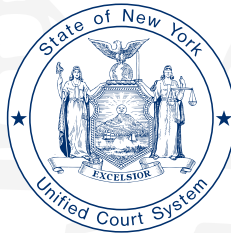




2021 OJI CHILD WELFARE AND SUBSTANCE USE DISORDER KEYNOTE SERIES

# IS PREVENTION AN ETHICAL OBLIGATION?

JUNE 23, 12:30 TO 2:00 PM



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EXCELSIOR

## **Ira J. Chasnoff, M.D.**

Ira J. Chasnoff, M.D., an award-winning author, researcher and lecturer, is president of NTI Upstream and a Professor of Clinical Pediatrics at the University of Illinois College of Medicine in Chicago. He is one of the nation's leading researchers in the field of child development and the effects of environmental factors on the developmental trajectory of children and adolescents. Dr. Chasnoff's work encompasses community approaches to the integration of behavioral health services into primary health care, the schools, and the other multiple systems that serve children and families. Dr. Chasnoff has served several U.S. presidential administrations on committees and commissions dedicated to organizing and coordinating services for children and families across the nation and developing policies and procedures for addressing the needs of children at risk from prenatal and postnatal trauma. He also has worked with a wide range of States across the nation and internationally in developing universal outreach and early intervention programs for children and families affected by trauma or substance abuse.

Dr. Chasnoff received his medical degree from the University of Texas Health Science Center at San Antonio, which in 1991 awarded him its first *Distinguished Alumnus Award*. He is the author of numerous research articles regarding the long-term cognitive, behavioral, and learning outcomes of high-risk children, and his article on racial and social class bias in the health care system has been cited as a landmark study by the American College of Obstetricians and Gynecologists. Dr. Chasnoff has authored 15 books, which have received numerous awards and one of which has been recognized by The Hague International Court. Dr. Chasnoff's books explore the biological and environmental factors that impact the ultimate development of high-risk children and presents practical strategies for helping children reach their full potential at home and in the classroom. His newest book, *Guided Growth*, has received international acclaim for its in-depth discussion of educational and behavioral interventions for children and teens with Fetal Alcohol Spectrum Disorders and early trauma. Dr. Chasnoff has been a regular contributor to *Psychology Today*, writing about high-risk children and their families. The recipient of several awards for his work with women, children, and families, Dr. Chasnoff for several years has been selected by a poll of physicians across the nation for listing in *America's Best Doctors*, cited for his ability to translate complex medical and psychosocial issues into relevant policy that guides the delivery of quality services. Dr. Chasnoff has been active in establishing comprehensive family intervention programs for children in Australia, Denmark, Portugal, Canada, Vietnam, the former Soviet Union, and across the United States and has lectured on this topic around the world.

## **Honorable Caren Loguercio**

Judge Loguercio began serving on the bench of the Suffolk County Family Court in January 2011 and handles child protective proceedings, Family Treatment Court, custody and visitation, family offense and adoption cases. Judge Loguercio is a member of both the Attorney for the Children Advisory Committee of Suffolk County and Mental Health Professionals Screening Committee for the Second Department. Judge Loguercio was appointed as an Acting Supreme Court Justice in January of 2015.

Judge Loguercio was also recently appointed to the statewide Family Treatment Court strategic planning committee and as lead judge to the Child Welfare Court Improvement Project. Judge Loguercio is a member of the Suffolk County Bar Association and was elected to the Board of Directors in June of 2018. Judge Loguercio is the former co-chair of both the Family Court Committee and Professional Ethics and Civility Committee, a member of the Bench Bar Committee, the Suffolk County Women's Bar Association and the Suffolk County Matrimonial Bar Association where she served as a member of the Board of Directors from 2012 to 2016 and President (2013-2014). Judge Loguercio has lectured for the New York State Bar Association, Suffolk County Bar Association Academy of Law, Suffolk County Women's Bar Association, Suffolk County 18-b panel and for Touro Law School and has served as editor for the Suffolk Lawyer Family Law Section.

Prior to being elected, Judge Loguercio was the Principal Law Clerk to Supreme Court Justice Emily Pines and served as an Assistant Town Attorney for the Town of Brookhaven. Judge Loguercio graduated from the University of Florida with a bachelor's degree in criminal justice in 1989 and a Juris Doctorate, with honors, in 1992.

In 2017, Judge Loguercio was named Judge of the Year by the Suffolk County Court Officers Benevolent Association. In 2011, Judge Loguercio was named Public Citizen of the Year by the Suffolk County Chapter of the Association of Social Workers. In 2007, the Town of Brookhaven honored Judge Loguercio as Woman of the Year in Law.

Judge Loguercio previously served as a member of the Board of Directors of the Osteogenesis Imperfecta Foundation, a not-for-profit corporation dedicated to helping the lives of people afflicted with this brittle bone disease and Every Child's Dream, a not-for profit corporation which assists children and families living in homeless shelters on Long Island.

# The Child Abuse Prevention and Treatment Act: Knowledge of Health Care and Legal Professionals

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**Ira J. Chasnoff**

*NTI Upstream*

**Gail Barber**

*Iowa Children's Justice*

*Iowa Supreme Court*

**Jody Brook**

*University of Kansas*

*School of Social Welfare*

**Becci A. Akin**

*University of Kansas*

*School of Social Welfare*

The purpose of this study was to determine if health care and court professionals were aware of the mandatory reporting requirements associated with the Child Abuse Prevention and Treatment Act (CAPTA) and the correct markers for prenatal substance exposure (PSE). Discipline-specific surveys revealed that approximately 82% of health care and 71% of court personnel

were unaware of the legislation and that few knew of the correct markers of PSE.

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The Child Abuse Prevention and Treatment Act (CAPTA), first passed in 1974, was amended in 2003 and then again in 2010 (CAPTA Reauthorization Act of 2010, 2010) to include required referral of newborns affected by prenatal exposure to alcohol or illegal drugs to each state's child protection services (CPS). Most recently, in 2016, the Comprehensive Addiction and Recovery Act (CARA), in response to the current epidemic of Neonatal Abstinence Syndrome, amended CAPTA to remove the term "illegal" when denoting reportable substance exposure (Comprehensive Addiction and Recovery Act, 2016). Although most child welfare reporting laws reside at the state level, CAPTA specifically requires that state plans contain assurances that there is a state law or statewide program that includes:

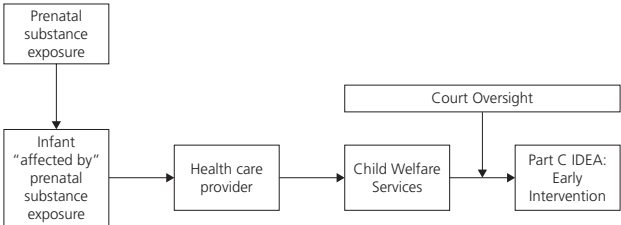
...policies and procedures (including appropriate referrals to child protection service systems and for other appropriate services) to address the needs of infants born and identified as being affected by substance abuse or withdrawal symptoms resulting from prenatal drug exposure, or a Fetal Alcohol Spectrum Disorder (*sic*), 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.... (Child Abuse Prevention and Treatment Act, sections 106[b][2][B][ii] and [iii]).

CAPTA does not establish a definition under federal law of what constitutes child abuse or neglect, nor does it require prosecution for any illegal action. Rather, the purpose of referral to CPS is to ensure that the child is linked to the state's early intervention services provided under Part C of the federal Individuals with Disabilities Education Act (IDEA) and that a plan of safe care for the infant is developed. The need for such legislation is based on research that demonstrates the benefits of early intervention for infants affected by prenatal exposure to alcohol and illicit drugs, especially if these children are recognized early and receive early intervention services (Streissguth et al., 1991; Bono et al., 2005).

Prenatal exposure to substances of abuse can cause significant impairment in neonatal health outcomes as well as structural and functional changes in the developing fetal brain. Prenatal alcohol exposure can produce microcephaly and a broad spectrum of significant abnormalities of various brain structures (Astley et al., 2009) that can result in a wide range of neurobehavioral deficits in the newborn. In addition, cocaine, methamphetamine, opiates, and other illicit drugs, as well as tobacco, can have significant adverse effects on pregnancy and the child: increased rates of preterm labor and delivery, low birth weight infants, dysmorphism, neonatal seizures, and erratic neonatal neurobehavioral patterns (Chasnoff, Burns, Schnoll, & Burns, 1985; Coles, Platzman, Smith, James, & Falek, 1992; Lester et al., 2002; DeMoraes Barros et al., 2006). The current epidemic of opiate abuse has led to increasing numbers of newborns undergoing Neonatal Abstinence Syndrome (Finnegan, Kron, Connaughton, & Emich, 1975) with neurological, gastrointestinal, and respiratory difficulties.

Essentially, the CAPTA legislation promotes a chain of events that links health care, child welfare, and early intervention agencies, with the courts providing oversight of the child welfare system (see Figure 1). This flow across systems is grounded in health care providers' recognition of newborns "affected by" prenatal exposure to alcohol and other substances and knowledge of juvenile court professionals as to the effects of prenatal substance exposure (PSE) and the need to

**Figure 1. Cross-systems Flow for CAPTA Regulations Regarding Prenatal Substance Exposures**



link affected infants to early intervention services. The objectives of this study were to determine if children's health care providers and juvenile court professionals were aware of the CAPTA legislation and knew the signs and symptoms of an infant's having been affected by PSE that would require referral to early intervention services via CPS.

## Methods

Iowa Children's Justice, a division of State Court Administration, Judicial Branch of Iowa, conducted a series of comprehensive training programs for children's health care and juvenile court personnel. The training programs addressed the impact of prenatal substance exposure on infants, children, and adolescents and were guided by three learning objectives:

*At the completion of the training, participants would be able to:*

1. Name five signs and symptoms seen in newborns affected by prenatal substance exposure.
2. Explain the purpose of the CAPTA legislation as related to referral of infants affected by prenatal substance exposure and describe the procedures for this referral.
3. Discuss the effectiveness of early intervention for infants affected by prenatal substance exposure.

A previous case-control study, funded by the Health Resources Services Administration, documented the efficacy of this training program, with participants in the training group scoring significantly higher on knowledge acquisition regarding PSE than a matched group of controls (Chasnoff & Wells, 2010).

Prior to each training session, one of two surveys designed specifically for children's health care providers or juvenile court personnel was distributed to each attendee, who completed the questionnaire and returned it to the conference staff before the training began.<sup>1</sup> These

<sup>1</sup> A copy of the surveys can be found at [www.ntiupstream.com/CAPTA](http://www.ntiupstream.com/CAPTA).

survey questionnaires asked respondents of their knowledge regarding CAPTA and to identify items known to be indicators of prenatal substance exposure, based on well-established markers contained in the peer reviewed literature (Chasnoff, Burns, Schnoll, & Burns, 1985; Coles, Platzman, Smith, James, & Falek, 1992; Lester et al., 2002; DeMoraes Barros et al., 2006; Finnegan, Kron, Connaughton, & Emich, 1975). De-identified data collected on the questionnaire were entered into SPSS for analysis. Descriptive and bivariate analyses were conducted to address the study's objectives. To examine whether health care and legal professionals differed in regards to the factors that should prompt referral of infants affected by PSE, chi-square tests were conducted. Analyses of Variance (ANOVA) was used to consider, within each professional group, whether there was an association between age or professional experience and the factors for referral to CPS. Finally, for each professional group, chi-square tests were used to examine whether the course of action differed by illicit drug versus alcohol exposure. Two-sided  $p$  values  $< .05$  were considered statistically significant. Procedures for this study were approved by the University of Kansas Institutional Review Board.

## Results

Participants were voluntary attendees at a series of perinatal substance abuse conferences conducted in Iowa. Over 90% of attendees completed the survey and turned it in to conference staff before the training program began. Physicians (pediatricians and family practitioners) and obstetric, neonatal, and pediatric nurses comprised the children's health care personnel, and attorneys and judges made up the juvenile court personnel (see Table 1).

The multiple-choice survey asked respondents what factors determined that an infant had been "affected by" PSE, thus requiring referral to CPS. Of nine possible responses, the most common indicator cited by both medical and court professionals was a positive urine toxicology in the mother at the time of delivery or in the infant at birth, followed



**Table 1. Sample Characteristics**

	<b>Total Sample Number (%)</b>	<b>Health Care Number (%)</b>	<b>Legal Number (%)</b>
<b>Total</b>	170	74	96
<b>Gender</b>			
Male	45 (27%)	9 (12%)	36 (38%)
Female	124 (73%)	65 (88%)	59 (62%)
No response	1 (< 1%)		1 (1%)
<b>Age (years)</b>			
Range	22–71	22–69	27–71
Mean	46.6	44.0	48.7
<i>SD</i>	12.3	13.2	11.2
<b>Profession</b>			
Nurse	53 (31%)	53 (71%)	
Physician	21 (12%)	21 (28%)	
Attorney	76 (45%)		76 (79%)
Judge	20 (12%)		20 (21%)
<b>Length of Time in Profession (years)</b>			
Range	0–45	0–45	1–43
Mean	18.1	17.7	18.5
<i>SD</i>	12.2	13.0	11.5

*Abbreviation:* *SD*, standard deviation.

by maternal self-report of substance use (see Table 2). Relatively few health care providers or court personnel considered common medical indicators (Chasnoff, Burns, Schnoll, & Burns, 1985; Coles, Platzman, Smith, James, & Falek, 1992; Finnegan, Kron, Connaughton, & Emich, 1975; Hurd et al., 2005; Varner et al., 2009) of PSE or neurobehavioral deficits (Lester et al., 2002; DeMoraes Barros et al., 2006) in the newborn as an indication of the infant’s having been affected by prenatal substance exposure. Rather, 15% of health care providers stated that the mother’s socioeconomic status was a factor in deciding if a referral to CPS was warranted. Overall, 4% of health care providers stated they never referred a child to CPS in spite of known PSE, and 9% of

**Table 2. Factors Requiring an Infant’s Referral to CPS**

Substance	Total Sample (n = 170)		Health Care (n = 74)		Legal (n = 96)		Chi-Square
	n	%	n	%	n	%	
<b>Illegal Drugs</b>							
Mother’s Urine	102	60.0	46	62.2	56	58.3	$\chi^2(1) = 0.522$ $p = .613$
Newborn Urine	117	68.8	52	70.3	65	67.7	$\chi^2(1) = 0.128$ $p = .721$
Growth Disturbance	26	15.3	16	21.6	10	10.4	$\chi^2(1) = 4.050$ $p = .044$
Behavioral Disturbance	23	13.5	17	23.0	6	6.3	$\chi^2(1) = 9.989$ $p = .002$
Poor Muscle Tone	13	7.6	9	12.2	4	4.2	$\chi^2(1) = 3.783$ $p = .052$
Previous CPS Removal	55	32.4	29	39.2	26	27.1	$\chi^2(1) = 2.798$ $p = .094$
Mother’s Psychosocial Risk	14	8.2	11	14.9	3	3.1	$\chi^2(1) = 7.621$ $p = .006$
Mother Self-Report	97	57.1	40	54.1	57	59.4	$\chi^2(1) = 0.483$ $p = .487$
Family/Friend Report	55	32.4	24	32.4	31	32.3	$\chi^2(1) = 0.000$ $p = .984$
Never Report to CPS	12	7.1	3	4.1	9	9.4	$\chi^2(1) = 1.803$ $p = .179$
<b>Alcohol</b>							
Growth Disturbance	37	21.8	25	33.8	12	12.5	$\chi^2(1) = 11.117$ $p = .001$
Behavioral Disturbance	34	20.0	27	36.5	7	7.3	$\chi^2(1) = 22.261$ $p = .000$
Poor Muscle Tone	18	10.6	14	18.9	4	4.2	$\chi^2(1) = 9.606$ $p = .002$
Previous CPS Removal	40	23.5	24	32.4	16	16.7	$\chi^2(1) = 5.773$ $p = .016$

(continued)

**Table 2. Factors Requiring an Infant’s Referral to CPS (Continued)**

Substance	Total Sample (n = 170)		Health Care (n = 74)		Legal (n = 96)		Chi-Square
	n	%	n	%	n	%	
Mother’s Psychosocial Risk	15	8.8	10	13.5	5	5.2	$\chi^2(1) = 3.583$ $p = .058$
Mother Self-Report	66	38.8	33	44.6	33	34.4	$\chi^2(1) = 1.838$ $p = .175$
Family/Friend Report	37	21.8	17	23.0	20	20.8	$\chi^2(1) = 0.112$ $p = .737$
Never Report to CPS	39	22.9	11	14.9	28	29.2	$\chi^2(1) = 4.835$ $p = .028$

*Abbreviation:* CPS, Child Protective Services.

juvenile court personnel responded that there are no reporting requirements related to PSE.

Significant differences were observed between medical and court professionals’ opinions as to the indications for reporting (see Table 2). Medical professionals were significantly more likely to perceive an infant was “affected by” prenatal exposure to illicit drugs based on growth disturbance, behavioral disturbance, and the mother’s psychosocial risk than were judges and lawyers. As for prenatal alcohol exposure, medical professionals were significantly more likely to consider an infant affected if there was evidence of growth disturbance, behavioral disturbance, poor muscle tone, or previous CPS removal than were legal professionals. Further analysis revealed that neither the health care practitioner’s age nor years of professional experience affected patterns of response among any of the indicators for reporting prenatal exposure. In contrast to health care providers, however, ANOVA revealed that both age and professional experience of court personnel were associated with selection of markers of an infant’s having been affected by prenatal substance exposure. Court personnel who selected infant growth disturbance were significantly older than court personnel who did not select that factor ( $F(1) = 6.475, p < .02$ ), and court professionals who selected poor muscle tone had significantly more years in practice

( $F(1) = 9.167, p < .01$ ). Overall, significantly more legal professionals than medical professionals stated that prenatal alcohol exposure was never an indication for reporting to CPS.

Health care providers were asked what action they took upon identifying an infant affected by prenatal substance exposure (see Table 3). Providers were significantly more likely to refer an infant to CPS if illicit drug exposure occurred as compared to prenatal alcohol exposure. It also was significantly more likely that alcohol-exposed infants would never be reported to CPS as compared to infants with prenatal exposure to illicit drugs.

Court personnel also demonstrated a significantly different approach to infants prenatally exposed to alcohol as compared to those exposed to illicit drugs, and 29% and 9%, respectively, stated that there is no CPS reporting requirement for infants affected by prenatal exposure to alcohol or illicit drugs ( $\chi^2(1) = 17.145, p < .001$ ). Similar to health care

**Table 3. Health Care Providers' Course of Action upon Identification of an Infant Affected by Prenatal Alcohol vs. Illicit Drug Exposure**

Type of Referral	Health care providers' response		
	Yes %	$\chi^2$	$p$
Treatment Referral		$\chi^2(1) = 38.91$	< .001
For Alcohol Use	56.8		
For Drug Use	62.2		
Hospital Social Work Referral		$\chi^2(1) = 45.18$	< .001
For Alcohol Use	56.8		
For Drug Use	62.2		
Early Intervention Referral		$\chi^2(1) = 39.41$	< .001
For Alcohol Use	50.0		
For Drug Use	50.0		
Contact CPS		$\chi^2(1) = 29.47$	< .001
For Alcohol Use	31.1		
For Drug Use	44.6		

*Abbreviation:* CPS, child protective services.

personnel, the courts were more likely to refer the mother for substance abuse treatment and the infant for early intervention services for illicit drug exposure as compared to alcohol exposure (see Table 4).

Overall, when questioned about their knowledge of CAPTA, 82.4% of children’s health care providers and 70.8% of juvenile court personnel responded that they were unaware of any CAPTA regulations requiring referral of infants affected by prenatal exposure to alcohol or illicit drugs to CPS or responsibility for linking these infants to early intervention services.

**Limitations**

Some limitations of the current work should be noted prior to fully considering the findings. This study relied on a convenience sample of professionals who voluntarily attended a statewide training, thus limiting the generalizability of the findings. The attendees were individuals who had an existing identified interest in prenatal substance use and thus their level of knowledge may not represent the general population. Also, the survey instrument was unique in that, while the survey

**Table 4. Court Professionals’ Course of Action upon Identification of an Infant Affected by Prenatal Alcohol vs. Illicit Drug Exposure**

Resource Referral Type	Legal professionals’ response		
	Yes %	$\chi^2$	<i>p</i>
Treatment Referral		$\chi^2 (1) = 64.357$	< .001
For Alcohol Use	63.5		
For Drug Use	65.6		
Early Intervention Referral		$\chi^2 (1) = 66.088$	< .001
For Alcohol Use	57.3		
For Drug Use	57.3		
No Action		$\chi^2 (1) = 65.535$	< .001
For Alcohol Use	10.4		
For Drug Use	9.4		

instrument contained identified indicators of PSE supported in the peer reviewed literature, the presenters of the workshop aggregated these indicators into one survey instrument for purposes of this measurement. Despite these limitations, the authors believe that the information gleaned from the study provides meaningful baseline information about an important and vastly understudied topical area.

## Discussion

Implementation of the CAPTA legislation is grounded in children's health care providers' recognizing infants who have been "affected" by prenatal substance exposure and the courts' ability to assess the implications of that effect as decisions are made that can make a difference in the long term developmental trajectory of the child (Streissguth et al., 1991; Bono et al., 2005). Numerous studies have demonstrated that cocaine, methamphetamine, opiates, and other illicit drugs as well as alcohol, marijuana, and tobacco can have significant adverse effects on neonatal outcome, varying with the specific substance(s) the pregnant woman has used (Chasnoff, Burns, Schnoll, & Burns, 1985; Coles, Platzman, Smith, James, & Falek, 1992; Finnegan, Kron, Connaughton, & Emich, 1975; Hurd et al., 2005; Varner et al., 2009). In addition, neonatal neurobehavioral effects often are present in the newborn, with the infant exhibiting poor motor performance, tonic problems, and difficulties with self-regulation, including sleep and feeding problems (Chasnoff, Burns, Schnoll, & Burns, 1985; Coles, Platzman, Smith, James, & Falek, 1992; Lester et al., 2002; DeMoraes Barros et al., 2006). These deficits have significant implications for the infants' everyday functioning, as the neuropsychological, behavioral, and cognitive deficits exhibited by children prenatally exposed to substances can affect their long-term development and socialization and lead to secondary conditions that interfere with appropriate academic, behavioral, and developmental progress (Streissguth et al., 1991). On the other hand, early recognition of problems and appropriate intervention early in life significantly improve long-term outcomes (Streissguth et al., 1991; Bono et al., 2005).

While the intention of the CAPTA legislation is to ensure infants with prenatal substance exposure receive needed resources in a timely manner, a fundamental barrier exists. As demonstrated in the current study, few children's health care professionals and juvenile court personnel are even aware of CAPTA reporting requirements, thus undermining implementation of the legislation.

The purpose of the original 2003 amendment to the CAPTA legislation was to establish a national standard for child protection interventions related to prenatal substance exposure (Weber, 2006). However, the CAPTA statute is unclear as to what "affected by" prenatal substance exposure means, and both within and across states, there is no consistent agreement as to how "affected" should be defined. This lack of consistency allows for subjectivity that negates the purpose of the legislation.

The majority of physicians in the present study rely primarily on toxicology results to guide decision-making as to whether to refer an infant to CPS or not. However, urine toxicologies are quite limited in their ability to identify exposed infants (Lester et al., 2001); alcohol, for the most part, is not included in toxicology screening panels, and even if it is, it is rarely found in the urine. As to the mother's use of illicit substances during pregnancy, that use, for the most part, has to have been within the previous 24 to 48 hours in order for the urine to be positive. Meconium toxicologies provide a wider window, documenting use throughout the third trimester of pregnancy (Lester et al., 2001). But, again, alcohol is rarely included in the routine meconium toxicology. In addition, by the time meconium toxicology results are available, in most instances the mother and infant already have been discharged from the hospital.

Implementation of the CAPTA legislation also is complicated in that the child health care professionals in the current study viewed the obligation to report differently for alcohol as opposed to illicit drug exposure. Adding to this, there is a general lack of consensus as to the possible harm of prenatal marijuana exposure on the child's health and developmental outcome, although multiple studies have documented the ill effects (Chasnoff, 2017). Tobacco use during pregnancy has

unequivocally been shown to be harmful (Varner et al., 2014), but the CAPTA legislation is silent on this point, although tobacco would fall into the most recent legislation, as written, requiring reporting of all infants affected by substances.

Studies dating back to the earliest days of requiring physicians to report child abuse have documented health care professionals' reluctance to have child welfare involved with a family (Chang et al., 1976), and this factor most likely continues to influence physician decisions today. From a social justice perspective, multiple studies, including this one, have demonstrated a significant social class difference in reporting infants due to prenatal substance exposure (Chasnoff, Landress, & Barrett, 1990; Van Ryn & Burke, 2000). In the current study, 15% of health care providers included socioeconomic status as a risk factor to be considered when deciding whether or not to report a prenatally exposed child to CPS. At the level of hospital policies, there are ongoing inconsistencies as to what substances are reportable and what maternal characteristics should drive decisions to report infants to child protection agencies (Weber, 2006; Burke, 2007).

From the perspective of prevention, universal screening of and therapeutic interventions for pregnant women at risk of substance use would reduce the medical and developmental risks related to prenatal substance exposure. Numerous studies have demonstrated the improved pregnancy, neonatal, and child outcomes associated with interventions during pregnancy that help the woman cease substance use as early in pregnancy as possible (Chasnoff et al., 1989; Goler et al., 2008). Establishing a universal screening, brief intervention, and referral to treatment (SBIRT) system accrues many benefits to mother and child (U.S. Prevention Services Task Force Ratings, 2003) and avoids the racial and social class biases that can disrupt prenatal care (Chasnoff, Landress, & Barrett, 1990; Van Ryn & Burke, 2000). As it stands now, current CAPTA legislation, rather than promoting prenatal identification of risk and referral to treatment, delays intervention until after potential harm is done, and in some ways may encourage punitive rather than therapeutic approaches postnatally (Weber, 2006).



The CAPTA legislation is an attempt to create a consistent approach to ensuring that infants affected by prenatal substance exposure have access to early intervention services. To achieve this shared purpose, several recommendations can be made:

1. Federal guidance—A consensus at the federal level is needed to guide states in defining the term “affected by.” This consensus statement should describe research-based objective criteria that identify newborns as “affected by” prenatal substance exposure and provide guidance as to when a newborn should be brought to the attention of child protective services.
2. State child welfare systems—At the state level, based on the guidance developed at the federal level, consistent standards are required as to the child welfare system’s response to families and their newborns affected by prenatal substance exposure. Consideration should be given to the fact that many of the complications related to perinatal substance use can be caused by other factors. For instance, how can a decision be made as to whether an infant’s being born low birth weight is due to an impoverished mother’s having poor access to healthy nutrition or due to the mother’s use of substances during pregnancy? In the first instance, the child would be referred directly to early intervention services; in the second, that referral would, based on CAPTA, necessarily have to go through CPS.
3. Professional education—Collaborative education of professionals across disciplines and agencies is needed to bring consensus to the CAPTA legislation as it is implemented at the local level. This education should address the impact of prenatal substance exposure; the need for early recognition and intervention; and the intent, requirements, and processes for implementation of CAPTA.
4. Public health approach—Rather than involving child welfare services in all cases, the question must be asked if direct referral from

the physician to early intervention would not be a better policy requirement. Of course, if it appears that there is risk of harm for the child, CPS must be notified. But in many cases, developing a direct link between the health care provider and early intervention would alleviate the potential disruption of the provider/patient relationship, resolve health care providers' reluctance to make a referral to CPS, and remove the stigma families often feel when referred to CPS, making them more likely to keep their appointment for early intervention services. In a follow-up to this study, we learned that only 4% of families referred by Iowa CPS to early intervention actually kept their appointment for an early intervention assessment. Linking the referral directly from the health care provider to the early intervention services may enhance the efficiency of the system and improve ultimate access for the families.

## Conclusions

CAPTA is designed to operate across multiple systems (see Figure 1). However, a study funded by the Administration for Children and Families, the federal agency responsible for oversight of CAPTA, demonstrated that two of the greatest challenges impeding implementation of CAPTA were in fact collaboration across agencies and development of effective protocols for identifying exposed newborns (Price et al., 2012). This was evident in the current study, which documented that physicians and court personnel rarely agreed as to the indications of an infant's having been affected by prenatal exposures. These inconsistencies are rooted in federal legislation that has created a statute grounded in a legal standard (required reporting) but that gives a wide range of discretion to both physicians and court personnel as to how the statute actually is to be interpreted and thus implemented.

In the end, CAPTA can provide a cross-systems structure for preventing and intervening in risk for child abuse and neglect. However,

the ambiguous language contained in the legislation impedes its success. In order to facilitate family stability, appropriate reunification, and prevent child abuse and neglect, pregnant women at risk of substance use and infants affected by prenatal substance exposure must be identified early and have full access to treatment and intervention services that will promote family and child well-being.

## References

- Agency for Healthcare Research and Quality. (2012). *U.S. Preventive Services Task Force ratings: Strength of recommendations and quality of evidence*. Rockville, MD: Agency for Healthcare Research and Quality.
- Astley, S. J., Olson, H. C., Kerns, K., Brooks, A., Coggins, T. E., Davies, J., Dorn, S., Gendler, B., Jirikowic, T., Kraegel, P., Maravilla, K., & Richards, T. (2009). Neuropsychological and behavioral outcomes from a comprehensive magnetic resonance study of children with fetal alcohol spectrum disorders. *Canadian Journal of Clinical Pharmacology*, *16*, e178–e201.
- Bono, K. E., Bolzani Dinehart, L. H., Claussen, A. H., Scott, S. G., Mundy, P. C., & Katz, L. F. (2005). Early intervention with children prenatally exposed to cocaine: Expansion with multiple cohorts. *Journal of Early Intervention*, *27*(4), 268–284.
- Burke, K. D. (2007). Substance-exposed newborns: Hospital and child protection responses. *Children and Youth Services Review*, *29*(12), 153–1519.
- The CAPTA Reauthorization Act of 2010, 42 U.S.C. §5101–5119 (2010).
- Chang, A., Oglesby, A. C., Wallace, H. M., Goldstein, H., & Hexter, A. C. (1976). Child abuse and neglect: Physicians' knowledge, attitudes, and experiences. *Public Health Briefs*, *66*(12), 1199–1201.
- Chasnoff, I. J., Burns, W. J., Schnoll, S. H., & Burns, K. A. (1985). Cocaine use in pregnancy. *The New England Journal of Medicine*, *313*(111), 666–669.
- Chasnoff, I. J., Griffith, D. R., MacGregor, S., Dirkes, K., & Burns, K. A. (1989). Temporal patterns of cocaine use in pregnancy: Perinatal outcome. *JAMA*, *261*(12), 1741–1744.
- Chasnoff, I. J., Landress, H. J., & Barrett, M. E. (1990). The prevalence of illicit-drug or alcohol use during pregnancy and discrepancies in mandatory reporting in Pinellas County, Florida. *New England Journal of Medicine*, *322*(17), 1202–1206.
- Chasnoff, I. J., & Wells, A. M. (2010). *Fetal Alcohol Spectrum Disorders: Prevention and Intervention*. Report to the Health Resources and Services Administration. Washington, DC: U.S. Department of Health and Human Services.

- Chasnoff, I. J. (2017). Medical marijuana laws and pregnancy: Implications for public health policy. *American Journal of Obstetrics & Gynecology*, *216*(1), 27–30.
- Coles, C. D., Platzman, K. A., Smith, I., James, M. E., & Falek, A. (1992). Effects of cocaine and alcohol use in pregnancy on neonatal growth and neurobehavioral status. *Neurotoxicology and Teratology*, *14*(1), 23–33.
- Comprehensive Addiction and Recovery Act of 2016, 42 U.S.C. §201 (2016).
- de Moraes Barros, M. C., Guinsburg, R., de Araújo Peres, C., Mitsuhiro, S., & Chalem, E., Laranjeira, R. R. (2006). Exposure to marijuana during pregnancy alters neurobehavior in the early neonatal period. *Journal of Pediatrics*, *149*(6), 781–787.
- Finnegan, L. P., Kron, R. E., Connaughton, J. F., & Emich, J. P. (1975). Assessment and treatment of abstinence in the infant of the drug-dependent mother. *International Journal of Clinical Pharmacology and Biopharmacy*, *12*(1–2), 19–32.
- Goler, N. C., Armstrong, M. A., Talliac, C. J., & Osejo, V. M. (2009). Substance abuse treatment linked with prenatal visits improves perinatal outcomes: A new standard. *Journal of Perinatology*, *29*(2), 181.
- Hurd, Y. L., Wang, X., Anderson, V., Beck, O., Minkoff, H., & Dow-Edwards, D. (2005). Marijuana impairs growth in mid-gestation fetuses. *Neurotoxicology and Teratology*, *27*(2), 221–229.
- Lester, B. M., Tronick, E. Z., LaGasse, L., Seifer, R., Bauer, C. R., Shankaran, S., ... Maza, P. L. (2002). The maternal lifestyle study: Effects of substance abuse exposure during pregnancy on neurodevelopmental outcome in 1-month-old infants. *Pediatrics*, *110*(6), 1182–1192.
- Lester, B. M., ElSohly, M., Wright, L. L., Smeriglio, V. L., Verter, V. L., Bauer, C. R., ... Maza, P. L. (2001). The maternal lifestyle study: Drug use by meconium toxicology and maternal self-report. *Pediatrics*, *107*(20), 309–317.
- Price, A., Bergin, C., Luby, C., Watson, E., Squires, J., Funk, K., ... Little, Christina. (2012). Implementing Child Abuse Prevention and Treatment Act (CAPTA) requirements to serve substance-exposed newborns: Lessons from a collective case study of four program models. *Journal of Public Child Welfare*, *6*(2), 149–171.
- Streissguth, A., Aase, J. M., Clarren, S. K., Randels, S. P., LaDue, R. A., & Smith, D. F. (1991). Fetal Alcohol Syndrome in Adolescents and Adults. *JAMA*, *265*(15), 1917–2028. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/2008025>
- Streissguth, A. P., Aase, J. M., Clarren, S. K., Randels, S. P., LaDue, R. A., & Smith, D. F. (1991). Fetal alcohol syndrome in adolescents and adults. *JAMA*, *265*(15), 1961–1967.
- Stroud, L. R., Paster, R. L., Goodwin, M. S., Shenassa, E., Buka, S., Niaura, R., ... Lipsitt, L. P. (2010). Maternal smoking during pregnancy and neonatal behavior: A large-scale community study. *Pediatrics*, *123*(5), 848.

- Van Ryn, M., & Burke, J. (2000). The effect of patient race and socio-economic status on physicians' perception of patients. *Social Science and Medicine*, 50(6), 813–828.
- Varner, M. W., Silver, R. M., Rowland Hogue, C. J., Willinger, M., Parker, C. B., Thorsten, V. R., ... Reddy, U. M. (2015). Association between stillbirth and illicit drug use and smoking during pregnancy. *Obstetrics and Gynecology*, 123(1), 113–125.
- Weber, E. M. (2007). Child welfare interventions for drug-dependent pregnant women: Limitations of a non-public health response. *UMKC Law Review*, 75(3), 789–845.

## OBSTETRICS

# Medical marijuana laws and pregnancy: implications for public health policy

Ira J. Chasnoff, MD

An increasing number of states are passing or considering medical marijuana laws. The goal of this paper is to address the public health system's responsibility to educate physicians and the public about the impact of marijuana on pregnancy and to establish guidelines that discourage the use of medical marijuana by pregnant women or women considering pregnancy.

## Patterns of marijuana use in pregnancy

The prevalence of marijuana use during pregnancy ranges from 2% to 5% in most studies but is reported as high as 15–28% among young, urban, socioeconomically disadvantaged women.<sup>1</sup> Importantly, the mean potency of marijuana in terms of its content of 9-carboxy- $\Delta^9$ -tetrahydrocannabinol, the psychoactive ingredient in marijuana, has increased steadily over the past 30 years.<sup>2</sup>

Although no epidemiological studies of the use of marijuana during pregnancy provide information as to the source of the women's access to marijuana, a recent report from the US Drug Testing Laboratories (Chicago, IL), examined Colorado's 2012 ballot initiative allowing large-scale marijuana production and statewide distribution and studied its impact on patterns of maternal marijuana use.<sup>3</sup> The ballot

Although there is much to learn yet about the effects of prenatal marijuana use on pregnancy and child outcome, there is enough evidence to suggest that marijuana, contrary to popular perception, is not a harmless drug, especially when used during pregnancy. Consequently, the public health system has a responsibility to educate physicians and the public about the impact of marijuana on pregnancy and to discourage the use of medical marijuana by pregnant women or women considering pregnancy.

**Key words:** marijuana, medical marijuana, pregnancy

initiative was passed in November 2012 and went into effect January 2014.

Based on local hospital protocols, meconium specimens from newborns across the nation that were determined to be at high risk of prenatal drug or alcohol exposure were collected and forwarded to the US Drug Testing Laboratories for analysis. Data were analyzed for the presence of marijuana in specimens originating from hospitals within the state of Colorado vs specimens sent from the rest of the United States during the first 9 months of the years 2012 and 2014. Positive samples were confirmed for 9-carboxy- $\Delta^9$ -tetrahydrocannabinol using gas chromatography–mass spectrometry.

The rates of positive meconium samples for marijuana were similar at each of the time points in the 2 populations, with an approximately 10% increase in the rate of positive marijuana samples in Colorado and in the rest of the country. More importantly, however, although the concentration of marijuana in exposed neonates' meconium for the US-wide population demonstrated little change across the 2 time periods, the exposed neonates in Colorado experienced substantially more exposure to marijuana in the postlegalization period as indicated by a significant increase (Mann-Whitney,  $P = .013$ ) in the concentrations of 9-carboxy- $\Delta^9$ -tetrahydrocannabinol, from a mean concentration of  $213 \text{ ng/g} \pm 230.9 \text{ ng/g}$

(median,  $142 \text{ ng/g}$ ) in 2012 to  $361 \text{ ng/g} \pm 420.3 \text{ ng/g}$  (median,  $212 \text{ ng/g}$ ) in 2014.<sup>3</sup>

## Consequences of marijuana use in pregnancy

Although increased rates of stillbirths<sup>4</sup> and low-birthweight neonates<sup>5–8</sup> have been documented in pregnancies complicated by prenatal marijuana use, these findings are partially confounded by tobacco use, which is relatively common among women who use marijuana during pregnancy. However, the known action of exogenous cannabinoids could explain the consistent neurological and neurodevelopmental outcomes that have been documented in infants and children prenatally exposed to marijuana.<sup>9</sup>

Marijuana is highly lipid soluble and crosses the placenta and the blood-brain barrier with ease, accumulating in fetal tissues, particularly the brain.<sup>10,11</sup> In the adult central nervous system, 9-carboxy- $\Delta^9$ -tetrahydrocannabinol interferes with the endocannabinoid signaling system, responsible for modulating synaptic neurotransmitter release to regulate motor control, memory, and other brain functions.<sup>12</sup>

Components of the endocannabinoid system are present during embryonic central nervous system development as early as 16–22 days' gestation in humans.<sup>13</sup> It is at this time that the neural plate and neural tube, the basic scaffold for the forebrain, midbrain, and hindbrain, are established. A large study

From the Department of Clinical Pediatrics, University of Illinois College of Medicine, and NTI Upstream, Chicago, IL.

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Corresponding author: Ira J. Chasnoff, MD.  
[irachasnoff@gmail.com](mailto:irachasnoff@gmail.com)

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conducted by the US National Birth Defects Prevention Center documented a significantly increased risk for anencephaly when the fetus is exposed to marijuana during the first month of gestation.<sup>14</sup> This risk was isolated to the period when the neural tube is closing, 1–4 weeks after conception.

The function of the endocannabinoid system during the preneuronal phase in humans has not been well delineated. However, a long line of research has demonstrated its important role in

shaping neuronal circuitry in the developing fetus as well as modulating development of various neurotransmitter systems, primarily the catecholaminergic and opioidergic systems.<sup>15–17</sup> Gestational exposure to exogenous cannabinoids, as found in marijuana, may target the cannabinoid receptor CB<sub>1</sub>, disrupting migration, differentiation, and synaptic communication in the developing neurotransmitter system.<sup>18–21</sup>

There also is evidence that intrauterine exposure to marijuana impairs dopamine D2 mRNA expression in the amygdala and in the nucleus accumbens at around 18–22 weeks' gestation.<sup>22</sup> The resulting defective dopamine D2 signaling in these centers, which play a role in cognitive and emotional functioning, is consistent with the neurobehavioral deficiencies that have been observed in newborns exposed to marijuana.

These deficits primarily reflect impaired regulatory control: irritability, tremors, and poor habituation<sup>23</sup>; difficulty with arousal and state regulation<sup>24,25</sup>; and sleep disturbance.<sup>26</sup> Although 2 studies<sup>27,28</sup> found no neurobehavioral differences between marijuana-exposed and nonexposed infants in the early neonatal period, it has been postulated that these 2 studies differed from the others because of sociocultural differences as well as the varying statistical treatments of the different confounding factors.<sup>25</sup>

Numerous studies have documented neurodevelopmental deficits in older children, adolescents, and young adults who were prenatally exposed to marijuana.<sup>29–36</sup> These studies once again are consistent with 9-carboxy- $\Delta^9$ -tetrahydrocannabinol's action on the developing fetal central nervous system. Longitudinal follow-up of children in a large prospective study found a consistent pattern of deficits in cognitive functioning. At 6 years of age, prenatal marijuana exposure was linked to lower verbal reasoning scores and deficits in composite, short-term memory, and quantitative intelligence scores.<sup>29</sup>

In this same cohort at 10 years of age, negative effects of prenatal marijuana

exposure had a significant impact on design memory and screening index scores of the Wide Range Assessment of Memory and Learning,<sup>30</sup> and the exposed children had lower test scores on school achievement.<sup>31</sup> In addition, by age 10 years, prenatal marijuana exposure was significantly related to increased hyperactivity, impulsivity, and inattention problems as well as significantly increased rates of child depressive symptoms.<sup>32,33</sup>

Child depressive symptoms and attention problems in these children at age 10 significantly predicted delinquency at 14 years.<sup>34</sup> Fried and Smith,<sup>35</sup> in a review of several well-controlled longitudinal studies, showed that prenatal marijuana exposure was related to a significantly increased rate of difficulties with executive functioning, an aspect of regulatory control that is key to learning and to managing behavior.

A study of functional MRIs in a group of 18–22 year old young adults who had been prenatally exposed to marijuana revealed altered neural functioning that impacted short-term memory.<sup>36</sup> Further animal and human studies are needed, especially studies that can overcome the common limitations found in the majority of studies that investigate teratogenic agents in humans, specifically the inability to conduct randomized, controlled, prospective studies and the reliance on retrospective self-report regarding amounts and patterns of marijuana use.

### Policy implications

Although there is much to learn yet about the effects of prenatal marijuana use on pregnancy and child outcome, there is enough evidence to suggest that marijuana, contrary to popular perception, is not a harmless drug, especially during pregnancy. Twenty-four states and Washington, DC, have passed medical marijuana legislation<sup>37</sup> (Table).

In general, the legislation in all states removes state-level criminal penalties on the use, possession, and cultivation of marijuana by patients who possess written documentation from their physician advising that they would derive benefit from the medical use of marijuana. Only Oregon has legislation

**TABLE**  
**States with medical marijuana laws<sup>37</sup>**

State	Date of passage of original legislation
Alaska	November 1998
Arizona	November 2010
California	November 1996
Colorado	November 2000
Connecticut	May 2012
District of Columbia	May 2010
Delaware	May 2011
Hawaii	June 2000
Illinois	May 2013
Maine	November 1999
Maryland	April 2014
Massachusetts	November 2012
Michigan	November 2008
Minnesota	May 2014
Montana	November 2004
Nevada	November 2000
New Hampshire	May 2013
New Jersey	January 2010
New Mexico	March 2007
New York	June 2014
Oregon	November 1998
Pennsylvania	April 2016
Rhode Island	January 2006
Vermont	May 2004
Washington	November 1998

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that requires a point-of-sale warning at dispensaries regarding cannabis use in pregnant or breast-feeding women.<sup>38</sup> The Colorado Department of Health has posted recommended screening questions for women who are pregnant and recommends discussing the importance of the cessation of marijuana during pregnancy or, at a well-woman visit, if a woman desires to become pregnant.<sup>39</sup>

The number of physicians who are prescribing marijuana to pregnant women across the various states is unknown, but professional organizations have recognized the need to address the issue. The American Medical Association announced in 2015 that it would advocate for regulations and pregnancy warning labels on medical and recreational marijuana,<sup>40</sup> and in July 2015 the Committee on Obstetric Practice of the American College of Obstetricians and Gynecologists published a policy statement that discouraged obstetricians and gynecologists from “prescribing or suggesting the use of marijuana for medicinal purposes during preconception, pregnancy, and lactation.”<sup>1</sup>

From a public health perspective, state departments of health, in collaboration with state licensing boards, should take several steps to educate and inform the public and professionals on the possible impact of marijuana’s use during pregnancy and to discourage such use including the following:

- Medical marijuana legislation should include public, professional, and legislative education about the impact of marijuana on pregnancy and child outcome.
- Informational materials should be available at all sites that prescribe or sell marijuana, and a government warning label, similar to alcohol, regarding marijuana use and pregnancy should be posted.
- Physicians who plan to write marijuana prescriptions should be required to obtain continuing medical education credits that address marijuana and pregnancy.
- Guidelines for physicians writing marijuana prescriptions should be

developed, including asking all women of child-bearing age about the possibility of a current pregnancy and offering a pregnancy test to all women of child-bearing age prior to giving a prescription for marijuana.

From a research perspective, randomized controlled studies of the effectiveness of marijuana as a medication need to include women, and rates of marijuana use in pregnancy before and after new medical marijuana legislation need to be assessed further. It appears that in the short term, legalization of marijuana use did not significantly increase the rate of marijuana use among pregnant women in Colorado.<sup>3</sup> However, those women who were using marijuana were either using greater quantities of marijuana or marijuana with higher concentrations of 9-carboxy- $\Delta^9$ -tetrahydrocannabinol.

Importantly, prospective, longitudinal studies of child cognitive and neurocognitive development need to be undertaken to further assess the impact of prenatal marijuana exposure, and studies of family functioning and child safety are necessary to understand environmental factors that may affect the child if a family member is using or abusing marijuana.

As states continue to legalize marijuana, making it more accessible, increased use across the general population could lead to increased rates of prenatal marijuana exposure, especially because most women do not realize they are pregnant during the first weeks after conception.

From a public health perspective, at the very least, we must acknowledge that marijuana’s use during pregnancy has potential risks, and we need to incorporate guidelines into the new and emerging marijuana laws that recognize and communicate that risk. Marijuana use is fast fading from the legal agenda, but its use, especially during pregnancy, remains a public health issue. ■

## REFERENCES

1. American College of Obstetricians and Gynecologists. Marijuana use during pregnancy

and lactation. ACOG Committee Opinion no. 637. *Obstet Gynecol* 2015;126:234-8.

2. Mehmedic Z, Chandra S, Slade D, et al. Potency trends of  $\Delta^9$ -THC and other cannabinoids in confiscated cannabis preparations from 1993 to 2008. *J Forens Sci* 2010;55:1209-17.

3. Jones JT, Baldwin A, Shu I. A comparison of meconium screening outcomes as an indicator of the impact of state-level relaxation of marijuana policy. *Drug Alcohol Depend* 2015;156:e104-5.

4. Varner MW, Silver RM, Hogue CJR. Association between stillbirth and illicit drug use and smoking during pregnancy. *Obstet Gynecol* 2014;123:113-25.

5. Hayatbakhsh MR, Flenady VJ, Gibbons KS, et al. Birth outcomes associated with cannabis use before and during pregnancy. *Pediatr Res* 2012;71:215-9.

6. Hurda YL, Wanga X, Anderson V, Beck O, Minkoff H, Dow-Edwards DY. Marijuana impairs growth in mid-gestation fetuses. *Neurotoxicol Teratol* 2005;27:221-9.

7. El Marroun H, Tiemeier H, Steegers EAP, et al. Intrauterine cannabis exposure affects fetal growth trajectories: the Generation R study. *J Am Acad Child Adolesc Psychiatry* 2009;48:1173-81.

8. English DR, Hulse GK, Milne E, Holman CD, Bower CI. Maternal cannabis use and birth weight: a meta-analysis. *Addiction* 1997;92:1553-60.

9. Trezza V, Cuomo V, Louk J, Vanderschuren MJ. Cannabis and the developing brain: insights from behavior. *Eur J Pharmacol* 2008;585:441-52.

10. Harbison RD, Mantilla-Plata B. Prenatal toxicity, maternal distribution and placental transfer of tetrahydrocannabinol. *J Pharmacol Exp Ther* 1972;180:446-53.

11. Hutchings DE, Martin BR, Gamagaris Z, Miller N, Fico T. Plasma concentrations of delta-9-tetrahydrocannabinol in dams and fetuses following acute or multiple prenatal dosing in rats. *Life Sci* 1989;44:697-701.

12. Pertwee RG. Ligands that target cannabinoid receptors in the brain: from THC to anandamide and beyond. *Addict Biol* 2008;13:147-59.

13. Psychoyos D, Vinod KY. Marijuana, Spice “herbal high,” and early neural development: implications for rescheduling and legalization. *Drug Test Anal* 2013;5:27-45.

14. vanGelder MM, Reefhuis J, Caton AR, Werler MM, Druschel CM, Roeleveld N. Maternal periconceptional illicit drug use and the risk of congenital malformations. *Epidemiology* 2008;20:60-6.

15. Mulder J, Aguado T, Keimpema E, et al. Endocannabinoid signaling controls pyramidal cell specification and long range axon patterning. *Proc Natl Acad Sci USA* 2008;105:8760-5.

16. Hernandez M, Berrendero F, Suarez I, et al. Cannabinoid CB(1) receptors colocalize with tyrosin hydroxylase in cultured fetal mesencephalic neurons and the action increases the



levels of this enzyme. *Brain Res* 2000;857:56-65.

**17.** Harkany T, Keimpema E, Barabas K, Mulder J. Endocannabinoid functions controlling neuronal specification during brain development. *Mol Cell Endocrinol* 286: S84-90.

**18.** Harkany T, Guzman M, Galve-Roperch I, Berghuis P, Devi LA, Mackie K. The emerging functions of endocannabinoid signaling during CNS development. *Trends Pharmacol Sci* 2007;28:83-92.

**19.** Galve-Ropeth I, Aguado T, Rueda D, Velasco G, Guzman M. Endocannabinoids: a new family of lipid mediators involved in the regulation of neural cell development. *Curr Pharm Des* 2006;12:2319-25.

**20.** Guzman M, Sanchez C, Galve-Ropeth I. Control of the cell survival/death decision by cannabinoids. *J Mol Med* 2001;78:613-25.

**21.** Bernard C, Milh M, Morozoy YM, Ben-Ari Y, Freund TF, Gozlan H. Altering cannabinoid signaling during development disrupts neuronal activity. *Proc Natl Acad Sci USA* 2005;102:9388-93.

**22.** Wang X, Dow-Edwards D, Anderson V, Minkoff H, Hurd YL. In utero marijuana exposure associated with abnormal amygdala dopamine D2 gene expression in the human fetus. *Biol Psychiatry* 2004;56:909-15.

**23.** Fried PA, Makin JE. Neonatal behavioral correlates of prenatal exposure to marijuana, cigarettes, and alcohol in a low risk population. *Neurotoxicol Teratol* 1987;9:1-7.

**24.** Coles CD, Platzman KA, Smith I, James ME, Falek A. Effects of cocaine and alcohol use in pregnancy on neonatal growth and

neurobehavioral status. *Neurotoxicol Teratol* 1992;14:23-33.

**25.** Maraes Barros MC, Guinsburg R, Araujo Peres C. Exposure to marijuana during pregnancy alters neurobehavior in the early neonatal period. *J Pediatr* 2006;149:781-7.

**26.** Scher MS, Richardson GA, Coble PA, Day NL, Stoffer DS. The effects of prenatal alcohol and marijuana exposure: disturbances in neonatal sleep cycling and arousal. *Pediatr Res* 1988;24:101-5.

**27.** Richardson GA, Day NL, Goldschmidt L. Prenatal alcohol, marijuana and tobacco use: infant mental and motor development. *Neurotoxicol Teratol* 1995;17:479-89.

**28.** Dreher MC, Nugent K, Hudgins R. Prenatal marijuana exposure and neonatal outcomes in Jamaica: an ethnographic study. *Pediatrics* 1994;93:254-60.

**29.** Goldschmidt L, Richardson GA, Willford J, Day NL. Prenatal marijuana exposure and intelligence test performance at age 6. *J Am Acad Child Adolesc Psychiatry* 2008;47:254-63.

**30.** Jastak S, Wilkinson GS. *Manual for the Wide Range Achievement Test*, revised. Wilmington (DE): Jastak Associates; 1984.

**31.** Goldschmidt L, Richardson GA, Cornelius MD, Day NL. Prenatal marijuana and alcohol exposure and academic achievement at age 10. *Neurotoxicol Teratol* 2004;26:521-32.

**32.** Richardson GA, Ryan C, Willford J, Day NL, Goldschmidt L. Prenatal alcohol and marijuana exposure: effects on neuropsychological outcomes at ten years. *Neurotoxicol Teratol* 2002;24:309-20.

**33.** Goldschmidt L, Day NL, Richardson GA. Effects of prenatal marijuana exposure on child behavior problems at age 10. *Neurotoxicol Teratol* 2000;22:325-36.

**34.** Day NL, Leech SL, Goldschmidt L. The effects of prenatal marijuana exposure on delinquent behaviors are mediated by measures of neurocognitive functioning. *Neurotoxicol Teratol* 2011;33:129-36.

**35.** Fried PA, Smith AM. A literature review of the consequences of prenatal marijuana exposure: an emerging theme of a deficiency in aspects of executive function. *Neurotoxicol Teratol* 2001;23:1-11.

**36.** Smith AM, Longo CA, Fried PA, Hogan MJ, Cameron I. Effects of prenatal marijuana on visuospatial working memory: an fMRI study in young adults. *Neurotoxicol Teratol* 2006;28:286-95.

**37.** ProCon.org. (March 14, 2016). 24 legal medical marijuana states and DC. Available at: <http://medicalmarijuana.procon.org/view.resource.php?resourceID=000881>. Accessed June 4, 2016.

**38.** Oregon Health Authority. Public Health Division, Medical Marijuana Dispensary Program. Information bulletin 2015-04. Sept. 10, 2015, page 3. Available at: [www.oregon.gov/oha/mmj/Bulletins/Informational%20Bulletin%202015-04%20Early%20Retail%20Sales.pdf](http://www.oregon.gov/oha/mmj/Bulletins/Informational%20Bulletin%202015-04%20Early%20Retail%20Sales.pdf).

**39.** Colorado Department of Health. Available at: [www.colorado.gov/pacific/sites/default/files/MJ\\_RMEP\\_Pregnancy-Breastfeeding-Clinical-Guidelines.pdf](http://www.colorado.gov/pacific/sites/default/files/MJ_RMEP_Pregnancy-Breastfeeding-Clinical-Guidelines.pdf). Accessed June 4, 2016.

**40.** American Medical Association. Marijuana use in pregnancy. Policy Statement, Nov. 16, 2015. Available at: [www.ama-assn.org](http://www.ama-assn.org). Accessed June 18, 2016.

# Misdiagnosis and Missed Diagnoses in Foster and Adopted Children With Prenatal Alcohol Exposure

Ira J. Chasnoff, MD, Anne M. Wells, PhD, Lauren King, MA

## abstract

**OBJECTIVE:** The purpose of this article is to assess the rate of misdiagnosis and missed diagnoses of fetal alcohol spectrum disorders (FASD) among a population of foster and adopted youth referred to a children's mental health center.

**METHODS:** Data were collected from a sample of 547 children who underwent a comprehensive multidisciplinary diagnostic evaluation. Utilizing current diagnostic criteria, children were diagnosed, as appropriate, with fetal alcohol syndrome, partial fetal alcohol syndrome, alcohol-related neurodevelopmental disorder, or alcohol-related birth defects. Changes in rates of alcohol exposure-related diagnoses and cooccurring mental health disorders pre- and postassessment were analyzed by using McNemar's test for dependent proportions.

**RESULTS:** Among 156 children and adolescents who met criteria for a diagnosis within the fetal alcohol spectrum, 125 had never been diagnosed as affected by prenatal alcohol exposure, a missed diagnosis rate of 80.1%. Of the 31 who had been recognized before referral as affected by prenatal alcohol exposure, 10 children's FASD diagnoses were changed within the spectrum, representing a misdiagnosis rate of 6.4%. The remaining 21 (13.5%) children's diagnoses stayed the same. There also were significant changes in the rate of mental health diagnosis, and learning disorders, communication disorders, and intellectual disability, objective signs of neurocognitive damage, were not recognized in a significant number of children with FASD.

**CONCLUSIONS:** Within this clinical sample, 86.5% of youth with FASD had never been previously diagnosed or had been misdiagnosed. These high rates of missed diagnoses and misdiagnosis have significant implications for intervention and therapeutic services.

**WHAT'S KNOWN ON THIS SUBJECT:** Researchers speculate that children with fetal alcohol spectrum disorders often are not recognized or diagnosed correctly.

**WHAT THIS STUDY ADDS:** This is the first study to assess the rate of missed diagnoses and misdiagnosis in foster and adopted children with fetal alcohol spectrum disorders.

*Children's Research Triangle, Chicago, Illinois*

Dr Chasnoff served as principle investigator and lead author for this study; Dr Wells served as the data analyst for this study and participated directly in writing this article; and Ms King was responsible for data management and participated directly in writing this article.

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Address correspondence to Ira J. Chasnoff, MD, Children's Research Triangle, 70 E. Lake St, Suite 1300, Chicago, IL 60601. E-mail: [ichasnoff@cr-triangle.org](mailto:ichasnoff@cr-triangle.org)

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In children with a history of prenatal alcohol exposure, the diagnosis of fetal alcohol syndrome (FAS) is based on 3 criteria: prenatal and/or postnatal growth retardation, central nervous system impairment, and characteristic facial dysmorphism.<sup>1-3</sup> However, neurodevelopmental deficits among children who have confirmed prenatal exposure to alcohol but who do not meet diagnostic criteria for FAS are much more common. Partial FAS<sup>4</sup> (pFAS) and alcohol-related neurodevelopmental disorder (ARND)<sup>4</sup> are the most common diagnoses, and alcohol-related birth defects (ARBDs)<sup>4</sup> is relatively rare.<sup>5</sup> In 2004, a group of federal agencies developed a consensus definition of a more comprehensive term, fetal alcohol spectrum disorders (FASD). The term FASD is “an umbrella term describing the range of effects that can occur in an individual whose mother drank during pregnancy.”<sup>3</sup>

Unfortunately, many children and adolescents with FASD go unrecognized and untreated<sup>6-8</sup>; this is due to multiple factors, including unknown maternal history of alcohol use during pregnancy,<sup>9,10</sup> lack of consistent facial dysmorphism and growth impairment across all diagnoses within the fetal alcohol spectrum,<sup>3,4,10,11</sup> and the high rate of cooccurring mental health disorders.<sup>12</sup> Within our clinic’s population at the Children’s Research Triangle (CRT), it was noted that large numbers of children with FASD had been incorrectly diagnosed before referral. The purpose of this article is to assess the rate of misdiagnosis and missed diagnoses of FASD among a population of children and adolescents referred to a children’s mental health center for assessment and treatment. We hypothesized that the majority of children with FASD referred to the center would not have been diagnosed with FASD at the time of referral and that, of those that were

identified as having an alcohol diagnosis within FASD, a significant number would have been inaccurately diagnosed.

## METHODS

The clinic at CRT is a mental health center specializing in the assessment and treatment of high-risk populations of children and adolescents, especially those in the child welfare system. CRT is not an “FAS clinic”; there are no screening criteria for referral to the clinic, but the most common reason for referral is behavioral problems. Almost all of the children are referred through the Illinois Department of Children and Family Services (DCFS), and all children are in the care of a pediatrician or other children’s primary health care provider, as required by DCFS. An average of 200 children per year undergo a comprehensive medical, mental health, and neurodevelopmental assessment at the center. Approximately 30% of the children evaluated each year receive a diagnosis within the fetal alcohol spectrum.

## Study Inclusion

All foster and adopted children 4 to 18 years of age who have undergone comprehensive evaluation for any reason at CRT were eligible for inclusion in the study. An office clerk and interns who had no knowledge of history or diagnosis of any of the children pulled a sample of 547 charts from ~3000 charts.

## Child Assessment

The initial evaluation for each child consisted of a full pediatric, neurologic, and dysmorphism examination conducted by 1 of 2 board-certified pediatricians with extensive experience in diagnosing, assessing, and treating children with FASD. Each child’s prenatal alcohol exposure (yes/no) was verified through documentation in the child’s birth, medical, child welfare, and/or

adoption records. In addition, maternal use of tobacco and illicit drugs as documented through maternal admission of use or positive toxicology for the mother or newborn was recorded. Information regarding dosage and frequency of maternal alcohol, tobacco, and/or illicit drug use was not available for most children.

Before 2003, the pediatricians assessed the child’s facial features based on published dysmorphic abnormalities consistent with FAS.<sup>1,3</sup> After 2003, a digital facial photograph of each child was taken following the guidelines established by Astley and Clarren,<sup>13</sup> and measurements of palpebral fissure length and intercanthal distance were calculated via the photograph by using the recommended formulae. The philtrum and lip Ranks (Ranks 1 through 5) were assigned by the pediatrician during the examination based on the established grading system<sup>13</sup> and were confirmed through computer-generated upper lip circularity calculations. After the medical examination, the child and family underwent a clinical interview with a licensed psychologist, and the child was evaluated under the direction of a doctoral level psychologist utilizing instruments that assess child psychological and neurodevelopmental functioning across several domains.

## Measures

The neurodevelopmental battery with which each child was evaluated included age appropriate instruments and approaches that assessed neurocognitive functioning, including general intelligence, memory, executive functioning, and speech and language; academic achievement; self regulation, including sensory processing, social skills, and behavior; and adaptive behaviors.<sup>14-16</sup>

## Diagnostic Assignment

Based upon the completed comprehensive evaluation, children were assigned an alcohol exposure-

related diagnosis that is consistent with the University of Washington's 4-digit code system<sup>10</sup> for diagnosis:

- Growth retardation: current or past weight and/or height less than third percentile adjusted for age and gender. We tightened the growth criteria from 10th percentile, as recommended in the Centers for Disease Control and Prevention diagnostic and referral guidelines,<sup>3</sup> to third percentile, as recommended by Astley,<sup>10</sup> because of the high rates of diagnostic misclassification.<sup>2,17,18</sup> Recent studies have documented that use of third percentile for growth assessment neurodevelopmentally differentiates children within the fetal alcohol spectrum.<sup>10,15</sup> Also, growth criteria below third percentile align more closely with the original definition of FAS.<sup>1</sup>
- Facial dysmorphism: abnormal measurements of the upper lip (rank 4 or 5) and the philtrum (rank 4 or 5) and shortened palpebral fissures based on direct measurement or, after 2003, according to analysis of facial features utilizing the Lip-Philtrum Guide and digital facial photograph based on the criteria of Astley and Clarren.<sup>13,19</sup>
- Central nervous system abnormalities: demonstration of structural, neurologic, or functional central nervous system deficits<sup>20</sup> as documented by the presence of microcephaly (current head circumference below third percentile for age and gender) and/or functional deficits demonstrated as global cognitive delays with performance below the third percentile on standardized testing or 3 or more domains of neurodevelopmental functioning more than 2 SDs below the normed mean on standardized measures of neurocognitive, self-regulatory, or adaptive functioning.

Based on these standards, children who met all physical criteria for

growth impairment and facial dysmorphism as well as neurodevelopmental deficits were assigned a diagnosis of FAS. Children with confirmed prenatal alcohol exposure, facial dysmorphism, and neurodevelopmental deficits but with normal growth (height and weight) patterns were diagnosed as pFAS. Children with confirmed exposure, normal growth and neurodevelopmental functioning but major structural abnormalities were diagnosed as ARBDs. Children who had confirmed exposure and met criteria for neurodevelopmental deficits but did not meet criteria for facial dysmorphism and/or growth were classified as ARND. This approach is consistent with the newly published *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* mental health criteria for neurobehavioral disorder with prenatal alcohol exposure (ND-PAE),<sup>21</sup> which is the terminology that will replace the term ARND.

#### Data Analytic Approach

Frequency tables and case summaries were generated for each of the hypothesized questions by using SPSS 21.0 (IBM SPSS Statistics, IBM Corporation). Pre- and postassessment proportions of diagnoses were analyzed by using McNemar's test for dependent proportions, again using SPSS 21.0. This statistical test is used on paired nominal data, which are contained in a 2×2 table in studies that have a before and after component.<sup>22</sup>

The Institutional Review Board of DCFS and the Western Institutional Review Board approved all procedures for this archival study.

## RESULTS

### Descriptive Data

All children in the sample were in a foster or adoptive home at the time of referral and evaluation. Among the

547 children included in the study, the mean age was 9.36 years (SD = 3.93), and 63.8% were boys. Racial (50.6% African American, 1.3% Asian, 32.2% white, 0.7% Native American, 12.2% biracial, 0.2% other, 2.8% unknown) and ethnic distribution (11.7% Hispanic, 82.8% non-Hispanic, 5.9% unknown) reflected the general distribution of children under DCFS supervision.

### Referral Diagnoses

By far, the most common reason for referral of the 547 children to the CRT clinic was "behavioral problems." At referral, diagnoses related to prenatal alcohol exposure were relatively rare, with 36 children (6.6%) being referred with a diagnosis of FAS and 15 children (2.7%) being referred with a diagnosis of ARND. The most common mental health diagnosis at the time of referral (Table 1) was attention-deficit/hyperactivity disorder (ADHD), followed by posttraumatic stress disorder, conduct disorder, oppositional defiant disorder, and reactive attachment disorder.

### Diagnoses After Evaluation

After the comprehensive multidisciplinary evaluation at CRT and using the diagnostic criteria presented in the Methods section, 156 children (28.5%) met criteria for a diagnosis within FASD: 93 with FAS, 1 with pFAS, 61 with ARND, and 1 with ARBD. In this way, the sample was representative of our overall clinic population in that ~30% of the children evaluated at CRT meet criteria for a diagnosis within FASD. The subset of children with FASD was similar as to mean age and distribution by gender and race/ethnicity to the referral sample of children. Mental health diagnoses assigned after comprehensive assessment changed significantly for the 156 children with FASD (Table 2) and demonstrated a wide range of disorders.



**TABLE 1** Changes in Mental Health Diagnoses of Children With FASD After Assessment (*N* = 156)

Diagnosis	Referral Diagnosis		Postassessment Diagnosis		<i>P</i>	Effect Size Cramer's $\nu$
	<i>N</i>	%	<i>N</i>	%		
	ADHD	42	26.4	88		
Adjustment disorder	3	1.9	20	12.6	.001	0.05
Anxiety disorder	3	1.9	15	9.4	.004	0.11
Autism/pervasive developmental disorder	0	0.0	8	5.0	—	
Bipolar Cyc	3	1.9	3	1.9	.999	0.32
Bipolar NOS	6	3.7	3	1.9	.453	0.22
Communication disorder	6	3.7	20	12.5	.007	0.02
Depression NOS	5	3.2	12	7.7	.065	0.36
Developmental disorder	2	1.3	8	5.0	.109	0.03
Developmental delay	8	5.0	5	3.1	.508	0.29
Learning disability	7	4.4	23	14.5	.002	0.08
Mental retardation	7	4.4	24	15	<.001	0.34
Mood disorder NOS	3	1.9	6	3.8	.508	0.03
Oppositional defiant disorder	8	5.0	4	2.5	.344	0.15
Psychotic disorder	2	1.3	5	3.1	.375	0.30
Posttraumatic stress disorder	10	6.3	26	16.3	.001	0.38
Reactive attachment disorder	9	5.6	14	8.8	.383	0.02
Sensory integration disorder	6	3.8	25	16.0	<.001	0.28
Sleep disorder	1	0.6	7	4.4	.031	0.37
Other diagnosis	7	4.4	20	12.6	.011	0.10

Em dash denotes the following: Because there were no children with this diagnosis at referral, McNemar's test could not be calculated. NOS, not otherwise specified.

### Changes in Diagnosis Pre- and Postassessment

Among the 547 children, 51 were referred with a diagnosis within the fetal alcohol spectrum; of these children, 20 did not meet criteria for any diagnosis within FASD, and 31 children retained an alcohol exposure-related diagnosis, a significant reduction ( $P < .001$ , Cramer's  $\nu = 0.73$ ) in the number of children with a diagnosis related to FASD. Specific diagnoses within FASD also demonstrated a high rate of error. Of the 36 children who had been diagnosed with FAS, only 16 of these children met criteria for FAS

after their full assessment, 7 met criteria for ARND, and 13 qualified for no alcohol-related diagnosis. Of 15 children with a referral diagnosis of ARND, 5 retained a diagnosis of ARND after comprehensive assessment, 2 received a diagnosis of FAS, and 8 received no alcohol-related diagnoses.

Of the 156 children who after full assessment received a diagnosis within FASD, only 31 (19.9%) had been referred to the clinic with a diagnosis related to prenatal alcohol exposure; 80.1% of children with FASD had not been recognized.

After comprehensive assessment by the multidisciplinary clinical team,

there was a significant change in mental health diagnoses for the 156 children with FASD (Table 1). Of note, learning disorders, communication disorders, and intellectual disability, objective signs of significant neurocognitive damage, had not been recognized in a large majority of the children with these disabilities. Among the 156 children with confirmed FASD, 147 (94.2%) received a cooccurring mental health diagnosis, with 104 (66.7%) having 2 or more mental health diagnoses in addition to the alcohol exposure-related diagnosis.

### Changes in Treatment Pre- and Postassessment

The multidisciplinary assessment led to a significant change in therapeutic approaches for the 156 children with FASD (Table 2). After the evaluation, significantly fewer children required the developmental therapy, physical therapy, and speech/language therapy that they had been receiving and instead needed services, especially family therapy, sensory integration treatment, and psychotherapy, that they previously had not been receiving (Table 2). In addition to the therapeutic modalities presented in Table 2, after assessment, a number of children with FASD required further medical interventions; 27 children (17.3%) required extensive dental work and 8 (5.1%) children needed an ophthalmology evaluation. Also, attachment therapy was recommended for 33 children (21.2%), and educational services were recommended for 109 (69.9%) of the children with FASD.

Recommendations for medication use also changed from the time of referral to the time after diagnosis of FASD. At the time of referral, 11 of the 156 children and youth were on stimulant medications to treat ADHD. After assessment, stimulant medications were recommended to only 1 of these individuals. Twenty-two other children and youth with FASD who

**TABLE 2** Changes in Therapeutic Services for Children With FASD After Assessment (*N* = 156)

Therapeutic Service	Referral		Postassessment		<i>P</i>	Cramer's $\nu$
	<i>N</i>	%	<i>N</i>	%		
	Occupational therapy	40	25.7	2		
Physical therapy	19	11.9	4	2.6	<.001	0.06
Developmental therapy	19	11.9	0	0	—	
Speech/language	45	28.8	16	10.2	<.001	0.09
Family therapy	15	9.5	25	15.7	.002	0.13
Sensory integration	18	11.3	76	48.7	<.001	0.21
Psychotherapy	54	34.6	42	26.9	.008	0.02

Em dash denotes the following: Because there were no children receiving this therapy post assessment, McNemar's test could not be calculated.

had not presented on medications were prescribed stimulant medications for ADHD after assessment.

Of the 156 children and youth with FASD, before referral 8 had been prescribed psychotropic medications. Of those 8 youth, 1 child's prescription stayed the same and 7 children were taken off psychotropic medication. Six additional youth with FASD were placed on psychotropic medications. Eighteen of the youth came into the clinic with other medication prescriptions, of which 14 were taken off their medication and 4 maintained their medication. Eleven additional youth were placed on new medication.

## DISCUSSION

Within this clinical sample, the higher number of children with FAS as opposed to pFAS or ARND most likely is due to the fact that foster and adopted children with severe behavioral disorders frequently are referred to CRT's clinic. However, even with this severity of behavioral problems, 86.5% of children and adolescents with FASD had never been previously diagnosed or had been misdiagnosed. The majority of these youth (80.1%) had a missed diagnosis, whereas the remaining 6.4% of youth had a misdiagnosis (ie, their diagnosis within the FASD spectrum was changed). These findings suggest that FASD frequently go unrecognized; thus, education is most needed in overall awareness of FASD, with additional emphasis on differential diagnosis within the spectrum. This is especially pertinent as ND-PAE replaces ARND as a diagnostic term. In the current study, all 155 children diagnosed as having FAS, pFAS, and ARND met newly published criteria for ND-PAE.<sup>21</sup>

There are several barriers to early recognition and accurate diagnosis of children and adolescents with FASD. The frequent lack of clear

physical findings in children affected by alcohol exposure,<sup>3,5,10,11</sup> the historically confusing language and diagnostic terminology applied to alcohol-affected children,<sup>23</sup> and the perceived stigma against addressing alcohol use by pregnant women<sup>24</sup> most likely contributed to the majority of affected children and adolescents in the current study having been misdiagnosed or missed completely. A survey of American Academy of Pediatrics members indicated that only 50% of respondents felt prepared to make a diagnosis within the fetal alcohol spectrum.<sup>24</sup> Further, children's health providers do not routinely consider prenatal alcohol exposure in the differential diagnosis of behavioral and learning problems.<sup>25</sup>

In the current study, ADHD was the most common referral diagnosis for children who ultimately were diagnosed with FASD. Previous studies have demonstrated that anywhere from 40% to 75% of children with FASD are diagnosed with ADHD.<sup>15,26,27</sup> However, there are qualitative differences in the types of attention problems seen in children with FASD as compared with children with ADHD.<sup>28-30</sup> Children with ADHD and FASD have been shown to have greater deficits in verbal comprehension and perceptual reasoning than children having ADHD without prenatal alcohol exposure.<sup>28</sup> Another study of children with FASD compared with children with ADHD revealed that children with FASD were more likely than the ADHD group to engage in sociopathic behaviors, such as lying and stealing.<sup>29</sup> Greenbaum et al<sup>30</sup> found that children with FASD demonstrate a behavioral profile distinct from children with ADHD, especially related to difficulties in social cognition and emotion processing. These differences and the lack of recognition of FASD have significant implications for the pharmacologic

and therapeutic approach to treating the child, since studies have demonstrated differential response to medication for children with FASD.<sup>31,32</sup>

The cooccurring mental health disorders in individuals affected by prenatal alcohol exposure have implications for therapy.<sup>33,34</sup> In the current study, the majority of children and youth ultimately diagnosed with FASD required significant alteration of therapeutic services. The therapies most commonly delivered by early intervention and school systems (developmental, speech/language, occupational, and physical therapies) were the therapies the children with FASD were most frequently receiving. However, upon receiving a comprehensive evaluation, significant numbers of the children and youth did not need these therapies but required more intense forms of mental health therapy addressing attachment difficulties, behavioral difficulties, and sensory processing deficits, as well as the need for the child and family to participate in some form of psychotherapy. In addition, the need for specialized educational services in the school and dental care often had been overlooked for the children who ultimately were diagnosed with FASD.

This study has limitations in that the target population consists of a foster or adopted population referred for a behavioral or mental health assessment. Thus, conclusions may not be generalized to the general population of children with FASD. In addition, it is important to note that some of the mental health diagnoses and the recommended changes to therapeutic approaches may have been due to the child's aging and corresponding evolution of symptoms. Further, children's varying presentations at the assessment session, incomplete access to records, changes in reporting by parents and other auxiliary informants, and foundational differences in training

among professionals (psychologists, psychiatrists, and pediatricians) may also influence the interpretation of diagnostic criteria. However, issues such as intellectual disabilities, communication disorders, and learning disorders (problems that are relatively common among individuals with FASD) do not change over time, are less subjective than many of the mental health diagnoses, and provide concrete evidence of neurologic damage, but were not recognized in a number of the children.

Once children and adolescents with FASD are recognized, there must be an immediate effort to obtain diagnostic and therapeutic services. Early diagnosis, especially before the age of 6 years, coupled with earliest intervention is 1 of the strongest correlates with an improved outcome for the child long-term.<sup>35</sup> Delayed or incorrect diagnosis, especially among children who do not have the sentinel facial dysmorphology associated with FAS, may lead to a higher incidence of secondary disabilities<sup>36</sup> and greater need for special education services.<sup>37</sup> The role of the pediatrician and other children's health care providers is clear: early recognition of the child or adolescent with FASD, referral to a provider who can conduct a full evaluation, and participation in the development of a targeted treatment plan that incorporates mental health treatment, behavioral management strategies, and special education services.

## CONCLUSIONS

Although FASD have long been recognized as a leading cause of intellectual disabilities, behavior problems, learning disabilities, and cooccurring mental health disorders,<sup>1,2,5,9,12-15,20,31,32</sup> children and adolescents who have been affected by prenatal alcohol exposure often go undiagnosed or are misdiagnosed. Pediatricians and other children's health care providers

have the opportunity to screen children and youth in their practices for FASD and ensure that affected individuals receive the targeted range of services they may need.

## REFERENCES

1. Jones KL, Smith DW. Recognition of the fetal alcohol syndrome in early infancy. *Lancet*. 1973;302(7836):999-1001
2. Stratton K, Howe C, Battaglia F. *Fetal Alcohol Syndrome: Diagnosis, Epidemiology, Prevention, and Treatment*. Washington, DC: National Academy Press; Institute of Medicine; 1996
3. Bertrand J, Floyd LL, Weber MK; Fetal Alcohol Syndrome Prevention Team, Division of Birth Defects and Developmental Disabilities, National Center on Birth Defects and Developmental Disabilities, Centers for Disease Control and Prevention (CDC). Guidelines for identifying and referring persons with fetal alcohol syndrome. *MMWR Recomm Rep*. 2005;54(RR-11):1-14
4. Institute of Medicine. Stratton KR, Howe CJ, Battaglia FC, et al. Diagnosis and Clinical Evaluation of Fetal Alcohol Syndrome. In: Stratton KR, Howe CJ, Battaglia FC, eds. *Fetal Alcohol Syndrome: Diagnosis, Epidemiology, Prevention and Treatment*. Washington, DC: National Academy Press; 1996:63-80
5. May PA, Gossage JP, Kalberg WO, et al. Prevalence and epidemiologic characteristics of FASD from various research methods with an emphasis on recent in-school studies. *Dev Disabil Res Rev*. 2009;15(3):176-192
6. O'Connor M, McCracken MD, Best A. Under recognition of prenatal alcohol exposure in a child inpatient psychiatric setting. *Ment Health Aspects Dev Disabil*. 2006;9:105-109
7. Stoler JM, Holmes LB. Under-recognition of prenatal alcohol effects in infants of known alcohol abusing women. *J Pediatr*. 1999;135(4):430-436
8. Elias E. *Improving Awareness and Treatment of Children With Fetal Alcohol Spectrum Disorders and Co-occurring Psychiatric Disorders*. Washington, DC: The Disability Service Center; 2013
9. Benz J, Rasmussen C, Andrew G. Diagnosing fetal alcohol spectrum disorder: history, challenges and future directions. *Paediatr Child Health (Oxford)*. 2009;14(4):231-237
10. Astley SJ. Comparison of the 4-digit diagnostic code and the Hoyme diagnostic guidelines for fetal alcohol spectrum disorders. *Pediatrics*. 2006; 118(4):1532-1545
11. Sampson PD, Streissguth AP, Bookstein FL, Barr HM. On categorizations in analyses of alcohol teratogenesis. *Environ Health Perspect*. 2000;108(suppl 3):421-428
12. O'Connor MJ, Paley B. Psychiatric conditions associated with prenatal alcohol exposure. *Dev Disabil Res Rev*. 2009;15(3):225-234
13. Astley SJ, Clarren SK. Measuring the facial phenotype of individuals with prenatal alcohol exposure: correlations with brain dysfunction. *Alcohol Alcohol*. 2001;36(2):147-159
14. Astley SJ, Olson HC, Kerns K, et al. Neuropsychological and behavioral outcomes from a comprehensive magnetic resonance study of children with fetal alcohol spectrum disorders. *Can J Clin Pharmacol*. 2009;16(1):e178-e201
15. Chasnoff IJ, Wells AM, Telford E, Schmidt C, Messer G. Neurodevelopmental functioning in children with FAS, pFAS, and ARND. *J Dev Behav Pediatr*. 2010; 31(3):192-201
16. Kodituwakku PW. Defining the behavioral phenotype in children with fetal alcohol spectrum disorders: a review. *Neurosci Biobehav Rev*. 2007;31(2):192-201
17. Aase JM. Clinical recognition of FAS: difficulties of detection and diagnosis. *Alcohol Health Res World*. 1994;18:5-9
18. Abdelmalik N, van Haelst M, Mancini G, et al. Diagnostic outcomes of 27 children referred by pediatricians to a genetics clinic in the Netherlands with suspicion of fetal alcohol spectrum disorders. *Am J Med Genet A*. 2013;161A(2):254-260
19. Astley SJ, Clarren SK. Diagnosing the full spectrum of fetal alcohol-exposed individuals: introducing the 4-digit diagnostic code. *Alcohol Alcohol*. 2000; 35(4):400-410
20. National Task Force on Birth Defects and Developmental Disabilities, Centers for Disease Control and Prevention. *Fetal Alcohol Syndrome: Guidelines for Referral and Diagnosis*. Washington, DC: US Department of Health and Human Services; 2004

21. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. Arlington, VA: American Psychiatric Association; 2013
22. Agresti A. *An Introduction to Categorical Data Analysis*. 2nd ed. Hoboken, NJ: John Wiley & Sons, Inc; 2007
23. Peadon E, Fremantle E, Bower C, Elliott EJ. International survey of diagnostic services for children with fetal alcohol spectrum disorders. *BMC Pediatr*. 2008; 8:12
24. Elliott EJ, Payne J, Haan E, Bower C. Diagnosis of foetal alcohol syndrome and alcohol use in pregnancy: a survey of paediatricians' knowledge, attitudes and practice. *J Paediatr Child Health*. 2006;42(11):698–703
25. Gahagan S, Sharpe TT, Brimacombe M, et al. Pediatricians' knowledge, training, and experience in the care of children with fetal alcohol syndrome. *Pediatrics*. 2006;118(3). Available at: [www.pediatrics.org/cgi/content/full/118/3/e657](http://www.pediatrics.org/cgi/content/full/118/3/e657)
26. Fryer SL, McGee CL, Matt GE, Riley EP, Mattson SN. Evaluation of psychopathological conditions in children with heavy prenatal alcohol exposure. *Pediatrics*. 2007;119(3). Available at: [www.pediatrics.org/cgi/content/full/119/3/e733](http://www.pediatrics.org/cgi/content/full/119/3/e733)
27. Rasmussen C, Benz J, Pei J, et al. The impact of an ADHD co-morbidity on the diagnosis of FASD. *Can J Clin Pharmacol*. 2010;17(1):e165–e176
28. Glass L, Ware AL, Crocker N, et al; Collaborative Initiative on Fetal Alcohol Spectrum Disorders (CIFASD). Neuropsychological deficits associated with heavy prenatal alcohol exposure are not exacerbated by ADHD. *Neuropsychology*. 2013;27(6):713–724
29. Nash K, Rovet J, Greenbaum R, Fantus E, Nulman I, Koren G. Identifying the behavioural phenotype in fetal alcohol spectrum disorder: sensitivity, specificity and screening potential. *Arch Women Ment Health*. 2006;9(4):181–186
30. Greenbaum RL, Stevens SA, Nash K, Koren G, Rovet J. Social cognitive and emotion processing abilities of children with fetal alcohol spectrum disorders: a comparison with attention deficit hyperactivity disorder. *Alcohol Clin Exp Res*. 2009;33(10):1656–1670
31. Coles CD, Platzman KA, Raskind-Hood CL, Brown RT, Falek A, Smith IE. A comparison of children affected by prenatal alcohol exposure and attention deficit, hyperactivity disorder. *Alcohol Clin Exp Res*. 1997;21(1):150–161
32. Oesterheld JR, Kofoed L, Tervo R, Fogas B, Wilson A, Fiechtner H. Effectiveness of methylphenidate in Native American children with fetal alcohol syndrome and attention deficit/hyperactivity disorder: a controlled pilot study. *J Child Adolesc Psychopharmacol*. 1998;8(1):39–48
33. Kodituwakku PW, Kalberg W, May PA. The effects of prenatal alcohol exposure on executive functioning. *Alcohol Res Health*. 2001;25(3):192–198
34. Steinhausen HC, Willms J, Spohr HL. Long-term psychopathological and cognitive outcome of children with fetal alcohol syndrome. *J Am Acad Child Adolesc Psychiatry*. 1993;32(5):990–994
35. Streissguth AP, Bookstein FL, Barr HM, Sampson PD, O'Malley K, Young JK. Risk factors for adverse life outcomes in fetal alcohol syndrome and fetal alcohol effects. *J Dev Behav Pediatr*. 2004;25(4): 228–238
36. Streissguth AP, Barr HM, Kogan J, Bookstein FL. *Understanding the occurrence of secondary disabilities in clients with fetal alcohol syndrome (FAS) and fetal alcohol effects (FAE). Final report to the Centers for Disease Control and Prevention*. Seattle, WA: University of Washington, Fetal Alcohol and Drug Unit; 1996
37. Autti-Rämö I. Twelve-year follow-up of children exposed to alcohol in utero. *Dev Med Child Neurol*. 2000;42(6):406–411



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