LSHSS

## Clinical Forum

# Neurobiology and Neurodevelopmental Impact of Childhood Traumatic Stress and Prenatal Alcohol Exposure

**Jim Henry**
**Mark Sloane**
**Connie Black-Pond**
Western Michigan University, Kalamazoo

Over the past 10 years, research on the developmental impact of prenatal alcohol exposure and childhood traumatic experience has rapidly increased due to heightened awareness of their pernicious effects on the developmental process. Yet, no published research exists on the compounding influence of coexisting prenatal alcohol exposure and postnatal trauma. The current study sought to analyze the impact of prenatal alcohol exposure and postnatal trauma compared with trauma alone on specific neurodevelopmental domains including language, memory, motor functions, visual processing, attention, and behavior.

Both prenatal alcohol exposure and postnatal trauma impact two core developmental processes—neurophysiological growth of the brain, nervous system, and endocrine system, along with psychosocial development, including personality formation, social conduct, and capacity for relationships (Putnam, 2006; Riley & McGee, 2005; Streissguth, 1997a). Developmental domains affected include language, social cognition and communication, cognition, impulse control, memory, attention, and executive functioning (Institute of Medicine [IOM], 1996; O'Malley, 2001; Pearce & Pezzot-Pearce, 1997; Perry, 2006). Differences in the behavioral and emotional impact of prenatal alcohol exposure and postnatal trauma

ABSTRACT: **Purpose:** Research reveals that prenatal alcohol exposure and child trauma (i.e., abuse, neglect, sexual abuse) can have deleterious effects on child development across multiple domains. This study analyzed the impact on childhood neurodevelopment of prenatal alcohol exposure and postnatal traumatic experience compared to postnatal traumatic experience alone. Although the harmful effects of both have been well documented individually, there is no research documenting the concurrent effects of prenatal alcohol exposure and postnatal trauma on a child's developmental process.
**Method:** Transdisciplinary assessment of the children included the core disciplines of medicine, speech-language pathology, occupational therapy, social work, and psychology. Medical examination, standardized developmental and intelligence testing, projective tools, parent questionnaires, and psychosocial interviews provided information in the primary developmental areas.
**Results:** Findings indicated that children who had been exposed prenatally to alcohol along with postnatal traumatic experience had lower intelligence scores and more severe neurodevelopmental deficits in language, memory, visual processing, motor skills, and attention than did traumatized children without prenatal alcohol exposure, as well as greater oppositional/defiant behavior, inattention, hyperactivity, impulsivity, and social problems.
**Clinical Implications:** Successful teacher and speech-language pathologist interventions with traumatized children with prenatal alcohol exposure demand a paradigm shift that requires the development of new perspectives and ongoing training.

KEY WORDS: child trauma, developmental delays, language delays, prenatal alcohol exposure


0161-1461/07/3802-0099

are often clinically indistinguishable. However, examining the effects separately aids in unraveling the complexity when both conditions occur simultaneously.

## CHILDHOOD TRAUMATIC EXPERIENCE

Research on child development strongly suggests that children who experience multiple chronic traumatic events, including abuse, neglect, and sexual abuse, often develop relational disturbances, deficits with language and cognition, dysregulation of mood and behavior, and social/emotional disturbances (Cook, Blaustein, Spinazzola, & van der Kolk, 2003; Kaufman, Plotsky, Nemeroff, & Charney, 2000; Putnam, 2006). The experience of trauma physiologically affects core regulatory systems so that the modulation and processing of sensory experience is compromised, increasing sensitivity to or need for sensory stimuli and preventing integration of sensory experience (Glod, Teicher, Hartman, Harakal, & McGreenery, 1997; Perry, 1997, 2006; Schore, 2001). Changes in the hypothalamic-pituitary-adrenal axis (HPA axis), which is the body's critical stress response system, prevent modulation of the resulting frustration that accompanies sensory dysregulation, rendering a child incapable of self-regulation of affective states and self-control over behavior (Putnam, 2006).

Speech-language pathologists (SLPs) have considerable exposure to traumatized children and the complexity of their neurodevelopmental, social/emotional, and behavioral impairments. A recent study noted that preschool children who had been exposed to traumatizing violence were more than seven times more likely than children who had not been exposed to be referred for speech-language pathology services (Kernic et al., 2002). Traumatized children often have delays with grammar and vocabulary comprehension and production; conversational skills; receptive and expressive syntactic skills; and semantic skills, including difficulties with multiple word and sentence meanings (Hyter, Henry, Atchison, Sloane, & Black-Pond, 2003). Impairments with social communication skills are also common, including those necessary to exchange information; initiate and develop social relationships; cope with changing environmental demands; and assert one's needs, desires, and preferences (Coggins, Friet, Morgan, & Wikstrom, 1998; Walker, Schwarz, Nippold, Irvin, & Noell, 1994). Katz (1992) reviewed research that was conducted from 1975 to 1992, finding that both physically abused children and neglected children evidenced language delays and disorders, with those of neglected children being more severe.

Delays with the development of language functions, behavioral control, and emotional regulation translate into poor academic and social experiences (Cohn, Miller, & Tickle-Degnen, 2000; Dunn, 2001; Dunn & Westman, 1997; Miller, Reisman, McIntosh, & Simon, 2001; Parham & Mailloux, 1995). Behaviorally and emotionally, the experience of trauma increases a person's vulnerability to stressors, including severe reactivity to mild stressors (Gunnar & Donzella, 2002). The capacity to problem solve may disintegrate, resulting in disorganized states, extreme helplessness, confusion, withdrawal, or rage (Crittenden, 1998; Kagan, 2004; Teicher, Andersen, Polcari, Anderson, & Navalta, 2002), Experience of trauma may manifest as overcompliance and resistance to change (Crittenden & DiLalla, 1988) or aggression and oppositional defiant disorder (Ford, Taylor, & Warner-Rogers, 2000), all of which further reduce the potential for positive social and academic outcomes.

## FETAL ALCOHOL EXPOSURE

The term fetal alcohol spectrum disorder (FASD) is a recent addition to diagnostic classification that refers to a wide range of conditions associated with prenatal alcohol exposure. Included under the umbrella term FASD are the full fetal alcohol syndrome (FAS); the "partial syndrome," previously called fetal alcohol effects (FAE) but now referred to as alcohol-related neurodevelopmental disorder (ARND; Bertrand et al., 2004); and a new, somewhat controversial category sometimes labeled prenatal exposure to alcohol (PEA), in which children with significant prenatal alcohol exposure have no facial evidence of FAS, presumably due to alcohol abstinence during the time that facial features are forming embryologically (Mattson, Riley, Gramling, Delis, & Jones, 1997).

Although facial features associated with FAS are familiar to most clinicians, research indicates that the level of dysmorphology and the level of neuropsychiatric impairment are not necessarily related (Riley, McGee, & Sowell, 2004; Riley & McGee, 2005). Because children with ARND and PEA often lack the physical features that are used to diagnose FAS, clinicians may minimize or even dismiss the role of fetal alcohol exposure in these children's development. Consequently, children with ARND and PEA are at risk of not receiving interventions that adequately address their level of brain impairment (O'Malley, 2000; Sampson, Streissguth, Bookstein, & Barr, 2000; Streissguth, 1997b).

Secondary disabilities, such as disruptions in education, substance abuse, unemployment, problems parenting, homelessness, victimization, mental health crises, legal problems, and premature death (Streissguth et al., 2004), are a result of a combination of organic brain deficits and additional environmental stressors that are commonly associated with FASD (Streissguth & O'Malley, 2000). Research suggests that these secondary disabilities often become more impairing as the individual ages (Streissguth, 1992), and have far-reaching implications, including the tendency for FASD females, who often have limited capacity to parent, to themselves give birth to prenatally exposed children (Streissguth & O'Malley, 2000). Experts agree that early diagnosis of and consistent treatment for FAS/ARND is crucial for minimization and/or prevention of later life crises (Streissguth, 1992, 1997a; Streissguth & O'Malley, 2000).

FAS and ARND appear to have a behavioral phenotype for children (Streissguth, 1997a), but there are also important differences among individual children. Additionally, a child may vary greatly in his or her repertoire of competencies and delays (Malbin, 1993). Clinicians who work with these children must be aware that fetal alcohol exposure can affect central nervous system (CNS) development in a number of different ways that can ultimately manifest in a wide variety of cognitive and behavioral challenges for the child with FAS/ARND (Malbin, 1993; Riley et al., 2004; Streissguth & O'Malley, 2000). Despite the dearth of empirical knowledge on effective treatment strategies for FASD, many experts concur that children with FAS/ARND are best served by multidisciplinary treatment that considers overall development (Riley & McGee, 2005; Weiner & Morse, 1994).

## ISSUES IN ASSESSMENT

Recent advances in neurobiology (including neuroimaging) have greatly impacted the assessment and treatment of children and

adolescents who have been traumatized and prenatally exposed to alcohol. The evidence base has increased exponentially in the past 10 years and has provided many mechanisms to explain problematic behavior in this population. The CNS regions commonly involved affect three main domains: attachment, affect regulation, and information processing. Detailed explanation of exact brain mechanisms is well beyond the scope of this review, but a brief summary of the involved brain structures is provided in Table 1.

Often overlooked is the striking similarity between the CNS (reflected in various neuroimaging studies) abnormalities of traumatized children (Bremner, Southwick, & Charney, 1999; DeBellis & Van Dillen, 2005; Teicher et al., 2002) and those of children with FASD (Riley et al., 2004), as well as the behavioral phenotypes of these two groups (DeBellis & Van Dillen, 2005; Streissguth, 1997a). This apparent overlap has not yet been addressed in the medical or behavioral science literature, but further exploration is critically important. Research contamination, that is, researchers from both groups inadvertently studying the same clinical population, is one possible explanation for these similarities in CNS abnormalities and clinical presentation.

An important, yet often overlooked, factor in the assessment and management of traumatized and prenatally exposed children with severe behavioral problems involves the neurogenetic contribution from both biological parents. Many inheritable conditions with profound behavioral implications in children and adolescents (e.g., attention deficit hyperactivity disorder, bipolar disorder, major depression, and anxiety disorders) are frequently reported in biological parents whose children are placed in foster care. These and other mental health disorders are also extremely common in adults who have significant alcohol and substance use/abuse. Therefore, any discussion regarding problematic behavior in children and adolescents who have been traumatized and exposed to alcohol must include these genetic factors. This genetic component should also be an essential consideration when performing assessments and formulating treatment plans for these afflicted children.

Both prenatal alcohol exposure and postnatal trauma have pernicious effects on children's neurophysiology and psychosocial development, but no published research has existed on the compounding influence of coexisting prenatal alcohol exposure and postnatal trauma. The current study sought to analyze the impact of prenatal alcohol exposure and postnatal trauma compared with trauma alone on specific neurodevelopmental domains.

## METHOD

### Sample

Referrals for this study came primarily from the child welfare systems within a 13-county area in southwestern Michigan. This region includes two large urban areas with the majority of the counties being rural. Referral sources were primarily state or private

**Table 1.** Central nervous system brain structures that are affected by trauma and prenatal alcohol exposure.

| *Region of brain* | *Purpose* |
|---|---|
| Attachment | |
| Neurotransmitters | Oxytocin, vasopressin, estrogen, norepinephrine, dopamine, and others – chemical messengers that allow different brain structures to communicate |
| Hypothalamic-pituitary-adrenal (HPA) axis | Multi-organ network that allows the organism to respond swiftly and proficiently to perceived threat (fight/flight/freeze response) |
| Amygdala | Primary role in threat detection—initiates the Fight/flight/freeze response and is extensively connected to many other brain structures |
| Hippocampus | Involved in new memory acquisition and learning |
| Corpus callosum | Critical connecting structure between two cerebral hemispheres; involved in emotional regulation and smoothly integrating the two halves of the brain |
| Fusiform face area (FFA) | Necessary for facial recognition; critical for infant recognition of caregiver |
| Affect regulation – control of emotions | |
| Locus ceruleus | Vital area in brainstem (pons) involved in alertness and arousal |
| Thalamus | Central relay station in the middle of the brain where sensory input is screened and distributed to other parts of the brain |
| Corpus callosum | (See above) |
| Striatum, nucleus accumbens | Reward center of the brain |
| Orbitofrontal cortex | Regulates emotion, social behavior, and conscious decision making |
| Information processing | |
| Amygdala and hippocampus | Involved in new memory formation |
| Anterior cingulate | Associated with conflict monitoring, resolution, and executive function |
| Orbitofrontal cortex | Essential for conscious decision making |

social service agencies serving children with significant histories of traumatic experience.

Children 6 to 16 years of age ($N$ = 274) were included in this retrospective study. Descriptive statistics for the sample are provided in Table 2. This age range corresponds with the the Pediatric Early Elementary Examination (PEEX 2; Levine, 1996a) and the Pediatric Examination of Educational Readiness at Middle Childhood (PEERAMID 2; Levine, 1996b) neurodevelopmental testing instruments described below, with 115 children in the 6–8;11 (years;months) range (PEEX 2) and 159 within the 9–15;11 range (PEERAMID 2). The mean age for the entire sample was 9;8, with a median of 10 years. Fifty-two percent of the children were males. Racial composition included 69% Caucasian, 19% African American, and 12% biracial. This racial distribution is consistent with the racial distribution within southwestern Michigan. However, there was a statistically significant race difference within the sample. The majority of these children lived with foster parents (54%), with the remainder of the children's living arrangement divided between relatives (18%), biological parents (14%), and adoptive parents (14%) (see Table 2).

Ninety-seven percent of the children were determined to be moderately to severely traumatized. The severity of traumatization was determined by senior Children's Trauma Assessment Center (CTAC) clinicians on the basis of the known effects of trauma, the extent of internalized and externalized distress as measured by rating scales and clinical interview, history, caregiver concerns, resilience factors, and neurodevelopmental status. The Traumagenic Impact of Maltreatment Rating (James, 1989) was completed by the senior clinicians, making the determination of traumatization on a 1–10 scale from absent to severe. Meeting formal posttraumatic stress disorder (PTSD) criteria was not required because this *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–IV;* American Psychological Association, 1994) diagnosis was believed to be too exclusionary for determining past traumatization.

Forty percent of the children in our sample met the criteria for FASD (including the full FAS as well as ARND), which represents the spectrum of conditions that are related to prenatal exposure (Bertrand, Floyd, Weber, O'Connor, & Riley, 2004). The University of Washington (UW) FAS Diagnostic and Prevention Network (FAS DPN) model was used in determining whether a child was classified as FASD. The University of Washington FAS DPN model was developed in the mid 1990s to allow a more systemic and quantitative approach to FASD diagnosis. This model involves analysis of four key domains, including growth, facial FASD characteristics, CNS dysfunction/damage, and level of alcohol exposure using a 4-digit classification system (one digit corresponding to each of the domains listed above), with 256 possible combinations. This system is one of several formal FAS diagnostic protocols available to clinicians. To be considered FASD, a child was required to meet UW criteria for sentinel physical findings for FAS, as determined by the CTAC behavioral/developmental pediatrician, in addition to demonstrating three areas of significant CNS delay, as determined by neurodevelopmental testing, history, and observation. Sentinel physical findings refers to growth deficiency at the 3rd or 4th level of 4 levels, and/or the presence of the facial phenotype at the 3rd or 4th of 4 levels. Actual documentation of biological mothers' disclosure of alcohol use during pregnancy was rare because 86% of the children were not living with their biological parents, thus limiting access to reliable prenatal histories.

**Table 2.** Demographic variables for the total sample ($N$ = 274).

| *Variable* | *Percentage of sample* |
|---|---|
| Mean age | 9;8 |
| Gender | |
| Male | 52 |
| Female | 48 |
| Race | |
| Caucasian | 69 |
| African American | 19 |
| Biracial | 12 |
| Living arrangement | |
| Foster parents | 54 |
| Relatives | 18 |
| Biological parents | 14 |
| Adoptive parents | 14 |
| Experienced trauma | 97 |
| Met criteria for fetal alcohol spectrum disorder (FASD) | 40 |
| Trauma without FASD | 60 |

***Note.*** 9;8 is in years;months.

## Instruments

The PEEX 2 (Levine, 1996a) and PEERAMID 2 (Levine, 1996b) are standardized neurodevelopmental/neurobehavioral assessments of children and adolescents between the ages of 6–8;11 (PEEX 2) and 9–14;11 (PEERAMID 2), having been normed on a national sample of students within those age ranges. Reliability values are not available for these tests because they do not yield an overall score or subtest scores, but instead are designed to generate a narrative description of a child's neurodevelopmental profile. The tests are administered according to a standardized manual in order to meet standards of reliability. The instruments are subdivided into five sections: fine motor, language, gross motor, memory, and visual processing. Within each domain, two tasks below age norms indicate a moderate delay for that specific domain, and three or more tasks below age norms indicate a major delay for that domain. Areas of function that are comprehensively assessed include attention, memory, neuromotor function, visual–spatial processing, temporal–sequential function, and higher level cognition. The range of language skills measured includes phonology, syntax, semantics, word retrieval, discourse processing, sentence comprehension, inferencing, summarization, and expressive fluency. All of the items on the PEEX 2 and PEERAMID 2 are related directly to specific aspects of the school experience and help to evaluate key neurodevelopmental functions needed for optimal learning, adaptation to school, and academic productivity. These instruments do not yield a total score or label for the student; instead, they contribute to the creation of a profile of CNS strengths and weaknesses. This strength and weakness profile allows the assessment team to generate specific recommendations for managing the student at home and/or at school.

Efficient neurodevelopmental function of children and adolescents is critical to effective and efficient learning and behavioral functioning. The PEEX 2 and PEERAMID 2, which typically take 60–90 min to administer, were originally developed to assist primary care pediatricians in the formulation of effective neurobehavioral strategies for children with learning differences who may

have not been eligible for traditional special education services. These instruments have proven very useful for this specific population of children, who often are not able to successfully complete a traditional neuropsychological evaluation, which can take 6–8 hr to finish, due to significant arousal problems and affective dysregulation. These screening instruments allow construction of a neurodevelopmental strength and weakness profile that provides a behavioral template and greatly facilitates the development of effective treatment strategies.

The Kaufman Brief Intelligence Test (K–BIT; Kaufman & Kaufman, 1990) is a brief, individually administered instrument that measures the verbal and nonverbal intelligence of individuals ranging in age from 4 to 90 years. The K–BIT is composed of two subtests, Vocabulary (including expressive vocabulary and definitions) and Matrices, along with an overall score, known as the K-BIT IQ composite. The Vocabulary subtest measures verbal, school-related skills (crystallized thinking) by assessing a person's word knowledge and verbal concept formation. The Matrices subtest measures nonverbal skills and the ability to solve new problems (fluid thinking) by assessing an individual's ability to perceive relationships and complete analogies. All Matrices items are motor free and involve pictures or abstract designs rather than words. Age-based standard scores having a mean of 100 and a standard deviation of 15 are provided for the composite and both subtests. Relevant to this study, scores from 90 to 109 are considered average, with scores from 80 to 89 being below average. Reliability coefficients specific to the population in this study range from .79 to .95. ($M$ = .90), with the lowest value specific to reliability for 6-year-olds on the Matrices subscale. The K–BIT norming sample is similar to several full-length intelligence and achievement tests (e.g., WISC–III [Wechsler, 2002], KAIT [Kaufman & Kaufman, 1993], WRAT–3 [Moseley, 2003], PIAT–R [Markwardt, 1989], and K–TEA [Kaufman & Kaufman, 2004]), permitting direct comparisons to scores earned on these instruments. Because of the shorter time period needed for administration, it is well suited for children with neurodevelopmental delays.

The Conners' Rating Scales—Revised (CRS–R; Conners, 1997) measures psychopathology and problem behavior in children and adolescents, and different versions are used to evaluate problem behaviors by obtaining reports from parents/caregivers and from teachers. Normed on a North American sample of more than 8,000 children and adolescents (ages 3–17), the CRS–R provides a battery of behavioral subscales that correspond to the *DSM–IV.* The CRS–R: Long Form (CPRS–R:L) is completed by parents/caregivers and contains 80 items within 14 subscales. The Conners' Teacher Rating Scale—Revised: Long Form (CTRS–R:L) is completed by teachers and contains 59 items within 13 subscales. Raw scores are summed and converted into standardized $t$ scores that have a mean of 50 and a standard deviation of 10. $t$ scores over 65 indicate significant problems, with those at 66 to 70 considered moderately atypical and those over 70 considered markedly atypical. In terms of reliability, internal consistency coefficients range from approximately .75 to .90, and 6- to 8-week test–retest reliability coefficients range from approximately .60 to .90.

## Data Collection

Data were collected both before and during the assessment testing of children. Children's testing occurred over a 1- to 2-day period depending on the child's traveling distance, ability to maintain attention, and/or dysregulation. The instruments were administered by CTAC clinicians. Following the assessment, detailed written reports (between 15 and 20 pages) were constructed to provide information to the caregiver, referring agency, and court. The majority of the caregivers met with CTAC staff to review and discuss assessment results and recommendations and to address any questions and concerns. Testing scores and information from each assessment report were entered into SPSS by trained data managers.

## Procedures

The sample was divided into two groups: children with trauma/no FASD and children with trauma/FASD. The two groups were compared with respect to performance on the PEEX 2/PEERAMID 2, K–BIT, and CRS–R. Results from the PEEX 2 and PEERAMID 2 were collapsed into two categories: no problem/mild problem and moderate/major problems. Chi-squared analyses were used to determine if the percentage of children with moderate/major problems differed among children with trauma/FASD and trauma/no FASD. The K–BIT and CRS–R instruments are both continuous scales; thus, $t$ tests were used to compare the means for children with trauma/FASD and trauma/no FASD. $t$ tests were also used to compare each group's mean score on the K–BIT to the national mean.

# RESULTS

Table 3 displays the descriptive statistics for the two groups: trauma/no FASD and trauma/FASD. Forty percent of the children with moderate to major trauma met the criteria for FASD ($N$ = 113), with 60% ($N$ = 161) not meeting the criteria for FASD. The demographics were similar between the trauma/FASD and trauma/no FASD groups with the exception of racial composition and living arrangement. The percentage of children with trauma/FASD who were Caucasian (80%) was greater than that of Caucasian children with trauma/no FASD (61%). For African American children, the opposite was found, with 26% of the trauma/no FASD

**Table 3.** Demographic variables within groups.

| *Variable* | *Trauma/No FASD* N = *161* | *Trauma/FASD* N = *113* |
|---|---|---|
| Mean age | 10;1 | 9;5 |
| Gender | | |
| Male | 52% | 50% |
| Female | 48% | 50% |
| Race | | |
| Caucasian* | 61% | 80% |
| African American* | 26% | 9% |
| Biracial | 13% | 12% |
| Living arrangement | | |
| Foster parents | 63% | 60% |
| Relatives | 19% | 14% |
| Biological parents | 12% | 8% |
| Adoptive parents | 6% | 18% |

*Two-tailed $p$ = .003.

**Table 4.** Percentage of children with moderate/major delays on the PEEX 2/PEERAMID 2.

| *Domain* | *Trauma/No FASD* | *Trauma/FASD* | *Two-tailed* p *value* |
|---|---|---|---|
| Attention | .74 | .89 | .004 |
| Memory | .71 | .87 | .005 |
| Receptive language | .57 | .81 | .000 |
| Expressive language | .50 | .72 | .001 |
| Visual processing | .60 | .71 | |
| Fine motor | .48 | .60 | |
| Graphomotor | .48 | .60 | |
| Gross motor | .24 | .30 | |

group being African American and only 9% of the trauma/FASD group being African American. With respect to living arrangement, 18% percent of the trauma/FASD group were living in adoptive homes; only 6% of the trauma/no FASD group were living in adoptive homes. The sample did not include children with no trauma/FASD because virtually all of the children who were assessed had experienced at least moderate trauma.

Table 4 displays the percentage of children with moderate/major delays on the PEEX 2/PEERAMID 2 tests for the two groups. Consistent with the literature on prenatal alcohol exposure and trauma (DeBellis & Van Dillen, 2005; Riley & McGee, 2005), the domains of greatest deficits were attention, memory, and language (receptive, expressive). A greater percentage of children with trauma/FASD exhibited moderate to major delays in each of the domains as compared to children with trauma only, at a 0.01 level of significance.

The mean score for the K–BIT for each group was compared to the national norm, with statistically significant differences between each of the groups and the national norm at the .001 level. Table 5 displays the mean scores and effect sizes for the K–BIT for each group. Effect sizes ranged from small (.22) to moderate (.42) (Cohen, 1988). Both K–BIT subtests along with the K–BIT composite had statistically significant differences between the means for the two groups, with verbal problem-solving capacity having the lowest overall mean for both groups. Consistent with the PEEX 2/PEERAMID 2 data, children with trauma/FASD had greater intellectual/cognitive deficits than did those with trauma/no FASD.

The results of the CPRS–R:L, which was completed by parents/caregivers, are displayed in Table 6. Effect sizes ranged from very small (.06) to medium (.50) (Cohen, 1988). Across all domains, children with trauma/FASD had greater difficulties than did those with only trauma. Eight of the CPRS–R:L subgroup means for children with trauma/FASD were within the markedly atypical range (scores > 70), indicating a significant problem. Six of the CPRS–R:L subgroup means for children with trauma/no FASD were in the moderately atypical range (scores between 66–70), indicating a significant problem. *t* tests revealed that eight CPRS-R:L behavioral areas between these two groups had statistically significant differences.

In contrast to the CPRS–R:L, the CTRS–R:L revealed no behavioral areas in the markedly atypical range for either group of children (see Table 7). Effect sizes ranged from not detectable (0) to medium (.46) (Cohen, 1988). The CTRS–R:L revealed 10 of 13 behavior categories as moderately atypical, indicating possible clinical significance for children with trauma/FASD. The children with trauma/no FASD had no behaviors in the moderately atypical range, although 8 of the 13 behavior categories were within 1–3 points of reaching this range. Five behavioral areas had statistically significant differences between the two groups.

## DISCUSSION

Statistical analysis revealed that prenatal alcohol exposure and postnatal trauma (FASD/trauma) had significant potential to drastically alter normal childhood development. The combination of prenatal alcohol exposure and trauma was more devastating to neurodevelopment (as demonstrated by the PEEX 2/PEERAMID 2 findings) than trauma alone. Four of the eight neurodevelopmental constructs measured revealed a greater percentage of children with trauma/FASD having moderate/major delays as compared to children with trauma/no FASD.

Lower intelligence scores in these children compound the neurodevelopmental findings. Children with trauma/FASD had scores on the K–BIT that were statistically lower than those of children with trauma/no FASD. Further, the mean scores for the children with trauma/FASD were in the below average range (Kaufman & Kaufman, 1990). Both groups are at risk for learning disabilities, but their learning problems may not be easily identifiable via traditional special education formulas designed to determine eligibility for cognitive impairment and learning disability. Some children, however, receive special education services through other labels, including emotionally impaired and/or otherwise health impaired.

When neurodevelopmental delays for traumatized children with or without FASD are under-identified, this leads to a substantial risk of underestimating a child's need for school intervention. Language deficits, in particular, are likely to contribute to the child's poor coping strategies, which often result in negative undesirable behaviors. Due to language deficits, SLPs will be assessing and providing services to these children in the school setting. Educators,

**Table 5.** Kaufman Brief Intelligence Test (K–BIT) mean scores and standard deviations.

| | *Trauma/No FASD* | | *Trauma/FASD* | | *Two-tailed* | |
|---|---|---|---|---|---|---|
| *Subtest* | M | SD | M | SD | p *value* | *Effect size* |
| Vocabulary (Verbal) | 92 | 16 | 87 | 13 | .007 | .31 |
| Matrices (Nonverbal) | 94 | 20 | 89 | 22 | .04 | .22 |
| Composite | 94 | 14 | 88 | 14 | .01 | .42 |

**Table 6.** Conners' Parent Rating Scale—Revised mean scores and standard deviations.

| | *Trauma/No FASD* | | *Trauma/FASD* | | *Two-tailed* | |
|---|---|---|---|---|---|---|
| *Behavioral category* | M | SD | M | SD | p *value* | *Effect size* |
| Oppositional | 68[a] | 15 | 72[b] | 15 | .04 | .26 |
| Cognitive problems/inattention | 62 | 19 | 69[a] | 20 | | .35 |
| Hyperactivity | 68[a] | 16 | 73[b] | 16 | | .31 |
| Anxious/shy | 61 | 15 | 63 | 16 | | .13 |
| Perfectionism | 53 | 12 | 54 | 9 | | .08 |
| Social problems | 64 | 17 | 70[a] | 16 | .02 | .35 |
| Psychosomatic | 60 | 15 | 61 | 16 | | .06 |
| ADHD index | 65 | 15 | 72[b] | 14 | .004 | .47 |
| Restless/impulsive | 67[a] | 15 | 73[b] | 15 | .01 | .40 |
| Emotional lability | 63 | 15 | 65 | 14 | | .13 |
| Global index | 67[a] | 13 | 73[b] | 13 | .02 | .46 |
| *DSM–IV* inattention | 64 | 14 | 71[b] | 14 | .004 | .50 |
| *DSM–IV* hyperactive/Impulsive | 70[a] | 15 | 75[b] | 15 | .03 | .33 |
| *DSM–IV* Total | 68[a] | 15 | 72[b] | 14 | .005 | .27 |

***Note.*** ADHD = attention deficit hyperactivity disorder.
[a]moderately atypical *t* score, indicating a significant problem. [b]markedly atypical *t* score, indicating a significant problem.

as well as SLPs, are likely to be continually challenged by these children who have complex multiple neurodevelopmental needs that frequently exceed the school staff's ability and training.

Observations of these challenging behaviors through reports by parents and teachers on the CRS–R support these findings. The fact that the mean scores on the CPRS–R:L for children with FASD/trauma were in the clinically significant range in 8 of 14 behavioral categories strongly suggests that these extreme behaviors are actually typical for children with trauma/FASD. Although the results of the CTRS–R:L were not as extreme as those of the CPRS–R:L, there were still six behavioral areas that were moderately atypical. Both scales suggest that both caregivers and teachers are likely to perceive these children as far more oppositional, hyperactive, restless/impulsive, inattentive, socially inept, and exhibiting more global behavioral problems than the general population of children. Caregivers and teachers are also at risk for misinterpreting these children's behaviors through the traditional paradigm of being "willfully disobedient."

In a study of teachers of children who had been exposed prenatally to drugs, Watson and Westby (2003) found that "educator's attitudes and beliefs about the nature of children's behavioral difficulties determined the behavior management strategies they used (p. 208)." The majority of educators blamed the children or their parents for the children's poor performance and challenging behaviors. The dominant beliefs were that the "children had the ability to control their own behaviors, and they just chose to misbehave" (Watson & Westby, p. 209), or that the children's behaviors were outcomes of inadequate childrearing. Only a few seemed "to understand the physical, cognitive, and emotional bases for the behaviors" (Watson & Westby, p. 209). Words such as "defiant" and "lazy" frequently become the standard lexicon to describe these children. In response to these children, educators

**Table 7.** Conners' Teacher Rating Scale—Revised mean scores and standard deviations.

| | *Trauma/No FASD* | | *Trauma/FASD* | | *Two-tailed* | |
|---|---|---|---|---|---|---|
| *Behavioral category* | M | SD | M | SD | p *value* | *Effect size* |
| Oppositional | 64 | 18 | 66[a] | 16 | | |
| Cognitive problems/inattention | 60 | 15 | 66[a] | 12 | .006 | .40 |
| Hyperactivity | 63 | 15 | 67[a] | 14 | | .27 |
| Anxious/shy | 60 | 13 | 62 | 12 | | .15 |
| Perfectionism | 53 | 11 | 52 | 11 | | .00 |
| Social problems | 60 | 16 | 64 | 15 | | .25 |
| ADHD index | 64 | 14 | 69[a] | 14 | .02 | .36 |
| Restless/impulsive | 64 | 13 | 69[a] | 13 | .03 | .38 |
| Emotional lability | 63 | 16 | 67[a] | 15 | | .25 |
| Global index | 65 | 15 | 69[a] | 14 | | .27 |
| *DSM–IV* inattention | 63 | 15 | 67[a] | 15 | .009 | .27 |
| *DSM–IV* hyperactive/Impulsive | 63 | 14 | 67[a] | 13 | | .29 |
| *DSM–IV* Total | 63 | 13 | 69[a] | 13 | .01 | .46 |

[a]moderately atypical *t* score, indicating a significant problem.

are likely to use traditional child management strategies that rely heavily on increased consequences to produce behavior change. These strategies assume that these children have the neurodevelopmental abilities and skills of adaptation, flexibility, and frustration tolerance to alter their behaviors (Greene & Ablon, 2006).

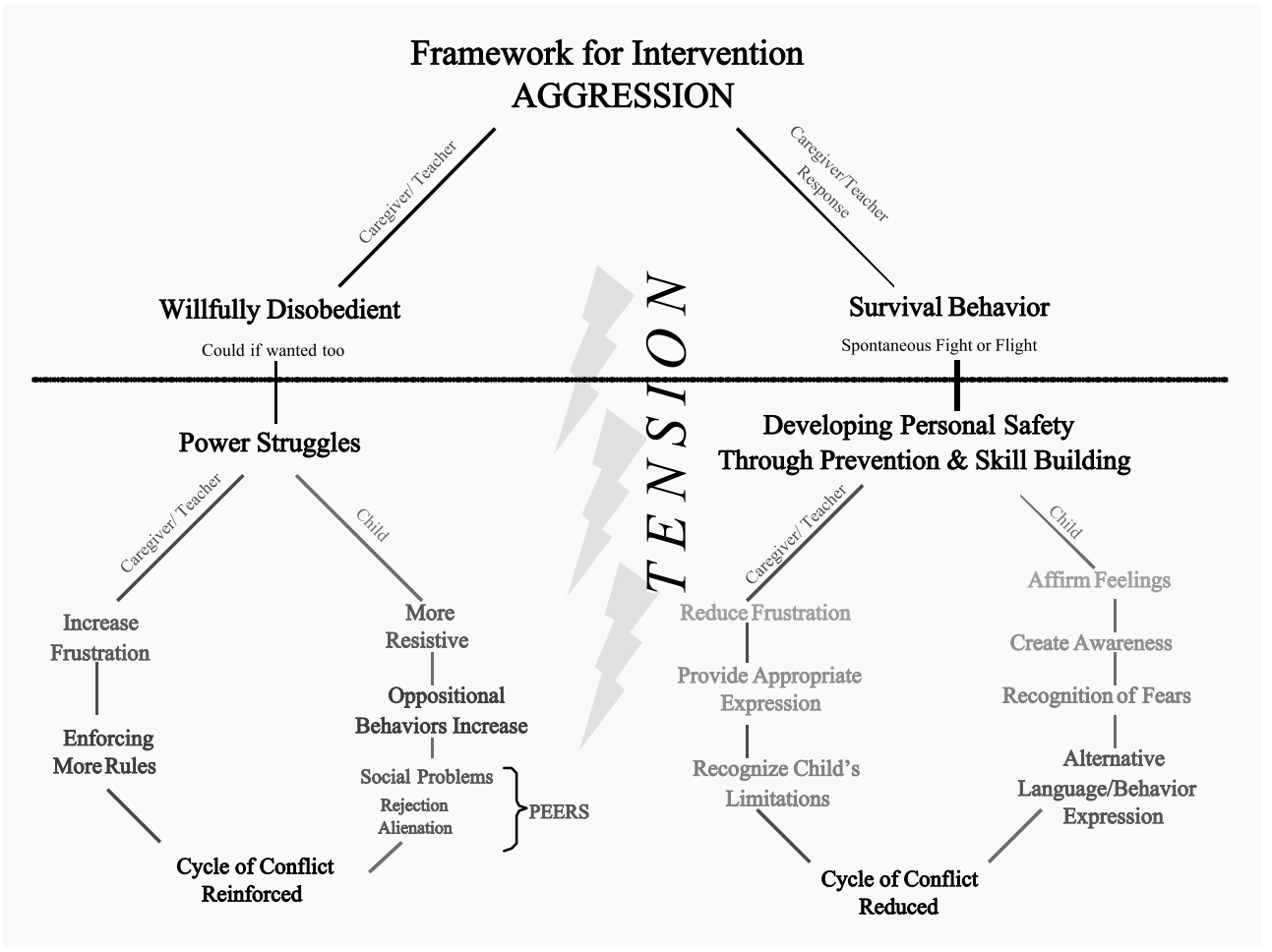
A more accurate understanding of problematic behaviors of traumatized children with and without FASD demands a "paradigm shift." The paradigm shift recognizes the impact of prenatal alcohol exposure and trauma on children's neurodevelopment functioning. Rather than viewing these children as willfully disobedient, the new paradigm appreciates their difficulties in affect regulation, coping skills, and problem solving. Their hyperactivity, opposition/defiance, aggression, emotional disengagement, and avoidance of responsibility are often produced by traumatic stress reactions.

## CLINICAL IMPLICATIONS

Knowledge of the limited coping strategies of children with prenatal alcohol exposure and/or trauma requires alternative interventions that provide the maximum opportunity for these children to be successful within multiple domains. A brain-behavior-based paradigm acknowledges that the etiologies of challenging behaviors are rooted in poor executive functioning, cognitive inflexibility, limited social communication, deficits in language processing, affect dysregulation, and traumatic stress. These children most often do not respond to typical models of traditional discipline. Figure 1 indicates how adult perspectives on the etiology of challenging behaviors directly affect their responses to the children. Perceiving children's behavior as willfully disobedient (left side of Figure 1) often leads to frequent power struggles. In order to affect child behavior change, adults attempt to exert power through rule reliance and increased consequences (Bloom, 2005). However, traumatized children with and without FASD are not likely to respond positively to more stringent rules and punishments (Bloom, 2005; Greene & Ablon, 2006; Perry, 2006). Consequently, a negative cycle of conflict that results in continual power struggles, heightened adult frustration, and increased oppositional child behaviors is likely to be further cemented and reinforced.

In contrast, the brain-behavior-based paradigm (right side of Figure 1) recognizes the neurodevelopmental impact of prenatal alcohol exposure and/or trauma. These interventions and strategies consider a child's limitations in remembering and following

**Figure 1.** Framework for intervention for trauma/no FASD and trauma/FASD children (Henry, Black-Pond, & Sloane, 2004).



directions, their need for increased support in learning new skills, and their vulnerability to stress. These children often unconsciously reenact their trauma by spontaneously responding with fight/flight/freeze self-protective survival behaviors. The goal of the brain-behavior-based paradigm is to provide physical and psychological safety, which is a prerequisite for affect management, skill building, trauma resolution, and cognition (Perry, 2006). Creating safer relationships that affirm children's feelings through appreciation of their overwhelming affect is essential to building trust and managing traumatic stress. Thus, under this new paradigm, adults provide and model new language for children that accurately identifies and allows for expression of their affective states. Adults then use challenging behaviors as opportunities for teaching these children how to respond differently. As children become more proficient at recognizing and expressing their anger and unresolved trauma, oppositional and aggressive behaviors decrease, and children gain internal resources that create conscious connections between their traumatic histories and their current behaviors. Through skill building and self awareness, the children are better able to modulate their affect and behavior. As a result, relationships with adults become more reciprocal/transactional and less stressful, which encourages trust (Greene & Ablon, 2006).

SLPs are often in the unique position of working individually with these children. They have the opportunity to provide relational safety, recognize the need for further assessment, and model effective communication that enhances skill building. Further, they can support the implementation of the alternate paradigm with classroom teachers and administrators.

A tension exists between the two paradigms (the lightning bolt within the middle of Figure 1) that represents the continual pull to the more traditionally embedded rule-based authoritative philosophy that labels difficult children as willfully disobedient. Systems of care (school, foster care, courts) commonly operate on these assumptions, subsequently interfering in the development of emotional safety, healthy relationships, increased self-esteem, and academic success. In addition, support for implementing and sustaining alternative models that recognize the unique needs of these affected children is limited. Mainstream criticisms viewing such strategies as enabling of "bad behavior" and increasing "child manipulation," especially when child progress is slow, encourage the return to traditional interventions.

Finally, recognition that school professionals are not specifically trained to understand and intervene with children with FASD/trauma and traumatized children is important. Furthermore, schools struggle to achieve the necessary staffing that provides the individualized attention that most of these children require on a daily basis. Subsequently, caregivers, educators, and SLPs require additional support for their own stress reduction and coping strategies to integrate the new paradigm. Such multiple challenges demand that multiple systems collaborate closely with school personnel to provide intense resource support if these children are to be academically and socially successful.

## REFERENCES


**American Psychological Association.** (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

**Bertrand, J., Floyd, R. L., Weber, M. K., O'Connor, M., & Riley, E. M., & National Task Force on FAS/FAE.** (2004). *Fetal alcohol syndrome: Guidelines for diagnosis & referral*. Atlanta, GA: Centers for Disease Control & Prevention.

**Bloom, S.** (2005, April). *Sanctuary model*. White paper presented at the National Children's Traumatic Stress Network Annual National All-Network Meeting, Alexandria, VA.

**Bremner, J. D., Southwick, S. W., & Charney, D. S.** (1999). The neurobiology of post-traumatic stress disorder: An integration of animal and human research. In P. A. Saigh & J. D. Bremner (Eds.), *Post-traumatic stress disorder: A comprehensive text* (pp. 103–143). New York: Allyn & Bacon.

**Coggins, T. E., Friet, T., Morgan, T., & Wikstrom, M.** (1998). *Washington state FAS DPN: Communicative behavior assessment*. Seattle: University of Washington Publication Services.

**Cohen, J.** (1988). *Statistical power analysis for the behavioral sciences* (2nd ed). Hillsdale, NJ: Erlbaum.

**Cohn, E., Miller, L. J., & Tickle-Degnen, L.** (2000). Parental hopes for therapy outcomes: Children with sensory modulation disorders. *American Journal of Occupational Therapy, 54*(1), 36–43.

**Conners, K.** (1997). *Conners' Rating Scales—Revised User's manual*. North Tonawanda, NY: Multi-Health Systems.

**Cook, A., Blaustein, M., Spinazzola, J., & van der Kolk, B. (Eds.).** (2003). *Complex trauma in children and adolescents*. Retrieved January 17, 2005, from http://www.NCTSNet.org

**Crittenden, P. M.** (1998). Dangerous behavior and dangerous contexts: A thirty-five year perspective on research on the developmental effects of child physical abuse. In P. Trickett (Ed.), *Violence to children* (pp. 11–38). Washington, DC: American Psychological Association.

**Crittenden, P. M., & DiLalla, D. L.** (1988). Compulsive compliance: The development of an inhibitory coping strategy in infancy. *Journal of Abnormal Child Psychology, 16,* 585–599.

**DeBellis, M. D., & Van Dillen, T.** (2005). Childhood post-traumatic stress disorder: An overview. *Child & Adolescent Psychiatric Clinics of North America, 14,* 745–772.

**Dunn, W.** (2001). The sensations of everyday life: Empirical, theoretical, and pragmatic considerations. *American Journal of Occupational Therapy, 55,* 608–620.

**Dunn, W., & Westman, K.** (1997). The sensory profile: The performance of a national sample of children without disabilities. *American Journal of Occupational Therapy, 51,* 25–34.

**Ford, T. J., Taylor, E., & Warner-Rogers, J.** (2000). Sustained release methylphenidate. *Child Psychology and Psychiatry, 5,* 108–114.

**Glod, C. A., Teicher, M. H., Hartman, C. R., Harakal, T., & McGreenery, C. E.** (1997). Enduring effects on early abuse on locomotor activity, sleep, and circasian rhythms. *Annals of the New York Academy of Sciences, 821,* 465–467.

**Greene, R., & Ablon, S.** (2006). *Treating explosive kids: The collaborative problem solving approach*. New York: Guilford Press.

**Gunnar, M., & Donzella, B.** (2002). Social regulation of the cortisol levels in early human development. *Psychoneuroendocrinology, 27*(1–2), 199–220.

**Henry, J., Black-Pond, C., & Sloane, M.** (2004, April). *Interventions with traumatized children*. Presentation at Child Welfare Institute, Kalamazoo, MI.

**Hyter, Y., Henry, J., Atchison, B., Sloane, M., & Black-Pond, C.** (2003, November). Children affected by trauma and alcohol exposure: A profile of the southwestern Michigan children's trauma assessment center. *The ASHA Leader, 14,* pp. 6–7.

**Institute of Medicine.** (1996). *Fetal alcohol syndrome: Diagnosis, epidemiology, prevention, and treatment*. Washington, DC: National Academy Press.

**James, B.** (1989). *Treating traumatized children*. Lexington, MA: Lexington Books.

**Kagan, R.** (2004). *Rebuilding attachments with traumatized children: Healing from losses, violence, abuse, and neglect*. Birmingham, NY: Haworth Maltreatment and Trauma Press.

**Katz, K. B.** (1992). Communication problems in maltreated children: A tutorial. *Journal of Childhood Communication Disorders, 14*(2), 147–163.

**Kaufman, A. S., & Kaufman, N. L.** (1990). *Kaufman Brief Intelligence Test*. Circle Pines, MN: American Guidance Service.

**Kaufman, A. S., & Kaufman, N. L.** (1993). *Kaufman Adolescent and Adult Intelligence Test*. Circle Pines, MN: American Guidance Service.

**Kaufman, A. S., & Kaufman, N. L.** (2004). *Kaufman Test of Educational Achievement, Second Edition, Comprehensive Form*. Circle Pines, MN: AGS Publishing.

**Kaufman, J., Plotsky, P. M., Nemeroff, C. B., & Charney, D. S.** (2000). Effects of early adverse experiences on brain structure and function: Clinical implications. *Biological Psychiatry, 48*(8), 778–790.

**Kernic, M. A., Holt, V. L., Wolf, M. E., McKnight, B., Huebner, C. E., & Rivara, F. P.** (2002). Academic and school health issues among children exposed to maternal intimate partner abuse. *Archives of Pediatrics and Adolescent Medicine, 156*(6), 549–555.

**Levine, M. D.** (1996a). *Pediatric Early Elementary Examination*. Cambridge, MA: Educators Publishing Service.

**Levine, M. D.** (1996b). *Pediatric Examination of Educational Readiness at Middle Childhood*. Cambridge, MA: Educators Publishing Service.

**Malbin, D.** (1993). *Fetal alcohol syndrome fetal alcohol effects: Strategies for professionals*. Center City, MS: Hazelden.

**Markwardt, F.** (1989). *Peabody Individual Achievement Test—Revised*. Bloomington, MN: Pearson Assessments.

**Mattson, S. M., Riley, E. M., Gramling, L., Delis, D. C., & Jones, K. L.** (1997). Heavy prenatal alcohol exposure with or without physical features of FAS leads to IQ deficits. *J Pediatr, 131,* 718–721.

**Miller, L. J., Reisman, E., McIntosh, D. N., & Simon, J.** (2001). An ecological model of sensory modulation: Performance of children with fragile X syndrome, autistic disorder, attention-deficit/hyperactivity disorder, and sensory modulation dysfunction. In S. S. Roley, E. I. Blanche, & R. Schaaf (Eds.), *Understanding the nature of sensory integration with diverse populations* (pp. 57–85). San Antonio, TX: Therapy Skill Builders.

**Moseley, D.** (2003). *Word Recognition and Phonics Skills Test (WRaPs) Specimen Skill Set* (2nd ed.). London, UK: Hudder Education.

**O'Malley, K.** (2000). Neuropsychiatric implications and long term consequence of FASD. *Seminars in Clinical Neuropsychiatry, 5,* 177–190.

**Parham, D., & Mailloux, Z.** (1995). Sensory integrative principles in intervention with children with autistic disorder. In J. Case-Smith, A. S. Allen, & P. N. Pratt (Eds.), *Occupational therapy for children* (pp. 329–382). St. Louis, MO: Mosby.

**Pearce, J. W., & Pezzot-Pearce, T. D.** (1997). *Psychotherapy of abused and neglected children*. New York: Guilford Press.

**Perry, B. D.** (1997). Incubated in terror: Neurodevelopmental factors in the 'cycle of violence.' In J. Osofsky (Ed.), *Children, youth and violence: The search for solutions* (pp. 124–148). New York: Guilford Press.

**Perry, B. D.** (2006). Applying principles of neurodevelopment to clinical work with maltreated and traumatized children: The neurosequential model of therapeutics. In N. B. Webb (Ed.), *Working with traumatized children in child welfare* (pp. 27–52). New York: Guilford Press.

**Putnam, F. W.** (2006). The impact of trauma on child development. *Juvenile and Family Court Journal, 57*(1), 1–11.

**Riley, E. P., & McGee, C. L.** (2005). Fetal alcohol spectrum disorders: An overview with emphasis on changes in brain and behavior. *Experimental Biology and Medicine, 230,* 357–365.

**Riley, E. P., McGee, C. L., & Sowell, E. R.** (2004). Teratogenic effects of alcohol: A decade of brain imaging. *American Journal of Medical Genetics Part C: Seminars in Medical Genetics, 127C,* 35–41.

**Sampson, P. D., Streissguth, A. P., Bookstein, F. L., & Barr, H.** (2000). On categorizations in analyses of alcohol teratogenesis. *Environmental Health Perspectives, 108*(Suppl. 3), 421–428.

**Schore, A. N.** (2001). The effects of early relational trauma on right brain development, affect regulation, and infant mental health. *Infant Mental Health Journal, 22*(1/2), 299–328.

**Streissguth, A.** (1992). Fetal alcohol syndrome and fetal alcohol effects: A clinical perspective of later developmental consequences. In I. Zagon & T. Slotkin (Eds.), *Maternal substance abuse and the developing nervous system* (pp. 5–25). San Diego, CA: Academic Press.

**Streissguth, A.** (1997a). *The challenge of fetal alcohol syndrome: Overcoming secondary disabilities*. Seattle: University of Washington Press.

**Streissguth, A.** (1997b). *Fetal alcohol syndrome: A guide for families and communities*. Baltimore: Paul H. Brookes.

**Streissguth, A. P., Bookstein, F. L., Barr, H. M., Sampson, P. D., O'Malley, K., & Young, J. K.** (2004). Risk factors for adverse life outcomes in fetal alcohol syndrome and fetal alcohol effects. *Journal of Developmental and Behavioral Pediatrics, 25*(4), 228–238.

**Streissguth, A., & O'Malley, K.** (2000). Neuropsychiatric implications and long-term consequences of fetal alcohol spectrum disorders. *Seminars in Neuropsychiatry, 5,* 177–190.

**Teicher, M. H., Andersen, S. L., Polcari, A., Anderson, C. M., & Navalta, C. P.** (2002). Developmental neurobiology of childhood stress and trauma. *Psychiatric Clinics of North America, 25*(2), 397–426.

**Walker, H., Schwarz, I., Nippold, M., Irvin, K., & Noell, W.** (1994). Social skills in school age children and youth: Issues and best practices in assessment and intervention. *Topics in Language Disorders, 14,* 70–82.

**Watson, S. M. R., & Westby, C. E.** (2003). Prenatal drug exposure: Implications for personnel preparation. *Remedial and Special Education, 24*(4), 204–214.

**Wechsler, D.** (2002). *Wechsler Preschool and Primary Scale of Intelligence* (3rd ed.). San Antonio, TX: The Psychological Corporation.

**Weiner, L., & Morse, B. A.** (1994). Intervention and the child with FAS. *Alcohol Health & Research World, 18*(1), 67–72.

Received November 22, 2005
Revision received May 22, 2006
Accepted September 25, 2006
DOI: 10.1044/0161-1461(2007/010)

Contact author: Jim Henry, 1000 Oakland Drive, 3rd floor, Kalamazoo, MI 49008. E-mail: james.henry@wmich.edu