

Addressing Fetal Alcohol Spectrum Disorders (FASD)

A Review of the Literature*

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TREATMENT IMPROVEMENT PROTOCOL (TIP) SERIES

58

*This document is available online only (<http://store.samhsa.gov/>) and supports TIP 58, Addressing Fetal Alcohol Spectrum Disorders (FASD).



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Part 3, Section 1—Literature Review

Overview

This Treatment Improvement Protocol (TIP) is designed for use by behavioral health service providers and practitioners to address Fetal Alcohol Spectrum Disorders (FASD). Professionals in almost any healthcare and social service field can also apply the information in this TIP when working with women at risk of an alcohol-exposed pregnancy (AEP) or individuals who may have an FASD.

The dearth of treatment approaches for and practitioner knowledge of FASD has prompted the development of this TIP. All too often the care that is given to an individual with an FASD is not matched to the person's needs and strengths, which increases the risk for secondary disabilities (Streissguth and Kanter, 1997). The brain damage suffered prenatally is permanent and impacts the individual's cognitive and behavioral abilities. Consequently, typical treatment approaches do not always work as they assume more complex processing abilities. This TIP provides an understanding of the range of behavioral deficits, screening processes, prevention approaches, needs and services for families as well as strategies for intervention.

Intervention and prevention go hand in hand. Intervention with women of childbearing age is prevention of AEP and possibly FASD. Intervention with women with an alcohol-exposed child can prevent subsequent AEPs. Intervention with an individual with an FASD can prevent adverse life outcomes. Increased awareness among primary and behavioral health providers to the deleterious effects of prenatal alcohol exposure may reverse the current undetected and misdiagnosed scenarios and expand appropriate the service delivery, resulting in improved outcomes for individuals, families, and programs.

TIP Organization

This TIP consists of three parts. Parts 1 and 2 are bound together in a printed volume and Part 3 is available only online.

Part 1 of the TIP is for behavioral health providers and consists of three chapters:

- Chapter 1 discusses approaches to preventing FASD; that is, assisting women who are in treatment settings and are pregnant or may become pregnant to remain abstinent from alcohol. In providing these guidelines, this TIP adopts the Institute of Medicine (IOM) model for prevention, which sees prevention as a step along a continuum that also incorporates treatment and maintenance.
- Chapter 2 discusses methods for identifying individuals in treatment who have or may have an FASD, referring them for diagnosis where possible, and providing appropriate interventions to meet their needs.
- Chapter 3 provides clinical vignettes designed to realistically portray the provider–client interactions that might take place when providing FASD prevention or interventions.

Part 2 is an implementation guide for program administrators and consists of two chapters.

- Chapter 1 lays out the rationale for the approach taken in chapter 2 and will help readers understand how administrators can provide support for programs and counselors as they address FASD. It is hoped that this knowledge will enhance the ability of treatment programs to address FASD concurrently with other behavioral health needs; addiction, mental health issues, etc.
- Chapter 2 provides detailed information on how to achieve high-quality implementation of the recommendations in **Part 1**.

Part 3 of this TIP is a literature review on the topic of FASD and is available for use by clinical supervisors, interested counselors, and administrators. *Part 3* includes literature that addresses both clinical and administrative concerns. To facilitate ongoing updates, the literature review will only be available online.

The following topics are addressed in Part 3:

- Review of literature pertaining to clinical issues of Part 1 of this TIP
 - Prevalence
 - Cost of Care
 - Screening
 - Diagnosis
 - Teratogenic Science
 - Behavioral Deficits
 - Co-Occurrence
 - Interventions
 - Research
- Review of screening reimbursement codes, Affordable Care Act (ACA), and Child Abuse Prevention and Treatment Act (CAPTA)
- Information about the methodology used to perform the literature search (see Appendix A)
- A select list of abstracts of core sources
- A general bibliography

Fetal alcohol syndrome (FAS) is a *specific* diagnosable birth defect caused by alcohol use during pregnancy. FASD describes a range of disorders all associated with prenatal alcohol exposure. It is an umbrella term describing the *range* of effects that can occur in an individual whose mother drank alcohol during pregnancy. The range of deficits includes brain damage; physical, cognitive, emotional, behavioral, and/or learning disabilities. **FASD is not a diagnosis.**

Prevalence of FASD

In 2005 the Office of the U.S. Surgeon General issued a press release stating, “it is estimated that for every child born with FAS, three additional children are born who may not have the physical characteristics of FAS but still experience neurobehavioral deficits resulting from prenatal alcohol exposure that affect learning and behavior.”(U.S. Department of Health and Human Services [HHS], 2005).

The actual prevalence of FAS and FASD has proved difficult to uncover for several reasons. One of the primary reasons is the paucity of diagnostic services. Another reason is that primary care physicians do not tend to recognize the possibility of FAS and FASD in their patients. Other reasons include the knowledge and capacity to diagnose. In addition, diagnoses other than FAS under the FASD spectrum (such as Alcohol Related Neurodevelopmental Disorder [ARND] and Partial Fetal Alcohol Syndrome [PFAS]) are not included in all diagnostic systems, specifically the Centers for Disease Control and Prevention (CDC) Fetal Alcohol Syndrome Surveillance Network (FASSNet) system that only records FAS (Bertrand et al., 2004). In addition, identification of FAS can be difficult because the facial dysmorphism that characterizes the condition may not be obvious and deficits in the central nervous system may not be clearly manifested at birth (Little, 1990; Floyd, O'Connor, Sokol, Bertrand, and Cordero, 2005).

The less severely dysmorphic forms of FASD, however, are complex, involving multiple indicators of physiology, development, and behavior, many of which are neither obvious nor easily identified at birth (Clarren, Randels, Sanderson, and Fineman, 2001; Floyd et al., 2005; May et al., 2009). In a population of 1,400 individuals with prenatal alcohol exposure attending a FASD diagnostic clinic, for every child diagnosed with FAS, 8 other children have been diagnosed with another FASD (i.e., a neurobehavioral disorder without the physical features of FAS) (Astley, 2010). While there are accurate screening tools available, including the FASD 4-Digit Code, there is no universal assessment currently in use. Researchers are generating estimates in general and high-risk populations both nationally and internationally (Astley, Stachowiak, Clarren, and Clausen, 2002; May, 2009; May et al., 2011). Other barriers to estimating the prevalence include the negative stereotypes and perceptions associated with maternal alcohol use during pregnancy and the difficulty in confirming prenatal alcohol exposure. ARND requires a confirmed exposure because the condition does not include the highly specific FAS facial phenotype.

Researchers have taken different approaches to studying prevalence including passive surveillance, clinic-based ascertainment and active case ascertainment. In 2009, May and colleagues reviewed FAS and FASD prevalence literature and examined the advantages and disadvantages of each approach.

Passive Surveillance

The first and most common method is passive surveillance. In the passive surveillance method, information is obtained through birth defects registries and physician records or school records. This method is inexpensive, easy to implement, and uses existing systems for data collection (May et al., 2009). The primary limitation of passive surveillance is it underestimates the true prevalence. This method is used and funded by the CDC FASSNet (Druschel and Fox, 2007; CDC, 2002). FASSNet uses several sources to obtain information on cases of FAS. Sources include birth defects monitoring programs, Medicaid files, early intervention programs, and developmental and genetic clinics (CDC, 2002). The CDC funded projects for FASSNet in Alaska, Arizona, Colorado, New York, and Wisconsin from 1995–1997. A report on their findings in 2002 stated that the prevalence in “Alaska, Arizona, Colorado, and New York ranged from 0.3 to 1.5 per 1,000 live-born infants...” (CDC, 2002).

Another FASSNet study from Druschel and Fox (2007) examined prevalence in two New York counties. They found the prevalence rate of FAS in Erie County to be 0.90 per 1,000 live births

and Monroe County to be 0.21 cases per 1,000 live births in 1995–1999. They suspected that their results in Monroe County may have been influenced by less “active” participation of data sources, and clinician difficulty obtaining documentation of facial features for diagnosis. They also state that FASSNet data sources, such as birth defects registries, were more biased toward newborns. In addition, 30 percent of the New York FASSNet cases were diagnosed after 2 years of age and most of those cases were captured through genetic clinics (Drushel and Fox, 2007).

Morleo et al. (2011) found that use of hospital records as tools for estimation of prevalence may lead to under-reporting of FAS and FASD. They found that regional data on alcohol-related hospital admissions for women of child-bearing age were not connected with higher levels of FASD. Morleo et al. (2011) found that the 41 percent increase in regional data on alcohol related hospital admissions for women of child-bearing age during 2002–2003 and 2007–2008 did not result in an increase in the reported numbers of FASD-related conditions. Morleo et al. (2011) states that the results suggest that incidence is under-reported by hospital admission data.

Clinic/Hospital Based

The second method May et al. (2009) examined was clinic-based studies of prevalence of FAS and FASD. Clinic-based studies of FASD are usually initiated in the prenatal care unit of hospitals with subsequent evaluation of the offspring of those pregnancies (May and Gossage, 2001). This allows for design control and consistency of record keeping (May et al., 2009). They also allow access to pregnant women so information can be collected on maternal use of alcohol and drugs, diet, and other variables. Abel and Sokol (1987) reviewed 18 clinic-based studies and reported a prevalence rate for FAS of 2.2 per 1,000 live births. Of nine North American clinic-based studies they reviewed, five of the studies were in minority populations in low-income areas and had overall larger sample sizes. In 1995, Abel expanded the literature search conducted earlier and revised the prevalence rate of FAS to 1.95 per 1,000 live births. Studies in the United States reviewed were with low-income minority populations. These results are difficult to extrapolate to the general population of the United States as these clinics are considered high risk for a number of reasons and may not reflect the general population. May and Gossage (2001) also point out that women with the highest risk of having a child with FAS may not use prenatal clinics regularly or may not attend at all.

Hospital-based surveillance of FAS and ARND at birth will miss about half of the newborns with FAS and all of the newborns with ARND. A diagnosis of ARND requires confirmation of severe brain dysfunction. A newborn is too young to assess brain function. FAS requires microcephaly and/or severe brain dysfunction. Only the subset of newborns with microcephaly will be detectable at birth (Astley 2011). May et al. (2009) report that it is almost impossible to diagnose FASD cases in the first 6 weeks of life, “...only the most severe cases are obvious cases and diagnosable throughout the infant period” (p. 179).

Active Case Ascertainment

Another method of estimating prevalence is active case ascertainment. Active case ascertainment efforts are a more thorough, prospective approach to estimating prevalence. These studies actively screen and diagnose children at risk for FASD (May and Gossage, 2009; Astley et al., 2002; Astley, 2004b). Active case ascertainment methods provide a more accurate estimate of FASD in selected populations as long as there is a large participation rate of the selected populations.

Advantages to this approach include being able to find children at the appropriate age for diagnosis and provide more applicable findings (May et al., 2009; Stratton, Howe and Battaglia, 1996). May et al. (2009) summarized data from three small (pilot) in-school case ascertainment studies in the United States. Those data suggest an FASD prevalence rate of 16.5 per 1,000 younger school children. Data using similar methods in South Africa and Italy suggest higher mean FASD prevalence rates of 72.3 and 35.7, respectively, per 1,000 younger school children (May et al., 2009). May et al. 2011 also used similar methods to estimate the rate of FASD for two health districts near Rome. They estimated the rate of FASD to be between 23.1 to 62.6 per 1,000 (or 2.3 percent to 6.3 percent) in those communities (May et al., 2011).

Stratton et al. (1996) points out that the disadvantages of active surveillance may include the high cost and labor required to collect data from a population significant enough to yield results, and it is a selective process because only those with the condition who participate will be included. Despite the challenges often faced in active surveillance efforts, a long-term active surveillance of FAS was conducted in a foster care population in Washington State from 1999 to 2009 (Astley et al., 2002; Astley, 2004b). All children entering the public foster care system had a digital facial photograph taken that was evaluated using the FAS Facial Photographic Analysis Software developed by Astley. All children with the full FAS facial phenotype (Rank 4, as defined by the 4-Digit Code) screened positive and received a full FASD diagnostic evaluation. Ninety-five percent of the children who screened positive for the FAS facial phenotype received a diagnosis of FAS. The prevalence of FAS in this foster care population was 1/100; ten times higher than the prevalence in the general population (Astley et al., 2002). There was a 98 percent participation rate of the foster population in this active surveillance program.

After an analysis of existing studies May et al. (2009) concluded that passive surveillance produced the lowest rates of prevalence among the three methods. They stated that method may not fully capture all cases of FASD because FASD is harder to identify.

Cost of Care

The financial cost of FAS/FASD to society is staggering. Amendah, Grosse, and Bertrand (2011) estimated that annual medical spending on children with FAS and found it was nine times higher than children without an FAS diagnosis. They used the MarketScan Multi-State Medicaid database from 2005 to determine their estimate. Children with FAS had an average annual medical expense of nearly \$17,000. Children with an FAS and a record of intellectual disability had a mean annual medical expense of \$39,000 (Amendah et al., 2011).

Lupton, Burd, and Harwood (2004) estimated that the lifetime cost of care for an individual with FAS is up to \$2.44 million after adjusted for inflation (Bureau of Labor Statistics). This estimate includes costs for medical services and productivity losses. The findings of Lupton et al. (2004) were used to develop an **online cost calculator tool**, which is available online at the online clinic of Dr. Larry Burd (<http://online-clinic.com/>). The cost calculation is based on North Dakota costs of healthcare, special education, juvenile justice, and other services (Lupton et al., 2004).

In 1992, Astley, Bailey, Talbot, and Clarren (2000b) conducted a 5-year FAS primary prevention study in Washington State to assess the feasibility of using a FASD Diagnostic clinic as a center for identifying and targeting primary prevention intervention to high-risk women (namely women who had given birth to a child with FAS). Their approach documented the cost of raising a child with FAS was roughly 30 times the cost of preventing FAS in the child (Astley et al., 2000b).

Cost studies of FASD are less common. Thanh, Jonsson, Dennett, and Jacobs (2011) performed a meta-analysis of cost studies in the United States and Canada. They estimate the annual U.S. cost of FASD for 2009 to be \$5 billion, the annual individual cost of FASD to be \$20,460.95, and the lifetime individual cost to be \$1.5 million. This estimate includes healthcare services, educational and social services, correctional services, and others (Thanh et al., 2011; www.x-rates.com used for currency conversion at 2009 price level).

The cost of care estimates vary for a number of reasons:

- Different prevalence rate estimates can be used
- Differences in estimates of types, utilization rates, and costs of medical care and other services used by individuals affected by FASD
- Inclusion of residential support cost as some studies only estimate these cost up to age 21, while others estimate cost of care up to age 65
- Inflation
- Advancing current knowledge of FASD compared to more dated studies
- Inclusion of lost productivity (Lupton et al., 2004)

Screening Women at Risk

The risk of an alcohol-exposed pregnancy impacts all women of childbearing age regardless of ethnicity, socio-economic status, or race. The National Task Force on Fetal Alcohol Syndrome and Fetal Alcohol Effect recommended that providers routinely screen all women of childbearing age for alcohol abuse disorders (CDC, 2009).

Screening can be performed in a number of settings. A critical factor in screening is the use of a validated tool for women. The T-ACE and TWEAK are two tools that have been designed for women in the prenatal setting. The CRAFFT was designed and validated for adolescents. Tables 1 and 2 list a number of screening tools for women.

Table 1. Evidence-Based Screening Tools for Women at Risk (validated for women at risk; not for women with an FASD). See Appendix B of Part 1 of the TIP for Full Screening Tool.

Tool	Description	Source
T-ACE	Brief (< 2min) Designed for use in prenatal settings Easy to score No training required	Sokol, Martier and Age, 1989; additional info from Savage et al., 2003; Sarkar et al., 2009

	<ul style="list-style-type: none"> • Validity varies across ethnic populations • Identifies primarily heavy and late-stage drinkers 	
TWEAK	<ul style="list-style-type: none"> • Brief (< 2min) • Designed for use in prenatal settings • Easy to score • Optimal for racially diverse groups • Validity varies across ethnic populations • Identifies primarily heavy and late-stage drinkers 	Russell, 1994; additional info from Savage et al., 2003; Sarkar et al., 2009; Anthony, Austin, and Cormier, 2010
CRAFFT	<ul style="list-style-type: none"> • Designed and validated for detecting a range of alcohol problems with adolescents 	Knight et al., 1999; additional information from Knight, Sherrit, Harris, Shrier, and Chang, 2002.

The complete T-ACE, TWEAK and CRAFFT screening tools are included in Appendix B of Parts 1 and 2.

Table 2. Additional Tools for Screening Women at Risk

Tool	Description	Source
4P's Plus	<ul style="list-style-type: none"> • Brief • Modifies 4P's screening tool • Designed to identify light to heavy prenatal use of alcohol, illicit drugs, and tobacco in obstetric settings • Demonstrated high validity in urban settings, reliably and effectively screens women including women usually missed by other perinatal screening methods 	Chasnoff et al., 2005; additional information from Chasnoff, Wells, and Bailey, 2007
5P's (Modified)	<ul style="list-style-type: none"> • Brief • Modifies 4P's to discuss drinking during pregnancy • Inclusion of peer question makes it suitable for screening pregnant teenagers • Lack of validation through study 	Kennedy, Finkelstein, Hutchins, and Mahoney, 2004; additional info from Anthony et al., 2010
Quick Drinking Screen (QDS)	<ul style="list-style-type: none"> • Brief • Use when daily drinking data is not available • Lack of validation through study 	Sobell and Sobell, 2003; additional info from Dum et al., 2009
Ten Question Drinking History (TQDH)	<ul style="list-style-type: none"> • Designed for use in prenatal settings • A longer screening tool • Lack of validation through study • May miss binge and heavy drinking 	Weiner, Rosett, and Edlin, 1982; additional info from Savage et al., 2003
Timeline Followback (TLFB)	<ul style="list-style-type: none"> • Validated across broad age range • A longer screening tool • Not specifically validated for pregnant women 	Sobell and Sobell, 1995; additional info from Dum et al., 2009

Diagnostics

There is currently no universal approach to diagnosis. Five diagnostic systems for FAS/FASD are currently in use in North America. All five systems include an assessment of the unique FAS facial characteristics, often referred to as dysmorphic features. They are:

- Short palpebral fissure (width of the opening of the eye, *See Figure 1*),
- Thin vermilion border of the upper lip, and
- Smooth philtrum (the vertical groove between the nose and upper lip, *See figure 1*).

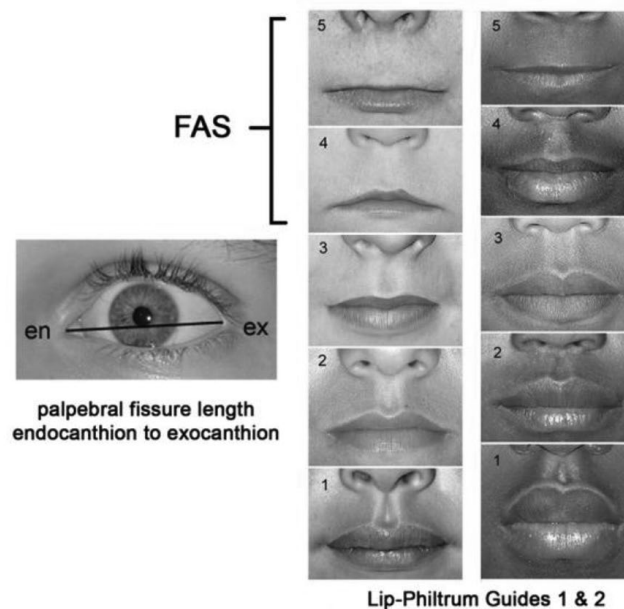


Figure 1. Palpebral fissure and Lip-Philtrum Guide.

With permission from Susan Astley Ph.D., University of Washington.

The facial features are some of the key diagnostic features of FAS along with Central Nervous System (CNS) involvement, and growth retardation. In addition to facial features, growth retardation, central nervous system involvement, and confirmation of alcohol exposure are also part of the evaluation. Please refer to Part 1, Appendix E of this TIP for a side-by-side comparison table of these five systems (Table developed by Astley 2011).

Central to all of the schema for the diagnosis of an FASD is the recognition of specific dysmorphia and, more importantly, clear evidence that the behavioral and adaptive problems faced by the patient are in whole or in part due to diffuse brain damage and not to environmental or psychiatric conditions alone. Thus the diagnostic evaluation requires a team approach in all systems combining a medical evaluation with a neurocognitive assessment and an appropriate determination of alcohol exposure *and*, finally, an exclusion of other possible diagnoses.

The five diagnostic systems are:

- The Institute of Medicine's (IOM) established guidelines in 1996 for diagnosing FAS (with and without confirmed prenatal alcohol exposure), PFAS, ARND, and Alcohol Related Birth Defects (ARBD)
- The University of Washington established the FASD 4-Digit Diagnostic Code in 1997 for diagnosis of FAS, partial FAS, Static Encephalopathy/Alcohol Exposed and Neurobehavioral Disorder/Alcohol Exposed. The 3rd edition of the Code was printed in 2004 (Astley and Clarren, 1997; Astley, 2004a)
- The CDC's National Task Force on FAS and FAE established guidelines in 2004 for diagnosis of FAS. (Bertrand et al., 2004)
- Hoyme and colleagues published a clarification of the IOM Guidelines in 2005 for diagnosis of FAS, PFAS, ARND, and ARBD
- Chudley and colleagues established the Canadian FASD diagnostic guidelines in 2005 for diagnosis of FAS, PFAS, and ARND that blends the nomenclature of the IOM and methodology of the 4-Digit Code

Institute of Medicine's Guidelines

In 1996, the IOM issued diagnostic criteria for FAS, PFAS and introduced two other alcohol-related conditions, ARND and ARBD. The IOM system has two categories for FAS with confirmed maternal intake of alcohol at an excessive level as defined by Stratton et al. (1996) and without confirmed maternal alcohol exposure. For a diagnosis of FAS, the IOM Guidelines require the presence of a characteristic pattern that includes features such as short palpebral fissures, flat upper lip, flattened philtrum, and flat midface (Stratton et al., 1996). The diagnostic criteria for FAS and PFAS include requirements for growth retardation in at least one of the following areas:

- Low birth weight for age
- Low weight for height
- Decelerating weight (not due to nutrition) (Stratton et al., 1996)

The IOM Guidelines outline areas for CNS neurodevelopmental abnormalities:

- Decreased cranial size at birth
- Structural brain abnormalities (such as microcephaly, partial/complete agenesis of the corpus callosum, cerebellar hypoplasia)
- Neurological hard or soft signs (as appropriate for age) such as impairment in fine motor skills, hearing loss, poor tandem gait, or poor hand-eye coordination (Stratton et al., 1996)

Diagnostic criteria for FAS require evidence of at least one of the CNS neurodevelopmental abnormalities described.

A diagnosis of PFAS using the IOM Guidelines requires confirmed maternal intake of alcohol at an excessive level as defined by Stratton et al. (1996) and the presence of some components of the facial anomalies. The requirements for evidence of one of the following: growth retardation, CNS neurodevelopmental abnormalities, or a complex pattern of behavior/cognitive abnormalities. The behavior or cognitive abnormalities can include: "learning difficulties; deficits in school performance; poor impulse control; problems in social perception; deficits in higher

level receptive and expressive language; poor capacity for abstraction or metacognition; specific deficits in mathematical skills; or problems in memory, attention or judgment” (Stratton et al., 1996).

The IOM Guidelines also include two other terms related to maternal alcohol exposure, ARND and ARBD. Both conditions require confirmed maternal intake of alcohol at an excessive level (Stratton et al., 1996). Neither diagnosis requires evidence of growth retardation or facial abnormalities. ARND requires presence of CNS neurodevelopmental abnormalities and/or a complex pattern of behavior/cognitive abnormalities. The guidelines for ARBD require at least one congenital abnormality listed in the guidelines, possibly in the cardiac, skeletal, renal, ocular, auditory, or other area (Stratton et al., 1996).

The University of Washington’s FASD 4-Digit Diagnostic Code

The four digits of the FASD 4-Digit Code reflect the magnitude of expression of the four key diagnostic features of FASD, in the following order: (1) growth deficiency, (2) FAS facial phenotype, (3) CNS structural/functional abnormalities, and (4) prenatal alcohol exposure. 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 classical presence of the FASD feature.

The 4-Digit Code produces four diagnostic sub-classifications under the umbrella of FASD: FAS, PFAS, Static Encephalopathy/Alcohol Exposed (SE/AE), and Neurobehavioral Disorder/Alcohol Exposed (ND/AE). The 4-Digit Code is the only diagnostic system with an FAS facial phenotype confirmed to be highly specific to prenatal alcohol exposure and FAS. The specific criteria for each diagnostic classification are presented in Part 1 of this TIP. In general, the diagnostic classifications are defined as follows:

- Fetal Alcohol Syndrome (alcohol exposed):
Individuals with growth deficiency (height and/or weight at or below the 10th percentile); the full FAS facial phenotype (all three of the following features: palpebral fissure length at or below the 3rd percentile, smooth philtrum and thin upper lip); significant structural, neurological, and/or functional CNS abnormalities; and confirmed prenatal alcohol exposure (Astley, 2004a).
- Fetal Alcohol Syndrome (alcohol exposure unknown):
Individuals with the growth, face and CNS features of FAS but the alcohol exposure is unknown (Astley, 2004a).
- Partial Fetal Alcohol Syndrome (alcohol exposed):
Individuals with significant structural, neurological, and/or functional CNS abnormalities, most (but not all) of the growth or facial features of FAS, and a confirmed history of prenatal alcohol exposure (Astley, 2004a).
- Static Encephalopathy/Alcohol Exposed:
Individuals with confirmed prenatal alcohol exposure who present with severe CNS structural or functional abnormalities, but no FAS facial phenotype (Astley, 2004a).
- Neurobehavioral Disorder/Alcohol Exposed:
Individuals with confirmed prenatal alcohol exposure and moderate CNS dysfunction, but no FAS facial phenotype (Astley, 2004a).

The most recent Diagnostic Guide for the 4-Digit Diagnostic Code can be accessed here: <http://depts.washington.edu/fasdpn/pdfs/guide2004.pdf>. In addition, the FASD 4-Digit Code Caregiver Interview Checklist is provided in Part 1, Chapter 2, of this TIP and in Appendix D. The checklist is provided as a means for collecting information about significant delay/impairments across a wide array of functional domains. This qualitative information gathered from caregivers is used to complement the more rigorous, quantitative neuropsychological assessment obtained through a standardized neuropsychological assessment. It is not presented as a validated FASD screening tool.

The CDC's National Task Force on FAS and FAE

In 2004, the National Center on Birth Defects and Developmental Disabilities Centers at the CDC in coordination with National Task Force on Fetal Alcohol Syndrome and Fetal Alcohol Effect released guidelines for diagnosis of FAS (Bertrand et al., 2004). Diagnostic criteria were based on facial anomalies, growth problems, and CNS abnormalities. The CDC system requires the same three facial features as the 4-Digit Code, but relaxed the palpebral fissure length criteria to below the 10th percentile. Adjusted height or weight should be at or below the 10th percentile. CNS abnormalities must be present as well as structural issues, neurological issues, or functional deficits must be present. Structural issues that may be present include an adjusted head circumference below the 10th percentile or clinically significant brain abnormalities. Neurological issues that may be present include seizures, or other soft neurological signs outside normal limits. Functional issues that may be present include global cognitive deficit (decreased IQ) or significant developmental delay in young children. Deficit in three or more functional domains may also be used as criteria for diagnosis. Domains include cognitive or developmental discrepancies; executive functioning; motor; social skill problems; attention/hyperactivity; and other such as sensory, language, or memory (Bertrand et al., 2004). The guidelines can be accessed online at http://www.cdc.gov/ncbddd/fasd/documents/fas_guidelines_accessible.pdf.

Hoyme et al. (2005 Revision to the IOM Guidelines)

In 2005, Hoyme et al. clarified the IOM guidelines. Under these guidelines FAS can be diagnosed with either confirmed maternal intake of alcohol at an excessive level as defined by Hoyme et al. (2005) or unknown maternal alcohol exposure. The criteria for FAS use the same three facial features as the 4-Digit Code, but relax the criteria to require only two of the three features and relaxed the palpebral fissure length criteria to be at or below the 10th percentile. The Hoyme et al. guidelines use the University of Washington Lip-Philtrum Guide (Astley, 2004a). In addition to the facial characteristics, evidence of prenatal and/or post natal growth retardation must be present. This is determined by height and weight. Lastly, deficit in brain growth must be evident through structural abnormalities and/or small head circumference (Hoyme et al., 2005). Brain function is not addressed in the FAS criteria.

The PFAS diagnostic criteria have much of the same requirements as the FAS requirements. PFAS can be diagnosed with either confirmed maternal intake of alcohol at an excessive level or unknown maternal alcohol exposure. The facial criteria for PFAS are the same as the requirements for FAS. A diagnosis of PFAS requires evidence of at least one of the following areas:

- Prenatal and/or post natal growth retardation (determined by height and weight)

- Deficient brain growth or morphogenesis evident through structural abnormalities and/or small head circumference
- Complex pattern of cognitive or behavioral abnormalities not consistent with developmental level must be evident or explained by a genetic predisposition, family background, or environment (Hoyme et al., 2005)

This complex pattern includes:

...marked impairment in the performance of complex tasks (complex problem solving, planning, judgment, abstraction, metacognition, and arithmetic tasks); higher-level receptive and expressive language deficits; and disordered behavior (difficulties in personal manner, emotional liability, motor dysfunction, poor academic performance, and deficient social interaction. (Hoyme et al., 2005, p. 44).

The diagnostic criteria for ARND require confirmed maternal alcohol exposure. One of the following must be present:

- Deficit in brain growth must be evident through structural abnormalities
- Deficient brain growth or morphogenesis evident through structural abnormalities and/or small head circumference
- Complex pattern of behavioral or cognitive abnormalities not consistent with developmental level

The criteria for ARBD require confirmed maternal alcohol exposure and two or more facial anomalies. In addition, one or more congenital or structural defects, including malforms and dysplasias, must be present or one or more of the following categories: cardiac, skeletal, renal, eyes, or ears. Alternatively, if the patient displays only display minor anomalies as specified by Hoyme et al. (2005), two or more must be present. The term ARBD is not recognized by the Canadian Guidelines for diagnosis or the University of Washington's FASD 4-Digit Code (Chudley et al., 2005; Astley 2004a). According to Chudley et al. (2005), it is recognized that alcohol is a teratogen may be responsible for numerous birth defects. However, "...in the absence of other features of FAS or brain deficits, it is difficult to attribute causation" to prenatal alcohol exposure (Chudley et al., 2005).

The Canadian Guidelines for diagnosis

In 2005, a subcommittee of the Public Health Agency of Canada's National Advisory Committee on FASD examined current approaches for diagnosis and made recommendations for diagnosis of FASD-related conditions in Canada. The committee recommended that the 4-Digit Diagnostic Code, "should be used to describe, assess and measure objectively alcohol exposure, growth, facial features and brain damage" and that, "terminology in the IOM criteria should be used to describe the diagnosis" (Chudley et al., 2005).

For diagnosis of FAS, there must be evidence of prenatal or postnatal growth impairment, which includes one of the following criteria at or below the 10th percentile:

- Low birth weight or length
- Height or weight
- Disproportionately low height to weight ratio (Chudley et al., 2005)

All three facial anomalies must be present using University of Washington's Lip-Philtrum Guide. There must be confirmed impairment in three or more of the CNS domains specified. The domains include:

- Hard and soft neurologic signs
- Brain structure; cognition
- Communication; academic achievement
- Memory
- Executive functioning and abstract reasoning
- Attention deficit/hyperactivity
- Adaptive behavior
- Social skills
- Social communication (Chudley et al., 2005)

FAS can be diagnosed with confirmed maternal alcohol exposure or unknown exposure. (Chudley et al., 2005).

For diagnosis of PFAS, at least two facial anomalies must be present. As with diagnosis of FAS, for PFAS there must be confirmed impairment in three or more of the CNS domains specified. Growth retardation is not required for diagnosis. Maternal alcohol exposure must be confirmed (Chudley et al., 2005). The Canadian diagnostic criteria for ARND require confirmed maternal alcohol exposure and confirmed impairment in three or more of the CNS domains specified (Chudley et al., 2005). The Canadian guidelines for diagnosis can be accessed online here http://www.cmaj.ca/content/172/5_suppl/S1.full.

ARND—Consensus Statement

In 2011, the Interagency Coordinating Committee on Fetal Alcohol Spectrum Disorders (ICCFASD) organized a conference to address “Recognizing Alcohol-Related Neurodevelopmental Disorder in primary Health Care of Children.” The goal of the meeting was to reassess whether sufficient evidence currently exists to encourage screening and diagnosis of ARND in primary health care of children and to make recommendations and identify future directions. A multidisciplinary panel used the evidence presented to them by experts in the field to develop answers to principal issues that were presented in the form of five questions. The consensus statement that was subsequently developed and released on February 9, 2012 is a compilation of the answers to the questions. The five questions follow. The answers are provided in Appendix A of this document.

Question 1: What is ARND; how can it be diagnosed (classical, current diagnostic schemes, in practice today)?

Part A: Evidence of CNS Developmental Abnormalities

Part B: Evidence of a Complex Pattern of Behavior and Cognitive Abnormalities

Question 2: Can ARND be differentiated from other disorders?

Question 3: What prenatal alcohol exposure evidence is necessary for an ARND diagnosis?

Question 4: What signs/symptoms will be useful as screening criteria?

Question 5: What are the treatment needs for those diagnosed with ARND?

Teratogenic Science and the Brain

A teratogen is an environmental agent (drug or other physical substance) capable of interfering with the development of a fetus, causing birth defects. Alcohol is a potent teratogen that affects the development of the brain. To understand the magnitude of the insult to the brain from alcohol, an understanding of the brain structure and function is necessary. Studies of the brain in children affected by FASD allow researchers to examine which areas of the brain have been affected. Continued research is needed to fully understand the functional significance of damaged areas of the brain in individuals affected by FASD. Identifying which brain areas are impacted by alcohol and knowing the corresponding function of that brain area may enable improved interventions. Figure 2 illustrates several parts of the brain impacted by alcohol exposure.

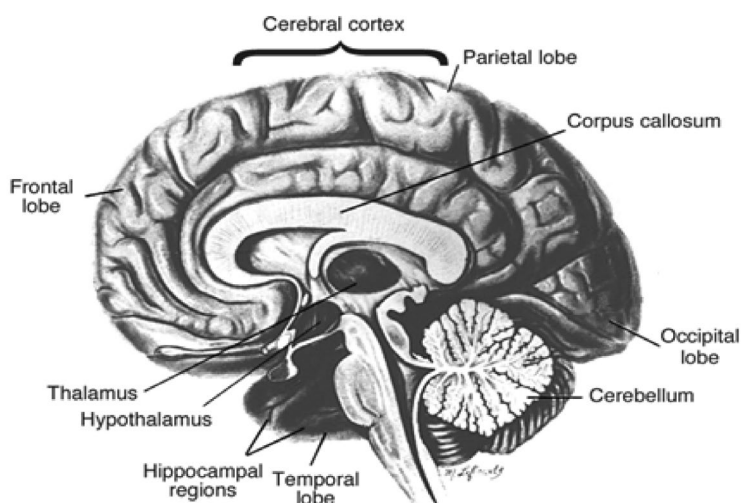


Figure 2. Schematic drawing of the human brain.

Source: Oscar-Berman, M. and Marinlovic, K. Alcoholism and the brain: An overview. *Alcohol Research and Health* 27(2):125–133, 2003. <http://pubs.niaaa.nih.gov/publications/arh27-2/125-133.htm>.

Cerebral cortex

The cerebral cortex is a thin layer of tissue just below the surface of the cerebrum. This is the area dense in neurons, the cells that do most of the information processing within the brain (NINDS, 2001). Studies report that neuronal loss is the main cause of reduced brain mass and lifelong neurobehavioral disturbances resulting from early ethanol exposure. (Han et al., 2005;

Medina, 2011). Ethanol exposure may also lead to persistent impairments in the function of the surviving neurons (Medina 2011).

The cerebral cortex is referred to as gray matter because the nerves that make up this area do not have the insulation that makes the other areas of the brain appear white (NINDS, 2001). The other areas of the brain are referred to as white matter. The white matter is largely structural elements and connections from one area of the central nervous system to others. The cerebral cortex showed increase in thickness of up to 1.2mm in subjects affected by FASD compared with the control group in the lateral brain surface in frontal, temporal, occipital, and parietal cortices (Sowell et al., 2008).

Overall volume/microcephaly

According to the National Institute of Neurological Disorders and Stroke (NINDS) (2008), “Microcephaly is a medical condition in which the circumference of the head is smaller than normal because the brain has not developed properly or has stopped growing.” The NINDS Web site states that depending on the severity of the syndrome that accompanies the microcephaly, children may have mental retardation, delayed motor functions, hyperactivity, seizures, difficulties with coordination and balance, and among other neurological issues (NINDS, 2008). Some children may only experience mild disability with normal intelligence (NINDS, 2008). Not all areas of the brain are equally affected by alcohol exposure but there is a reduction in the size of the cranial vault and brain size. (Sowell 2001; Astley et al., 2009b; Astley, 2010).

Head circumference at or below the 10th percentile is a criteria but not a requirement for FAS diagnosis in the revised IOM Criteria and CDC guidelines (Bertrand et al., 2004; Hoyme et al, 2005). Microcephaly (a head circumference at or below the 3rd percentile) is a criteria but not a requirement for FAS, PFAS, and static encephalopathy/alcohol-exposed diagnosis in the 4-Digit Code guidelines (Astley, 2004a).

In a study of 1,400 patients with prenatal alcohol exposure, Astley (2010) documented the prevalence of microcephaly among those with FAS/PFAS (45 percent) was twice as high as those diagnosed with Static Encephalopathy/Alcohol Exposed (severe brain dysfunction without the FAS facial phenotype). In an MRI study of children with FASD, Astley, Aylward et al. (2009b) documented a significant trend of decreasing total brain volume with increasing severity of FASD diagnostic classification. Significant correlations were observed between the size of brain regions and level of prenatal alcohol exposure, magnitude of FAS facial phenotype, and level of CNS dysfunction.

Chen, Coles, Lynch, and Hu (2011) studied the effects of prenatal alcohol exposure in young adults in three groups: dysmorphic feature and prenatally exposed to alcohol, nondysmorphic and prenatally exposed to alcohol and control. In the two groups that had been prenatally exposed to alcohol, their results indicated the smaller the brain, the lower the IQ. Additionally they found a negative correlation between the level of prenatal alcohol exposure and whole brain volume. When they compared prenatal alcohol exposure effects on the brain with prenatal alcohol exposure effects on general physical growth, they found that the effect on brain development was more affected than general physical growth until the young adult age.

Archibald et al. (2001) compared a group of individuals with FAS with a control group. They found the decrease in white matter was more significant than the decrease in grey matter in individuals with FAS. This may seem counterintuitive given microcephaly is a characteristic of prenatal alcohol exposure. Sowell et al. (2008) suspect that this developmental process may not happen normally in children with FASD possibly resulting in disrupted and slower communication between nerve cells. Other research suggests that alcohol-exposed patients had relative increases in gray matter and decreases in white matter in the temporal and parietal lobes (Sowell 2001). While Archibald et al. (2001) found that white matter hypoplasia (incomplete development) was found to be more significant than gray matter hypoplasia.

In other parts of the brain, Archibald et al. (2001) found that the cerebellum was disproportionately affected in size in individuals with FAS. Astley, Aylward et al. (2009b) found that the absolute volume of the hippocampus was significantly smaller across study groups with FASD compared to the control group.

Cerebrum

The cerebrum is divided into two hemispheres that are divided into four lobes with specific functions. The frontal lobes are located directly behind the forehead and they are involved in decision-making, planning, and rational thinking (NINDS, 2001). Parietal lobes receive and process sensory information, like taste, touch, movement, and temperature from the body. They also function in reading and arithmetic (NINDS, 2001). Occipital lobes are involved in sight and visual memory. The temporal lobes process auditory information, development and retrieval of memories, and integration of memory with sensations like sound, sight, taste, and touch (NINDS, 2001).

Astley, Aylward et al. (2009b) confirmed the frontal lobe was significantly and disproportionately smaller in children with FAS/PFAS than in children with prenatal alcohol exposure, severe brain dysfunction, but no FAS facial phenotype. The FAS/PFAS group was the only group with the full FAS facial phenotype. Morphogenesis of the middle and upper face is heavily influenced by signals emanating from the forebrain to the frontonasal prominence (Astley, Aylward et al., 2009b).

Poor decision making is linked to the frontal lobe. Individuals with frontal lobe damage show similar risky and maladaptive behaviors as those with FASD. PAE has a negative effect on the frontal cortex, thus putting individuals with FASD at increased risk for engaging in problematic behaviors. (Rasmussen and Wyper, 2007).

O'Hare et al. (2009) compared children and adolescents with FASDs with typically developing children and adolescents using fMRI and verbal working memory tests. Individuals were matched for task performance on verbal working memory tests. Individuals affected by FASD showed increased activation in bilateral dorsal, left inferior parietal, and bilateral posterior temporal regions during the verbal working memory tests compared with normal individuals. O'Hare et al. (2009) conclude that "frontal parietal processing during verbal [working memory] is less efficient in alcohol-exposed individuals."

Sowell, Mattson et al. (2008) had similar findings. They used MRI to measure which areas of the brain activated during a verbal recall test. They concluded that their results suggest that

subjects affected by FASD may rely more on the frontal structure of the brain compared with the control group, possibly to compensate for damage in other brain regions responsible for verbal learning and recall (Sowell, Mattson et al., 2008).

Hippocampus and amygdala

The hippocampus is located deep inside the temporal lobe of the brain. The hippocampus is involved in memory. Research on the effect of prenatal alcohol exposure on the hippocampus and amygdala has been somewhat inconclusive. However, Astley, Aylward et al. (2009b) report the mean absolute volume of the hippocampus decreased significantly—16 percent smaller—progressing across four study groups from control to three different FASD groups.

The amygdala is an almond shaped mass of nuclei located deep within the temporal lobe of the brain near the front of the hippocampus. The amygdala is associated with many functions including emotion, learning, attention, memory, perception, fear, and reward learning (Baxter and Murray, 2002). Archibald et al. (2001) found the amygdala was relatively spared compared to other regions of the brain in study participants with FAS. However, Chen et al. (2011) found that left amygdala was specifically affected by prenatal alcohol exposure.

Cingulate gyrus

The cingulate gyrus is between the corpus collosum and the cerebral cortex and it has two segments: the anterior cingulate, which is concerned with vocalization, emotional and motoric functioning involving the hands, and regulating autonomic and endocrine activities; and the posterior cingulate, which is involved in visual-spatial and tactile analysis as well as motor output and memory.

Bjorkquist, Fryer, Reiss, Mattson, and Riley (2010) studied a group of children with histories of heavy prenatal alcohol exposure and a control group using MRI. They found significantly smaller amounts of cingulate grey matter, white matter, and overall tissue when alcohol-exposed children were compared with their peers. Reduction in white matter was more affected. White matter reduction occurred regardless of FAS diagnosis in the alcohol-exposed group. They concluded that damage to the cingulate may contribute to deficits in cognitive control, attention, and emotion regulation.

Basal ganglia

The basal ganglia are a set of nerve cell clusters that includes the putamen, globus pallidus, and caudate nucleus. The basal ganglia are involved in motor and cognitive abilities, including executive functions (Mattson et al., 2001). Studies have shown that in individuals with prenatal alcohol exposure the basal ganglia have decreased volume. Mattson et al. (1996) conducted an MRI study on children diagnosed with FAS and compared them to a control group. The results indicated significant size reductions of the basal ganglia in the children with FAS (Mattson et al., 1996).

Prenatal alcohol exposure not only affects the size of parts of the brain, but it can also distort their shape. Some studies have found the symmetry of the caudate nucleus of the basal ganglia was affected. The caudate nucleus is associated with learning, mental flexibility and behavioral inhibition (Cortese et al., 2006). Willford et al. (2010) studied 45 young adults comprising three groups—subjects whose mothers did not drink at all during pregnancy, subjects whose mothers

drank during the first trimester only, and subjects whose mothers drank throughout pregnancy. Differences in the caudate volume between the three groups were not found to be significant. However, they found that the more the alcohol an individual was exposed to prenatally, the greater the effect was on their caudate nucleus. The amount of alcohol affected the magnitude and direction of the asymmetry of the caudate nucleus in the individuals prenatally exposed to alcohol. Willford and colleagues state that:

“...anomalies associated with the interconnections within the caudate can adversely affect the larger network of brain regions that underlie complex cognitive behavior. Disruptions in neural circuitry can result in poor coordination between brain regions and may impact the speed of processing.” (Willford et al., 2010, p. 7).

The Chen et al. (2011) study found the left and right caudate nuclei were both disproportionately affected by prenatal alcohol exposure. Astley, Alyward et al. (2009b) report the mean relative volume of the caudate in both the FAS/PFAS and static encephalopathy/alcohol-exposed groups were comparable with one another but significantly small (12 and 14 percent, respectively) than the mean of the control group.

Corpus callosum

The corpus callosum is the collection of nerve fibers that connects the two hemispheres of the brain. It is what allows the two sides of the brain to communicate with each other (Mattson, Schoenfeld, and Riley, 2001). The corpus callosum has been found to be smaller in individuals with FASD and significantly displaced in three-dimensional space. It was also reported that a negative relationship exists between verbal learning and the location of the corpus callosum in an FASD group (Sowell 2001).

Abnormalities in the corpus callosum have been associated with deficits in attention, intellectual functioning, executive and psychosocial functioning, verbal memory, and reading. All of these areas are impaired in individuals prenatally exposed to alcohol (Mattson, Schoenfeld, and Riley, 2001). Astley, Alyward et al. (2009b) found the corpus callosum to be significantly shorter and smaller in test groups with FASD relative to the control group. The Chen et al. (2011) study found that the posterior portion of the corpus callosum was specifically affected by prenatal alcohol exposure. Dodge et al. (2009) found that heavy prenatal alcohol exposure is associated with less efficient inter-hemispheric transfer and corpus callosum function is especially sensitive to the effects of prenatal alcohol exposure.

Cerebellum

The cerebellum is wrinkled ball of tissue located at the base of the brain by the spinal cord and brain stem (NINDS, 2001). The cerebellum is involved with motor and cognitive skills (Mattson, Schenfeld, and Riley, 2001). Damage to the cerebellum has been associated with learning deficits as well as balance and coordination. These areas are all impaired in individuals prenatally exposed to alcohol (Mattson et al., 2001).

O'Hare et al. (2005) examined the structure between the right and left hemispheres of the cerebellum, known as cerebellar vermis, and found abnormalities in the size and location of the vermis in individuals with FASD. The anterior vermis showed the strongest negative correlation

with verbal learning performance in the alcohol-exposed group. (Nunez, Roussotte, and Sowell, 2011; O'Hare et al., 2005).

An Astley, Aylward et al. (2009b) study of brain abnormalities revealed that the mean absolute midsagittal areas of the cerebellar vermis and lobules I–V were significantly smaller in the FAS/PFAS group than the mean of each area in the control group (12 and 15 percent, respectively).

Birth Outcomes

Armstrong et al. (2003) reported that women who screened positive for alcohol use during pregnancy experienced increased rates of assisted ventilation, low birth weight, preterm delivery, and increased admissions to NICUs. Preterm birth was associated with moderate and high levels of prenatal alcohol consumption even when drinking ceased before the second trimester (O'Leary, Nassar, Kurinczuk, and Bower, 2009).

Cognitive and Behavioral Impact

Since the brain is physically altered in so many ways in so many areas, it then becomes clear why brain function is altered in so many ways as well. The impact of alcohol exposure is keenly expressed in the cognitive abilities and behavior manifestations of an individual with FASD. While the following five topics—behavior, education, social skills, adaptive functioning, and executive functioning—are presented individually for the purpose of research review, they are all interrelated.

Behavioral deficits

The affects of alcohol exposure can sometimes be identified at birth but is typically not recognized until the child is a toddler or school age. Some of the signs of dysfunction in infancy due to prenatal alcohol consumption include irritability, jitteriness, increased levels of activity, and slow response/poor alertness (Streissguth and Kanter, 1997; Mattson and Riley 1998; McGee and Riley 2007; Troese et al., 2008). Infants exposed to alcohol during pregnancy can also display sleeping, eating, and motor problems (Coles, Smith, Fernhoff, and Falek, 1984; Troese et al., 2008).

Behavior difficulties identified in early childhood include sustained attention, emotional reactivity, increased activity levels, irritability, temper tantrums, mood changes, noncompliance, and impulsivity (Kelly, Day, and Streissguth, 2000; Olson and Montague, 2011). In a study of 1,400 patients with prenatal alcohol exposure, Astley (2010) documented behavioral problems to be in the clinical range across the full spectrum of FASD. These deficits can affect the parent stress level and impact the early mother–child interactions. O'Connor and Paley (2009) studied the quality of the mother–child relationship, as measured by the mother's supportive presence. Mothers who were less emotionally responsive to their children had children reporting higher levels of depressive symptoms. "The effects of alterations in child behavior and the mother–child relationship may be one of the most significant results of prenatal alcohol exposure". (O'Connor and Paley 2009, p. 226).

Educational impact

The cognitive problems associated with fetal alcohol exposure include deficits in learning, language, motor, visuospatial, manual dexterity, and executive functioning abilities (Mattson and Riley, 1998; Olson, Feldman, Streissguth, Sampson and Bookstein, 1998; Astley, Olson et al., 2009; Astley, 2010). Dysfunctional classroom behavior, poor verbal, memory ability, low arithmetic skills and low overall academic achievement on standardized tests have been associated with prenatal alcohol exposure as well (Streissguth, Bookstein, and Barr, 1996). Prenatally exposed children tend to perform worse than controls on relatively complex tasks, regardless of the domain of functioning. The more severe the FASD diagnosis, the more difficult the task was for the child (Aragon, 2008; Astley, Olson et al., 2009; Astley, 2010).

Many of the tasks that are sensitive to prenatal alcohol exposure rely on speed of response either by using reaction time or imposing time constraints on the participant (Burden, Jacobson, and Jacobson, 2005). Reaction time is an indicator of cognitive processing speed. Alcohol-exposed children demonstrated slower overall choice reaction time and significantly slower premotor reaction time during choice reaction time tasks (Simmons, Wass, Thomas, and Riley, 2002). The working memory deficit appears to be accounted for, in part, by the prenatal alcohol effect on processing speed (Burden et al., 2005).

Baddeley and colleagues defined working memory in terms of a three component system used for short-term storage and manipulation of information required for diverse cognitive tasks. The “visuospatial sketchpad” is for holding and manipulating visual-spatial information, the “phonological loop” is for maintaining and rehearsing verbal information, and the “central executive,” an attention controlling system, is involved in planning, selective attention, set shifting, and inhibition” (Baddeley and Hitch, 1974; Baddeley, 1992; Baddeley, 1996; Pei, Rinaldi, Rasmussen, Massey, and Massey, 2008). Kodituwakku, Handmaker, Cutler, Weathersby, and Handmaker (1995) proposed that a dysfunction exists in the ability of children with FASD to hold and manipulate information, with tasks that evaluated planning ability, and to manage goals in working memory. These effects would relate to the “visuospatial sketchpad” and the “central executive.” Mattson and colleagues (2002) identified memory deficits primarily in the process of encoding or manipulating information in short-term memory, rather than with long-term storage of information. Other researchers reported adverse effects primarily on complex tasks that engage working memory and include processing efficiency and specifically number processing (Kodituwakku, Kalberg, and May, 2001; Burden et al., 2005).

In an fMRI study of children with FASD, Astley, Alyward et al. (2009a) reported children across the full spectrum of FASD exhibited significant working memory deficits and altered activation patterns in brain regions that are known to be involved in working memory.

Youth with FASD are shown to have impairments in their understanding of spoken language and their ability to produce language forms at the level expected for their age (Conroy and Lane 2009). Their superficial talkativeness may lead others to overestimate their competence and level of understanding (Fast and Conroy, 2009). While they may be adept at parroting verbal information they have heard, their communication often lacks meaningful content. (Gibbard, Wass, and Clarke, 2003). Children exposed prenatally to alcohol performed significantly poorer than controls on measures of word comprehension and naming ability (Riley and McGee, 2005).

Astley and colleagues document language as a primary disability among children across the full spectrum of FASD (Astley, 2010; Astley, Olson et al., 2009).

The principal deficits seen in prenatal alcohol exposure for number processing relate to the core quantity system, which involves the processing of relative magnitude such as number comparison and proximity judgment (Jacobson, Dodge, Burden, Klorman, and Jacobson, 2011). The arithmetic function that is most heavily dependent on magnitude comparison is subtraction (Chochon, Cohen, van de Moortele, and Dehaene, 1999; Ischebeck et al., 2006). Interventions focusing on basic concepts relating to quantity and distance warrant particular consideration in remedial work with fetal alcohol-exposed children (Jacobson et al., 2011).

Older children performed worse relative to the norm than younger children on letter fluency, inhibition/switching, inhibition, and the word context test on the D-KEFS. Specific verbal executive function skills may be particularly difficult for older children with FASD leading children with FASD to fall further behind their peers with age. A distinctive profile emerged with children with FASD performing very poorly on the card sorting task but displaying relative strengths on the some visual-spatial executive function tasks (Rasmussen, 2009).

“The trouble with school is that you spend all of your time doing things you do poorly and no time doing things you do well” (Kyle, on growing up with FAS/FASD Streissguth and Kanter, 1997, p. 193).

In a secondary disability study of 415 people with FASD/FASD, 61 percent of those 12 years and older had had a disrupted school experience. Of this group, 29 percent had been expelled, and 26 percent had dropped out (Streissguth, Barr, and Bookstein, 1996). This increases the odds of additional secondary disabilities such as alcohol and drug problems, trouble with the law, etc. The disconnect from the school setting removes them from job, vocational, and life-skills training (Streissguth and Kanter, 1997).

Executive function

Executive function is the basis of many complex cognitive, emotional and social skills (Lezak, Howieson, Loring, Hannay, and Fischer, 2004). Research has identified alcohol-related deficits on measures of planning, set shifting and flexibility, fluency, concept formation, abstract reasoning, strategy use, verbal reasoning, working memory, and emotional regulation (Mattson, Goodman, Caine, Delis, and Riley, 1999; Kodituwakku, Kalberg et al., 2001; Schonfeld, Mattson, Lang, Delis, and Riley, 2001; Rasmussen, 2005; Astley, Olson et al., 2009; Astley, 2010).

Children with heavy prenatal alcohol exposure could be distinguished from non-exposed peers on the basis of scores for frequently used measures of attention; alcohol-exposed and comparison subjects were classified with 91.7 percent accuracy (Lee, Mattson, and Riley, 2004; Fryer et al., 2007). Alcohol-exposed children had more difficulty sustaining attention when processing visual stimuli than when processing auditory stimuli (Coles, Platzman, Lynch, and Freides, 2002).

Burd, Carlson, and Kerbeshian (2007) found that the most common mental disorder comorbid with FASD was ADHD, occurring in 48 percent of subjects of all ages. Astley (2010) also reported ADHD to be the most common co morbid mental disorder with FASD, occurring in

54 percent of 1,400 children with diagnosed prenatal alcohol exposure. Using the four-factor model of attention, FAS/FAE children had problems with encoding and shift which was not seen in ADHD children. These could be factors for distinguishing between the two groups (Coles et al., 1997).

Social skills

Although many of the social behaviors in children with FASD may be acceptable at a young age, these behaviors become increasingly inappropriate and noticeably different from peers as the children become older and suggest an arrest in development at about the 4- to 6-year age level (Thomas, Kelly, Mattson, and Riley, 1998).

Children with FASD showed particular difficulty interpreting the mental states of others and identifying emotions in faces, which were associated with their increased risk of behavior problems and poor social development (Greenbaum, Stevens, Nash, Koren, and Rovet, 2009). Social processing deficits reflect their difficulties understanding and interpreting social cues and considering the consequences of their actions, failure to learn from experience (Streissguth and Giunta, 1988). Additionally, they often lack social judgment, show poor social communication (Kodituwakku, 2007), have inappropriate interpersonal skills (Olson, Feldman, Streissguth and Gonzales, 1992), and problematic use of play and leisure time (Thomas, 1998). Clinical observations have identified problems with being overly friendly with strangers, socially immature and naïve (Olson and Montague, 2011). According to caregivers, 25 percent of patients had no friends and 10 percent had dysfunctional contacts (Freunsch and Feldman, 2011).

Conroy, Fast, and Loock (1997) studied youth who were being psychologically assessed in an inpatient unit and noted the significant behavior problems were socially inept/inappropriate and having a poor understanding of personal boundaries. Children and adolescents with FASD frequently engage in antisocial behaviors such as cheating and stealing (Olson et al., 1997; Nash et al., 2006) as well as inappropriate sexual behaviors and difficulty with managing anger (Streissguth and Kanter, 1997; Kelly et al., 2000).

In a study of ADHD among children prenatally exposed and those who were not, the most notable pattern was the lack of improvement with age in socialization and communication among prenatally exposed children. Children with only ADHD experienced a delay but improvement with age (Crocker, Vaurio, Riley, and Mattson, 2009).

Individuals with FASD may socialize with a maladaptive or socially deviant population who are more accepting of their behavior. This association increases the chance of these individuals getting into trouble with the law (Boland, Chudley, and Grant, 2003).

Adaptive functioning

Adaptive function is a person's competence in the real world. It is the ability to meet developmentally appropriate expectations of personal independence and social responsibility, including performance of everyday tasks, and adapt to changes in the environment. Childhood deficits in adaptive skills are reported to increase with age (Whaley et al., 2001). In a study of adaptive function by Jirikowic, Kartin, and Olson (2008), children with FAS performed significantly below that of their peers in all but three of fourteen subscales on the Scales of Independent Behavior-Revised. The three subscales that revealed strengths included gross motor, personal

self-care, and domestic skills. Awareness of specific strengths and needs can guide interventions that promote function and participation (Jirikowic et al., 2008). Adaptive functioning is affected in this population and many individuals identified as having FASD are unable to live or work independently (Astley, Olson et al., 2009; Astley, 2010). Assessment of adaptive behavior of individuals with FASD using the Vineland Adaptive Behavior Scale revealed that impairments in adaptive functioning “were more profound than the deficits observed in either IQ or Achievement Tests” (Streissguth, 2006, p. 6).

McGee, Fryer, Bjorkquist, Mattson, and Riley (2008) state that, “adequate social problem solving is essential for adaptive functioning.” In the McGee et al. (2008) study, “adolescents prenatally exposed to alcohol demonstrated significant impairment in social problem solving, reporting approaching problems, generating alternate solutions, making decisions, and implementing and verifying the chosen solution. They were more likely to endorse an avoidant, careless, or impulsive approach” (McGee et al., 2008, p. 429).

Brown et al. (2011) stated that, “preliminary research on suggestibility in FASD finds that this population is prone to social manipulation by others and may even be hypersuggestible” (p. 41). In a study of 62 adults with FASD, 92 percent of caregivers described the subject as vulnerable to manipulation (Clark, Lutke, Minnes, and Oullette-Kuntz, 2004). In a pilot study of eight middle school children with FASD researchers found a negative correlation between memory ability and interrogative suggestibility (Gruppuso, 2009). Other characteristics reported by caregivers that could compromise adaptive functioning include: impulsivity, unawareness of consequences of behavior, and deficient risk perception (Streissguth et al., 1998).

Freunsch and Feldman (2011) found the following:

Adults with FAS experience increased disruption and failure in their school and occupational career. Early diagnosis, stable life situation, adequate therapy forms and comprehensive supervision may attenuate the severe consequences of FAS in adulthood. Assisted living and supported employment may protect adult FAS patients from social and emotional distress, trouble with the law, and health risks. (p. 37).

Protective Factors

The odds of an “adverse life outcome” were reduced if the person with an FASD had an early diagnosis, lived in a stable nurturing home environment, and had not been a victim of abuse (Streissguth et al., 2004). “The provision of early intervention is critically important because the foundation for the development of self-regulatory skills is laid during the first four years of life” (Kodituwakku and Kodituwakku, 2011). Developmental neuroscience research has shown that the quality of mother-child interaction and the psychological state of the mother influence the child’s stress responses as measured by glucocorticoid levels (Albers et al., 2008; Lupien et al., 2009). Current intervention research addresses maximizing positive interactions and reducing parent stress (Grant, Ernst, Streissguth, and Stark, 2005; Olson, Oti, Gelo, and Beck, 2009).

In a study of 1,400 patients with prenatal alcohol exposure, Astley (2010) reports 9.3 percent presented with no CNS dysfunction despite having prenatal alcohol exposure as high as those diagnosed with FAS. In this unaffected group, physical and sexual abuse was 2- to 5-fold less prevalent than in the FASD groups. In addition, tobacco use, illicit drug use, neglect, and out of

home placement were also less prevalent and prenatal care was more prevalent. Early identification and treatment have been demonstrated to be protective against more serious psychiatric outcomes (Streissguth, Barr, and Bookstein, 1996).

In a study that examined the risk and protective factors associated with behavior problems of children and adolescents following prenatal alcohol exposure, length of time spent in residential care was the most pervasive risk factor associated with internalizing, externalizing, and total behavior problems (Fagerlund et al., 2011). Adverse home environment of alcohol-exposed children has been associated with poorer outcomes in language and social communications (Coggins, Timler, and Olswang, 2007). Foster care and adoption are frequent experiences for children with FAS/FASD with varying reported rates between 23.7 percent and 73.3 percent (May, Hymbaugh, Aase, and Samet, 1983; Caruso and ten Bensel, 1993). In the Greenbaum et al. (2009) study that included 33 children with an FASD, 60 percent of the population was in some form of care. Astley (2010) reported 70 percent of 1,400 children with a prenatal alcohol exposure experienced foster/adoption placements with an average of three home placements before the age of 10 years.

Co-Occurrence

Mental health

Mental health problems are prominent in individuals with FASD throughout the lifespan. Environmental influences, for better or for worse, can impact the trajectory. (O'Connor and Paley, 2009). PAE can act as a significant risk factor in the emergence of early onset psychopathology. While environmental factors including the mother-child interaction, abuse and neglect, transient home placements, and maternal death might play a role in mediating the effects of prenatal alcohol exposure on child depressive symptoms, direct effects of alcohol also appear to mediate this relationship. These findings indicate that problems such as anxiety and depression are not only significant issue among adults and children with an FASD, but also may have a neurobiological basis. This suggests that mental disorders may be a primary rather than secondary disability (Helleman, Sliwowska, Verma, and Weinberg, 2010).

In a study of 473 patients diagnosed with either FAS or fetal alcohol effects, over 90 percent of the patients had a mental health problem with 52 percent documenting depression (Streissguth, Barr et al., 1996). Fryer et al. (2007) observed a greater proportion of depressive disorders in alcohol-exposed subjects compared with control subjects. According to Fryer et al. (2007), "children with heavy prenatal alcohol exposure experienced higher rates (97 percent) of many common DSM-IV axis I psychiatric disorders, compared with matched comparison children (40 percent)" (p. e737). In a sample of 23 children with PAE between the ages of 5 and 13, 87 percent met criteria for a psychiatric disorder, 61 percent of the group received a mood disorder diagnosis, 35 percent for bipolar, 26 percent for major depressive disorder, and 13 percent for ADHD. (O'Connor et al., 2002). Olson, O'Connor and Fitzgerald (2001) reported elevated rates of depression, anxiety, and suicidal thoughts. Of that group of children only 40 percent of the alcohol-exposed sample had been evaluated for or received a psychiatric diagnosis. In a study of 1,400 patients with prenatal alcohol exposure 75 percent presented with one or more mental disorders (Astley, 2010). Astley, Bailey, Talbot, and Clarren's (2000a) study of 80 birth mothers

of children with FAS found that 96 percent had between one and 10 mental disorders, 77 percent had PTSD, and 59 percent had a major depressive episode.

Suicide

In a review of the Surgeon General's suicide risk factors, Huggins, Grant, O'Malley, and Streissguth (2008) identified nine of the sixteen risk factors as aligning with clinical characteristics of FASD. In a cross-sectional study of adolescents and adults with PAE (Streissguth, Barr et al., 1996), 43 percent reported suicide threats and 23 percent reported a history of suicide attempts over the course of the lifetime. In a small sample size of 11 adults with FASD 55 percent, or 6 individuals, had attempted suicide. "Attempters were more likely to have mental disorders, substance abuse, a history of trauma or abuse, financial stressors, and unstable social supports compared to non-attempters". (Huggins et al., 2008, p. 33).

Standard protocols for assessing and intervening with clients who have FASD and are at risk for suicide need to consider communication impairments as well as the psychiatric and emotional deficits that may or may not easily present themselves. Thorough assessment and follow-up monitoring in response to first attempters is necessary to prevent self-harm becoming a regular coping mechanism and to reduce the risk of a subsequent attempt (Huggins et al., 2008).

Sleep disturbance

Wengel, Hanlon-Dearman, and Fjeldsted (2011) state that, "the active process of sleep onset and maintenance is dependent on a normally functioning central nervous system" (p. 390). Healthy development of the thalamus and hypothalamus is critical for the control of sleep and studies have shown that brain damage in FASD can affect the structure and metabolism of these regions (Fagerlund et al., 2006; Lebel et al., 2008). Chen et al. (2012) states that "any level of respiratory abnormalities may also result in repeated cortical arousals during sleep, compounding sleep fragmentation" (p. 18). In a study of 19 children with FASD and 12 control children, Wengel et al. (2011) reported a significant correlation between sleep disturbances and sensory processing. They also found a negative correlation between wake time and auditory processing, multisensory processing, and sedentary behavior.

In a study of 34 children with FASD, 85 percent had marked sleep disturbance (Chen, Olson, Piccano, Starr, and Owens, 2012). The most common disturbances are difficulty falling asleep, frequent awakening during the night, night terrors, and early waking (Jan et al., 2010). Other identified disturbances include increased bedtime resistance, shortened sleep duration, and increased sleep anxiety. (Wengel et al., 2011).

According to Jan et al. (2010), "many children diagnosed with FASD have longstanding sleep disturbance which interferes with their daily activities, cognition, behavior, health and management. Without appropriate treatment of the sleep difficulty, the effectiveness of all interventions may be markedly reduced" (p. 1). It was reported that sleep deprivation predisposes children to impairments in mood, behavior, cognition, physical health, and executive functioning and also increases irritability and depression (Stores, 1999).

In a study that surveyed 100 caregivers of children with FASD over a 7-day period, 82 reported a sleep problem such as sleep walking, waking more than twice during the night, night terrors, and daytime fatigue. Sleep difficulties are related to the severity of cognitive loss and brain

disturbance rather than to a specific diagnosis of disability (Jan et al., 2007). Sleep disturbances can also impact the sleep of the caregiver as well as the mother-child relationship as well as the family unit due to additional challenges and frustration they present.

Substance abuse

Adolescents whose mothers drank during pregnancy reported higher levels of alcohol use and related problems at 14 years of age (Baer, Barr, Bookstein, Sampson, and Streissguth, 1998). The relationship between PAE, heavy drinking, and alcohol-related problems persisted into early adulthood (Baer, Sampson, Barr, Connor, and Streissguth, 2003). After controlling for other factors, adolescents whose mothers consumed three or more drinks per drinking occasion during pregnancy were at increased risk of drinking more alcohol in a binge pattern than those whose mothers consumed less alcohol (Alati et al., 2008).

Astley et al. (2000a) found that of the 80 birth mothers of children with FAS in the study, 86 percent had alcohol abuse and “94 percent did not want to reduce their use because it helped them cope, while 72 percent did not want to reduce because they were in an abusive relationship and 79 percent were too depressed to do anything about it” (p. 513).

Victimization

The interaction of prenatal alcohol exposure and exposure to childhood trauma may result in more severe neurodevelopmental deficits (Henry, Sloane, and Black-Pond, 2007). In a Greenbaum et al. (2009) study that included 33 children with an FASD, 91 percent of the population had experienced some form of physical abuse or neglect. According to caregivers of 60 adults with FAS, 76 percent had fallen victim to criminal act or exploitation and abuse. A regression analysis to identify variables that may predict victimization in adulthood revealed that the number of changes of living situation proved to be the most adequate predictor. Commonly reported victimization includes sexual abuse, violence, attacks, and extortion (Freunsch and Feldman, 2011).

Astley et al. (2000a) found that of 80 birth mothers of children with FAS, 73 percent reported childhood or adult sexual abuse and 95 percent had been physically or sexually abuse during their lifetimes. Once again, alcohol is associated with a method of coping (Astley et al., 2000a).

Efficacy of Intervention

There are few programs that are specific to the prevention of FASD and intervention for individuals with an FASD. In 2007, SAMHSA issued a request for proposal (RFP) through the FASD Center for Excellence for the purpose of integrating evidence-based FASD programs into local service delivery organizations. Twenty-three subcontracts were awarded.

Eight diagnosis and intervention subcontracts were awarded to improve the functioning and quality of life of children, youth, and their families by diagnosing those with an FASD and providing interventions based on the diagnosis. Funding for these subcontracts ended in Spring 2012.

SAMHSA FASD Diagnosis and Intervention Projects

Child Mental Health Providers

- Mississippi Department of Mental Health
- COMHAR, Inc.
- Child Guidance Center, Inc.
- Community Assessment Referral Education (CARE)

Dependency Court/Delinquency Court Projects

- Arkansas Department of Human Services-Division of Children and Family Services
- White Earth Tribal Council
- Hennepin County Human Services and Public Health Department
- 17th Judicial District, Colorado Judicial Department

Fifteen FASD prevention subcontracts were awarded to decrease the incidence of FASD by implementing evidence-based programs to eliminate alcohol consumption by pregnant women. Funding for all 15 programs ended in 2012. The target audience was pregnant women who drink or women of childbearing age in alcohol or substance abuse treatment. Three programs were selected for implementation under the prevention component of the RFP because they had been clinically studied and produced statistically significant results. See Table 3.

SAMHSA FASD Prevention Projects

Alcohol Screening and Brief Intervention Projects in WIC Programs

- Illinois Department of Human Services
- South Dakota Division of Alcohol and Drug Abuse
- Public Health Dayton and Montgomery County
- Memorial Hospital of South Bend, Indiana

Alcohol Screening and Brief Intervention Projects in Home Visitation Programs

- Arizona Dept. of Health Services
- Child and Family Services of New Hampshire
- Aberdeen Area Tribal Chairman's Health Board

Project CHOICES Interventions in Substance Abuse Treatment Programs

- New York Office of Alcoholism and Substance Abuse
- Texas Office for Prevention of Developmental Disabilities
- San Diego Youth and Community Services
- ARC Community Services, Inc.
- Pine Belt Mental Healthcare Resources
- Serving Children and Adolescents in Need, Inc.

Parent-Child Assistance Program (PCAP) in Substance Abuse Treatment Programs

- Southern California Alcohol and Drug Programs, Inc.
- Michigan Bureau of Substance Abuse and Addiction Services (formerly Michigan Office of Drug Control Policy)

Table 3. Evidence-Based Programs Replicated by SAMHSA

Target population	Service Settings	Eligibility Criteria	Intervention Components	Client Outcomes
Parent-Child Assistance Program (PCAP)				
Pregnant and postpartum (up to 6 months) women	Home visitation and community case management	Self-report of heavy alcohol or illicit drug use during pregnancy, and ineffective or non-engagement with social services. Addiction Severity Index, 5th Ed., administered at intake and exit, with supplemental questions examining pregnancy, substance use, contraception, and service utilization.	Paraprofessional home visitation designed to prevent future births of children prenatally exposed to alcohol through goal setting and case management targeting contraception use; alcohol use, and linkages to community services and programs. Case management is provided at least twice monthly for up to 3 years following initial entry into the program.	Grant et al. 2005: At 36-month program exit, effective in increasing completed substance abuse treatment, abstinence, delivery of unexposed children, and use of contraception across sites and over time. Other findings include increased maternal employment and increased connection with services. Hensley, 2011: Effective in increasing abstinence at 6-month, 12-month, and 18-month follow-up, as well as increased contraceptive use at 18-month follow-up.
Project CHOICES				
Women of childbearing age between the ages of 18–44	Residential and outpatient substance abuse treatment, community mental health treatment, jails, community-based teen programs for girls	Self-reports of being sexually active, not pregnant but able to conceive, high-risk drinking (8 or more drinks per week or 4 or more drinks in one occasion) in the past 30 days, and ineffective use of or no contraception.	Four counseling sessions plus a contraceptive counseling visit.	Project CHOICES Intervention Research Group, 2003: Reduced risk of AEP among 68.5 percent of participants at 6-month follow-up. Floyd et al. 2007: At 3, 6, and 9 months women receiving the

				<p>intervention were two-fold more likely to be at reduced risk for AEP than the controls. Binge drinking episodes reduced from 30 at baseline to 7 at 9 months follow-up; median number of drinks per week at baseline was reduced from 36 drinks to 2.3 drinks per week at 9 months.</p> <p>Hensley, 2011: Program shown to be effective at reducing binge drinking and increasing abstinence among women in residential treatment and community settings at end-of-program, 6-month follow-up, and 12-month follow-up.</p>
Alcohol Screening and Brief Intervention (SBI)				
Pregnant women	WIC and Healthy Families (home visitation)	Self reports of active drinking (1 or more days with 1 or more drinks) in the past 30 days OR high risk score (2 or more) on the TWEAK or T-ACE based on responses to questions assessing alcohol tolerance.	10- to 15-minute brief intervention session utilizing a scripted workbook.	O'Connor and Whaley, 2007: Study divided participants into brief intervention and assessment-only groups. Women in the former were five times more likely to report abstinence after intervention compared with women in assessment-only. Newborns of mothers who received brief

				<p>intervention had higher birth weights and lengths, and lower mortality, than those of women in assessment-only.</p> <p>Hensley, 2011: Reports of abstinence at third trimester follow-up were 99 percent for clients assessed at enrollment as at-risk based on prior alcohol tolerance levels, and 93 percent for clients who reported alcohol use in the past 30 days at enrollment.</p>
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Table 4. Additional Evidence-Based Programs

Target population	Service Settings	Eligibility Criteria	Intervention Components	Client Outcomes
Single Session Brief Intervention (With Partner)				
Pregnant women and their partners	Obstetric settings	1) Positive score on T-ACE, 2) at risk for an AEP (any alcohol consumption in the 3 months before study enrollment while pregnant, or consumption of at least one drink per day in the 6 months before study enrollment, or drinking during a previous pregnancy, 3) gestation less than 28 weeks, and 4) Agreement to study terms: randomization to	1) Knowledge assessment with feedback, 2) contracting and goal-setting, 3) behavioral modification, and 4) summary. Total time of intervention: Approx 25 minutes.	Chang et al. 2005: Women with the greatest levels of drinking at the time of intervention showed the greatest levels of reduction, and effects of the intervention were significantly enhanced when the participant had a support partner of their choosing involved.

Pregnant women and their partners	Obstetric settings	treatment, postpartum interview, selection of a partner to participate in diagnostic interview, brief intervention, and postpartum interview.	1) Knowledge assessment with feedback, 2) contracting and goal-setting, 3) behavioral modification, and 4) summary. Total time of intervention: Approx 25 minutes.	Chang et al. 2005: Women with the greatest levels of drinking at the time of intervention showed the greatest levels of reduction, and effects of the intervention were significantly enhanced when the participant had a support partner of their choosing involved.
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Brief Intervention and Goal Setting (With Partner)

Pregnant women	Women's hospital	1) Positive score on T-ACE, 2) being at risk for an AEP (defined as any alcohol consumption in the 3 months before study enrollment while pregnant, or consumption of at least one drink per day in the 6 months before study enrollment, or drinking during a previous pregnancy, 3) gestation less than 28 weeks, and 4) Agreement to study terms, including randomization to treatment, postpartum follow-up interview, selection of a partner to participate in diagnostic interview, brief intervention if so randomized, and postpartum interview.	RCT. Single session brief intervention with partner included: 1) Knowledge assessment with feedback, 2) contracting and goal-setting, 3) behavioral modification, and 4) summary. Total time of intervention: Approx 25 minutes.	Chang, McNamara, Orav, and Wilkins-Haung, 2006: Women who chose abstinence for prenatal drinking goal were most likely to achieve or maintain abstinence (75 percent). Of those who were not abstinent at enrollment and chose abstinence, 50 percent achieved it. Of those who were not abstinent and chose cutting down, 0 percent achieved abstinence. Partners behaviors included assuming extra responsibilities or be supportive of woman's general well-being.
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Early Start				
Pregnant women	Obstetric setting	1) Completion of universal self-administered prenatal substance abuse screening questionnaire 2) clinician referral 3) self-referral 4) positive urine toxicology screen results on universal toxicology screening test. Four study groups included; screened/assessed/treated; screened/assessed; screened; controls—no evidence of use.	A retrospective cohort study. Integrating onsite substance abuse treatment with prenatal visits.	Goler et al. 2008: Outcomes were measurements of newborn health. Lower rates of neonatal-assisted ventilation, preterm delivery and low birth weights were reported.

Table 5. Programs under Study with Positive Outcomes

Target/ Study Population	Program Focus	Intervention Components	Client Outcomes
Families Moving Forward (FMF)			
Children 5–11 years old affected by FASD and their caregivers	Increase caregiver self-efficacy, respond to family needs, and decrease child problem behavior	Low-intensity supportive behavioral health consultation 16 sessions, 90-minutes each, over a 9–11 month period carried out by mental health providers, services can be provided in homes or clinical settings, brief expert consultation in from psychologists or occupational therapists, a curriculum and written materials have been developed.	Bertrand (2009): Caregivers receiving the intervention reported an improved sense of parenting self-efficacy post-treatment, and more parents receiving the intervention reported engaging in self-care behaviors compared to the control group and a decrease in challenging disruptive behaviors in the child. Principal Investigator: Heather Carmichael Olson, Ph.D.
Social Skills Training			
Children 6–12 years of age affected by FASD	Improvement of overall social skills and competence	Twelve 90-minute sessions delivered over 12 weeks, parents attended concurrent sessions, children are taught social skills through instruction	Bertrand (2009): Children showed a significant improvement in knowledge of appropriate social behavior; parents reported increase

	including social network formation, conflict avoidance and negotiation, interaction with peers and other skills	on simple rules of social behavior, at home rehearsal, homework, and coaching by parents during play between children.	in social skills and decrease in problem behaviors, which were maintained at 3-month follow-up; children's social skills improved significantly from post-treatment to 3-month follow-up. O'Connor et al. (2012): Children with and without FASD showed significant benefits from the treatment including knowledge of appropriate social behaviors and areas of behavioral adjustment, intellectual/school status, and relief from anxiety compared with a standard of care treatment in a community mental health setting. Principal Investigator: Mary J. O'Connor, Ph.D.
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Neurocognitive Habilitation

Children ages 6–11 in foster care or who have been adopted, diagnosed with FAS or ARND	Provide education and to increase families' ability to care for children, and focus on improvement of executive function in children	Twelve 75-minute neurocognitive habilitation group therapy sessions, caregivers participated in concurrent parent education groups, intervention administered by licensed clinical psychologists, licensed clinical social workers, and supervised doctoral students.	Bertrand (2009): Children demonstrated significant improvement in executive functioning skills compared with the control group; caregivers reported seeing improvement in regulatory strategies and techniques in children. Wells et al. (2012): Children showed improvements in executive and emotional problem solving compared to the control group. Principal Investigator: Ira J. Chasnoff, M.D.
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Sociocognitive Habilitation using Math Interactive Learning Experience (MILE)

Children ages 3–10 years affected by prenatal alcohol use	Improvement of mathematical and behavioral functioning of children, provide a	To establish readiness to learn, case management and psychiatric consultation are provided as needed, intensive short-term individual mathematics instruction for children and training for caregivers and teachers to	Bertrand (2009): Caregiver satisfaction with the program was very high; children in mathematics program had significantly higher gains in mathematics knowledge over group receiving only a psychoeducational treatment.
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	consistent method of mathematical instruction across therapeutic, school, and home settings	support mathematics learning.	Principal Investigator: Claire Coles, Ph.D.
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Additional Programs

Two additional programs, one with variation, that have been identified and show positive results are reported in Table 4. Four CDC programs under study that target interventions with children with FASD and show positive outcomes are reported in Table 5.

Research

Animal Studies

Assessment and Therapeutic Treatment—Animal studies

Animal studies allow control over dose, duration and pattern of alcohol exposure as well as environment, nutrition, and other substances of abuse. They also offer the opportunity for pharmacological intervention (Wilson and Cudd, 2011). Research examining the effects of alcohol at different developmental stages and at various doses has helped highlight the vulnerability of the prenatal brain to alcohol-mediated damage (O’Leary-Moore, Parnell, Godin, and Sulik, 2011).

Assessment

In an animal study by Hellemans, Verma, Yoon, Yu, and Weinberg (2008), results “suggest the possibility that fetal reprogramming of two systems by alcohol may underlie, at least partly, an enhanced susceptibility of fetal alcohol exposed offspring to depression/anxiety-like disorders in adulthood” (p. 154). One system in particular is the hypothalamic-pituitary-adrenal system, which is the interaction of the hypothalamus organ with the pituitary and adrenal glands, also known as the HPA axis, which is a major part of the neuroendocrine system that controls reaction to stress (Hellemans et al., 2008).

Several animal studies have documented correlations between midline facial anomalies and underlying brain abnormality caused by prenatal alcohol exposure (Sulik, Johnson, and Webb, 1981; Sulik and Johnston, 1982; Sulik, Lauder, and Dehart, 1984; Astley and Clarren, 1996, 2001; Sulik, 2005).

“Zebrafish larvae are transparent and develop external to the mother, allowing for visual study of the effects of alcohol on development” (Tanguay and Riemers 2008 in Wilson and Cudd, 2011, p. 93). Early exposure to ethanol in zebrafish has shown neurochemical changes in reduced levels of dopamine, DOPAC (*3,4,-Dihydroxyphenylacetic acid*), serotonin, and 5 HIAA (*5-Hydroxyindoleacetic acid*), which impair behavior and brain function (Buske and Gerlai, 2011).

“The sheep model offers the advantage of a long gestation which allows for a variety of drinking patters and gestational exposure” (Wilson and Cudd, 2011, p. 94). In utero brain development

in sheep matches human brain development relatively well, and prenatal binge alcohol exposure in sheep produces brain and behavioral effects consistent with FASD (Cudd, 2012). Balaraman, Lunde, Box, Cudd, and Miranda (2010) are using a sheep model to identify microRNA biomarkers of FASD. MicroRNA expression will change in response to prenatal alcohol exposure (Miranda 2010).

Animal studies have also shown that binge drinking of alcohol during pregnancy impairs eyeblink conditioning (EBC) (see section below, “Eyeblink Conditioning,” for more information) and that this impairment is likely mediated by a loss of neurons and a reduction in neural plasticity (Jacobson et al., 2008).

Several animal and clinical studies have documented correlations between midline facial anomalies and underlying brain abnormality caused by prenatal alcohol exposure (Johnston, 1975; Sulik and Johnston, 1982; Sulik et al. 1981, Sulik et al. 1984; Astley and Clarren, 1996, Astley and Clarren, 2001; Sulik, 2005).

Therapeutic Treatment of Motor Activity and Hyperactivity

Nunes et al. (2011) states the following:

Studies in rodent models of ADHD and FASD suggest that impairments in the cAMP signaling cascade contribute to the hyperactivity phenotype. Early alcohol exposure significantly increased activity and reduced cAMP levels in the hippocampus. The acute treatment of ethanol-exposed animals with vinpocetine 20mg/kg dose restored both their locomotor activity and cAMP levels to control levels. (p. 1).

A rat pup that neonatally consumed ethanol displayed locomotor hyperactivity, which was reversed by 1 and 3 mg of lobeline (Smith et al., 2012). The increased response rate to dexamphetamine may be explained by an animal study revealing that the prenatal alcohol exposure of physically-hyperactive rats acts on receptors that are the site of action for dexamphetamine (O'Malley and Nanson, 2002).

Treatment to Enhance Brain Plasticity

Alcohol exposure can also impair the activation of cAMP response element-binding protein (CREB), which is necessary for neuronal plasticity. Administration of an agent that inhibits the enzyme phosphodiesterase (PDE) can prolong CREB activation (Krahe Wang, and Medina, 2009). In a study that administered PDE inhibitors to ferrets one week after alcohol insult they found that the inhibitors increased CREB activation and restored plasticity (Medina, Krahe, and Ramoa, 2006; Krahe et al., 2009).

Treatment of neuronal cell loss

Ethanol exposure can lead to widespread neuronal death in the brain of a rat. This effect was reversed by treatment with 1,000mg of pyruvate (Ullah, 2011).

Treatment with Zinc

Zinc deficiency is a co-teratogen with alcohol. Dietary zinc supplementation of 200mg in mice throughout pregnancy ameliorated dysmorphology and postnatal mortality caused by ethanol exposure early in pregnancy (Summers, Rofe, and Coyle, 2009).

Treatment with Choline

Choline is an essential nutrient that influences brain and behavioral development. Low choline levels during mouse gestation contributed to changes in part of the brain as well as nerves and blood vessels (Mehedint, Craciunescu, and Zeisel, 2010). When there are certain metabolic inefficiencies and low-choline there is a greater risk for FASD (Zeisel, 2011). A magnetic resonance spectroscopy study conducted by Astley, Richards et al. (2009d) documented significantly lower choline levels among children with FAS/PFAS relative to children with ARND. Choline decreased significantly with decreasing frontal white matter volume (Astley, Richards et al., 2009). The effects of ethanol on pregnant mice appear to be mitigated by the administration of 250mg choline supplementation (Thomas, Abou, and Dominguez, 2009). Choline-treated animals prenatally exposed to alcohol demonstrated better performance than untreated controls on measures of spatial learning (Thomas, Bian, O'Bryan, O'Neil, and Quinn, and Dominguez, 2007) and discrimination learning (Thomas, La Fiette, Quinn, and Riley 2000).

Choline supplementation is also being studied following birth in rats. Ryan et al. (2008) reported that the administration of choline during postnatal days 11–20 or 21–30 could improve behavioral outcomes. In addition, supplementation during days 40–60, which is the equivalent to adolescence/young adult in rats, improved cognitive performance.

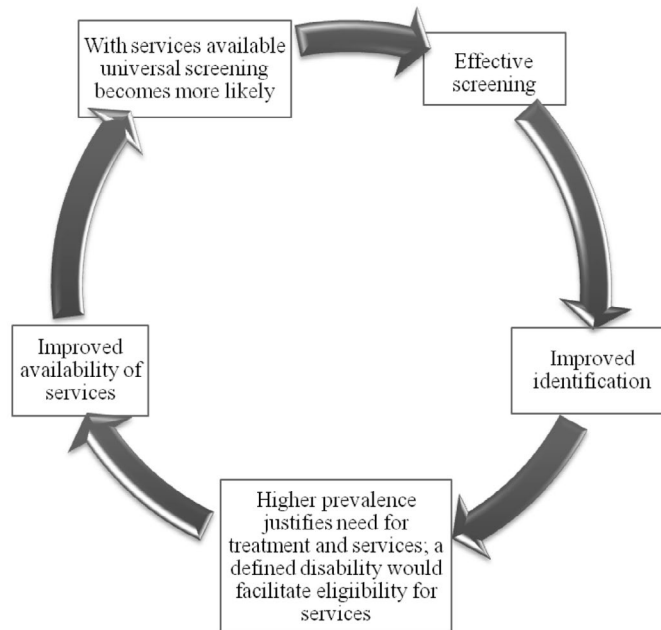


Figure 3. Impact of Effective Screening on Prevention

Research for Screening and/or Diagnosis

The development of an effective screening tool for FASD would allow for a more accurate identification of individuals affected. This in turn can enhance the awareness of FASD, improve

treatment availability and with improved availability of effective treatment, potentially improve the frequency of screening (see Figure 3).

Meconium Analysis

Meconium is the first fecal matter passed by newborns. Metabolism of alcohol in the fetal gastrointestinal tract produces fatty acid ethyl esters (FAEEs), which are then embedded in the meconium sample (Bearer et al., 2003). FAEEs do not readily cross the placenta; therefore, levels detected in meconium reflect fetal exposure to ethanol (Burd, Roberts, Olson, and Odendaal, 2007). Collection of meconium for FAEE analysis is a noninvasive method for detecting alcohol use by the mother primarily in the last half of pregnancy. However, the collection can only be done during the newborn period. A study compared alcohol prenatal exposure detection by questionnaire compared with biomarkers in meconium. Sixty-two meconium samples from mothers who denied alcohol consumption during pregnancy by questionnaire were analyzed. The objective analysis was made by determination of FAEEs as exposure biomarkers in meconium as biological matrix. In the meconium samples of 62 newborns from non-alcohol consuming mothers by questionnaire, 16.12 percent or 10 had positive FAEE values (≥ 2 nmol/g) (Manich et al., 2011).

In another study of 353 samples, 45 percent had a meconium concentration of ≥ 2 nmol/g, indicating heavy alcohol use, as compared to 15 percent identified from self-reporting (Garcia-Algar et al., 2008). Burd and Hofer (2008) conducted a meta-analysis of studies reporting use of meconium for the presence of FAEE as a marker. In the 10 studies they reviewed, a total of 2,221 subjects were screened, of which 455 or 20.5 percent had exceeded their respective study's criteria for exposure. Nine of the studies found a correlation between FAEE levels and maternal alcohol consumption while one found no correlation. The strategies used to assess PAE were quite variable and the diagnostic sensitivity and specificity were related to the analytical sensitivity and selectivity of the method.

While meconium analysis seems successful in identifying exposure, it cannot confirm exposures in the first trimester of gestation nor if the newborn is negatively affected by the alcohol. A predictive value of a positive result has not been established (Zelner, 2010). This mechanism can detect exposure during the latter half of pregnancy but not during the first trimester (Ostrea et al., 2006).

Hair Analysis

This is a noninvasive and easily collected method. Studies in neonates have been able to quantify FAEE in hair of infants exposed to excessive quantities of alcohol (Klein, 1999; Caprara, Klein, and Koren, 2005). The test must be done within 20 weeks post delivery.

Eyeblink Conditioning

Eyeblink conditioning (EBC) matches a conditioned stimulus with an unconditioned stimulus, a reflexive blink (Jacobson et al., 2008). It requires a complete neural circuit, including cerebellar Purkinje cells. Ethanol exposure induces cerebellar Purkinje cell loss. The rate of acquisition of eyeblink conditioning is reduced in proportion to cerebellar Purkinje cell loss. (Tran, Stanton, and Goodlett, 2007; Brown, Calizo, and Stanton, 2008).

Successful eyeblink performance relies on the higher brain centers in the frontal cortex and basal ganglia (Munoz and Everling, 2004). Deficits in the frontal cortex and basal ganglia have been reported in FASD (McGee, 2006). In a study that assessed eyeblink tasks in children with FASD, the results demonstrated that the greatest magnitude of difference in performance across the diagnostic subgroups occurred in a task which reflects deficits in executive function. (Munoz and Everling, 2004). Delay EBC has a high sensitivity for identifying individuals with a diagnosis of probably FAS and could be a potential biomarker for diagnosis of exposed children lacking the distinctive FAS dysmorphology (Jacobson et al., 2008). EBC is valuable because it can be used at different stages of development and it can be used in both animals and humans including very young children (Wilson and Cudd, 2011, p. 93).

Ultrasound

A pilot study conducted by Kfir et al. (2009) used ultrasound parameters for early detection of alcohol exposure. A second trimester ultrasound examination showed significant differences between exposed and non-exposed for femur length. In addition, CCD and FTD (brain measurements) were significantly shorter in the exposed group. A third trimester ultrasound was then conducted and showed that of the three measurements noted above, only the mean difference for FTD continued. It revealed that the OD brain measurement was significantly shorter in the exposed group in the third semester. This suggests the possibility of identifying impaired growth of the frontal cortex as early as 24 weeks. These findings were consistent with reduced frontal cortex measurements found by Wass, Persutte, and Hobbins (2001) using ultrasound.

Neuroimaging Studies

The use of neuroimaging can assist with identifying damage, biomarkers, dose and timing. It allows for in vivo assessment of neuroabnormalities (Astley, Richards et al., 2009). Advances in computational image analysis techniques have allowed reassessment of the image data and have provided new insight into the damage caused by heavy prenatal alcohol exposure (Riley, 2005, p. 360). Enhanced sensitivity resulting from improvements in MRI data acquisition techniques is producing new findings of previous studies (Nunez et al., 2011). Astley, Olson et al. (2009) report that in a typical FASD diagnostic clinic MRIs are typically only available when clinically indicated (e.g., evidence of neurological abnormalities). As such, only 204 (10 percent) of the 2,040 patients evaluated at the FAS DPN had a previous MRI evaluation summarized in their medical record and 76 percent of the 204 MRI evaluations were interpreted as normal by the patient neuroradiologist. Use of more sophisticated neuroimaging technology would greatly enhance identification of CNS abnormalities. The advantage to neuroimaging is that it is a non-invasive test that has no age restriction. Cost and availability are the disadvantages.

MRI - Structural MRI

The MRI allows for the very sensitive assessment of size, shape, volume, spatial orientation, and tissue composition of selected brain regions. The MRI could augment diagnosis of conditions under the umbrella of FASD, once population-based norms for structural development of the human brain are established. FASD MRI studies document significant reductions in the size of many but not all brain regions when comparing a group with FASD to a healthy control group. The greater the structural brain abnormality, the greater the dysfunction (Astley, Aylward 2009b). The Astley, Aylward et al. (2009b) FASD MRI study also confirms the FAS facial phenotype (as defined by the 4-Digit Diagnostic Code, Astley, 2004a) is expressed and measured

(e.g., Face Ranks 1, 2, 3, and 4) on a clinical continuum. The more severe the FAS facial phenotype is, the more severe the CNS structural and functional abnormalities. The FAS facial features serve as external biomarker for underlying brain damage/dysfunction. The FAS facial characteristics are not simply present or absent. Astley, Alyward et al. (2009b) confirmed that individuals with the full FAS phenotype have significantly and disproportionately smaller frontal lobe volumes and significantly more severe CNS dysfunction than individuals with prenatal alcohol exposure and no FAS facial features. Additionally, alcohol-exposed individuals with and without facial dysmorphology suffer damage to several parts of the brain. For example, Sowell et al. (2008), found in their sample of alcohol-exposed individuals that those with and without facial dysmorphology “exhibited statistically significant cortical thickness increases, making it clear that having facial dysmorphology is not a prerequisite for having brain dysmorphology”.

MRI rodent studies are showing damage from alcohol exposure at early developmental stages that in humans would occur prior to the time that pregnancy is typically recognized, underscoring the importance of pre-pregnancy counseling (O’Leary-Moore et al., 2011, p. 104).

MRI was conducted on six children with FAS and matched with seven normal controls. Results indicated significant size reductions of the basal ganglia in the children with FAS when compared with the control group (Mattson et al., 1996).

In a study of 31 youth, 21 with alcohol exposure and 10 without, using structural magnetic resonance imaging Bjorkquist et al. (2010) found significantly smaller amounts of cingulate grey matter, white matter, and overall tissue when alcohol-exposed children were compared with their peers. Reduction in white matter was more affected. White matter reduction occurred regardless of FAS diagnosis in the alcohol-exposed group. Bjorkquist et al. (2010) conclude that damage to the cingulate gyrus may contribute to deficits in cognitive control, attention, and emotion regulation.

FASD MRI studies conducted by Astley, Alyward et al. (2009b) confirm the FAS facial phenotype (as defined by the 4-Digit Diagnostic Code, Astley, 2004a) is expressed and measured (e.g., Face Ranks 1, 2, 3, and 4) on a clinical continuum. The more severe the FAS facial phenotype is, the more severe the CNS structural and functional abnormalities. The FAS facial features serve as an external biomarker for underlying brain damage/dysfunction. The FAS facial characteristics are not simply present or absent. Astley, Alyward et al. (2009b) confirmed that individuals with the full FAS phenotype have significantly and disproportionately smaller frontal lobe volumes and significantly more severe CNS dysfunction than individuals with prenatal alcohol exposure and no FAS facial features. Additionally, alcohol-exposed individuals with and without facial dysmorphology suffer damage to several parts of the brain. For example, Sowell et al. (2008), found in their sample of alcohol-exposed individuals that those with and without facial dysmorphology “exhibited statistically significant cortical thickness increases, making it clear that having facial dysmorphology is not a prerequisite for having brain dysmorphology”.

MRI—Functional MRI or fMRI

The fMRI is a potentially powerful tool that can assess regional brain activation in response to performance on specific cognitive, perceptual or motor tasks (Astley, Alyward et al., 2009a). The challenge in using the fMRI on a prenatally alcohol-exposed population is their smaller head size. This could potentially distort the results if not taken into account during analysis (Sowell

et al., 2001; Riley, McGee, and Sowell, 2004). Significant differences in brain activation patterns were observed between FASD and control groups during verbal learning (Sowell et al., 2007), verbal working memory (O'Hare et al., 2009), response inhibition (Fryer et al., 2007) spatial working memory (Malisza et al., 2005) nonspatial working memory and visual working memory (Astley, Olson, 2009c). In a study that included of both children and adults with FASD the pattern of responding was similar for both groups (Malisza et al., 2005).

O'Hare et al. (2009) compared 20 children and adolescents with FASD with 20 typically developing children and adolescents using fMRI and verbal working memory tests. Individuals were matched for task performance on verbal working memory tests. Individuals affected by FASD showed increased activation in several regions of the brain during the verbal working memory tests compared with normal individuals. The study concluded that processing in one part of the brain (frontal parietal) during verbal tests, which use working memory, is less efficient in alcohol-exposed individuals.

Diffusion Tensor Imaging (DTI)

DTI measures the diffusion of water molecules within white matter of the brain to produce images of white matter tracts and measures of white matter integrity (Basser, Marriello, and LeBihan, 1994; LeBihan, 1995). DTI may assist in identifying alcohol's adverse effects on the central nervous system development. In FASD research it has been used to define white-matter anomalies in the human whole brain, primarily in the corpus callosum (O'Leary-Moore et al., 2011). It is sensitive to microstructural abnormalities that have been observed in patients with FAS (Le, 1995). Studies that use DTI and 3D facial analyses based on MRM reconstruction are designed to identify subtle changes in facial morphology (Hammond et al., 2005).

In a study by Spottiswoode et al. (2011), DTI was used to assess the structural integrity of white matter microstructure (cerebellar penduncles) and the degree to which alcohol exposure deficits in eyeblink is mediated by structural impairment. Animal studies have shown that cerebellar penduncles are critical in eyeblink conditioning. Healthy, highly organized white matter is indicated by high fractional anisotropy (FA) values. The results showed a strong correlation between alcohol exposure and low FA values, specifically in perpendicular and not parallel diffusivity. This study suggests that the changes in white matter may in part be responsible for the poor eyeblink function associated with alcohol exposure.

Sowell, Johnson et al. (2008) found lower FA values in the lateral splenium and bilateral parietal white matter within an FASD group which was associated with poorer performance on visuo-motor integration tests.

Magnetic Resonance Spectroscopy (MRS)

An MRS is a technique that allows the noninvasive assessment of a number of neurochemicals in the brain and detection of subtle changes in brain biochemistry in the absence of gross dysmorphology (O'Leary-Moore et al., 2011). The three chemicals that can be measured using MRS include N-acetyl-aspartate, choline and creatine. Studies have shown changes in each of these neurochemicals following prenatal alcohol exposure.

Astley, Weinberger, Shaw, Richards, and Clarren (1995) conducted an MRS study in a nonhuman primate model of FASD in the 1990s. Fourteen years later the study was replicated in a

human population (Astley, Richards et al., 2009). Both studies presented with comparable findings; choline levels were significantly lower in the population with FAS/PFAS.

Brain metabolism of children with FASD is permanently altered. It may be useful as a biomarker for prenatal alcohol exposure (Astley et al., 1995; Cortese et al., 2006; Fagerlund et al., 2006; Astley, Richards et al., 2009).

Humans have widely varying dietary requirements for choline, in part explained by genetic variation (Zeisel, 2011). Magnetic resonance spectroscopy outcomes have demonstrated that the FAS/PFAS patient group had significantly low choline levels compared to controls. Decreased choline concentrations were associated with more severe FASD diagnoses and increased impairment in neuropsychological test performance (Astley, Richards et al., 2009; Biller, Bartsch, Homola, Solymosi, and Bendszus, 2009).

Magnetic Resonance Microscopy (MRM)

Magnetic resonance microscopy provides high resolution images up to a microscopic level. The MRM has been used with FASD mouse models to assess dose/timing and consequence of prenatal ethanol exposure (Parnell et al., 2009; Godin et al., 2010). According to Godin et al. (2010) the MRM was able “to readily show the range and severity of median forebrain deficiency that occurs in the absence of overt hindbrain dysmorphology” (p. 108). In addition, the MRM study identified cerebral cortical dysplasia (a spectrum of malformations of the cerebral cortex) and cerebral cortical heterotopias (the displacement from its normal position) resulting from acute gestational day (GD) 7 ethanol exposure in the mouse model (Godin et al., 2010).

2D Image Analysis of Facial Photographs

The “FAS facial phenotype” was first empirically identified/case-defined from direct measures of facial features back in 1995 (Astley and Clarren, 1995). The study population consisted of 194 children of all races, balanced on gender and age. A discriminant analysis identified smooth philtrum, thin upper lip, and short palpebral fissure as the cluster of features that best differentiated children with and without FAS. A subsequent study in 1996 (Astley and Clarren, 1996) demonstrated this FAS facial phenotype could be identified from 2D facial photographs with greater sensitivity and specificity. This FAS facial phenotype was incorporated into the FASD 4-Digit Diagnostic Code in 1997. In a 10-year, population-based foster care FAS screening program of 2,000 children (all races, birth to 18 years old), 95 percent of all children with this FAS Facial phenotype (as detected from a 2D digital facial photograph using the FAS Facial Photographic Analysis Software) were confirmed to have prenatal alcohol exposure and FAS (Astley et al., 2002).

3D Image Analysis

Often the FAS facial features in individuals are expressed subtly making identification difficult. These subtle features are difficult to identify with two-dimensional images. A current study is using a camera system to produce three-dimensional images to measure length, width and height of facial features of individuals in various age and ethnic groups. This may allow researchers to track how alcohol-exposed facial features change with age (Wetherill and Foroud, 2011).

A computational framework for surface-based morphometry (an imaging technique to localize shape changes related to different conditions) was applied to 3D analysis of facial morphology

in FAS. The results identified facial dysmorphology patterns in FAS that were consistent with prior findings (Stoler and Holmes, 2004; Hoyme et al., 2005; Wan et al., 2010).

Epigenetics

Epigenetics is the study of heritable changes in gene expression “caused by mechanisms other than changes in the underlying DNA sequence” (Chudley, 2011, p. 116). While epigenetics is only an emerging area of study, it has potentially significant ramifications for future understanding, prevention, and treatment of FASD. Epigenetic understandings may answer questions like such as: What is the nature and magnitude of the genetic effect of alcohol exposure? How does genetic variation interact with environmental factors? Understanding the genomic contribution is important in attributing risk (Ramsay, 2010).

As pointed out by Ramsay (2010), “many animal studies have shown that clinical severity following *in utero* ethanol exposure...is the result of complex gene-environment interactions that alter gene expression patterns, especially during development” (p. 2). Altering the pathways of genes during embryogenesis and development plays a potentially pivotal role in “the distinct characteristics associated with FASD—that is, CNS dysfunction, craniofacial abnormalities, and growth retardation” (Chudley, 2011, p. 115). What epigenetics suggests, accordingly to Chudley, is that “many of the effects of alcohol on gene expression...are more likely due to epigenetic effects, rather than to direct changes in DNA sequences” (p. 116).

Chudley goes on to explain the following:

The presence of imprinted genes can cause cells with a full parental complement of functional autosomal genes to specifically express one allele [a pair, or portion of a series, of genes], but not the other, and this will result in monoallelic expression of the imprinted loci [the location of a gene or gene sequence on a chromosome]. Genomic imprinting plays a critical role in fetal growth and behavioral development, and is regulated by DNA methylation [process by which groups are added to certain nucleotides in genomic DNA] and chromatin [A complex of nucleic acids—for example, DNA or RNA—and proteins, which condenses to form a chromosome during cell division] structure. (p. 117).

According to Ramsay (2010), the study of these interactions has provided evidence “supporting the involvement of epigenetic remodeling in alcohol teratogenesis” (p. 4). Ramsay, along with Reik, Dean and Walter (2001), point out that during development, there are essentially three main stages of generalized global epigenetic remodeling: 1) gametogenesis (formation of reproductive cells), where there is a wave of demethylation followed by sex-specific genetic imprinting and generalized methylation; 2) preimplantation, which is characterized by generalized DNA demethylation in the zygote (with the exception of imprinted loci); and 3) another wave of *de novo* methylation during gastrulation (the post-blastula stage of embryonic development). According to Ramsay (2010), there is ample evidence to suggest that epigenetic disturbances during these stages can result “in shifts towards increased or decreased gene expression, a phenomenon that would be in line with the broad range of clinical manifestations reflecting a dynamic and individual response to alcohol exposure” (p. 4).

The implications of better understanding the contribution of imprinting to the regulation of gene expression is significant. In terms of FASD, it suggests genetic factors on the part of both

the male and female that need to be considered when assessing susceptibility for having a child with an FASD, as well as the potential need to monitor alcohol use *before* pregnancy as well as during. This would greatly impact future research, as well as future efforts to prevent and/or treat FASD.

For additional information on epigenetics and FASD see Kobor and Weinberg, 2011, Chudley, 2011, and Ramsay, 2010.

Collaborative Initiative in Fetal Alcohol Spectrum Disorders (CIFASD)

The collaborative initiative is a cross-cultural assessment of FASD supported by NIAAA that coordinates basic, behavioral, and clinical investigators in a multidisciplinary research project to advance the FASD intervention and treatment field. The CIFASD consists of three core resource projects—administrative, informatics, and dysmorphology. These three projects facilitate collaboration through management oversight, communication, data access and integration. The two major research components are basic science and clinical.

Translational Studies of FASD Using a Sheep Model

The sheep model is being used to compare the effects of binge-like alcohol exposure during the period of brain development comparable to that of the human first trimester (1st-trimester model) with similar binge-like exposure that extends over the stages of brain development encompassing all three human trimesters (3-trimester model). The study is evaluating phenotypic measures used in the diagnosis of fetal alcohol syndrome—growth, facial dysmorphology, and brain and behavioral development. The study will test whether the pervasive effects on brain and neurobehavioral development that result from binge exposure will continue after the first trimester. The study will evaluate growth, facial morphometry, and effects on in vivo brain regional volumes using structural magnetic resonance imaging; assess neurobehavioral outcomes using eyeblink classical conditioning and spatial working memory; and assess neuroanatomical effects via neuronal counts in the cerebellum, hippocampal formation, and brainstem serotonin system. Another objective is to test the hypothesis that choline supplementation initiated periconceptually will attenuate the adverse effects of alcohol exposure in the 3-trimester sheep model.

Magnetic Resonance and Diffusion Tensor Imaging of a Mouse FASD Model

Magnetic Resonance Imaging (MRI) and Diffusion Tensor Imaging (DTI) will be applied to the study of an FASD mouse model to provide comprehensive documentation and discovery of the ethanol-induced CNS dysmorphology that results from prenatal ethanol exposure at embryonic and early fetal stages of development. The project will also define the facial dysmorphology that results from prenatal ethanol exposure during embryonic and/or early fetal stages and to relate their character and severity to accompanying abnormalities of the brain; and identify regions other than the brain or face that may serve as diagnostic indicators of prenatal ethanol exposure.

Mouse Model Neuro-Facial Dysmorphology: Translational and Treatments Studies

A mouse model that models human consumption and is known to produce FAS-like features resulting from prenatal alcohol exposure will be used to test the effects of differences in dose and timing of alcohol exposure on face and brain development. A combination of 3D imaging that includes micro-video and micro-resonance imaging (MRI) will be used to capture the detailed facial structure, micro-computational tomography (Micro-CT) to capture the underlying facial

bone, MRI for detailed brain dimensions, and diffusion tensor imaging (DTI) for nerve fiber tracks in the fetal period. A computational program will be compiled to detect features specifically as function of alcohol exposure. The association and dissociation of facial and brain dysmorphology as a function of dose and timing of alcohol exposure will be analyzed to better inform the diagnosis of FAS/ FASD. Portions of this study will also be clinical.

Clinical Component:

Spectrum of and Nutritional Risk Factors for FASD in Russia and Ukraine

This project will study the complete range of expression of FASD in relation to specific quantities, patterns, and gestational timing of alcohol exposure, and to evaluate the sensitivity of methods for earlier recognition of affected children. It will also assess the contribution of maternal nutritional status in relation to physical and neurobehavioral outcomes associated with prenatal alcohol exposure, as well as test the benefit of maternal second and third trimester supplementation with multinutrients with or without choline with respect to risk for FASD in the offspring. Lastly, the study will include a well-characterized sample of mothers/children with and without prenatal exposure to alcohol for testing of alternative and earlier methods of identifying affected children, including prenatal ultrasound and post-natal 3D facial imaging.

3D Facial Imaging in FASD

This project will include the collection of a longitudinal, multi-ethnic sample of individuals prenatally exposed to alcohol. This sample will allow the researchers to separate the effects of ethnic variation and developmental age from those due to alcohol exposure. The overarching goals will be to improve understanding of the dysmorphic features in FAS and FASD; enhance the capability for definitive diagnosis of FAS and the broader spectrum of FASD at different stages of the lifespan; and establish whether there is a relationship between FAS and FASD dysmorphic features and the specific underlying impairments in brain function.

A Multisite Neurobehavioral Assessment of Fetal Alcohol Spectrum Disorder

The primary aim of this project is to determine whether a neurobehavioral phenotype exists in children with fetal alcohol syndrome, whether the same phenotype exists in children with FASD who lack facial dysmorphology, and whether the phenotype can be used for differential diagnosis. Secondary aims, involving collaboration with other CIFASD projects and cores, are to determine the relationship between brain dysmorphology, facial dysmorphology, and neurobehavioral function.

Mapping the Brain, the Face, and Neurocognitive Function in FASD

This project will use quantitative brain mapping techniques with high-resolution structural and functional MRI collected both cross-sectionally and longitudinally from 80 children with FASD evaluated across three multi-cultural data collection sites (San Diego, Los Angeles, and Cape Town, South Africa). It will identify brain structural and functional abnormalities across the broad spectrum of FASD. This information will then be part of an assessment of the other components of the collaboration to create a multidimensional profile.

Regulations and Federal Legislation

Screening Reimbursement

Codes have been established that allow physicians to screen patients for alcohol abuse and provide behavioral interventions. As of Jan. 1, 2008 the CPT codes 99408 and 99409 for commercial insurance were created for physicians to use when billing for alcohol and substance abuse screening and intervention services. Two G-codes were created for substance abuse assessment and intervention for Medicare G0396 and G0397 and the codes for Medicaid are H0049 and H0050. The following **fact sheet** about Medicare reimbursement discusses what is covered and the type of health care providers who can screen and provide brief intervention.

The SAMHSA–Health Resources and Services Administration (HRSA) Center for Integrated Solutions provides a number of valuable resources about linking behavioral health and primary care. The link below provides access to a webpage on financing SBIRT. Once on the site you can stay at clinical practice and click on motivational interviewing and SBIRT or navigate to other tabs such as financing to learn about billing tools and private payers.

<http://www.integration.samhsa.gov/clinical-practice/sbirt/financing>

Affordable Care Act (ACA)

The ACA provides for comprehensive health insurance reform that will roll out over 4 years and beyond with many changes taking place by 2014. The overarching goal of the prevention component is to prevent diseases and promote wellness. The Act identifies targeted prevention activities that will be funded through commercial insurance, Medicare, and Medicaid. Initial changes to private health plans in 2010 included:

- Coverage for a range of preventive services.
- Ban on imposing cost sharing on patients receiving these services.
- Requirement for private insurance plans to provide coverage under four broad categories:
 - Evidence-based screenings
 - Evidence-based counseling
 - Routine immunizations
 - Preventive services for children, youth and women

In 2011, Medicare:

- Removed costs for preventive services,
- Provided an annual wellness visit and welcome to Medicare exam, and
- Began to cover preventive services, including tobacco cessation counseling and HIV screening.

Changes to Medicaid, within the ACA, include:

- An increased federal share for prevention services.

The community prevention activities will be funded through grant programs including the Prevention and Public Health Trust Fund and the Community Transformation Grants.

The *Prevention and Public Health Trust Fund* program for States provides incentives to Medicaid beneficiaries who participate in a program to develop a healthy life.

- Created by ACA to assist State and Community efforts to prevent illness and promote health.
- Funding is for \$15 billion over 10 years to address community and clinical prevention, public health infrastructure and primary care training.
- In FY2011, SAMHSA received \$88 million and funded programs that include SBIRT, Suicide Prevention, and Primary and Behavioral Health Care Integration.
- Funding for public health infrastructure is addressing capacity building, epidemiology and laboratory capacity, and public health training centers.
- Primary care training will address:
 - Advanced nursing education expansion
 - Assistant training
 - Nurse managed health clinics
 - State health care workforce development
- Much of the prevention work is done in partnership with states and communities to increase public health programs' effectiveness and efficiency and improve access to behavioral health services.

The *Community Transformation Grants* will provide the following:

- \$100 million in funding for up to 75 grants for State and local government agencies, Tribes, territories, and non-profit organizations.
- Funding is to address one of the five priority areas:
 - Tobacco-free living
 - Active living and healthy eating
 - Evidence-based quality clinic and other preventive services
 - Social and emotional wellness
 - Safe physical environments

Additional grants under ACA will include:

- Home visitation to moms and babies in high-risk communities.
- School-based health centers.
- Workforce provisions.

Additional information regarding provisions for women and children can be found on www.healthcare.gov, a Web site sponsored by HHS.

Child Abuse Prevention and Treatment Act (CAPTA)

President Obama signed Public Law 111-320 on December 20, 2010, authorizing another five years of the Child Abuse Prevention and Treatment Act (CAPTA). The fund source provides critical support to States and counties administering a child welfare program and has been a source for creative dependency court programs. FASD-related modifications CAPTA include a new category of referral and safe care plan requirement for newborns diagnosed with a "Fetal Alcohol Spectrum Disorder (FASD)." FASD is different from FAS and broadens the group of

children that might be identified. The expected outcome is that more newborns will be referred to state CPS programs.

http://www.acf.hhs.gov/programs/cb/laws_policies/cblaws/capta/capta2010.pdf

Modification to CAPTA

CAPTA Reauthorization Act of 2010 (P.L.111-320) modifies state grant eligibility requirements by adding FASD language.

ELIGIBILITY REQUIREMENTS—

1. STATE PLAN—

A. IN GENERAL—To be eligible to receive a grant a state shall submit to the Secretary a state plan that specifies the areas of the child protective services system that the state will address with amounts received under the grant.

2. CONTENTS—A state plan submitted shall contain a description of the activities that the state will carry out using amount received under the grant to achieve the objectives of this title, including—

A. an assurance that the state plan, to the maximum extent practicable, is coordinated with the state plan under part B of title IV of the Social Security Act (42 U.S.C. 621 et seq.) relating to child welfare services and family preservation and family support services;

B. an assurance in the form of a certification by the Governor of the state that the state has in effect and is enforcing a state law, or has in effect and is operating a statewide program, relating to child abuse and neglect that includes—

i. provisions or procedures for an individual to report known and suspected instances of child abuse and neglect, including a state law for mandatory reporting by individuals required to report such instances;

ii. policies and procedures (including appropriate referrals to child protection service systems and for other appropriate services) to address the needs of infants born with and identified as being affected by illegal substance abuse or withdrawal symptoms resulting from prenatal drug exposure, **or a Fetal Alcohol Spectrum Disorder**, including a requirement that health care providers involved in the delivery or care of such infants notify the child protective services system of the occurrence of such condition of such infants, except that such notification shall not be construed to—

I. establish a definition under Federal law of what constitutes child abuse or neglect; or

II. require prosecution for any illegal action.

iii. the development of a plan of safe care for the infant born and identified as being affected by illegal substance abuse or withdrawal symptoms or **Fetal Alcohol Spectrum Disorder**;

iv. procedures for the immediate screening, risk and safety assessment, and prompt investigation of such reports;

v. triage procedures, including the use of differential response, for the appropriate referral of a child not at risk of imminent harm to a community organization or voluntary preventive service;

vi. procedures for immediate steps to be taken to ensure and protect the safety of a victim of child abuse or neglect and of any other child under the same care who may

also be in danger of child abuse or neglect and ensuring their placement in a safe environment;

vii. provisions for immunity from prosecution under state and local laws and regulations for individuals making good faith reports of suspected or known instances of child abuse or neglect;

viii. methods to preserve the confidentiality of all records in order to protect the rights of the child and of the child's parents or guardians, including requirements ensuring that reports and records made and maintained pursuant to the purposes of this title shall only be made available to—

I. individuals who are the subject of the report;

II. Federal, state, or local government entities, or any agent of such entities, as described in clause (ix);

III. child abuse citizen review panels;

IV. child fatality review panels;

V. a grand jury or court, upon a finding that information in the record is necessary for the determination of an issue before the court or grand jury; and

VI. other entities or classes of individuals statutorily authorized by the State to receive such information pursuant to a legitimate state purpose;

ix. provisions to require a state to disclose confidential information to any Federal, state, or local government entity, or any agent of such entity, that has a need for such information in order to carry out its responsibilities under law to protect children from child abuse and neglect;

x. provisions which allow for public disclosure of the findings or information about the case of child abuse or neglect which has resulted in a child fatality or near fatality;

xi. the cooperation of state law enforcement officials, court of competent jurisdiction, and appropriate state agencies providing human services in the investigation, assessment, prosecution, and treatment of child abuse and neglect;

xii. provisions requiring, and procedures in place that facilitate the prompt expunging of any records that are accessible to the general public or are used for purposes of employment or other background checks in cases determined to be unsubstantiated or false, except that nothing in this section shall prevent State child protective services agencies from keeping information on unsubstantiated reports in their casework files to assist in future risk and safety assessment;

Appendix A

ICCFASD Consensus Statement on ARND (2011)

Question 1: What is ARND; how can it be diagnosed (classical, current diagnostic schemes, in practice today)?

Alcohol-related neurodevelopmental disorder (ARND) refers to a complex range of disabilities in neurodevelopment and behavior, adaptive skills, and self-regulation in the presence of confirmed prenatal alcohol exposure (PAE). ARND is one of the fetal alcohol spectrum disorders that also include fetal alcohol syndrome (FAS), which is additionally characterized by distinct facial features and growth retardation.

The term ARND was used in a 1996 report developed under the auspices of the Institute of Medicine (IOM; Stratton, 1996) to recognize the existence of neurodevelopmental disorders associated with confirmed PAE. Specifically, individuals with ARND do not present with the FAS facial phenotype (reduced palpebral fissure length, smooth philtrum, and thin upper vermilion border), but may present with structural and/or functional central nervous system (CNS) abnormalities, and may or may not present with growth deficiencies or decreased cranial size at birth. Acknowledging some degree of uncertainty that PAE caused the presenting adverse effects in any particular individual, the 1996 IOM report on FAS defined ARND as CNS neurodevelopmental abnormality evidenced by decreased cranial size at birth, or structural brain abnormalities, or neurological hard or soft signs in the presence of a pattern of confirmed excessive maternal prenatal alcohol use. Alternatively, ARND could be defined by evidence of a complex pattern of behavioral and cognitive abnormalities that are inconsistent with developmental level and cannot otherwise be explained by the genetic contribution of the biological parents, or by impairments in brain maturation conferred by adverse environmental factors. Alternative descriptors have emerged, including “neurodevelopmental disorder/alcohol exposed” and “static encephalopathy/alcohol exposed.”¹ In the ensuing years, animal research and human studies have helped families, clinicians, and researchers develop a deeper understanding of the relationship between PAE and neurodevelopmental manifestations subsequently evident in children, adolescents, and adults.

Part A: Evidence of CNS Developmental Abnormalities

The brain is susceptible to the neurotoxic effects of alcohol at all stages of gestation. Based on extensive, mutually reinforcing animal and clinical research, there appear to be patterns of significant structural and functional changes in the CNS attributable to PAE. Basic research suggests that numerous processes of neuronal development and functioning can be affected by PAE. Animal studies demonstrate that the timing, dose, and frequency of PAE differentially harm specific neuronal structures and brain circuits. Brain imaging and cognitive and behavioral studies have substantiated similar structural and functional alterations in humans.

Part B: Evidence of a Complex Pattern of Behavior and Cognitive Abnormalities

There is clear and compelling evidence from animal studies that PAE negatively affects behavior, cognition, motor function, self-regulation and adaptive function, executive function, activity, and mood in a complex way. Children with PAE frequently exhibit behavioral and emotional problems such as inattention, hyperactivity, anxiety, and mood de-regulation. These problems

may emerge early in life and continue to significantly impair an individual's functioning in numerous domains throughout the lifespan. Identified problems may be primary to PAE, be primary to a comorbid condition, or result from the contribution of and interaction among a number of factors, including interactions with the environment. Research with children suggests that PAE can be associated with general cognitive impairments and specific impairments in the following areas: information processing, attention, executive function, language, memory/learning, social cognition, number processing, and sensorimotor function. One or more behavioral and cognitive phenotypes specific to PAE have been elusive.

Identification of specific phenotypes is confounded by variability of exposure (dose, duration, and timing) and potential interactions among other factors, which may include qualities of the prenatal and postnatal environments, genetics, and exposure to other toxic substances.

Question 2: Can ARND be differentiated from other disorders?

Alcohol is a known teratogen and is strongly associated with a range of neurodevelopmental and behavioral disorders that may affect numerous domains of functioning across the lifespan. Emerging evidence from animal and human studies suggests that there is a constellation of symptoms attributable to PAE that may include (1) neurocognitive impairments, (2) self-regulatory challenges, and (3) impairments in adaptive functioning. However, differentiating ARND from other complex neurodevelopmental disorders can be challenging due to limited available studies attempting to distinguish ARND phenotype (s) from other disorders.

Even when a history of PAE is available, diagnosing ARND and distinguishing it from other complex developmental disorders requires prudent clinical judgment and consideration of other potential causes. In the future, we anticipate that clinicians will be assisted in making this diagnosis through advances in the identification of biomarkers sensitive to the detection of significant PAE and the development of tests that are both sensitive to and specific for alcohol induced neurobehavioral disorders. We recommend additional rigorous scientific investigation to further refine understanding of the cognitive, behavioral, neurologic, and psychiatric clinical profiles attributable to PAE, as well as of the patterns of development evidenced by individuals with PAE. We further recommend that investigations of other complex developmental disorders include inquiry about PAE to identify the contribution of PAE to the phenotypes of other developmental disorders.

Question 3: What prenatal alcohol exposure evidence is necessary for an ARND diagnosis?

An ARND diagnosis requires confirmed, significant PAE. Determination of alcohol exposure can be based on maternal self-report; the report of a spouse, partner, relative, or friend who observed the birth mother drinking alcohol during the index pregnancy; and/or documentation in medical or other records about maternal alcohol use during the index pregnancy.

Data from animal studies across multiple species confirm that, at the highest levels of alcohol exposure in the first trimester, facial abnormalities and brain maldevelopment occur in concert. However, alterations in brain development that subsequently affect behavior can occur with a range of alcohol dosages throughout gestation, even when the face and brain appear to be structurally normal. Variable patterns of maternal drinking, including binge drinking resulting in significant peak levels or sustained drinking resulting in significant cumulative exposures, may lead

to differential fetal outcomes. In addition, evidence suggests that variability in both maternal and fetal characteristics affects the potential for alcohol-induced alterations in brain development. Thus, because there is no known safe threshold for PAE, it is not currently possible to define a safe limit of alcohol consumption during pregnancy.²

Question 4: What signs/symptoms will be useful as screening criteria?

The U.S. Surgeon General recommends regular screening of every woman of childbearing age for alcohol use. Screening should be conducted by adult primary health care clinicians and obstetric caregivers to protect the health of women and any subsequent offspring. For children, pediatric primary health care clinicians should obtain medical records about PAE and other potential risks from the birth mother's obstetric caregiver. For children who are not living with their birth parents, clinicians should obtain any available records that may provide information about PAE or other relevant family history. Clinicians should query families in a nonjudgmental way about all risks to a child's development, including maternal alcohol use prior to and during pregnancy. We recommend that clinicians be trained regarding the most effective ways to ask about alcohol use to ensure that this practice is adopted as routine. Regular screening of parental alcohol use should continue as part of the process of child health supervision and developmental surveillance.

If PAE is confirmed, primary care clinicians should be alert for signs and symptoms that can occur during the child's development. Primary care clinicians should complete a comprehensive history for any child at risk for ARND that includes questions about developmental milestones, school functioning, peer and family relationships, adaptive and self-help skills, and specific areas of impairment, and they also should conduct a physical and neurological examination. A concern identified in any of these areas warrants a referral for a complete evaluation and follow-up. Absence of a concern should result in continued developmental surveillance of the child, as problems related to PAE may emerge during maturation, particularly during adolescence and young adulthood when latent ARND as well as other comorbidities commonly arise.

Question 5: What are the treatment needs for those diagnosed with ARND?

Given that the manifestations of PAE are heterogeneous, vary across development, and can be lifelong, treatment plans need to be multimodal and specific to the strengths and weaknesses of the affected individual and family across the lifespan. Treatment begins with support of the affected individual and family and education on the manifestations of ARND, risks for other problems, and available treatments. Treatments should draw on evidence-based practices as they pertain to an individual's specific needs. Treatment should be implemented flexibly, but with fidelity, in addressing the specific developmental strengths and weaknesses of the individual and family. Modifications of evidence-based treatments should only be considered when those treatments have been implemented with integrity over an adequate period of time and have failed to yield improvement.

Some interventions for ARND have targeted common manifestations of PAE, including problems with mathematics, attention, self-regulation, adaptive functioning and problem-solving, social impairment, and working memory. Other interventions have focused on individual and group-based skills development or training of caregivers in behavior management. Large, well

controlled behavioral intervention studies specific to ARND are needed. First, studies are needed to determine whether currently available evidence-based interventions for problems common to children without PAE yield similar benefits in children with ARND. Second, when evidence indicates a reduced or inadequate benefit, modifications of existing interventions or development of entirely new interventions need to be completed and evaluated. In addition, these research objectives also may be accomplished by assessing for PAE those participants with other mental disorders and developmental disabilities in treatment outcome studies. Finally, these studies should target children across the developmental spectrum, with special attention directed at the periods of challenging transitions from childhood to adolescence and from adolescence to adulthood.

Some medications have been shown to effectively treat emotional and behavioral problems in children. Although there is an impression that many of these medications may be less effective in children with PAE or may lead to an atypical response, the literature is sparse and inconclusive. High-quality randomized controlled trials of medication treatments are needed for individuals across the lifespan in order to identify which medications work best for the specific problems affecting individuals with ARND.

Many of the problems experienced by children with PAE manifest as academic impairments or other problems at school. Given the limited number of educational interventions specific to children with ARND, clinicians may need to draw on evidence-based educational interventions for other disorders. For any school-based educational, medical, or mental health intervention, it is particularly important to engage educators, school health and mental health professionals, and other staff in assessing, planning, and implementing the intervention with high fidelity to enhance the effectiveness of the specific intervention plan.

Problems in self-regulation and social and adaptive functioning can manifest as early as infancy and continue throughout the lifespan. For young children, it is important to educate caregivers and early intervention providers about the manifestations of ARND as well as the usefulness of specific intervention strategies for this age group. In addition, individuals with ARND can have poor decision-making skills, placing them at risk for a range of behavioral problems that may lead to contact with school-based professionals and other community service providers who may be unaware of how ARND can affect a child's functioning. It is therefore important to help affected individuals and families become self-advocates and to broadly educate the public and relevant professionals about behavior problems associated with ARND.

In conclusion, children and youth with PAE have a Special Health Care Need³ and should have ongoing developmental and behavioral surveillance by their primary health care clinician in a "medical home."⁴ This surveillance should continue throughout their lifespan to assess ongoing treatment and referral needs.

¹Currently, there are several commonly accepted diagnostic schemes and interpretations of the diagnostic guidelines presented in the 1996 IOM report on FAS (see Resources for Further Information and Application at the end of this statement for commonly accepted diagnostic schemes providing guidelines on diagnosing ARND). This panel is not advocating any particular system for the diagnosis of ARND or the use of these alternate descriptors.

²The 1996 IOM report on FAS describes necessary or required confirmed alcohol exposure as "a pattern of excessive intake characterized by substantial, regular intake or heavy episodic drinking. Evidence of this pattern may

include frequent episodes of intoxication, development of tolerance or withdrawal, social problems related to drinking, legal problems related to drinking, engaging in physically hazardous behavior while drinking, or alcohol-related medical problems such as hepatic disease.” New data are accumulating to suggest that ARND can occur at lower levels of alcohol exposure than indicated in the 1996 IOM report.

³ As adopted in 1998 by the American Academy of Pediatrics (AAP), children with Special Health Care Needs are those who have or are at increased risk for a chronic physical, developmental, behavioral, or emotional condition and who also require health and related services of a type or amount beyond that required by children generally. This definition originally was proposed in McPherson M, Arango P, Fox HB. A new definition of children with special health care needs. *Pediatrics*. 1998; 102:137–40.

⁴ The AAP defines a “medical home” as one in which the care of infants, children, and adolescents is delivered or directed by well-trained physicians who provide primary care and help to manage and facilitate essentially all aspects of pediatric care. The physician should be known to the child and family and should be able to develop a partnership of mutual responsibility and trust with them. Ideally, care is accessible, continuous, comprehensive, family centered, coordinated, compassionate, and culturally effective. The Affordable Care Act of 2010 endorses the medical home model throughout the lifespan.

Appendix B—Methodology

To identify the literature to support the development of this TIP, a search was conducted for fetal alcohol spectrum disorders information of primary interest to clinicians and program administrators.

The literature search began with the following questions:

- How can providers identify a fetal alcohol spectrum disorder in their patients and then most effectively address the disorder in the context of substance abuse treatment?
- How can providers most effectively contribute to the prevention of fetal alcohol spectrum disorders?
- How can screening and service delivery be enhanced to effectively address the fetal alcohol spectrum disorder disabilities resulting from an alcohol-exposed pregnancy?

For this review, the following terms or subject areas were used to search the National Library of Medicine's (NLM) PubMed database, the American Psychological Association's PsycInfo database, the National Institute on Alcohol Abuse and Alcoholism's Alcohol and Alcohol Problems Science Database (ETOH), and the Substance Abuse and Mental Health Services Administration's FASD Center for Excellence (CFE) database. The terms included:

- Prevalence
- Prevention
- Co-occurring
- Intervention
- Screening
- Disability
- Systems of Care

The initial search was conducted for literature published between 2000 and 2010 with limits for humans, English and Title/Abstract. PubMed allowed the use of a search string to further refine the search. An FAS string was formed by grouping similar terms together. The FAS string consisted of the following: "fetal alcohol syndrome," "fetal alcohol spectrum disorder," "fetal alcohol effects," "FASD," "foetal alcohol syndrome," "foetal alcohol spectrum disorder," "foetal alcohol effects," "prenatal alcohol exposure," and "alcohol-exposed pregnancy." The string was paired with each search term listed above resulting in a comprehensive search. The categories were part of the search for PubMed. The categorization was done after the search for the three other databases. Duplicates were progressively removed starting with PubMed and continuing through to ETOH.

For each of the citations found, reference information and abstracts were printed and then reviewed by two reviewers. Two reviewers were used to reduce any potential bias in the selection process. Reviewers eliminated citations in which the primary substance was not alcohol. The reviewers examined the results and identified articles for use in the TIP. Articles that were accepted by both reviewers were kept. Articles that were selected by only one reviewer were then directed to a third party for a decision. The final results identified 472 articles.

In addition to the database search for journal articles, the internet and FASD Center for Excellence database was also searched for books, monographs, curriculums, and policy and procedures using the search term Fetal Alcohol Syndrome. This list of publications was also reviewed by two reviewers. Selections by just one reviewer were then directed to a third party. These searches were repeated prior to submitting the TIP for clearance and will be performed every six months for a five-year period after clearance has been received.

In addition to the articles located by the electronic searches, some articles were retrieved because they were recommended by consensus panelists or were identified through references in the articles previously consulted.

The final list of references selected by the reviewers as the most relevant for the Review of Literature can be found in Section 2, Selected Abstracts and Books. A complete bibliography of all relevant literature located during the search appears in Section 3, General Bibliography.

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