? The CNS functional profiles of the FAS/PFAS, SE/AE, and ND/AE groups presented in Table 6 were generated from two primary sources of data: 1) past school/psychological assessments, and 2) current assessments conducted at the time of the FASD diagnostic evaluation. These school and clinic-based assessment protocols are more likely to target areas of deficit (rather than areas of strength) because the primary goals of these assessments are to determine if an individual qualifies for school-based services or meets established FASD diagnostic criteria. As a result, no two patients in the FAS DPN clinical dataset necessarily received the same test battery, and their test batteries likely focused more on their deficits than their strengths. This could lead to group profiles that under estimate the mean performance levels of each group as a whole. If every patient received the same age-appropriate test battery, and the battery assessed all areas of function, not just the areas with perceived deficits, how different might the profiles be? The recently completed FASD magnetic resonance research study provided an opportunity to answer this question.[24] Sixty-five children across the full continuum of FASD were randomly selected for enrollment into the magnetic resonance study from among these 1,400 WA FAS DPN patients. As a standard of research protocol, a single, comprehensive neuropsychological battery was administered to all 65 children.[24] The CNS functional profiles generated by the single, comprehensive research battery[24] were near identical to the functional profiles generated by the more variable, less comprehensive clinical batteries (Table 6). For example, the mean FSIQ[40] standard scores for the FAS/PFAS, SE/AE, and ND/AE groups in the magnetic resonance study were 77.5, 79.3, and 99.2 respectively. The mean Visual Motor Integration[41] total standard scores for the FAS/PFAS, SE/AE, and ND/AE groups in the magnetic resonance study were 76.2, 81.4, and 90.9 respectively. The mean Rey Complex Figure Test[42] Copy Raw Scores for the FAS/PFAS, SE/AE, and ND/AE groups in the magnetic resonance study were 17.4, 20.5, and 25.6 respectively. These outcomes suggest the CNS profiles presented in Table 6 were not markedly influenced by the variable clinical batteries used to generate them.

6. Do FASD diagnostic outcomes vary by level of prenatal alcohol exposure? Yes (Table 9). Individuals with FAS/PFAS had a significantly higher mean number of days per week (5.6) of prenatal alcohol exposure than individuals with a comparable level of CNS dysfunction, but no facial features (SE/AE) (4.3 days/week) ,or individuals with less severe CNS dysfunction and no facial features (ND/AE) (4.4 days/week). This same finding was observed in the FASD magnetic resonance study.[24]

7. Is the presence of other adverse exposures/events (e.g., prenatal exposure to illicit drugs, poor prenatal care, multiple home placements, physical/sexual abuse) associated with more severe developmental outcomes? Yes (Table 10). Prenatal alcohol exposure was rarely, if ever, the sole risk factor present among patients evaluated at the WA FAS DPN. One third of the population had no documented prenatal care. Ninety-three percent had other adverse prenatal exposures (e.g., tobacco, illicit drugs). Seventy percent were no longer in the care of their birth parents and had on average three out-of-home placements. At least 34% were physically abused and 24% sexually abused. Seventy-five percent had one or more mental health disorders documented in their medical record. The prevalence of adverse exposures and events was for the most part, comparably high across the three FASD groups (e.g., tobacco, illicit drug use, neglect, out-of-home placements). Occasionally the prevalence increased incrementally with increasing severity of FASD diagnostic outcome from NE/AE to SD/AE to FAS/PFAS (e.g., no prenatal care). Most striking, however, were the contrasts observed between the three FASD groups and Group 4 (the group with no evidence of CNS abnormality). Physical and sexual abuse was 2- to 5-fold more prevalent in the FASD groups than in Group 4. Children in the FASD

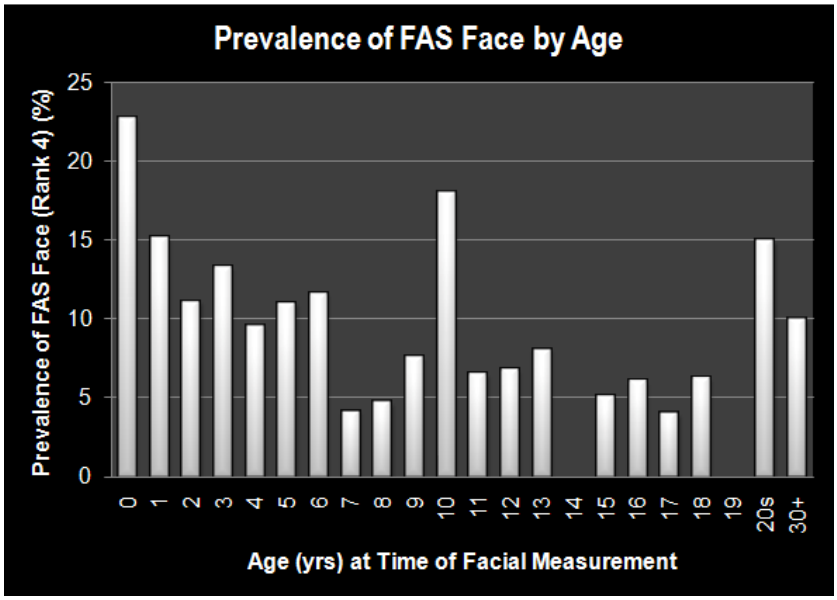
groups were twice as likely to be in adoptive care and significantly less likely to receive prenatal care than children in Group 4. Prenatal exposure to alcohol and other illicit drugs was comparably high across all four groups.

8. What proportion of individuals with prenatal alcohol exposure present with no evidence of CNS structural, neurological, or functional abnormalities? Were they exposed to less alcohol? Of the 1,400 subjects with prenatal alcohol exposure, 130 (9.3%) presented with no evidence of CNS abnormality (Table 1). Ninety-six of the 130 subjects in Group 4 presented with no growth or FAS facial features either. Although one might expect to see a lower reported alcohol exposure among this group, their reported level of exposure was comparable to that of the SE/AE and ND/AE groups. Three features that did distinguish this unaffected group from the other groups were their gender, age, and postnatal adverse experiences. The unaffected group was significantly more likely to be female (57.7%) than the other FASD clinical subgroup (FAS/PFAS 48.1%; ND/AE 41.6%; SE/AE 35.3%). And the unaffected group was significantly younger (46.2% were under 4 years of age) compared to 25.3% among the FAS/PFAS, 10.7% among the SE/AE, and 16.2% among the ND/AE. It is likely that some of the subjects in the unaffected group were classified as functionally within the normal range because they were too young to assess and rule-out higher level functional deficits. The only way an infant could meet the CNS functional criteria for SE/AE is with significantly delayed mental and motor development (e.g., Mental and Motor Developmental Index standard scores of 50 on the Bayley Scales of Infant Development[43]). But young age does not explain why the 130 patients in Group 4 did not meet the CNS functional criteria for ND/AE. A third factor that markedly differentiated the unaffected group from the three affected groups was adverse postnatal experiences. As reported above, the unaffected group was significantly less likely to experience high-risk (Rank 4) postnatal adverse events like physical or sexual abuse.

9. How often are other syndromes present in this patient population? Eighteen (1.3%) of the 1,400 subjects were diagnosed with other syndromes (Table 10). Only one of the seven clinics had a dysmorphologist on their interdisciplinary team. When the prevalence estimate was restricted to the 664 patients seen at the UW FAS DPN between 1993 and 1999 when a dysmorphologist served as the pediatrician on the team, 13 (1.9%) were identified with other syndromes. When syndromes other than FAS were suspected by the other pediatricians on the teams (the pediatricians who were not dysmorphologists or geneticists), the patients were referred to a geneticist. Of the 736 patients seen by the other pediatricians 5 (0.7%) were documented to have another syndrome and an additional 8 (1.1%) were suspected to have another syndrome and were referred to a geneticist. Thus, the pediatricians documented or suspected the same proportion of patients with other syndromes (1.8%) as was diagnosed when a dysmorphologist was on the team (1.9%). It is worth noting that one child diagnosed with FAS in the WA FAS DPN also had Down syndrome. Alcohol is a teratogen to all developing fetuses, including those with genetic disorders. The child presented with growth deficiency below the 2nd percentile on a growth chart for children with Down syndrome. The child presented with the facial features of Down syndrome and FAS. The facial features of Down syndrome are distinct from the facial features of FAS. The two phenotypes were readily apparent and easily distinguished. The child presented with microcephaly (3 SDs below the mean for boys with normal development, 1 SD below the mean for children with Down syndrome). The child presented with Bayley[43] Motor and Mental Index scores below 50; a level of developmental delay that can be observed in both Down syndrome and FAS. The birth mother was reported to have consumed alcohol daily throughout pregnancy.

10. The FASD literature suggests that infants and adults are less likely to present with the full FAS facial phenotype than school-aged children. Is there evidence of this in this dataset? No. The proportion of subjects who presented with the full FAS facial phenotype (Rank 4) by age group was as follows: birth to 3.9 yrs (36/258, 14%), 4 to 16.9 years (78/1001, 7.7%), and 17 to 53 years (12/141, 9.5%). The age group with the highest prevalence of the FAS facial phenotype was infants under one year of age (23%) (Figure 6).

FIG. 6 Proportion of patients in each age group who presented with the full Rank 4 FAS facial phenotype at the time (age) of their FASD diagnostic evaluation.



11. Growth deficiency has always been a hallmark of FAS/D. How prevalent is growth deficiency in this patient population? Only 34.1% of the 1,400 subjects presented with height and/or weight below the 10th percentile (Growth Ranks 2, 3 or 4). Only 7.9% presented with height and weight below the 3rd percentile (Growth Rank 4). Of the patients with FAS/PFAS, 35.7% presented with no growth deficiency (Growth Rank 1: height nd weight above the 10th percentile) and thus received a diagnosis of PFAS.

12. Who are the birth fathers of these children? The names of 76% of the birth fathers were known, compared to 95% of the birth mothers. The more severe the FASD diagnostic outcome, the less likely the birth father's name was documented in the child's records. Only 7.6% of them accompanied their child to the FASD diagnostic evaluation. The fathers were on average 29 years old at the birth of their child with FASD and 38 years old at the time the child was being diagnosed with FASD (Table 3). Thirty-nine percent did not finish high school, 45% completed high school, and 16% attended college. They were in general, older and more highly educated than the birth mothers. Approximately half of them reportedly had learning disabilities.

13. What proportion of patients were no longer in the care of their birth parents at the time of their FASD diagnostic evaluation? Seventy percent of the patients were no longer in the care of either birth parent at the time of their FASD diagnostic evaluation (Table 1). The average number of home placements across the 1,400 patients was 2.9 $\pm$ 3.1. Nineteen percent had four or more home placements.

14. Does a caregiver's impression of their child's behavior differ between FAS/PFAS, SE/AE, and ND/AE? Yes and No. Among the 1,270 caregivers who completed the Child Behavior Checklist[34] (for children 6 to 18 years of age), the prevalence and magnitude of behavior problems was comparably high across all FASD subgroups. Attention problems were reported most often (Table 7, Figure 2). When the results of the 2-hour, structured interview with the caregiver(s) (conducted by the medical doctor and psychologist on the day of the FASD diagnostic evaluation) were tabulated (Table 8, Figure 3), the

prevalence and magnitude of behavioral concerns often increased significantly and incrementally as one advanced from the ND/AE to SE/AE to FAS/PFAS group. It is important to note that these parent impressions of their child's behavior were recorded before the parent or the clinicians knew the FASD diagnostic outcome of the child.

FIG. 2 Child Behavior Check List[34] (CBCL/ 6-18) Syndrome Scales (*see* Table 7) among the 516 patients administered a CBCL when they were between 6 and 18 years of age. All abbreviations are defined in Table 7.

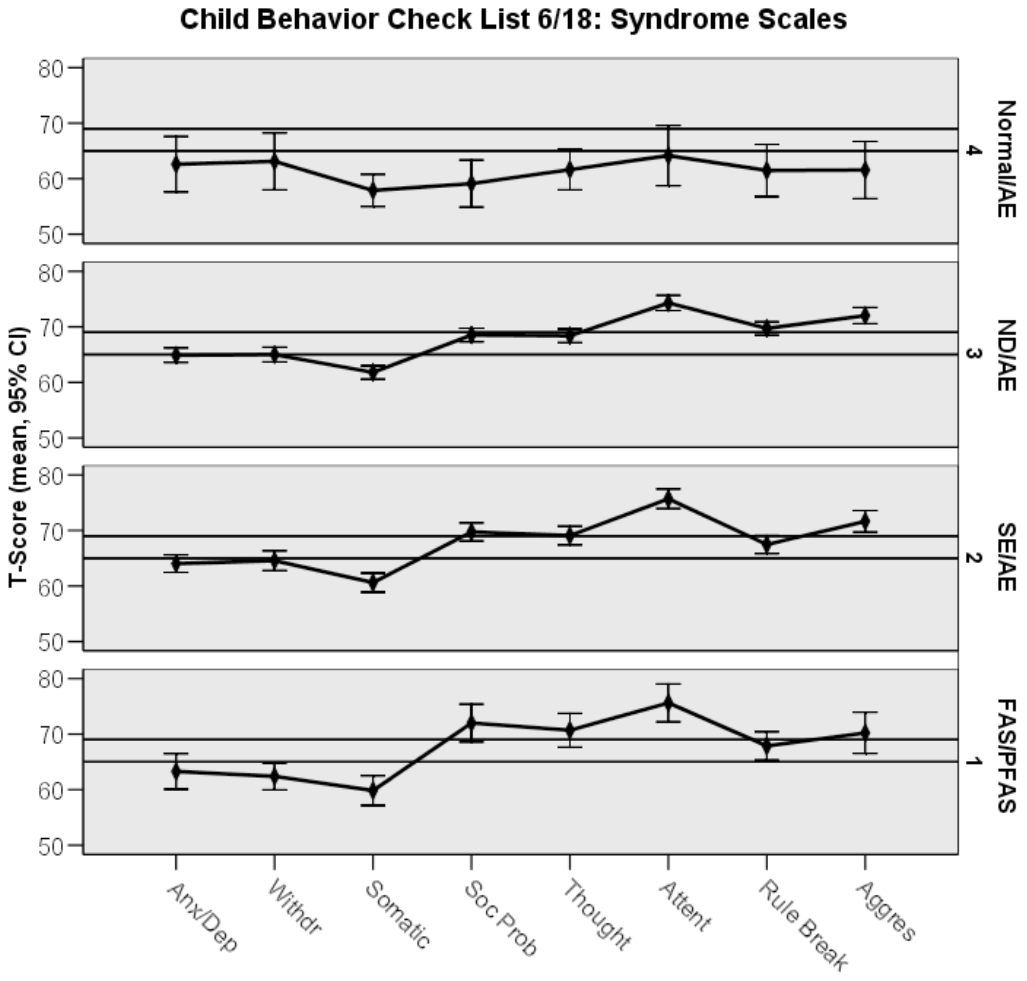


FIG. 3 Proportion of patients classified by the pediatrician as 'significantly delayed/impaired' in behaviors addressed in a 2-hour, structured caregiver interview administered jointly by the pediatrician and psychologist during the FASD diagnostic evaluation. This is a graphical presentation of the data presented in Table 8 to illustrate the cumulative increase in impairment as one advances across the study groups. Each color in a bar reflects the behaviors listed in Table 8 under each Domain. Abbreviations are defined in Table 8. The number printed in each colored section is the proportion of patients with significant impairment for that behavior. For example, the bar for the FAS/PFAS group in the Planning Domain reflects the following: blue square (24% present with significant impairment for "Needs considerable help organizing daily tasks"); red square (31.3% present with significant impairment for "Cannot organize time"); green square: (31.5% present with significant impairment for "Does not understand concept of time"); and purple square (26.5% present with significant impairment for "Difficulty carrying out multistep tasks").

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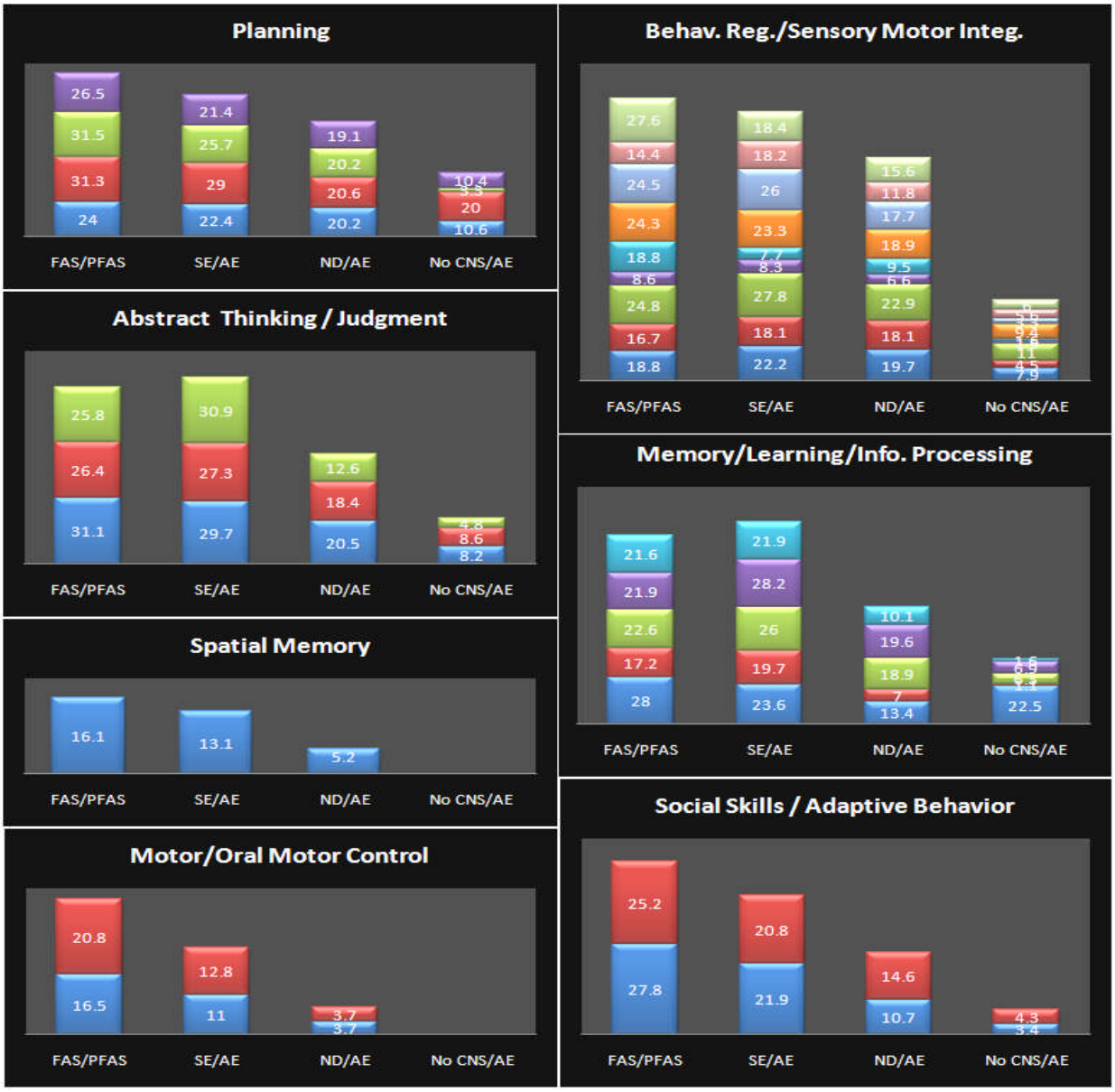

15. What is the prevalence of other mental health disorders in this patient population? Among the 1,064 patients, five or more years of age at the time of their FASD diagnostic evaluation, 82% had one or more mental health disorders documented in their medical records (Table 11). The most prevalent was ADD/ADHD (53.9%). Despite this high overall prevalence, the prevalence estimates for each disorder (based on review of medical records available to the FASD clinics) may substantially under-estimate the true prevalence of each disorder. Many of these disorders fail to be formally diagnosed and recorded in the medical record. When a representative subset of these children (n=65) were administered the Computerized Diagnostic Interview Schedule for Children[44] during their enrollment in the FASD magnetic resonance study[24], the prevalence estimates for many disorders were substantially higher. For example, Oppositional Defiant Disorder was reported in 6.8% of this clinical population, but was diagnosed in 52% of the subset that participated in the magnetic resonance study. Obsessive compulsive disorder was reported in 0.7% of this clinical population, but was diagnosed in 9.2% of the subset that participated in the magnetic resonance study.

16. Were patients satisfied with the interdisciplinary FASD diagnostic evaluation process? Were they provided information they were unable to obtain elsewhere? Was the 4-Digit Code approach to diagnosis easy to understand? Was their ability to access and benefit from recommended intervention services influenced by what diagnosis their child received under the umbrella of FASD? Would they recommend the clinic to other families with similar needs? A 10-question patient satisfaction survey has been sent to all patients evaluated at the UW FAS DPN clinic since 1993. The survey may be completed anonymously and comes with a stamped-addressed return envelope to maximize participation in the survey. Patients universally expressed high satisfaction for the FASD diagnostic services provided by the University of Washington (Table 12). Ninety-nine percent would recommend the Clinic to other families with similar needs. Ninety-one percent said they received information they were unable to obtain elsewhere, despite the fact the clinic is located in a large metropolitan area (Seattle) with many genetic, neurodevelopmental, and psychological evaluation services available. Eighty-six percent found the explanation of the diagnosis using the 4-Digit Code easy to understand. And perhaps most informative; family's whose child received a diagnosis of SE/AE or ND/AE were as likely to report successfully accessing and benefiting from recommended intervention services as family's whose child received a diagnosis of FAS/PFAS. This is in contrast to the oft stated belief that a family will not qualify for services if the diagnosis is not FAS/PFAS or at least given a name that implies alcohol is the causal agent (e.g., ARND). Overall, 82.1% of families reported being somewhat to very successful in finding the recommended intervention services and 83.7% reported these services met some to all of their needs.

Strengths and Limitations

The outcomes presented in this report reflect a very large, 13-year, statewide, clinical population of patients (newborn to adult) who all received an identical, interdisciplinary approach to FASD diagnostic evaluation using the FASD 4-Digit Code. By virtue of this, the outcomes presented in this report are highly representative of the study's intended target population (a statewide clinical population of individuals with prenatal alcohol exposure seeking an FASD diagnostic evaluation). The outcomes presented in this report should not be construed to represent the population of *all* individuals exposed to prenatal alcohol exposure. Even though the only requirement to obtain an FASD diagnostic evaluation in the WA FAS DPN is a confirmed prenatal alcohol exposure, alcohol-exposed individuals with developmental concerns are more likely to be referred to the clinic than alcohol-exposed individuals with no developmental concerns. Other features inherent to this clinical dataset should also be taken into consideration when interpreting the reported outcomes. 1) Data in the FAS DPN clinical dataset are obtained from a variety of sources (medical/educational/social service record review,

caregiver interview, and direct clinical evaluation). The accuracy of the data will vary by source. 2) No two patients have an identical dataset. The amount and type of data available on each patient varies by their age and the existence and availability of previous medical/educational assessments. 3) Prior medical and educational assessments may focus more on areas of concern than areas of strength. As a result, inclusion of these data sources could generate group profiles that over represent deficits. Overall, clinical datasets are an invaluable, ubiquitous resource that, when interpreted in the proper context can greatly inform and advance a field.

CONCLUSION

In summary, the existence of the WA FAS DPN diagnostic program and electronic database over the past 17 years confirms it is possible to establish and maintain a comprehensive statewide FASD diagnostic program and dataset. As demonstrated in this report, a broad array of clinical, research, and public health questions can be addressed with a FASD clinical dataset. The outcomes presented in this report reflect the experience of WA State. With the worldwide replication of this interdisciplinary approach to FASD diagnosis using the 4-Digit Code, the opportunity now exists, for the first time ever, to construct and validly compare clinical profiles across very diverse, geographically dispersed populations. This report serves as a formal appeal to FASD clinical programs worldwide to do just that. The benefits to individuals with FASD and their families would be immense.

Acknowledgements

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TABLE 1 FASD 4-Digit Diagnostic Categories within each of the four FASD diagnostic study subgroups.

Characteristic	FASD Diagnostic Subgroups										Statistics
	1. FAS/PFAS		2. SE/AE		3. ND/AE		4. Normal CNS/AE		Total		Chi-square Chi (p)
	N = 154		N = 394		N = 722		N = 130		N = 1400		
4-Digit Code FASD Diagnostic Categories (A-C, E-J): N (valid %)											
A. FAS / Alcohol Exposed	52	33.8							52	3.7	
B. FAS / Alcohol Exposure Unknown	7	4.5							7	0.5	
C. Partial FAS / Alcohol Exposed	95	61.7							95	6.8	
E. Sentinel Physical Findings / Static Encephalopathy / Alcohol Exposed			95	24.1					95	6.8	
F. Static Encephalopathy / Alcohol Exposed			299	75.9					299	21.4	
G. Sentinel Physical findings / Neurobehavioral Disorder / Alcohol Exposed					160	22.2			160	11.4	
H. Neurobehavioral Disorder / Alcohol Exposed					562	77.8			562	40.1	
I. Sentinel Physical Findings / Alcohol Exposed							34	26.2	34	2.4	
J. No Sentinel physical findings or CNS abnormalities detected / Alcohol Exposed							96	73.8	96	6.9	
Diagnostic outcomes across FAS DPN clinics: N (valid row %)											3.2 (.36)
University of Washington Core Clinic in Seattle	107	11.5	248	26.7	487	52.4	88	9.5	930	100	
Six other statewide FAS DPN clinics	47	10.0	146	31.1	235	50.0	42	8.9	470	100	

Abbreviations: Chi: chi-square test statistic across the four study groups. FAS: fetal alcohol syndrome. P: p-value. PFAS: partial FAS. ND/AE: Neurodevelopmental disorder/alcohol exposed. Normal CNS/AE: No central nervous system abnormalities/alcohol exposed. SE/AE: Static encephalopathy/alcohol exposed.

TABLE 2 Sociodemographic profiles across the four study groups.

Characteristic	FASD Diagnostic Subgroups										Statistics		
	1. 59 FAS/ 95 PFAS		2. SE/AE		3. ND/AE		4. Normal CNS/AE		Total		ANOVA		Chi-square
											Overall	Post Hoc	
	N = 154		N = 394		N = 722		N = 130		N = 1400		F (p)[A]	Duncan[B]	Chi (p)
Gender: N (valid%)													
male	80	51.9	255	64.7	422	58.4	55	42.3	812	58.0			22.8(.00)
Race (one race): N (valid%)													
White	87	56.5	182	46.2	357	49.4	58	44.8	684	48.9			30.2(.00)
Black	17	11.1	19	4.8	45	6.2	11	8.5	92	6.6			
American Indian/Native Alaskan	6	3.9	48	12.2	57	7.9	4	3.1	115	8.2			
Asian	0	0	0	0	0	0	0	0	0	0			
All others (including mixed race)	44	28.6	145	36.8	263	36.4	57	43.8	509	36.4			
Age at diagnosis (yr): N(row-column valid%)													
0 – 3.9	39	15.1-25.3	42	16.3-10.7	117	45.3-16.2	60	23.3-46.2	258	100-18.4			116(.00)
4 – 5.9	22	9.4-14.3	52	22.3-13.2	136	58.4-18.8	23	9.9-17.7	233	100-16.6			
6 – 10.9	53	11.0-34.4	157	32.6-39.8	251	52.1-34.8	21	4.4-16.2	482	100-34.4			
11 – 15.9	26	9.1-16.9	93	32.5-23.6	149	52.1-20.6	18	6.3-13.8	286	100-20.4			
16+	14	9.9-9.1	50	35.5-12.7	69	48.9-9.5	8	5.6-6.2	141	100-10.1			
Mean (SD)	8.9	8.3	10.1	6.1	8.9	5.5	6.7	7.0	9.0	6.2	10.2(.00)	3,12,4	
Minimum-Maximum	0.3	50.5	0.5	50.8	0.5	36.6	.02	48.1	0.02	50.8			
Patient's caregiver at diagnosis: N (valid%)													
Birth mother	26	17.3	79	21.4	152	21.9	36	28.6	293	21.9			[C] 6.5(.09)
Birth Father	10	6.7	34	9.2	47	6.8	11	8.7	102	7.6			
Other biological family member	25	16.7	39	10.6	123	17.7	25	19.8	212	15.8			
Adoptive parent	39	26.0	81	22.0	155	22.3	15	11.9	290	21.6			
Foster parent	35	23.3	80	21.7	149	21.4	29	23.0	293	21.9			
Social or caseworker	5	3.4	16	4.3	17	2.5	2	1.6	40	3.0			
Other	10	6.7	40	10.8	52	7.5	8	6.3	110	8.2			
Annual income < $35,000: N (valid%)	37	59.7	98	66.2	210	64.6	40	67.8	385	64.8			0.9(.82)

Abbreviations: Chi: chi-square test statistic across the four study groups, unless otherwise noted. F: F statistic. FAS: fetal alcohol syndrome. P: p-value. PFAS: partial FAS. ND/AE: Neurodevelopmental disorder/alcohol exposed. Normal CNS/AE: No central nervous system abnormalities/alcohol exposed. SD: standard deviation. SE/AE: Static encephalopathy/alcohol exposed. Notations: A. Numerator degrees of freedom = 3; denominator df = total sample size minus 4. B. The Duncan multiple comparisons range test is reported if the overall ANOVA is statistically significant; commas separate groups with homogeneous means at $p < 0.05$. C. Birth parent versus not birth parent.

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TABLE 3 Birth mother and birth father characteristics across the four study groups.

Characteristic	FASD Diagnostic Subgroups										Statistics		
	1. 59 FAS/ 95 PFAS		2. SE/AE		3. ND/AE		4. Normal CNS/AE		Total		ANOVA		Chi-square
											Overall	Post Hoc	
	N = 154		N = 394		N = 722		N = 130		N = 1400		F (p)[A]	Duncan[B]	Chi (p)
MOTHER													
Mother's name known	143	93.3	370	93.9	683	94.6	129	99.2	1325	94.6			6.8(.08)
Mother attended FASD evaluation: N (valid%)	26	17.3	79	21.4	152	21.9	36	28.6	293	21.9			5.2(.16)
Mother's age (yr)													
At child's birth: N mean (SD)	119	28.3 (6.6)	318	25.5 (6.1)	594	25.6 (6.3)	116	25.8 (5.9)	1147	25.9 (6.3)	6.6(.00)	1,234	
Min-Max	16.0	43.0	15.0	41.0	14.0	43.0	14.2	42.0	14.0	43.0			
At FASD diagnosis: N mean (SD)	119	37.1 (10.6)	318	35.4 (8.5)	594	34.5 (8.2)	116	32.4 (9.1)	1147	34.8 (8.7)	6.6(.00)	1,234	
Min-Max	22.1	81.5	19.3	77.6	17.5	75.3	16.0	79.1	16.0	81.5			
Maternal highest education level: N (valid%)													1.8(.61)
Did not finish high school	56	53.3	166	57.6	272	52.8	59	55.1	553	54.5			
Finished high school	37	35.2	91	31.6	171	33.2	26	24.3	325	32.0			
College	12	11.5	31	10.8	72	14.0	22	20.6	137	15.6			
Maternal learning disabilities: N (valid%)	57	56.4	168	60.6	291	59.5	47	50.0	563	58.6			6.4(.38)
Mother deceased: N (valid%)	9	12.3	17	8.8	34	9.6	2	2.6	62	8.9			5.1(.17)
Parity of index child: N mean (SD)	122	3.0 (1.8)	317	2.7 (1.8)	600	2.7 (1.7)	117	2.7 (1.7)	1156	2.7 (1.7)	1.0(.39)	--	
Min-Max	1	9	1	12	1	10	1	9	1	12			
Gravity of index child: N mean (SD)	83	3.5 (2.2)	174	3.1 (2.2)	322	3.2 (2.0)	52	2.9 (1.6)	631	3.2 (2.0)	1.2(.30)	--	
Min-Max	1	9	1	12	1	11	1	9	1	12			
FATHER													
Father's name known	105	68.2	299	75.9	555	76.9	107	82.3	1066	76.1			8.3(.04)
Father attended FASD evaluation: N (valid%)	10	6.7	34	9.2	47	6.8	11	8.7	102	7.6			2.4(.49)
Father's age (yr)													
At child's birth: N mean (SD)	62	31.5 (8.4)	189	29.4 (7.3)	369	28.7 (7.3)	70	27.8 (7.4)	690	29.0 (7.5)	3.5(.02)	1,234	
Min-Max	17	66	15	61	15	62	14	48	14	66			
At FASD diagnosis: N mean (SD)	62	40.6 (13.2)	189	39.6 (9.2)	369	38.1 (9.8)	70	35.1 (10.3)	690	38.4 (10.1)	4.5(.00)	123,4	
Min-Max	19	87	23	81	20	87	15	65	15	87			
Paternal highest education level: N (valid%)													
Did not finish high school	21	33.9	79	40.9	129	38.4	22	36.7	248	38.5			[C] 1.5(.68)
Finished high school	32	51.6	85	45.7	153	45.5	22	36.7	292	45.3			
College	9	14.5	25	13.4	54	16.1	16	26.6	104	16.2			
Paternal learning disabilities: N (valid%)	22	43.1	97	53.9	165	54.3	24	38.7	308	51.6			6.9(.33)

Abbreviations: Chi: chi-square test statistic across the four study groups, unless otherwise noted. F: F statistic. FAS: fetal alcohol syndrome. P: p-value. PFAS: partial FAS. ND/AE: Neurodevelopmental disorder/alcohol exposed. Normal CNS/AE: No central nervous system abnormalities/alcohol exposed. SD: standard deviation. SE/AE: Static encephalopathy/alcohol exposed. Notations: A. Numerator degrees of freedom = 3; denominator df = total sample size minus 4. B. The Duncan multiple comparisons range test is reported if the overall ANOVA is statistically significant; commas separate groups with homogeneous means at $p < 0.05$. C. Did versus did not finish high school.

TABLE 4 Growth and FAS facial outcomes across the four study groups.

Characteristic	FASD Diagnostic Subgroups										Statistics		
	1. 59 FAS/ 95 PFAS		2. SE/AE		3. ND/AE		4. Normal CNS/AE		Total		ANOVA		Chi-square
											Overall	Post Hoc	
	N = 154		N = 394		N = 722		N = 130		N = 1400		F (p)[A]	Duncan[B]	Chi (p)
GROWTH													
Growth Rank in 4-Digit Code: N (%)													
Rank 1	[C] 55	35.7	245	62.2	532	73.7	91	70.0	923	65.9			165(.00)
Rank 2	21	13.6	54	13.7	109	15.1	18	13.8	202	14.4			
Rank 3	35	13.6	52	13.2	58	8.0	19	14.6	164	11.7			
Rank 4	43	27.9	43	10.9	23	3.2	2	1.5	111	7.9			
Gestational age (wks): N mean (SD)	116	36.8 (3.2)	286	37.1 (3.5)	529	37.7 (3.0)	86	37.7 (2.7)	1017	37.4 (3.2)	4.2(.00)	12,234	
Birth weight percentile: N mean (SD)	124	33.2 (28.9)	284	48.0(32.6)	532	52.0(30.2)	93	45.7 (28.7)	1033	48.1 (31.1)	13.0(.00)	1,234	
Birth length percentile: N mean SD)	103	36.5 (34.7)	222	52.7 (34.5)	440	56.9 (31.9)	79	52.5 (30.9)	844	52.9 (33.4)	10.6(.00)	1,234	
Wgt percentile at diagnosis: N mean (SD)	143	33.6 (32.4)	367	47.6 (32.8)	661	53.9 (29.9)	119	53.3 (29.0)	1290	49.8 (31.6)	17.9(.00)	1,234	
Hgt percentile at diagnosis: N mean (SD)	143	25.1 (26.9)	364	38.3 (31.6)	664	42.6 (29.6)	119	39.5 (29.2)	1290	39.1 (30.3)	13.7(.00)	1,234	
FACE													
Face Rank in 4-Digit Code: N (%)													
Rank 1	0	0	93	23.6	210	29.1	55	42.3	358	25.6			816(.00)
Rank 2	0	0	301	76.4	413	57.2	58	44.6	772	55.1			
Rank 3	[D] 69	44.8	0	0	65	9.0	10	7.7	144	10.3			
Rank 4	85	55.2	0	0	[E] 34	4.7	[F] 7	5.4	126	9.0			
Mean PFL zscore: mean (SD)	-3.2	1.2	-2.6	1.6	-2.3	1.4	-1.9	1.5	-2.4	1.5	24(.00)	1,2,3,4	
Mean PFL $\leq$ -2 SD: N (valid%)	144	93.5	260	66.0	418	57.9	59	45.4	881	62.9			90(.00)
Innercanthal distance zscore: mean (SD)	-0.1	1.1	0.1	1.7	0.2	1.2	0.2	1.0	0.1	1.4	1.4(.24)		
Innercanthal distance $\geq$ 2SD: N (valid%)	4	5.3	20	9.3	19	5.7	4	8.5	47	7.0			3.2(.36)
Philtrum Smoothness Rank: N (valid%)													
1 (very deep)	0	0	99	25.4	178	24.9	57	44.2	334	24.2			408(.00)
2 (somewhat deep)	0	0	134	34.4	218	30.5	27	20.9	380	27.5			
3 (normal)	[G] 30	20.0	111	28.5	200	28.0	24	18.6	365	26.4			
4 (almost smooth)	76	50.7	34	8.7	101	14.1	14	10.9	225	16.3			
5 (completely smooth)	44	29.3	11	2.8	17	2.4	7	5.4	79	5.7			
Upper Lip Thinness Rank: N (valid%)													
1 (very thick)	0	0	121	31.1	180	25.2	36	27.9	337	24.1			248(.00)
2 (moderately thick)	0	0	105	27.0	178	24.9	34	26.4	317	22.6			
3 (normal)	[H] 30	19.4	106	27.2	165	23.1	31	24.0	332	23.7			
4 (moderately thin)	77	50.0	44	11.3	141	19.7	19	14.7	281	20.0			
5 (very thin)	43	29.1	13	3.3	50	7.0	9	7.0	115	8.3			

Abbreviations: Chi: chi-square test statistic across the four study groups, unless otherwise noted. F: F statistic. FAS: fetal alcohol syndrome. Hgt: height. P: p-value. PFAS: partial FAS. PFL: palpebral fissure length. ND/AE: Neurodevelopmental disorder/alcohol exposed. Normal CNS/AE: No central nervous system abnormalities/alcohol exposed. SD: standard deviation. SE/AE: Static encephalopathy/alcohol exposed. Wgt: weight. Wks: weeks.
Notations: A. Numerator degrees of freedom = 3; denominator df = total sample size minus 4. B. The Duncan multiple comparisons range test is reported if the overall ANOVA is statistically significant; commas separate groups with homogeneous means at $p < 0.05$. C. All 55 Rank 1 growths are PFAS. D. All 69 Rank 3 faces are PFAS; E. 25 too young to rule out CNS Rank 3 (< 8yrs). F. All 7 are too young to rule out CNS Rank 3 (<8yrs). G. All 30 Rank 3 philtrums are PFAS. H. All 30 Rank 3 lips are PFAS.

TABLE 5 CNS structural / neurological outcomes (4-Digit CNS Rank 4) across the four study groups.

Characteristic	FASD Diagnostic Subgroups										Statistics		
	1. 59 FAS/ 95 PFAS		2. SE/AE		3. ND/AE		4. Normal CNS/AE		Total		ANOVA		Chi-square
											Overall	Post Hoc	
	N = 154		N = 394		N = 722		N = 130		N = 1400		F (p)[A]	Duncan[B]	Chi (p)
CNS Rank in 4-Digit Code: N(valid%)													
Rank 1	0	0	0	0	0	0	130	100	130	9.3			2844(00)
Rank 2	0	0	0	0	722	100	0	0	722	51.6			
Rank 3	65	42.2	244	61.9	0	0	0	0	309	22.1			
Rank 4	89	57.8	150	38.1	0	0	0	0	239	17.1			
CNS functional Rank independent of Rank 4	N (valid%)	Age yrs mean(SD)	N (valid%)	Age yrs mean(SD)	N (valid%)	Age yrs mean(SD)	N (valid%)	Age yrs mean(SD)	N (valid%)	Age yrs mean(SD)			
Rank 1 (no dysfunction)	40 (26)	4.4 (5.8)	66 (16.8)	8.5 (8.2)	0 (0)	--	130(100)	6.7 (7.0)	236(16.9)	6.8 (7.3)			1740(00)
Rank 2 (mild dysfunction)	19 (12.3)	7.1 (5.1)	52 (13.2)	10.9 (9.5)	722(100)	8.9 (5.5)	0 (0)	--	793(56.6)	8.9 (5.9)			
Rank 3 (severe dysfunction)	95 (61.7)	11.2 (8.8)	276(70.1)	10.2 (4.5)	0 (0)	--	0 (0)	--	371(26.5)	10.5 (5.9)			
Duncan[B] comparing mean age between CNS Ranks 1,2,3: F(p)		F11.3(.00) Rank 12,3		F2.9(.06)						F26.8(.00) Rank 1,2,3			
CNS functional Rank among those with CNS Rank 4	N (valid%)	[C]Age yrs mean(SD)	N (valid%)	[C]Age yrs mean(SD)	N (valid%)	[C]Age yrs mean(SD)	N (valid%)	[C]Age yrs mean(SD)	N (valid%)	[C]Age yrs mean(SD)			
Rank 4 that is also Rank 1	40 (44.9)	[D]4.4 (5.8)	[F]66 (44.0)	8.5 (8.2)	n/a	n/a	n/a	n/a	106(44.4)	6.9 (7.6)			
Rank 4 that is also Rank 2	19 (21.3)	[E]7.1 (5.1)	[G]52(34.7)	10.9 (9.5)	n/a	n/a	n/a	n/a	71(29.7)	9.9 (8.7)			
Rank 4 that is also Rank 3	30 (33.7)	9.2 (5.0)	32 (21.4)	9.3 (3.1)	n/a	n/a	n/a	n/a	62(25.9)	9.3 (7.3)			
Duncan[B] comparing mean age between CNS Ranks 1,2,3: F(p)		7.0(.002) Rank12,23		1.3(.27)						4.0 (.02) Rank13,32			
Microcephaly: N (valid%)	69	44.8	99	25.3	0	0	0	0	168	12.1			[H]75(00)
OFC percentile: N, mean (SD)	152	24.0(28.0)	391	39.6(31.4)	715	52.0(23.8)	126	53.5(24.4)	1384	45.7(28.3)	57(00)	1,2,34	
Abnormal MRI among those Imaged: N (valid%)	7	26.9	18	30.5	0	0	0	0	25	18.1			
Seizure disorder: N (valid%)	10	6.5	32	8.1	0	0	0	0	42	3.0			
Why CNS Rank 4: N (valid%)	Of the 89 with Rank 4		Of the 150 with Rank 4						Of the 239 with Rank 4				
Microcephaly only	66	82.5	88	58.7	n/a	n/a	n/a	n/a	154	64.4			
Abnormal MRI only	2	0.3	13	8.7	n/a	n/a	n/a	n/a	15	6.3			
Seizure disorder only	7	0.9	18	12.0	n/a	n/a	n/a	n/a	25	10.5			
Microcephaly & abnormal MRI	3	0.4	0	0	n/a	n/a	n/a	n/a	3	1.3			
Microcephaly & seizures	0	0	9	6.0	n/a	n/a	n/a	n/a	9	3.8			
Abnormal MRI & seizures	2	0.3	2	1.3	n/a	n/a	n/a	n/a	4	1.7			
All 3	0	0	2	1.3	n/a	n/a	n/a	n/a	2	0.1			
Vision problems: N (valid%)	42	37.5	108	33.2	155	25.2	20	18.5	325	28.0			16(00)
Chronic hearing loss: N (valid%)	23	21.3	71	22.8	95	15.9	14	13.1	203	18.1			9.2(03)

Abbreviations: Chi2: chi-square test across the four study groups, unless otherwise noted. CNS: central nervous system. F: F statistic. FAS: fetal alcohol syndrome. Microcephaly: OFC <= -2SD. MRI: magnetic resonance image. OFC: Occipital frontal circumference. P: p-value. PFAS: partial FAS. ND/AE: Neurodevelopmental disorder/alcohol exposed. Normal CNS/AE: No central nervous system abnormalities/alcohol exposed. SD: standard deviation. SE/AE: Static encephalopathy/alcohol exposed. Yrs: years. Notations: A. Numerator degrees of freedom = 3; denominator df = total sample size minus 4. B. The Duncan multiple comparisons range test is reported if the overall ANOVA is statistically significant; commas separate groups with homogeneous means at p < 0.05. C. Of those with Rank 4 CNS, are those with Rank 1 too young to rule-out Rank 3? D. Only 5 (12%) of the 40 with Rank 1 function are >7yrs old. E. Only 4 (21%) of the 19 with Rank 2 Function are > 7 yrs old. F) 28 (42%) of the 66 with Rank 1 function are > 7 yrs old. G) 27 (52%) of the 52 with Rank 2 function were > 7 yrs old. H) Group 1 versus Group 2.

TABLE 6 CNS Functional outcomes (4-Digit CNS Ranks 1-3) across the four study groups.

Characteristic	FASD Diagnostic Subgroups										Statistics		
	1. 59 FAS/ 95 PFAS		2. SE/AE		3. ND/AE		4. Normal CNS/AE		Total		ANOVA Overall	ANOVA Post Hoc	Chi-square2
	N = 154		N = 394		N = 722		N = 130		N = 1400		F (p) [A]	Duncan[B]	Chi (p)
CNS functional Rank: N (valid%)													
Rank 1 (no dysfunction)	40	26.0	66	16.8	0	0	130	100	236	16.9			1740 (00)
Rank 2 (mild dysfunction)	19	12.3	52	13.2	722	100	0	0	793	56.6			
Rank 3 (severe dysfunction)	95	61.7	276	70.1	0	0	0	0	371	26.5			
Domain with Significant Dysfunction: N (valid%)													
Cognition	33	36.7	96	32.2	11	2.9	0	0	140	17.5			[c]120(.00)
Adaptation	47	70.1	109	72.7	65	36.1	0	0	221	53.2			[C]51(.00)
Achievement	32	43.2	132	56.4	35	12.5	0	0	199	32.8			[c]113(.00)
Executive Function, Memory	45	58.4	124	56.1	49	17.7	0	0	218	36.0			[C]93(.00)
Language	52	50.0	174	61.3	73	16.7	0	0	299	34.2			[c]157(.00)
Motor/Sensory	27	57.4	36	36.0	51	29.1	0	0	114	35.3			
Development	38	51.4	66	50.8	64	34.6	0	0	168	38.5			[c]35(.00)
ADHD	49	43.4	149	51.7	227	45.0	0	0	425	42.8			[c]4(.13)
Intelligence (WISC) FSIQ Std: N mean (SD)	88	77.8 (13.5)	274	78.8 (14.8)	347	93.4(13.0)	22	101.3(10.6)	731	86.3(15.7)	79.2(.00)	12,3,4	
FSIQ Std <= 70: N (valid%)	26	20.0	72	19.3	4	0.6	0	0	102	8.2			144(.00)
VIQ Std: N mean (SD)	67	78.4(14.0)	222	79.1(14.3)	242	92.0(12.8)	12	102.4(13.0)	543	85.2(15.2)	46.9(.00)	12,3,4	
VIQ Std <= 70: N (valid%)	20	15.4	65	17.4	9	1.4	0	0	94	7.5			106(.00)
PIQ Std: N mean (SD)	66	78.6 (13.8)	223	81.6(16.0)	235	94.3(13.4)	11	107.0(12.6)	535	87.3 (16.2)	44.6 (.00)	12,3,4	
PIQ Std <= 70: N (valid%)	16	12.3	54	14.5	8	1.3	0	0	78	6.3			85(.00)
Perceptual Organization Std: N mean (SD)	15	82.6 (17.0)	47	84.3 (13.2)	59	93.9 (13.6)	1	93.0	122	88.8 (14.6)	8.0 (.001)	[c] 12,3	
Verbal Comprehension Std: N mean(SD)	14	82.9 (13.8)	47	83.3 (13.9)	59	94.8 (12.9)	1	95.0	121	89.0 (14.5)	11.0 (.00)	[c] 12,3	
Freedom Distractibility Std: N mean(SD)	12	79.5 (14.5)	36	79.7 (12.9)	50	91.1 (13.1)	1	90.0	99	85.3 (14.3)	9.2 (.00)	[c] 12,3	
Information Sc: N mean (SD)	47	6.1 (2.9)	138	6.0 (2.8)	161	8.5 (2.7)	9	10.4 (2.8)	355	7.3 (3.1)	24.4 (.00)	12,3,4	
Similarities Sc Score: N mean (SD)	46	7.3 (3.2)	137	6.7 (3.2)	166	9.1 (3.1)	8	10.4 (2.3)	357	8.0 (3.4)	17.5 (.00)	12,34	
Arithmetic Sc: N mean (SD)	47	4.9 (2.4)	137	6.1 (2.7)	161	7.9 (2.5)	7	9.6 (3.7)	352	6.8 (2.8)	23.9 (.00)	12,3,4	
Vocabulary Sc: N mean (SD)	46	6.0 (3.4)	143	6.8 (3.1)	166	8.9 (2.8)	8	10.5 (3.2)	363	7.7 (3.2)	20.8 (.00)	12,34	
Comprehension Sc: N mean (SD)	44	6.5 (3.1)	132	6.5 (3.1)	159	8.9 (3.2)	7	10.6 (3.8)	342	7.7 (3.4)	17.5 (.00)	12,34	
Digit Span Sc: N mean (SD)	35	6.3 (2.9)	93	6.5 (2.6)	116	8.2 (2.6)	4	10.0 (6.2)	248	7.3 (2.8)	9.2 (.00)	123,34	
Picture Completion Sc: N mean (SD)	46	6.9 (3.3)	139	7.6 (2.8)	162	9.5 (2.9)	7	12.6 (3.2)	354	8.5 (3.1)	20.0 (.00)	12,3,4	
Picture Arrangement Sc: N mean (SD)	37	6.3 (3.2)	119	7.1 (3.6)	141	8.8 (3.1)	7	11.3 (3.1)	304	7.9 (3.5)	11.1 (.00)	12,23,4	
Block Design Sc: N mean (SD)	47	6.3 (3.3)	146	6.9 (3.4)	170	8.9 (3.2)	8	10.9 (2.4)	371	7.8 (3.4)	15.2 (.00)	12,3,4	
Object Assembly Sc: N mean (SD)	44	7.5 (3.3)	125	7.8 (3.6)	146	9.1 (2.9)	8	11.6 (2.8)	323	8.5 (3.3)	7.9 (.00)	123,4	
Coding Sc: N mean (SD)	35	5.8 (3.2)	113	6.5 (3.6)	142	8.3 (3.3)	6	8.8 (2.6)	296	7.4 (3.5)	8.8 (.00)	12,234	
Mazes Sc: N mean (SD)	6	3.8 (1.9)	25	6.6 (3.0)	31	9.0 (3.3)	1	15.0 (--)	63	7.7 (3.6)	8.0 (.00)	--	
Visual-Motor/Sensory QNST R:mean(SD)	28	37.2(16.9)	82	32.8(15.7)	114	24.2(13.2)	9	16.2(7.0)	233	28.5(15.4)	11.1 (.00)	12,3,4	
VMI Std: mean (SD)	37	77.3(11.4)	76	80.6(13.0)	140	89.6(10.2)	11	95.9(12.4)	264	85.5(12.5)	20.4 (.00)	12,3,4	
SSP Total = Definite Difference: N (valid%)	18	75.0	13	54.2	44	81.5	0	0	75	73.8			[c]6.4(.04)
Executive Function/Memory RCFT Copy R: mean(SD)	13	15.2(9.6)	40	18.8(9.9)	42	25.5(9.2)	3	32.7(1.5)	98	21.6(10.3)	7.0 (.00)	12,23,34	
RCFT 3min recall T: mean (SD)	13	25.2(5.6)	35	32.0(13.2)	39	42.5(14.7)	3	43.0(2.6)	90	36.0(14.4)	7.7 (.00)	12,234	
RCFT 30min recall T: mean (SD)	4	31.8(5.0)	13	28.1(13.0)	20	38.4(15.1)	2	45.5(2.1)	39	34.6(14.1)	2.0 (.13)	--	

Abbreviations: ADHD: attention deficit hyperactivity disorder. Chi: chi-square test statistic across the four study groups, unless otherwise noted. F: F statistic. FAS: fetal alcohol syndrome. FSIQ: full scale IQ. P: p-value. PFAS: partial FAS. ND/AE: Neurodevelopmental disorder/alcohol exposed. Normal CNS/AE: No central nervous system abnormalities/alcohol exposed. PIQ: Performance IQ. QNST: Quick Neurological Screening Test[45]. RCFT: Rey Complex Figure Test[42]. R: raw score. Sc: scaled score. SD: standard deviation. SE/AE: Static encephalopathy/alcohol exposed. SSP: Short Sensory Profile[46]. Std. standard score. T: t score. VIQ: Verbal IQ. VMI: Beery Buktenica Developmental Test of Visual Motor Integration[41]. WISC: Wechsler Intelligence Scale for Children[40]. Notations: A. Numerator degrees of freedom = 3; denominator df = total sample size minus 4. B. The Duncan multiple comparisons range test is reported if the overall ANOVA is statistically significant; commas separate groups with homogeneous means at p < 0.05. C. Only groups 1, 2 and 3 are compared.

TABLE 7 Child Behavior Check List (CBCL/ 6-18) outcomes (see Figure 2) among the 516 patients administered a CBCL when they were between 6 and 18 years of age.

Characteristic	FASD Diagnostic Subgroups										Statistics	
	1. 59 FAS/ 95 PFAS		2. SE/AE		3. ND/AE		4. Normal CNS/AE		Total		ANOVA	
											Overall	Post Hoc
	N = 154		N = 394		N = 722		N = 130		N = 1400		F (p)[A]	Duncan[B]
Problems: T-score[C]	N	Mean (SD)	N	Mean (SD)	N	Mean (SD)	N	Mean (SD)	N	Mean (SD)		
Internalizing	51	63.4(10.1)	154	64.5(10.9)	270	65.6(10.9)	25	60.8(14.1)	500	64.8(11.0)	1.9 (.14)	--
Externalizing	51	69.1(9.9)	154	69.6(10.9)	270	70.8(10.3)	25	60.3(13.2)	500	69.8(10.8)	7.6 (.000)	123,4
Total	51	71.4(8.9)	154	71.3(9.3)	270	72.1(9.0)	25	61.9(12.7)	500	71.3(9.5)	9.1 (.000)	123,4
Syndrome Scales: T-score[D]												
Anxious/Depressed	51	63.0(11.3)	153	64.0(9.9)	269	64.9(10.9)	25	62.6(12.1)	498	64.3(10.7)	0.8 (.53)	--
Withdrawn/Depressed	50	62.4(8.6)	153	64.6(11.2)	269	65.0(11.1)	25	63.1(12.4)	497	64.5(10.9)	0.9 (.42)	--
Somatic Complaints	51	60.0(9.3)	153	60.6(10.8)	269	61.8(10.0)	25	57.9(7.0)	498	61.0(10.1)	1.6 (.19)	--
Social Problems	50	72.0(12.0)	153	69.7(10.2)	269	68.5(10.2)	25	59.1(10.3)	497	68.8(10.7)	9.3 (.00)	123,4
Thought Problems	50	70.7(10.7)	153	69.1(10.6)	270	68.4(10.2)	25	61.6(8.8)	498	68.5(10.4	4.6 (.003)	123,4
Attention Problems	51	75.5(11.9)	153	75.7(11.0)	270	74.3(11.4)	25	64.2(13.1)	497	74.4(11.6)	7.6 (.000)	123,4
Rule-Breaking Behavior	51	67.9(8.9)	153	67.5(10.2)	269	69.7(10.0)	25	61.5(11.4)	498	68.4(10.2)	6.0 (.001)	123,4
Aggressive Behavior	50	70.2(13.1)	153	71.7(12.1)	269	72.0(12.2)	25	61.6(12.5)	497	71.2(12.4)	5.7 (.001)	123,4
Competence Scales: T-score [E]												
Activities	44	41.5(8.9)	135	42.8(8.8)	220	44.3(7.7)	18	46.1(7.2)	417	43.6(8.2)	2.5 (.05)	123,234
Social	44	36.0(9.2)	130	34.5(9.1)	211	36.4(9.8)	18	40.3(11.9)	403	35.9(9.7)	2.3 (.07)	123,34
School	37	28.3(6.4)	111	29.4(6.0)	181	31.9(6.2)	13	38.9(9.0)	342	31.0(6.6)	12.7 (.00)	12,23,4
Total	37	31.8 (10.0)	109	32.4 (8.2)	172	35.1 (7.6)	13	40.3 (9.4)	331	34.0 (8.4)	5.9 (.003)	123,4

Abbreviations: F: f-statistic. FAS: fetal alcohol syndrome. P: p-value. PFAS: partial FAS. ND/AE: Neurodevelopmental disorder/alcohol exposed. Normal CNS/AE: No central nervous system abnormalities/alcohol exposed. SD: standard deviation. SE/AE: Static encephalopathy/alcohol exposed. Notations: A. Numerator degrees of freedom = 3; denominator df = total sample size minus 4. B. The Duncan multiple comparisons range test is reported if the overall ANOVA is statistically significant; commas separate groups with homogeneous means at $p < 0.05$. C. Borderline clinical range (T score 60-63). Clinical range (T score > 63). D. Borderline clinical range (T score 65-69). Clinical range (T score > 69). E. (Activities, Social, School): Borderline clinical range (T score 31-35). Clinical range (T score <31); (Total): Borderline clinical range (T score 37-40). Clinical range (T score <37).

TABLE 8 Proportion of patients classified by the pediatrician as 'significantly delayed/impaired' across a spectrum of behaviors at the conclusion of a 2-hour, structured caregiver interview administered jointly by the pediatrician and psychologist during the FASD diagnostic evaluation (see Figure 3).

Patient Behaviors addressed in Caregiver Interview	FASD Diagnostic Subgroups										Statistic
	1. 59 FAS/ 95 PFAS		2. SE/AE		3. ND/AE		4. Normal CNS/AE		Total		Chi-square Groups 1,2,3
	N = 154		N = 394		N = 722		N = 130		N = 1400		Chi (p)
Domain Behavior	N	Valid %	N	Valid %	N	Valid %	N	Valid %	N	Valid %	
Planning											
Needs considerable help organizing daily tasks	24	24.0	63	22.4	92	20.2	5	10.6	184	20.8	2.9 (.57)
Cannot organize time	21	31.3	56	29.0	59	20.6	5	20.0	141	24.7	10.2 (.04)
Does not understand concept of time	17	31.5	39	25.7	48	20.2	1	3.3	105	22.2	11.8 (.02)
Difficulty carrying out multistep tasks	27	26.5	61	21.4	88	19.1	5	10.4	181	20.2	8.2 (.09)
Behavioral Regulation/Sensory Motor Integration:											
Poor management of anger/tantrums	24	18.8	74	22.2	127	19.7	8	7.9	233	19.3	7.9 (.10)
Mood swings	19	16.7	54	18.1	102	18.1	4	4.5	179	16.8	4.9 (.30)
Impulsive	28	24.8	87	27.8	137	22.9	9	11.0	261	23.6	6.7 (.15)
Compulsive	7	8.6	19	8.3	29	6.6	1	1.5	56	6.9	2.4 (.67)
Perseverative	18	18.8	18	7.7	42	9.5	1	1.6	79	9.4	11.5 (.02)
Inattentive	28	24.3	76	23.3	113	18.9	8	9.4	225	20.0	8.8 (.07)
Inappropriate activity level	27	24.5	70	26.0	92	17.7	2	3.3	191	19.9	8.8 (.07)
Lying/stealing	16	14.4	52	18.2	63	11.8	5	5.6	136	13.3	7.3 (.12)
Unusual high/low reactivity to sound/touch/light	27	27.6	42	18.4	60	15.6	4	6.0	133	17.1	10.2 (.03)
Abstract Thinking/Judgment:											
Poor judgment	28	31.1	81	29.7	98	20.5	4	8.2	211	23.7	18.2 (.00)
Cannot be left alone	19	26.4	56	27.3	64	18.4	3	8.6	142	21.5	19.4 (.00)
Concrete, unable to think abstractly	17	25.8	55	30.9	32	12.6	1	4.8	105	20.2	65.6 (.00)
Memory/Learning/Information Processing:											
Poor memory, inconsistent retrieval of learned information	33	28.0	77	23.6	72	13.4	18	22.5	200	18.8	26.7 (.00)
Slow to learn new skills	21	17.2	62	19.7	41	7.	1	1.1	125	11	78.9 (.00)
Does not seem to learn from past experiences	21	22.6	72	26.0	96	18.9	4	6.3	193	20	7.3 (.12)
Problems recognizing consequences of actions	21	21.9	75	28.2	97	19.6	4	6.9	197	21.5	8.2 (.08)
Problems w/information processing speed/accuracy	19	21.6	54	21.9	42	10.1	1	1.6	116	14.3	97.0 (.00)
Spatial Memory:											
Gets lost easily. Difficulty navigating from A to B	10	16.1	20	13.1	13	5.2	0	0	43	8.6	25.5 (.00)
Social Skills and Adaptive Behavior:											
Behaves at a level notably younger than chronological age	32	27.8	72	21.9	60	10.7	3	3.4	167	15.3	50.8 (.00)
Poor social/adaptive skills	30	25.2	70	20.8	91	14.6	4	4.3	195	16.7	18.3 (.00)
Motor/Oral Motor Control:											
Poor/delayed motor skills	20	16.5	34	11.0	21	3.7	0	0	75	6.9	61.4 (.00)
Poor balance	20	20.8	30	12.8	17	3.7	0	0	67	7.8	65.0 (.00)

Abbreviations: Chi: chi-square test statistic across Groups 1, 2 and 3. FAS: fetal alcohol syndrome. P: p-value. PFAS: partial FAS. ND/AE: Neurodevelopmental disorder/alcohol exposed. Normal CNS/AE: No central nervous system abnormalities/alcohol exposed. SE/AE: Static encephalopathy/alcohol exposed. Notations: Not all patients are old enough to demonstrate each of the behaviors listed above. Thus the valid % reflects the proportion of patients with significant impairment among those old enough to demonstrate the behavior.

TABLE 9 Alcohol exposure history across the four study groups.

Characteristic	FASD Diagnostic Subgroups										Statistics		
	1. 59 FAS/ 95 PFAS		2. SE/AE		3. ND/AE		4. Normal CNS/AE		Total		ANOVA		Chi-square
											Overall	Post Hoc	
	N = 154		N = 394		N = 722		N = 130		N = 1400		F (p)[A]	Duncan[B]	Chi (p)
Prenatal Alcohol Rank: N (valid%)													
Rank 1: Confirmed Absent	0	0	0	0	0	0	0	0	0	0			27 (.00)
Rank 2: Unk.	[C]7	4.5	0	0	0	0	0	0	7	0.5			
Rank 3: Confirmed; amount moderate or unk.	60	39.0	164	41.6	346	47.9	56	43.1	626	44.7			
Rank 4: Confirmed; amount high	87	56.5	230	58.4	376	52.1	74	56.9	767	54.8			
Before Pregnancy: N, mean (SD)													
Ave # drinks per drinking occasion	50	8.2(7.0)	162	9.8(10.1)	308	9.3(10.1)	67	10.9(12.8)	587	9.5(10.2)	0.8 (.50)		
Max # drinks per drinking occasion	52	12.0(9.4)	156	16.0(15.8)	264	14.8(14.7)	64	15.4(20.3)	536	14.9(15.4)	0.9 (.44)		
Ave # drinking days per week	72	5.5(2.0)	206	4.4(2.2)	373	4.7(2.2)	82	4.8(2.2)	733	4.7(4.8)	4.9(.002)	1,234	
Type of alcohol consumed: N (valid%)													
beer	63	40.9	140	35.5	273	37.8	55	42.3	531	37.9			2.6 (.46)
wine	20	13.0	58	14.7	100	13.9	19	14.6	197	14.1			0.3 (.96)
liquor	45	29.2	122	31.0	197	27.3	32	24.6	396	28.3			2.7 (.44)
During Pregnancy: N, mean (SD)													
Ave # drinks per drinking occasion	54	8.0(7.3)	176	8.2(8.4)	331	8.6(10.2)	69	9.1(14.2)	630	8.5(10.0)	0.2 (.89)		
Max # drinks per drinking occasion	56	12.5(10.0)	169	12.9(11.0)	275	13.3(13.9)	65	10.6(9.9)	565	12.8(12.3)	0.8 (.48)		
Ave # drinking days per week	81	5.6(2.1)	227	4.3(2.4)	409	4.4(2.3)	86	4.4(2.3)	803	4.5(2.3)	7.1(.000)	1,234	
Type of alcohol consumed: N (valid%)													
beer	64	41.6	153	38.8	280	38.8	60	46.2	557	39.8			2.9 (.41)
wine	22	14.3	58	14.7	102	14.1	18	13.8	200	14.3			0.1 (.99)
liquor	42	27.3	114	28.9	197	27.3	31	23.8	384	27.4			1.3 (.73)
Trimester of Alcohol Use: N (valid%)													[E] 4.2 (.24)
1st only	17	13.8	55	17.2	71	12.4	11	9.7	154	13.6			
1st and 2nd only	17	13.8	38	11.9	61	10.6	19	16.8	135	12.0			
[D] All 3	88	71.5	214	66.9	418	72.9	74	65.5	794	70.3			
Had an alcohol use problem: N (valid%)	127	93.4	315	86.5	622	93.1	113	91.6	1177	91.2			14 (.00)
Diagnosed with alcoholism: N (valid%)	90	82.6	241	76.3	545	79.8	102	87.2	887	79.8			7.3 (.06)
Received alcohol treatment: N (valid%)	86	78.9	214	70.4	412	72.8	95	81.2	807	73.6			6.9 (.08)
Source of alcohol information: N (valid%)													
Birth mother report	51	35.9	170	43.8	300	42.5	58	45.0	597	42.5			3.2 (.79)
Person who directly observed birth mother	55	38.7	133	34.3	248	35.0	44	34.1	480	35.1			
Other Source (med/legal/social reports)	36	25.4	85	21.9	160	22.6	27	20.9	308	22.5			

Abbreviations:: Chi: chi-square test statistic across Groups 1, 2 and 3, unless otherwise noted. FAS: fetal alcohol syndrome. P: p-value. PFAS: partial FAS. ND/AE: Neurodevelopmental disorder/alcohol exposed. Normal CNS/AE: No central nervous system abnormalities/alcohol exposed. SE/AE: Static encephalopathy/alcohol exposed. Unk: unknown. Notations: A. Numerator degrees of freedom = 3; denominator df = total sample size minus 4. B. The Duncan multiple comparisons range test is reported if the overall ANOVA is statistically significant; commas separate groups with homogeneous means at $p < 0.05$. C. All 7 unknown alcohol exposures have a full FAS diagnosis. D. When FAS/PFAS are split, 35 (81.4%) of the FAS group were exposed all 3 trimesters, compared to 53 (66.3%) of the PFAS group. E. All 3 trimesters versus less than 3 trimesters.

TABLE 10 Other prenatal and postnatal adverse exposures and events across the four study groups.

Characteristic	FASD Diagnostic Subgroups										Statistics		
	1. 59 FAS/ 95 PFAS		2. SE/AE		3. ND/AE		4. Normal CNS/AE		Total		ANOVA Overall	ANOVA Post Hoc	Chi-square
	N = 154		N = 394		N = 722		N = 130		N = 1400		F (p)[A]	Duncan[B]	Chi (p)
Prenatal Rank from 4-Digit Code: N (valid%)													
Rank 1: No risk	2	1.3	6	1.6	4	0.6	0	0	12	0.9			7.8 (.05)
Rank 2: Unknown Risk	27	17.9	55	14.2	89	12.4	19	14.6	190	13.7			
Rank 3: Some Risk	102	67.5	283	73.3	574	79.9	101	77.7	1060	76.5			
Rank 4: High Risk	20	13.2	42	10.9	51	7.1	10	7.7	123	8.9			
No prenatal care: N (valid%)	32	42.7	59	30.7	106	30.0	18	26.1	215	31.2			15.5 (.02)
Prenatal complications: N (valid %)	28	37.3	82	41.2	126	34.0	23	28.8	259	35.7			4.9 (.18)
Maternal learning disabilities: N (valid%)	57	56.4	168	60.6	291	59.5	47	50.0	563	58.6			3.7 (.29)
Paternal learning disabilities: N (valid%)	22	43.1	97	53.9	165	54.3	24	38.7	308	51.6			6.8 (.08)
Other syndromes All	2	1.3	10	2.5	4	0.5	2	1.5	18	1.3			8.0 (.05)
Binder	0	0	0	0	1	0.1	0	0	1	0.1			
Hemifacial microsomia	0	0	1	0.3	1	0.1	0	0	2	0.1			
Kabuki Makeup	0	0	1	0.3	0	0	0	0	1	0.1			
Marfan	0	0	1	0.3	0	0	0	0	1	0.1			
Schprintzen	0	0	1	0.3	0	0	0	0	1	0.1			
Sticklers	1	0.6	0	0	2	0.3	1	0.8	4	0.3			
Williams	0	0	1	0.3	0	0	1	0.8	2	0.1			
Kleinfelters	0	0	1	0.3	0	0	0	0	1	0.1			
Neurofibromatosis	0	0	1	0.3	0	0	0	0	1	0.1			
Amniotic band sequence	0	0	1	0.3	0	0	0	0	1	0.1			
Moebius sequence	0	0	2	0.5	0	0	0	0	2	0.1			
Down syndrome	1	0.6	0	0	0	0	0	0	1	0.1			
Other adverse prenatal exposures Any exposure	104	92.0	271	88.9	499	96.0	101	94.4	975	93.3			17.8 (.00)
Tobacco	84	57.1	238	61.5	455	63.6	84	64.6	861	62.4			2.6 (.46)
Marijuana	36	24.5	139	35.9	279	39.0	49	37.7	503	36.5			11.2 (.01)
Crack/Cocaine	58	39.5	124	32.0	279	39.0	60	46.2	521	37.8			9.9 (.02)
methamphetamines	15	10.2	26	6.7	52	7.3	9	6.9	102	7.4			2.0 (.57)
LSD/acid	6	4.1	15	3.9	23	3.2	3	2.3	47	3.4			[C] (.62)
Dilantin	1	0.7	1	0.3	6	0.8	0	0	8	0.6			[C] (1.0)
Postnatal Rank from 4-Digit Code: N(valid%)													
Rank 1: No risk	7	4.6	11	2.8	29	4.1	7	5.4	54	3.9			32.2 (.00)
Rank 2: Unknown Risk	18	11.9	42	10.8	87	12.2	33	25.4	180	13.0			
Rank 3: Some Risk	61	40.4	166	42.7	304	42.5	61	46.9	592	42.7			
Rank 4: High Risk	65	43.0	170	43.7	296	41.3	29	22.3	560	40.4			
Perinatal difficulties: N (valid%)	66	59.5	188	59.9	282	49.4	47	44.3	583	52.9			[D]17.7(.00)
Days in the birth hospital: N mean (SD)	57	9.6 (14.7)	155	8.1 (19.3)	256	7.0 (15.4)	43	3.8 (4.8)	521	7.4 (16.1)			1.3 (.28)
Physical abuse: N (valid%)	47	36.7	122	36.6	210	35.2	22	19.6	401	34.3			13.1 (.04)
Sexual abuse: N (valid%)	26	22.0	85	26.6	147	25.6	5	4.7	263	23.5			26.4 (.00)
Neglect: N (valid%)	86	65.6	235	67.3	399	63.9	65	56.5	785	64.4			4.6 (.21)
Total # of home placements: N mean (SD)	119	2.7 (2.3)	310	3.1 (3.5)	533	2.9 (3.3)	84	2.3 (1.3)	1046	2.9 (3.1)	1.5(.21)	--	

Abbreviations:: Chi: chi-square test statistic across the 4 study groups unless otherwise noted. FAS: fetal alcohol syndrome. P: p-value. PFAS: partial FAS. ND/AE: Neurodevelopmental disorder/alcohol exposed. Normal CNS/AE: No central nervous system abnormalities/alcohol exposed. SD: standard deviation. SE/AE: Static encephalopathy/alcohol exposed. Notations: A. Numerator degrees of freedom = 3; denominator df = total sample size minus 4. B. The Duncan multiple comparisons range test is reported if the overall ANOVA is statistically significant; commas separate groups with homogeneous means at $p < 0.05$. C. Fisher Exact Test: FASD groups versus Group 4. D. High risk versus all other risk groups.

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TABLE 11 Mental health disorders reported in the medical records of the 1,064 patients 5 or more years of age at the time of the FASD diagnostic evaluation across the four study groups.

Characteristic	FASD Diagnostic Subgroups										Statistics
	1. 59 FAS/ 95 PFAS		2. SE/AE		3. ND/AE		4. Normal CNS/AE		Total		Chi-square
	N = 154		N = 394		N = 722		N = 130		N = 1400		Chi (p)
Mental Health Disorders: N (valid%)											
One or more disorders	73	71.6	180	84.1	293	74.0	10	28.6	546	74.5	56 (.00)
ADD/ADHD	53	59.6	161	59.9	233	55.2	0	0	447	53.9	148 (.00)
Adjustment Disorder	4	2.6	8	2.0	29	4.0	3	2.3	44	3.1	3.9 (.27)
Antipersonality Disorder	0	0	0	0	1	0.1	0	0	1	0.1	--
Anxiety Disorder	2	1.3	10	2.5	8	1.1	0	0	20	1.4	5.8 (.12)
Reactive Attachment Disorder	6	3.9	19	4.8	27	3.7	2	1.5	54	3.9	2.9 (.41)
Bipolar/Manic Depression	4	2.6	10	2.5	13	1.8	3	2.3	30	2.1	0.8 (.85)
Conduct Disorder	2	1.3	16	4.1	24	3.3	1	0.8	43	3.1	5.3 (.15)
Depression	7	4.5	23	5.8	32	4.4	2	1.5	64	4.6	4.2 (.24)
Dysthymic Disorder	3	1.9	7	1.8	23	3.2	2	1.5	35	2.5	3.0 (.39)
Obsessive Compulsive Disorder	1	0.6	6	1.5	2	0.3	0	0	9	0.6	6.5 (.09)
Oppositional Defiant Disorder	8	5.2	39	9.9	72	10.0	1	0.8	120	8.6	15.0 (.00)
Post Traumatic Stress Disorder	10	6.5	32	8.1	49	6.8	4	3.1	95	6.8	3.9 (.27)
Suicidal	2	1.3	3	0.8	5	0.7	0	0	10	0.7	1.7 (.64)

Abbreviations:: Chi: chi-square test statistic across the 4 study groups. FAS: fetal alcohol syndrome. P: p-value. PFAS: partial FAS. ND/AE: Neurodevelopmental disorder/alcohol exposed. Normal CNS/AE: No central nervous system abnormalities/alcohol exposed. SE/AE: Static encephalopathy/alcohol exposed.

TABLE 12 Patient Satisfaction Survey outcomes from the University of Washington FAS DPN Clinic.

Characteristic	FASD Diagnostic Subgroups										Statistics
	1. 43 FAS/ 64 PFAS		2. SE/AE		3. ND/AE		4. Normal CNS/AE		Total		Chi-square
	N = 107		N = 248		N = 487		N = 88		N = 930		Chi (p)
Question on Patient Satisfaction Survey: N (valid%)											
1. Did we provide you with information you needed and were unable to get elsewhere?											
Yes	30	96.8	73	93.6	122	88.4	19	90.5	244	91.0	3.9 (.28)
2. Was the explanation of the patient's diagnosis easy to understand?											
Yes	36	90.0	71	81.6	133	85.8	22	91.7	262	85.6	2.5 (.48)
3. When you left Clinic, we recommended that you contact certain people and services to help you. How successful were you at finding these people and services?											
Very successful	15	46.9	33	55.0	55	44.0	9	52.9	112	47.9	3.7 (.30)
Somewhat successful	11	34.4	18	30.0	48	38.4	3	17.6	80	34.2	
4. If you were able to find the people and services we recommended to you, were they able to meet your needs?											
Yes, met all my needs	7	36.8	20	44.4	34	34.3	10	66.7	71	39.9	6.8 (.08)
Yes, met some of my needs	10	52.6	16	42.2	46	46.5	3	20.0	78	43.8	
No, they met none of my needs	1	5.3	3	6.7	6	6.1	0	0	10	5.6	
I was not able to find the people/services	1	5.3	3	6.7	13	13.1	2	13.3	19	10.7	
5. Would you recommend the FAS Clinic to other families with similar needs?											
Yes	32	100	75	98.7	143	98.6	21	100	271	98.9	0.7 (.87)
Duration of wait to get a diagnostic appointment. (yrs): mean SD	.53	.65	.59	.68	.56	.57	.49	.38	.56	.60	1.1 (.37)

Abbreviations:: Chi: chi-square test statistic across the 4 study groups. FAS: fetal alcohol syndrome. P: p-value. PFAS: partial FAS. ND/AE: Neurodevelopmental disorder/alcohol exposed. Normal CNS/AE: No central nervous system abnormalities/alcohol exposed. SD: standard deviation. SE/AE: Static encephalopathy/alcohol exposed.

TABLE 13 Selected contrasts between races.

Characteristic	Race (recorded as one race)										Statistics		
	1. Caucasian		2. Black		3. American Indian or Alaskan Native		4. Other (including mixed race)		Total		ANOVA		Chi-square
											Overall	Post Hoc	
	N = 684		N = 92		N = 115		N = 509		N = 1400		F (p)[A]	Duncan[B]	Chi (p)
FASD Diagnostic Group: N (valid%) 1. FAS/PFAS	87	12.7	17	18.5	6	5.2	44	8.6	154	11.0			30.1 (.00)
2. SE/AE	182	26.6	19	20.7	48	41.7	145	28.5	394	28.1			
3. ND/AE	357	52.2	45	48.9	57	49.6	263	51.7	722	51.6			
4. Normal CNS/AE	58	8.5	11	12.0	4	3.5	57	11.2	130	9.3			
Growth Rank: N (valid%) 1	436	63.7	60	65.2	87	75.7	340	66.8	923	65.9			12.9 (.17)
2	95	13.9	15	16.3	10	8.7	82	16.1	202	14.4			
3	88	12.9	12	13.0	10	8.7	54	10.6	164	11.7			
4	65	9.8	5	5.4	8	7.0	33	6.5	111	7.9			
Face Rank : N (valid%) 1	155	22.7	21	22.8	24	20.9	158	31.0	358	25.6			38.1 (.00)
2	372	54.4	46	50.0	79	68.7	275	54.0	772	55.1			
3	77	11.3	17	18.5	10	8.7	40	7.9	144	10.3			
4	80	11.7	8	8.7	2	1.7	36	7.1	126	9.0			
CNS Functional Rank: N (valid%) 1	106	15.5	20	21.7	12	10.4	98	19.3	236	16.9			14.7 (.02)
2	397	58.0	49	53.3	59	51.3	288	56.6	793	56.6			
3	181	26.5	23	25.0	44	38.3	123	24.2	371	26.5			
CNS Structural/Neurological Rank: N (valid%) 4	124	18.1	16	17.4	14	12.2	85	16.7	239	17.1			2.5 (.47)
Alcohol Rank : N (valid%) 1	0	0	0	0	0	0	0	0	0	0			23.5 (.00)
2	5	0.7	0	0	0	0	0	0	0	0			
3	278	40.6	39	42.4	39	33.9	270	53.0	626	44.7			
4	401	58.6	53	57.6	76	66.1	237	46.6	767	54.8			
Alcohol Use Before Pregnancy: N, mean (SD)													
Ave # drinks per drinking occasion	302	7.7(6.2)	45	8.5(6.9)	48	16.8(15.2)	192	10.8(13.1)	587	9.5(10.2)	13.3(.00)	124,3	
Max # drinks per drinking occasion	266	13.3(13.9)	43	12.7(9.1)	44	22.5(17.5)	183	16.0(17.4)	536	14.9(15.4)	5.2(.001)	124,3	
Ave # drinking days per week	383	4.7(2.2)	53	5.3(2.2)	55	4.1(2.3)	242	4.6(2.2)	733	4.7(2.2)	3.1 (.03)	341,12	
Alcohol Use During Pregnancy: N, mean (SD)													
Ave # drinks per drinking occasion	325	7.3(7.2)	45	8.2(6.2)	49	12.9(10.8)	211	9.4(13.4)	630	8.5(10.0)	5.4(.001)	124,3	
Max # drinks per drinking occasion	282	11.6(11.7)	41	13.4(8.2)	45	19.3(14.4)	197	12.9(13.0)	565	12.8(12.3)	5.1(.002)	124,3	
Ave # drinking days per week	415	4.6(2.3)	57	5.7(1.9)	57	3.6(2.3)	274	4.4(2.3)	803	4.5(2.3)	8.9(.00)	3,41,12	
Drank only in 1st trimester: N (valid%)	67	12.1	6	7.8	20	20.2	61	15.3	154	13.6			8.0 (.05)
Drank all 3 trimesters: N (valid%)	395	71.2	59	76.6	65	65.7	275	69.1	794	70.3			3.0 (.40)
Had an alcohol use problem: N (valid%)	569	90.5	66	82.5	107	94.7	435	92.8	1177	91.2			11.1(.01)
Diagnosed with alcoholism: N (valid%)	431	78.6	41	62.1	95	90.5	320	81.6	887	79.8			21.5 (.00)
Received alcohol treatment: N (valid%)	383	70.8	43	63.2	79	78.2	302	78.2	807	73.6			11.3 (.01)
Other adverse exposures in pregnancy: N (valid %)	488	92.6	77	96.3	65	87.8	345	94.8	975	93.3			6.3 (.09)
Child's age at diagnosis (yrs): N mean (SD)	684	9.3(6.9)	92	8.3(4.4)	115	9.5(5.5)	509	8.6(5.8)	1400	9.0(6.2)	2.2(.09)	--	
Mom's age(yr) at child's diagnosis: N mean (SD)	579	35.7(9.5)	73	35.1(6.1)	86	35.9(8.0)	412	33.4(7.9)	1147	34.8(8.7)	5.9(.001)	123,4	
Mom's age(yr) at child's birth: N mean (SD)	576	26.4(6.5)	73	26.7(5.3)	86	26.5(6.1)	412	24.9(6.1)	1147	25.9(6.3)	5.3(.001)	123,4	
Parity of index child: N mean (SD)	570	2.5 (1.5)	77	3.2 (1.8)	99	3.3 (2.1)	410	2.8 (1.8)	1156	2.7 (1.7)	7.8 (.00)	14,23	

Abbreviations: Chi: chi-square test statistic across the 4 racial groups. FAS: fetal alcohol syndrome. P: p-value. PFAS: partial FAS. ND/AE: Neurodevelopmental disorder/alcohol exposed. Normal CNS/AE: No central nervous system abnormalities/alcohol exposed. SD: standard deviation. SE/AE: Static encephalopathy/alcohol exposed. Notations: A. Numerator degrees of freedom = 3; denominator df = total sample size minus 4. B. The Duncan multiple comparisons range test is reported if the overall ANOVA is statistically significant; commas separate groups with homogeneous means at $p < 0.05$.

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