

## Clinical Forum

# A State of Double Jeopardy: Impact of Prenatal Alcohol Exposure and Adverse Environments on the Social Communicative Abilities of School-Age Children With Fetal Alcohol Spectrum Disorder

**Truman E. Coggins**

University of Washington, Seattle

**Geralyn R. Timler**

University of Buffalo, NY

**Lesley B. Olswang**

University of Washington, Seattle



Alcohol is the most frequently ingested teratogen in the world (Streissguth, 1997). A large body of descriptive and experimental research underscores

the broad range of harmful effects that teratogenic alcohol exposure exerts on human growth and development (Astley & Clarren, 2000; Mattson & Riley, 1998; Thomas, Kelly, Mattson, & Riley, 1998).

**ABSTRACT: Purpose:** This article is a retrospective examination of environmental risk, language performance, and narrative discourse data from a clinical database of school-age children with fetal alcohol spectrum disorder (FASD).

**Method:** A case-defined diagnostic approach for measuring and reporting the full spectrum of disabilities in children with prenatal alcohol exposure is presented. Demographic, environmental, language, and social communication (as reflected by narrative discourse) data are reported for a large cohort of children with FASD between the ages of 6;0 (years;months) and 12;0.

**Results:** Children with FASD are a heterogeneous group with varying levels of compromise. The data demonstrate a substantial comorbidity between the effects of prenatal alcohol exposure and

adverse caregiving environments. The data further reveal that school-age children with FASD often exhibit clinically meaningful deficits in language and social communication.

**Clinical Implication:** Children with FASD may be particularly vulnerable to language and social communication deficits as a result of prenatal alcohol exposure and atypical or adverse social interactive experiences. Comprehensive assessment is recommended. Dynamic and functional assessment paradigms may document the language and social communicative deficits in children with FASD and other clinical populations with complex neurodevelopmental profiles.

**KEY WORDS:** prenatal alcohol, language and communication, maltreatment

Because so many women drink alcohol during pregnancy, disabilities associated with alcohol have been estimated to occur in as many as 6 per 1,000 live births (Health Resources and Services Administration [HRSA], 2005; Institute of Medicine [IOM], 1996). Using this estimate, 2,000–12,000 of the projected 4 million children born each year in the United States are likely to have a fetal alcohol spectrum disorder (FASD). The incidence of FASD is greater than that of children born with chromosomal disorders, metabolic or exocrine disorders, or specific neurological disorders (Plumridge, Bennett, Dinno, & Branson, 1993). The term FASD describes the range of effects that can occur in an individual whose mother drank alcohol during pregnancy.

School-age children with FASD present complex clinical profiles. They often display peer-related social problems but, with appropriate expectations and supportive environments, do not typically have debilitating conduct disorders (Streissguth & Kanter, 1997). They often exhibit processing limitations and learning difficulties (Kerns, Don, Mateer, & Streissguth, 1997) but have been found to have intellectual abilities that are broadly within the normal range (Streissguth, Barr, Kogan, & Bookstein, 1996). One key deficit that these children frequently share is their difficulty using language in sophisticated social contexts (Coggins, Olswang, Carmichael Olson, & Timler, 2003). Because youngsters with compromised social communication lack pivotal resources for resolving the dynamic challenges associated with daily school activities, school-based speech-language pathologists (SLPs) are likely to be consulted.

The purpose of this article is to explore the language and social communicative deficits in a large cohort of school-age children with FASD. Toward this end, we first present a framework of social communication and summarize findings from the FASD literature that promoted its development. Next, we highlight environmental factors that place children with FASD at compound risk for neurodevelopmental disorders and social communication problems. Finally, we offer empirical evidence of environmental risk and language performance deficits in children with FASD. We believe that these data provide SLPs with a valuable perspective in understanding the challenging behaviors associated with this perplexing clinical population.

## A SOCIAL COMMUNICATIVE FRAMEWORK

The American Speech-Language-Hearing Association (ASHA) recently highlighted the responsibility that school-based SLPs have in providing services for children with social communication deficits (ASHA, 2000, p. 278). However, selecting and using behavioral measures to assess the interpersonal uses of language in children with complex profiles like FASD is a formidable assignment because social interactions vary from context to context and require dynamic allocation of resources in order to be successful.

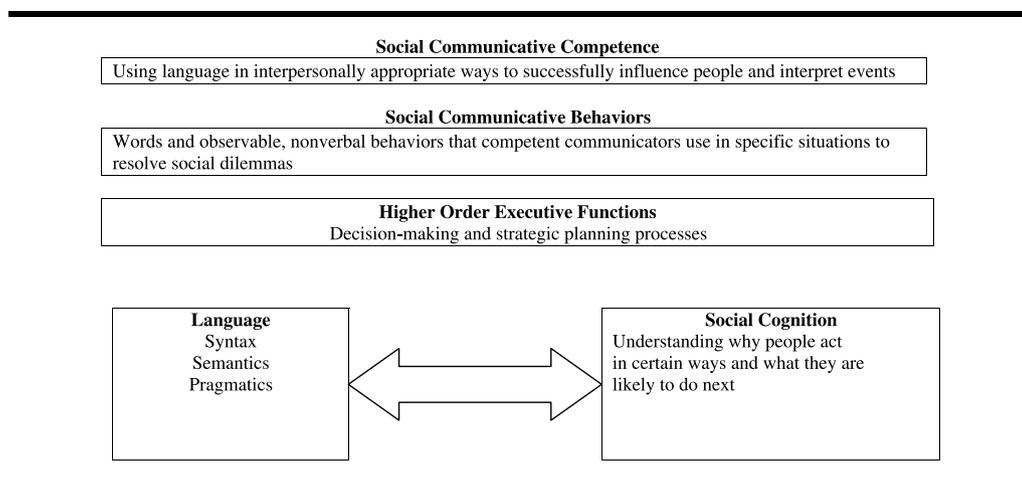
To this end, a promising framework has been proposed for assessing children with complex and diverse etiologies who exhibit compromises in social communication (Coggins et al., 2003). This conceptual framework, presented in Figure 1, is anchored to three interrelated developmental processes that children acquire and integrate in becoming competent communicators. These underlying processes include language, social cognition, and higher order executive functions. Because these processes ultimately determine children's social communicative behaviors, they are pivotal to social communicative competence.

Social communication is predicated on linguistic competence (Guralnick, 1999). Indeed, language is the primary means by which older children establish and maintain social relationships at home and school and with peers. The language variables that are of particular interest for school-age youngsters include knowledge of more sophisticated sentence construction and word knowledge as well as the ability to use this knowledge during real-time social interactions.

Because language is learned in dynamic social interactions with other people, children are naturally curious about the people around them. They try to make sense of social situations by figuring out why people act in particular ways and what they are likely to do next. The social cognitive component is, therefore, concerned with how children conceptualize and think about their social world—the people they observe, the relationships between people, and the groups in which people participate.

The remaining foundational process is executive function. The primary goals of the executive function are decision making and

**Figure 1.** A framework of social communication competence.



strategic planning (Singer & Bashir, 1999). Because a socially competent communicator must plan, integrate, and update his or her language and social cognitive abilities in accordance with the demands of particular situations, executive function is the overarching component of this model.

The dynamic relationship that exists among the underlying components is the essence of this social communication model. The effectiveness with which a child deploys specific behaviors of these foundational components during real-time social interactions reveals how well the underlying processes have been integrated. In the following sections, relevant findings with respect to these foundational components are summarized for school-age children with FASD.

## Language and FASD

Researchers who have studied developmental outcomes in children with FASD report that high levels of prenatal alcohol exposure disrupt the development and use of language (Mattson & Riley, 1998; Streissguth et al., 1996). This is not an unexpected discovery because alcohol is a teratogen that can alter brain structure and/or chemistry, and language development is highly correlated with brain maturation. Not surprisingly, much of the evidence to support this claim has been gathered using standardized tests that focus on how well these youngsters comprehend and/or produce the structure and content of their language (Abkarian, 1992; Becker, Warr-Leeper, & Leeper, 1990; Carney & Chermak, 1991; Church, Eldis, Blakley, & Bawle, 1997; Church & Kaltenbach, 1997; Gentry, Griffith, & Dancer, 1998; Janzen, Nanson, & Block, 1995; Weinberg, 1997). Although the results have revealed an array of performance profiles, no pattern of deficit has emerged.

A group of clinical researchers at the University of Washington has turned to more functional assessment strategies to describe the problems that are faced by children with FASD (Coggins, Friet, & Morgan, 1998; Hamilton, 1981; Thorne, Coggins, Carmichael Olson, & Astley, in press; Timler, Olswang, & Coggins, 2005). This research cadre has been interested in the ability of these children to use their language effectively to achieve important communicative goals and in obtaining information about underlying competence. Initial findings have revealed meaningful compromises with respect to how these children manage longer units of discourse during conversations (Hamilton, 1981) and narratives (Coggins et al., 2003).

The recent findings from a feasibility study bolster the argument that school-age children with FASD have narrative discourse deficits. Thorne et al. (in press) examined two independent parameters of narrative production in 16 school-age children with FASD and 16 age- and gender-matched peers with normal language. Narrative samples were coded for semantic elaboration of verbal and nominal concepts within the story and the degree to which unambiguous reference of nominal concepts was maintained as the story progressed. The former parameter involved the degree to which semantic concepts (e.g., "elk") were well specified or elaborated in the text as opposed to schematic concepts (e.g., "animal thing"). The reference parameter coded nominal and pronominal forms that storytellers used to introduce, maintain, or reintroduce concepts in the discourse (e.g., "There once was a boy who had a pet frog. He loved the frog very much.").

Results showed that both typically developing children and those with a diagnosis of FASD varied widely in the degree of semantic

elaboration they included in their stories. However, the children with FASD were significantly more likely to use pragmatically inappropriate (i.e., ambiguous) strategies for establishing and maintaining reference in their stories than were their typically developing peers (e.g., using definite nominal form *the* to introduce a concept into the story rather than indefinite form *a*). Thus, children in the FASD group were more likely to inappropriately distinguish between shared information and new information in their stories, resulting in greater ambiguity.

## Social Cognition and FASD

Social cognition is concerned with how children think about their social world—the people they observe, the relationships between people, and the groups in which people participate (Baron-Cohen, 2000; Tager-Flusberg, 1993). Caregivers have consistently reported that children with FASD seem unable to empathize and have genuine difficulty anticipating the consequences of their actions in social situations (Caldwell, 1993). Hinde's (1993) observations led her to argue that children with FASD do not understand "what is going on in social life and how they should behave in different situations" (p. 139).

The development of false-belief understanding, that is, the ability to make inferences about what other persons believe in specific situations, is regarded as an essential component of social cognition (Perner, 1991; Silliman et al., 2003; Wellman, 1990). Preliminary evidence has suggested that school-age children with FASD experience difficulty with false-belief tasks, even when false-belief tasks are presented in a simplified format (i.e., use of memory prompts, simple sentences, and forced-choice formats) (Coggins, 1997; Kodituwakku et al., 1997). Timler et al. (2005) suggested that this difficulty may, in part, be due to compromises that these children have in using mental state words to reference another person's perspective. Because effective use of these cognitive verbs is a critical measure of a child's ability to represent states of mind in themselves and others, children may not use language to describe what others may think or know during social interactions.

## Higher Order Executive Functions and FASD

Executive functions are higher order, decision-making, and planning processes that are invoked at the outset of a task and in the face of novel challenges (Singer & Bashir, 1999). Such processes permit children to disengage from the immediate context and reason about interpersonal goals. We have nested language and social cognition within higher order executive functions because socially competent communicators must integrate and/or modify their language and social cognitive abilities in accordance with the demands of particular situations.

Findings from a growing number of executive function investigations reveal that children with FASD have deficits in concept formation, response inhibition, and self-regulation (Jacobson & Jacobson, 2000; Kopara-Frye, Dehaene, & Streissguth, 1996; Mattson, Goodman, Caine, Delis, & Riley, 1999). Furthermore, executive function deficits appear to constrain the amount of information that children with high prenatal alcohol exposure can process when they are confronted with more complex challenges (Carmichael Olson, Feldman, Streissguth, Sampson, & Bookstein, 1998; Kerns et al., 1997; Kodituwakku, Handmaker, Cutler,

Weathersby, & Handmaker, 1995). Thus, it is not a leap of faith to suggest that processing deficits are likely to interfere with social performance and/or the use of complex language.

## Environmental Risk and FASD

Although high levels of prenatal alcohol exposure have a broad range of variable effects (Mattson & Riley, 1998; Streissguth et al., 1996), few investigators have systematically examined adverse environmental influences in children with FASD that may also compromise their developing nervous systems. This void is unfortunate because it is well known that adults who abuse alcohol often live in worlds that are disruptive and prone toward violence. The findings of Streissguth et al. (1996) and Willis and Silovsky (1998) have made clear the links between alcohol abuse and violence against others. As a result, children who live with dysfunctional adults are at considerable risk for neurobiological, psychophysiological, and/or psychological deficits (Cicchetti, 2004; Coster, Gersten, Beeghly, & Cicchetti, 1989; Kaufman, Plotsky, Nemeroff, & Charney, 2000; McFadyen & Kitson, 1996).

Lohmann and Tomasello (2003) found that early language experiences have a decided influence on children's underlying social cognitive behaviors. Cicchetti (2004) and Cicchetti, Rogosch, Maughan, Toth, and Bruce (2003) demonstrated that children with histories of maltreatment have distinct limitations in a "quintessential human characteristic" (Cicchetti et al., p. 1067); namely, their ability to interpret and predict the knowledge, intentions, and beliefs of other people. Thus, maladaptive social-interactive experiences, which often co-occur for children who have been compromised by prenatal alcohol exposure, are potent risk factors for theory-of-mind deficits.

Eigsti and Cicchetti (2004) argued that the socioemotional difficulties that are experienced by maltreated children "may be mediated or exacerbated by the observed language and communicative deficits" (p. 99). On the basis of their comparative review, Kelly, Day, and Streissguth (2000) concluded that prenatal alcohol exposure can alter the course of social communication. Thus, children with FASD may be particularly vulnerable to social communicative deficits as a result of both the teratogenic effects of prenatal alcohol exposure and the erratic and atypical social interactive experiences that are associated with a maltreating environment. To our knowledge, no one has yet seriously considered these comorbid conditions and the state of double jeopardy that prenatal alcohol and maltreatment may exert on a child's developing language and social communication.

## ENVIRONMENTAL RISK AND SOCIAL COMMUNICATION IN FASD: A RETROSPECTIVE STUDY

The purpose of this retrospective study is to explore environmental risk, language performance, and narrative discourse data for school-age children with FASD. The children were selected from an ACCESS database maintained by the Washington State Fetal Alcohol Syndrome Diagnostic and Prevention Network (FAS/DPN). Currently, the FAS/DPN database contains more than 1,700 clinical records. Because language and social communicative functioning have been documented in this database, as well as adverse environmental exposures and events, the FAS/DPN database is particularly germane to this clinical forum.

## METHODOLOGY

### Participants

Five hundred seventy-three school-age children were eligible to participate in this study. Each child had received a diagnosis of FASD from an experienced FAS/DPN interdisciplinary assessment team between January 1, 1993 and December 31, 2003 (see Clarren, Carmichael Olson, Clarren, & Astley, 2000). At the time of diagnosis, the participants ranged in age from 6;0 (years;months) to 12;11. The legal guardian for each participant provided written consent to use the resulting diagnostic data for research purposes.

**Documenting core characteristics of FASD.** All participants were diagnosed with FASD using a 4-digit diagnostic code (see Astley, 2004). The 4-digit diagnostic code is a case-defined diagnostic approach that uses quantitative scales to measure and report outcomes characterizing the full spectrum of disabilities of children who have been exposed to alcohol (Astley, 2004; Astley & Clarren, 2000, 2001; Chudley et al., 2005). The use of quantitative scales to measure the spectrum of disabilities in FASD and to design differential interventions is currently being explored (Thorne et al., in press; Timler & Olswang, 2001; Timler et al., 2005).

The 4-digit diagnostic code is presented in Figure 2. The 4-point numeric code scale in column one reflects the magnitude of expression of key FASD characteristics. Each characteristic assumes its own 4-point Likert scale (e.g., growth deficiency scale, alcohol

**Figure 2.** The 4-digit diagnostic code grid for quantifying core phenotype features of fetal alcohol spectrum disorder (i.e., growth deficiency, facial phenotype, brain damage, alcohol exposure) and associated prenatal exposure and postnatal risks (Astley, 2004).

Numeric code	Growth deficiency	Facial phenotype	Brain damage	Alcohol exposure	Prenatal risk	Postnatal risk
4	Severe	Severe	Definite	High	High	High
3	Moderate	Moderate	Probable	Some	Some	Some
2	Mild	Mild	Possible	Unknown	Unknown	Unknown
1	None	None	Unlikely	None	None	None

**Note.** From *Sensory Integration: Theory and Practice* by A. Bundy, S. Lane, & E. Murray, 2002. Copyright 2002 by F. A. Davis Company. Reprinted with permission.

exposure scale) and is ranked independently by members of an interdisciplinary assessment team. The core characteristics of this spectrum disorder are presented in columns two through four and include the growth deficiency scale, the facial phenotype scale, and the brain damage scale. A rank of 1 on any of these scales reflects complete absence of the FAS feature; a rank of 4 reflects a classic presentation of the feature.

The alcohol exposure scale is based on dose exposure patterns that cause fetal damage in animal models (Astley & Clarren, 2000). A rank of 4 on this scale is given when a woman consumed enough alcohol to cause drunkenness on a weekly basis throughout the first trimester of pregnancy. A rank of 1, on the other hand, is used when there is confirmed absence of drinking from conception to birth (see Astley, 2004, for detailed instructions in determining numeric codes for these key diagnostic features).

By way of example, consider the diagnostic outcomes of two hypothetical youngsters who received the following 4-digit codes: 4444 and 1111. The former code reflects the most severe expression of FASD; that is, significant growth deficiency, full presentation of FAS facial features, structural/neurological evidence of brain damage, and confirmed prenatal high levels of alcohol. The latter code (i.e., 1111) marks the other end of the diagnostic spectrum. This child's code signals normal growth, absence of distinctive facial features, no evidence of brain dysfunction, and confirmed absence of prenatal alcohol exposure. There are 256 possible 4-digit codes, and every combination has been observed in the Washington State FAS/DPN clinics, demonstrating "the continuous nature of alcohol's behavioral teratogenicity" (Mattson & Riley, 1998, p. 279).

**Documenting levels of other adverse exposures and events associated with FASD.** The last two scales in Figure 2 quantify adverse prenatal and postnatal exposures and events. These scales are crucial because there is no way of knowing if the concerns and/or limitations documented in this clinical population were *caused* by maternal consumption of alcohol. In point of fact, prenatal and postnatal risk each have the potential of being responsible for all, part, or none of the observed outcomes. The prenatal risk factor scale is reserved for alternative genetic conditions (e.g., Down syndrome) or teratogenic exposures (e.g., dilantin) that are known to produce physical abnormalities. The postnatal risk factor scale records environmental disruptions that have been documented to have significant adverse effects on cognitive, social, and/or communicative development (Cicchetti et al., 2003; Morriset, Barnard, Greenberg, Booth, & Spieker, 1990).

## Obtaining and Interpreting Data

Three data sets were culled from the clinical ACCESS database to examine the relationship between environmental risk and communicative performance. The three data sets were (a) an adverse environmental risk scale (i.e., postnatal risk), (b) a composite score for standardized language testing, and (c) narrative discourse performance data. Interobserver reliability data were not calculated for these clinical data sets. This limits to some degree the inferences and conclusions that can be developed from this retrospective analysis.

**Adverse postnatal risk factor.** A postnatal risk score was established by an interdisciplinary assessment team. Team members ranked the severity of environmental variables for each participant using the 4-point Likert scale in Figure 2. A participant's postnatal risk rank was based on a comprehensive review of pertinent

medical and social records as well as an in-depth caregiver interview that was conducted at the time of the diagnostic evaluation.

A child whose postnatal circumstances were replete with episodes of abuse and neglect received a rank of 4. Such disruptive conditions have significant adverse effects on development (Astley, 2004). Postnatal abuse and neglect that was less severe, yet could still compromise development across a broad spectrum, was assigned a rank of 3. A 2 rank signaled "unknown" risk. This rank was most often assigned in cases of adopted children, or those in foster care, where relevant information was unavailable. When a well-documented history confirmed an absence of adverse environmental events, a rank of 1 was used.

**Language severity scale.** Overall language performance data were collected and indexed on a language severity scale. The data for this scale were gathered from each participant using norm-referenced, standardized language tests. Because these formal language measures were collected during a 10-year period, not all children received the same standardized test. However, the tests that were administered are widely used, psychometrically sound, and considered appropriate by SLPs who typically use the scores from these tests to compare a particular child's language skills to those of same-age peers. The tests that were used included the Clinical Evaluation of Language Fundamentals (Semel, Wiig, & Secord, 1987, 1995), Test of Auditory Comprehension of Language (Carrow-Woolfolk, 1985, 1999), Test of Language Development (Newcomer & Hammill, 1988, 1997), Test of Language Competence (Wiig & Secord, 1989), and Test of Word Knowledge (Wiig & Secord, 1992).

Standardized test scores (e.g., *z* scores, scaled scores) were indexed on a 3-point Likert scale. Children who obtained scores above  $-1.25$  *SD* from the mean were considered within the normal range of performance (following Leonard, 1998; Owens, 1999; Paul, 1995). Children whose scores fell below this cutoff score were considered outside the bounds of normal variability and were placed into one of two categories. The mildly impaired performance category applied to children who obtained scores that ranged between  $-1.25$  *SD* and  $-2.00$  *SD* from test means. Children with test scores that fell more than  $-2.00$  *SD* from their respective means were categorized as moderately-to-severely impaired (see Fletcher & Miller, 2005 and McCauley, 2001 for a discussion of cutoff scores and inclusion criteria).

Standard scores were selected because they offer information about how each participant compared to an appropriate age group as well as information about the variability of that group (McCauley, 2001; Paul, 1995). To justify aggregating standard scores for comparative purposes, the normative sample for each test was reviewed (following McCauley & Swisher, 1984). The psychometric variables of interest included age, gender, race/ethnicity, geographic location, and parent education (McCauley, 2001). On visual inspection, the respective standardization samples did not appear to differ from each other in meaningful ways that could negatively affect the resulting standard scores. The measures were not only representative of the general U.S. population, but were also deemed a fair comparison for the children with FASD.

**Narrative discourse performance.** Samples of narrative discourse were collected. Narratives are ecologically valid ways of assessing a child's ability to produce meaningful language in socially integrated discourse (Owens, 1999). They require children to make sense of their world through inferencing and perspective taking, thereby providing an important alternative by which to

examine the social communicative processes of school-age children with FASD. Following this line of reasoning, a narrative might reasonably provide a more realistic impression of a child's integrated communicative abilities than will evidence that has been gathered from standardized tests under controlled conditions.

Younger school-age children (i.e., 6;0–7;11) retold *The Bus Story* (Renfrew, 1991). The *Bus Story* has been found to be positively related to a child's future language and literacy performance (Botting, 2002). This clinical measure explores a child's ability to track and modulate a variety of complex linguistic and pragmatic factors. An information score, based on the number of relevant story features and actions a child told, was calculated for each youngster (following Renfrew, 1991). Children who obtained an information score above the 10<sup>th</sup> percentile (i.e., approximately 1.25 *SD* from the mean) were considered within the expected range of performance; children who scored at or below the 10<sup>th</sup> percentile were considered impaired.

Older school-age children (i.e., 8;0–12;11) generated a spontaneous narrative using the wordless picture book, *Frog, Where Are You?* (Mayer, 1969) as the eliciting stimulus. Children became familiar with the general story line as they looked through the *Frog* book. Each child was allowed to use the picture book as a visual prompt while telling the *Frog* story; however, the respective clinician was always seated across the room from the child in order that he or she could not see the storybook. This decontextualized context (Curenton & Justice, 2004) obligated the child to clearly express essential story elements and events to the clinician solely through language. Norbury and Bishop (2003) noted that the stories that older children generate provide a more realistic impression of their "planning and expressive language abilities" (p. 291).

Each *Frog* story was analyzed for two narrative features: story cohesion and story coherence. *Story cohesion* explored whether youngsters were capable of linking a series of related events into a plot structure (Trabasso & Rodkin, 1994). *Story coherence* examined the "informativeness" of the narrative. These analyses were intended to reveal whether a particular child had sufficient command of these two complementary features to relate a satisfying narrative. Thus, a criterion-reference approach was adopted.

The cohesion analysis explored the child's ability to encode a "hierarchical representation" (Norbury & Bishop, 2003, p. 288) of essential story components. The *Frog* story is built around an initiating event that motivates the action of the narrative (i.e., pet frog escapes while boy is sleeping) and five subsequent subplots or story episodes that propel the characters through a series of searches to locate the missing frog. Each story episode contains three hierarchical components: (a) a goal, (b) attempts to achieve the goal, and (c) an outcome. To be credited for sufficient command or mastery of story cohesion, a child was obligated to encode the initiating event and at least two episodes complete with all three components (Coggins et al., 1998).

The coherence analysis explored whether the child possessed sufficient ability to communicate unambiguous information to the listener. An informative utterance established a clear (i.e., unambiguous) link to story entities and events, leaving no doubt in a listener's mind as to what was intended (following Coggins et al., 1998). To be sure, not every response produced by even accomplished narrators might reasonably be expected to always be unambiguous and informative. Thus, children who clearly encoded the essential elements and inferences to at least eight of the 24 *Frog* story picture stimuli (i.e., did not presuppose unwarranted knowledge on the part of the listener) were credited with sufficient informative ability.

## Demographic Data

Substantial prenatal alcohol exposure can adversely affect children in any social class or racial group. Table 1 presents a socio-demographic summary of the 573 school-age participants who were evaluated in the FAS/DPN clinics between 1993 and 2003. This subset of participants represents 36% of all individuals who received interdisciplinary assessments during this 10-year period ( $n = 1,539$ ).

A visual inspection of Table 1 reveals that males accounted for 60% of the sample. Although the ethnicity of biological parents included African American, American Indian, Canadian, and Alaskan Native, almost half (48%) of the parents were both Caucasian. At the time of the assessment, 30% of the youngsters lived with either their biological mother or father; 40% lived with adoptive or foster parents.

The age at which children received their clinical diagnosis is also presented in Table 1. Fifty-four percent of children between the ages of 6;0–8;11 were diagnosed with FASD. These data reveal a trend toward diagnosing children with significant fetal alcohol exposure earlier during the school-age years. An accurate and timely diagnosis is essential for maximizing access to resources while mitigating secondary disabilities associated with prenatal alcohol exposure (Streissguth et al., 1996).

## Spectrum of Clinical Outcomes

For purposes of this study, the 4-digit codes for the 573 participants were organized into four diagnostic categories. These categories and their frequency of occurrence are presented in Table 2. Children in the first category met the clinical diagnosis for FAS

**Table 1.** Summary of the sociodemographic variables for 573 school-age children with fetal alcohol spectrum disorder (FASD).

Characteristic	Frequency	
	Absolute	Proportional
Gender		
Male	346	.60
Female	227	.40
Race		
Both parents Caucasian	277	.48
At least 1 parent Black	80	.14
At least 1 parent American, Canadian, or Alaskan Native	139	.24
All others	77	.13
Caregiver at time of assessment		
Biological mother	109	.19
Biological father	60	.11
Foster parents	110	.19
Adoptive parent	119	.21
Other	150	.26
Age at diagnosis		
6;0–6;11 (years;months)	118	.21
7;0–7;11	93	.16
8;0–8;11	98	.17
9;0–9;11	71	.13
10;0–10;11	67	.12
11;0–11;11	74	.13
12;0–12;11	52	.09

**Table 2.** Four diagnostic categories and their frequency of occurrence for 573 school-age children with FASD.

<i>FASD diagnostic category</i>	<i>Absolute frequency</i>	<i>Proportional frequency</i>
Fetal alcohol syndrome	63	.11
Partial fetal alcohol syndrome		
Static encephalopathy	194	.34
Neurobehavioral disorder	290	.50
No central nervous system dysfunction	26	.05
Total	573	1.00

or partial FAS (i.e., most growth and facial features with abnormal brain functioning). As can be seen in Table 2, 11% of the school-age sample received 4-digit diagnostic codes that met the criteria for FAS or partial FAS.

Thirty-four percent of the sample had 4-digit codes that fell into the static encephalopathy category (with confirmed alcohol exposure). The term *encephalopathy* refers to “any significant abnormal condition of the structure or function of brain tissues” (Anderson, 2002, p. 595); the term *static* means that the abnormality is unchanging. Children in this diagnostic category were identified as having definite abnormalities in brain structure and/or function.

A structural abnormality (e.g., microcephaly) or a “hard” neurological finding (e.g., seizures) is the strongest clinical evidence of static encephalopathy. This level of evidence justified a rank of 4 for brain damage, indicating significant structural abnormalities or “hard” neurological signs (e.g., seizures not due to a postnatal insult). It was, however, far more common for children in this sample to receive a brain rank of 3, indicating abnormal brain functioning. A rank of 3 was assigned to individuals with clinically meaningful deficits in three or more domains of brain functioning (Astley, 2004; Chudley et al., 2005). The domains of particular interest include intelligence, adaptation, academic achievement, language, and neuropsychology. In our clinical experience, using three (or more) clinically meaningful deficits in three (or more) different domains as evidence of diffuse brain damage has solid content validity.

The third category in this classification system is neurobehavioral disorder. The data in Table 2 reveal that 50% of these school-age youngsters presented with histories of behavioral, cognitive, and/or developmental problems, suggestive of central nervous system damage. However, there was no convincing evidence in defense of structural, neurological, or functional deficits, even though these children had confirmed prenatal alcohol exposure.

The fourth category included children with confirmed alcohol exposure but no discernable evidence of central nervous system dysfunction. This category accounted for only 5% of the sample population. Even though these children did not exhibit any functional or developmental problems, some presented with sentinel physical findings. The term *sentinel* refers to key physical findings of FASD that include a unique cluster of minor facial anomalies and/or growth deficiency (i.e., a ranking of 3 or 4 on the 4-digit code).

### Adverse Environmental Experience

Comprehensive postnatal (i.e., environmental) risk data were available for 393 of the 573 school-age children with FASD (i.e., 69%). These adverse environmental factors have been summarized

**Table 3.** The spectrum of adverse environmental risks for 393 school-age children with FASD using the 4-digit diagnostic code (Astley, 2004).

<i>Numeric code (rank)</i>	<i>Level of postnatal risk</i>	<i>Total</i>
4	High	173 (.44)
3	Some	162 (.41)
2	Unknown	39 (.10)
1	Unremarkable	19 (.05)

in Table 3. Of the nearly 400 school-age children in the sample population, 173 (i.e., 44%) had postnatal risk scores of 4. This level of adversity has been shown to disrupt, if not alter, children’s ability to conceptualize and make sense of their social world (Cicchetti et al., 2003). A nearly equal number (i.e., 162) and proportion (i.e., 41%) of children had a postnatal risk score of 3. Children at this level of risk also had documented cases of abuse and/or neglect, with probable adverse effects on development. When postnatal data were unavailable, a rank of 2 was used. This category rank accounted for 10% of the sample data. This ranking occurred most frequently with children who had been adopted, and to a lesser degree, with children in foster care. Only 5% ( $n = 19$ ) of the children in this clinical population received a rank of 1 as a result of well-documented histories where adverse environmental events were absent.

### Language Performance

Adverse postnatal risk and language severity data for the 393 school-age children are presented in Table 4. These data reveal a continuum of language outcomes for children who were exposed to prenatal alcohol and who experienced adverse environments. Inspection of Table 4 reveals that 120 youngsters (i.e., 31%) obtained an overall language performance score that placed them in the mildly impaired range; a compromise, according to McCauley (2001) that is “worthy of attention” (p. 221). It is particularly interesting to note that 84% of these youngsters (i.e., 101/120) had experienced clinically meaningful levels of abuse and/or neglect (i.e., postnatal risk score 3 or 4) in addition to their prenatal alcohol exposure.

Nearly 40% of this sample (i.e., 148) had standard language scores that placed them in the moderately-to-severely impaired category. This level of impairment identifies children who many clinically oriented writers consider outside the range of normal variability (see McCauley, 2001; Owens, 1999; Paul, 1995). Of note, 84% (i.e., 124/148) of youngsters with this level of language impairment had postnatal risk scores of 3 or 4—precisely the same proportion presented by the children with mild language impairment.

Despite the compound risk of prenatal alcohol exposure and atypical social interactive experiences associated with a maltreating environment, 32% ( $n = 125$ ) of this clinical sample achieved standardized language scores within the expected range of performance. The proportion of these youngsters with postnatal risks of 3 or 4 (i.e., 88%) is consistent with the data presented above for youngsters with mild and moderate-to-severe impairments.

### Narrative Discourse Performance

Adverse postnatal risk and narrative discourse data were available for 313 of the 393 FAS/DPN children. Depending on their age,

**Table 4.** Adverse environmental risk and language severity performance data for 393 school-age children with FASD.

Numeric code	Level of postnatal risk	Language severity			Total
		Mildly impaired (-1.25 to -2.00 SD)	Moderately-to-severely impaired (>-2.00 SD)	Normal performance range (-1.25 SD & above)	
4	High	51	65	57	173
3	Probable	50	59	53	162
2	Unknown	12	17	10	39
1	Unremarkable	7	7	5	19
		120	148	125	393

children either retold a narrative or spontaneously generated their own oral narrative. There were 115 school-age children between the ages of 6;0 and 7;11 who retold *The Bus Story* (Renfrew, 1991). Spontaneously generated *Frog, Where Are You?* (Mayer, 1969) narratives were gathered from 198 children between the ages of 8;0 and 12;11. Table 5 presents the results of these narrative analyses.

**Story retell.** The amount of pertinent *Bus Story* information that each child recalled was tallied. As summarized earlier, a child whose score fell at or below the 10<sup>th</sup> percentile was considered to have an impaired ability in recounting pertinent information. The results of this analysis are presented in the upper panel of Table 5 along with levels of postnatal risk data that categorize adverse environmental conditions.

The information scores were evenly divided between youngsters: 57 youngsters achieved a score that reflected an impaired aptitude (i.e., at or below the 10<sup>th</sup> percentile criterion), and 58 produced an adequate number of informational units. More than 90% of youngsters who retold *The Bus Story* (i.e., 106/115) had also experienced episodes of abuse and/or neglect of sufficient magnitude to receive a postnatal risk score of 3 or 4 from the assessment team. Although these levels of environmental disruption put children at clear risk for developmental difficulties, the adverse events do not appear to be systematically related to information scores. This finding suggests that information scores are not sufficient by

themselves to distinguish the full spectrum of compromises in FASD. Botting (2002) has argued that a more detailed examination of subordinate clauses during story retelling may provide a more accurate discrimination.

**Story generation.** Generating a satisfying narrative is a complex task that demands the skillful integration of multiple linguistic, social cognitive, and strategic planning (Reilly, Losh, Bellugi, & Wulfeck, 2004). To determine whether older school-age children with FASD possessed the requisite cohesive (i.e., basic story elements) and coherent (i.e., informativeness) abilities for relating a good *Frog* narrative, we combined these story features data into an overall narrative performance score. Children who coded the initiating event plus two or more story episodes AND communicated unambiguous information to their listener on at least eight of the *Frog* story pictures were considered to have adequate narrative performance ability. Children who did not reach this criterion were viewed as having inadequate narrative performance ability.

Narrative performance *Frog* story data are presented in the lower panel of Table 5. The data reveal that a subgroup of school-age children with FASD ( $n = 54$ , 27%) had sufficient cohesion and coherence ability to produce an integrated story capturing story elements and conveying information effectively to their listener. However, more than 2-1/2 times as many children did not meet this performance standard ( $n = 144$ , 73%). This finding indicates that older

**Table 5.** Adverse environmental risk data and story retell information scores for 115 school-age children with FASD between the ages of 6;0-7;11 (upper panel). Adverse environmental risk data and overall narrative performance scores for 198 school-age children with FASD between the ages of 8;0-12;11 (lower panel).

Numeric code	Level of postnatal risk	Information score The Bus Story (Renfrew, 1991)		
		>10 <sup>th</sup> percentile	≤10 <sup>th</sup> percentile	
4	High	38 (.66)	21 (.37)	59 (.51)
3	Probable	20 (.34)	27 (.47)	47 (.41)
2	Unknown	—	6 (.11)	6 (.05)
1	Unremarkable	—	3 (.05)	3 (.03)
		58 (1.00)	57 (1.00)	115 (1.00)

Numeric code	Level of postnatal risk	Narrative performance score <i>Frog, Where Are You?</i> (Mayer, 1969)		
		Adequate	Inadequate	
4	High	25 (.46)	65 (.45)	90 (.45)
3	Probable	19 (.35)	63 (.44)	82 (.41)
2	Unknown	4 (.08)	10 (.07)	14 (.07)
1	Unremarkable	6 (.11)	6 (.04)	12 (.06)
		54 (1.00)	144 (1.00)	198 (1.00)

school-age children with FASD are quite likely to experience enervating compromises in both the referential aspects (i.e., representation of main story elements) and pragmatic aspects (i.e., ability to determine and convey relevant information) of narrative production.

Adverse environmental risk data in Table 5 reveal profiles similar to those of the younger school-age youngsters. The overwhelming majority of older children had documented instances of postnatal abuse and neglect with probable (numeric rank 3) or highly likely (numeric rank 4) effects on development. Although these atypical social experiences are potent risk factors for language and social communication deficits, they are not linked to narrative discourse performance in a straightforward manner that can easily be revealed in a descriptive study. The data reveal that 81% of FASD children with concerning atypical social experiences had adequate narrative performance scores. A similar proportion of postnatal risk (89%) is reflected for children whose *Frog* stories were judged inadequate.

## DISCUSSION

Children in this retrospective study provide convincing evidence of the comorbidity between FASD and adverse environmental conditions. Although teratogenic levels of prenatal alcohol exposure can disrupt the development and use of language, the sequelae of abuse and neglect is also likely to be a debilitating factor. These comorbid conditions appear to conspire in this clinical population to seriously compromise higher level language and/or social communicative abilities. The magnitude of the problem appears robust.

The findings from this investigation reveal that children with FASD are disproportionately subject to negative or unpredictable caregiving environments. On the basis of our clinical encounters in the Washington State FAS/DPN, it is not uncommon for these children to undergo multiple home placements during their formative years. It is also not uncommon that the biological parents of these children present with co-occurring affective illnesses.

Equally concerning are those children who are living with caregivers who continue to abuse alcohol, thereby placing the children at considerable risk for physical, sexual, and/or emotional abuse. Cicchetti and Rizley (1981) reported that children who experienced three or more of types of abuse (i.e., physical, sexual, emotional) and/or neglect were most likely to present deviations in brain structure and function. Moreover, Eigsti and Cicchetti (2004) found that children of maltreating mothers had "less complex" language than did a group of nonmaltreated peers in more naturalistic social contexts.

Social communication looms as a key deficit in children with FASD. To be sure, the mechanism for how early chaotic environments serve to disrupt social communication is not yet well understood. Certainly, living in an unpredictable environment where positive, nurturing, and responsive interactions are minimal would seem to adversely affect children's ability to self-regulate and predict other's moods, intentions, and actions. In this context, prenatal alcohol exposure and adverse environments would be expected to have deleterious effects on children's social-cognitive skills and higher order executive functions and, in turn, the words and actions that children use to inform others and manage social relationships. As such, we believe that deficits in using language are reflections of underlying compromises in how social cognition, language,

and executive functions fuse together to meet the demands of varying social interactions.

Children with FASD, perhaps more than any clinical population, live in an extended state of double jeopardy due to the timing, quantity, and pattern of maternal drinking and the frequently co-occurring adverse effects of dysfunctional caregiving. In this article, we have provided SLPs with a perspective for understanding this complex clinical profile and how these two conditions seem to co-occur and conspire to disrupt language and social communication. Because multiple risks contribute to multiple deficits, we endorse a comprehensive assessment of social communication even when children perform within the normal range on standardized language measures. Clinicians should seriously consider more integrative tasks in their assessments that mimic the demands of everyday social interactions such as narratives and observations of peer interactions (Olswang, Svensson, Coggins, Beilinson, & Donaldson, 2006).

Assessment strategies for eliciting and sampling integrated performance of school-age children in real time are beginning to appear in the literature (Olswang, Coggins, & Timler, 2001). These approaches recognize the dynamic and multidimensional nature of communication, particularly the interaction between the child and the social environment. The shift toward more dynamic and interactive assessment paradigms appears to be a promising means of documenting the language and social communication deficits in children with complex profiles.

## ACKNOWLEDGMENTS

This project was facilitated by Grant 6T73MC00041-11-01 from the Interdisciplinary Leadership Training in Neurodevelopmental and Related Disabilities, HRSA MCH Bureau, John F. McLaughlin, MD, Principal Investigator; Grant P30 HD0227 from the National Institute of Child Health and Human Development, Michael Guralnick, PhD, Principal Investigator; and Grant 8945-0 from the FAS Diagnostic & Prevention Network, Washington State Division of Alcohol and Substance Abuse, Susan J. Astley, PhD, Principal Investigator. Special thanks to Kristin Daniels and our colleagues at the University of Washington FAS/DPN.

## REFERENCES

- Abkarian, G. (1992). Communication effects of prenatal alcohol exposure. *Journal of Communication Disorders, 25*, 221-240.
- Anderson, D. (2002). *Mosby's medical nursing and allied health dictionary* (6th ed.). St. Louis, MO: Mosby.
- American Speech-Language-Hearing Association. (2000). *Guidelines for the roles and responsibilities of school-based speech-language pathologists*. Rockville, MD: Author.
- Astley, S. (2004). *Diagnostic guide for fetal alcohol spectrum disorders* (3rd ed.). Seattle: University of Washington Fetal Alcohol Syndrome Diagnostic and Prevention Network.
- Astley, S., & Clarren, S. (2000). Diagnosing the full spectrum of fetal alcohol exposed individuals: Introducing the 4-digit diagnostic code. *Alcohol & Alcoholism, 35*, 400-410.
- Astley, S., & Clarren, S. (2001). Measuring the facial phenotype of individuals with prenatal alcohol exposure: Correlations with brain dysfunction. *Alcohol & Alcoholism, 36*, 147-159.
- Baron-Cohen, S. (2000). Theory of mind and autism: A fifteen year review. In S. Baron-Cohen, H. Tager-Flusberg, & D. Cohen (Eds.), *Understanding*

- other minds: Perspectives from developmental cognitive neuroscience (pp. 3–20). New York: Oxford University Press.
- Becker, M., Warr-Leeper, G., & Leeper, H.** (1990). Fetal alcohol syndrome: A description of oral motor, articulatory, short term memory, grammatical, and semantic abilities. *Journal of Communication Disorders*, 23, 97–124.
- Botting, N.** (2002). Narrative as a tool for the assessment of linguistic and pragmatic impairments. *Child Language and Teaching Therapy*, 18(1), 1–21.
- Bundy, A., Lane, S., & Murray, E.** (2002). *Sensory integration: Theory and practice*. Philadelphia: F.A. Davis.
- Caldwell, S.** (1993). Nurturing the delicate rose. In J. Kleinfeld & S. Wescott (Eds.), *Fantastic Antone succeeds! Experiences in educating children with fetal alcohol syndrome* (pp. 97–129). Anchorage: University of Alaska Press.
- Carmichael Olson, H., Feldman, J. J., Streissguth, A. P., Sampson, P. D., & Bookstein, F. L.** (1998). Neuropsychological deficits in adolescents with fetal alcohol syndrome: Clinical findings. *Alcoholism: Clinical and Experimental Research*, 20, 1998–2012.
- Carney, L., & Chermak, G.** (1991). Performance of American Indian children with fetal alcohol syndrome on the Test of Language Development. *Journal of Communication Disorders*, 24, 123–134.
- Carrow-Woolfolk, E.** (1985). *Test of Auditory Comprehension of Language-R*. Austin, TX: Pro-Ed.
- Carrow-Woolfolk, E.** (1999). *Test of Auditory Comprehension of Language-3*. Austin, TX: Pro-Ed.
- Chudley, A., Conry, J., Cook, J., Looock, C., Rosales, T., & LeBlanc, N.** (2005). Fetal alcohol spectrum disorder: Canadian guidelines for diagnosis. *Canadian Medical Association Journal*, 172(Suppl. 5), 1–21.
- Church, M., Eldis, F., Blakley, B., & Bawle, E.** (1997). Hearing, language, speech, vestibular, and dentofacial disorders in fetal alcohol syndrome. *Alcoholism: Clinical and Experimental Research*, 2, 227–237.
- Church, M., & Kaltenbach, J.** (1997). Hearing, speech, language, and vestibular disorders in the fetal alcohol syndrome: A literature review. *Alcoholism: Clinical and Experimental Research*, 21, 495–512.
- Cicchetti, D.** (2004). An odyssey of discovery: Lessons learned through three decades of research on child maltreatment. *American Psychologist*, 58, 731–741.
- Cicchetti, D., & Rizley, R.** (1981). Developmental perspectives on the etiology, intergenerational transmission, and sequelae of child maltreatment. *New Directions for Child Development*, 11, 32–59.
- Cicchetti, D., Rogosch, F., Maughan, A., Toth, S., & Bruce, J.** (2003). False-belief understanding in maltreated children. *Developmental and Psychopathology*, 15, 1067–1091.
- Clarren, S., Carmichael Olson, H., Clarren, S., & Astley, S.** (2000). A child with fetal alcohol syndrome. In M. Guralnick (Ed.), *Interdisciplinary clinical assessment of young children with developmental disabilities* (pp. 307–326). Baltimore: Paul H. Brookes.
- Coggins, T.** (1997, July). *Assessment of language and social communication in FAS*. Paper presented at the Prevention and Management of Fetal Alcohol Syndrome and Prenatal Substance Abuse Conference, Breckenridge, CO.
- Coggins, T., Friet, T., & Morgan, T.** (1998). Analyzing narrative productions in older school-age children and adolescents with fetal alcohol syndrome: An experimental tool for clinical applications. *Clinical Linguistics & Phonetics*, 12, 221–236.
- Coggins, T., Olswang, L., Carmichael Olson, H., & Timler, G.** (2003). On becoming socially competent communicators: The challenge for children with fetal alcohol exposure. *International Review of Research in Mental Retardation*, 27, 121–150.
- Coster, W., Gersten, M., Beeghly, M., & Cicchetti, D.** (1989). Communicative functioning in maltreated toddlers. *Developmental Psychopathology*, 25, 1020–1029.
- Curenton, S., & Justice, L.** (2004). African American and Caucasian preschoolers' use of decontextualized language: Literate language features in oral narratives. *Language, Speech, and Hearing Services in Schools*, 35, 240–253.
- Eigsti, I., & Cicchetti, D.** (2004). The impact of child maltreatment on expressive syntax at 60 months. *Developmental Science*, 7, 88–102.
- Fletcher, P., & Miller, J.** (2005). *Developmental theory and language disorders*. Amsterdam: John Benjamins.
- Gentry, B., Griffith, L., & Dancer, J.** (1998). Prenatal alcohol exposure and communication, behavior and nonverbal intelligence of 3 school-age children. *Perceptual and Motor Skills*, 86, 1089–1090.
- Guralnick, M.** (1999). Family and child influences on the peer-related social competence of young children with developmental delays. *Mental Retardation and Developmental Disabilities*, 5, 21–29.
- Hamilton, M.** (1981). *Linguistic abilities of children with fetal alcohol syndrome*. Unpublished doctoral dissertation. University of Washington, Seattle.
- Health Resources and Services Administration.** (2005). *Women's health USA 2005*. Rockville, MD: U.S. Department of Health and Human Services.
- Hinde, J.** (1993). Early intervention for alcohol-affected children. In J. Kleinfeld & S. Wescott (Eds.), *Fantastic Antone succeeds! Experiences in education children with fetal alcohol syndrome* (pp. 131–147). Anchorage: University of Alaska Press.
- Institute of Medicine.** (1996). Division of Biobehavioral Sciences and Mental Disorders, Committee to Study Fetal Alcohol Syndrome. In K. Stratton, C. Howe, & F. Battaglia (Eds.), *Fetal alcohol syndrome: Diagnosis, epidemiology, prevention, and treatment* (pp. 82–99). Washington, DC: National Academic Press.
- Jacobson, S., & Jacobson, J.** (2000). Teratogenic insult and neuro-behavioral function in infancy and childhood. In C. Nelson (Ed.), *Minnesota symposia on child psychology* (pp. 61–112). Hillsdale, NJ: Erlbaum.
- Janzen, L., Nanson, J., & Block, G.** (1995). Neuropsychological evaluation of preschoolers with fetal alcohol syndrome. *Neurotoxicology and Teratology*, 17, 273–279.
- Kaufman, J., Plotsky, P., Nemeroff, C., & Charney, D.** (2000). Effects of early adverse experiences on brain structure and function: Clinical implications. *Biological Psychiatry*, 48, 778–790.
- Kelly, S., Day, N., & Streissguth, A.** (2000). Effects of prenatal alcohol exposure on social behavior in humans and other species. *Neurotoxicology and Teratology*, 22, 143–149.
- Kerns, J., Don, A., Mateer, C., & Streissguth, A.** (1997). Cognitive deficits in nonretarded adults with fetal alcohol syndrome. *Journal of Learning Disabilities*, 30, 685–693.
- Kodituwakku, P., Handmaker, N., Cutler, S., Weathersby, E., & Handmaker, S.** (1995). Specific impairments in self-regulation in children exposed to alcohol prenatally. *Alcoholism: Clinical & Experimental Research*, 19, 1558–1564.
- Kodituwakku, P., May, P., Ballinger, L., Harris, M., Aase, J., & Aragon, A.** (1997, July). *Executive controlling functioning and theory of mind in children prenatally exposed to alcohol*. Paper presented at the Prevention and Management of Fetal Alcohol Syndrome and Prenatal Substance Abuse Conference, Breckenridge, CO.
- Kopara-Frye, K., Dehaene, S., & Streissguth, A.** (1996). Impairments of number processing induced by prenatal alcohol exposure. *Neuropsychologia*, 34, 1187–1197.

- Leonard, L. (1998). *Children with specific language impairment*. Cambridge, MA: MIT Press.
- Lohmann, H., & Tomasello, M. (2003). The role of language in the development of false belief understanding: A training study. *Child Development, 74*, 1130–1144.
- Mattson, S., & Riley, E. (1998). A review of the neurobehavioral deficits in children with fetal alcohol syndrome or prenatal exposure to alcohol. *Alcoholism: Clinical and Experimental Research, 22*, 279–294.
- Mattson, S. N., Goodman, A. M., Caine, C., Delis, D. C., & Riley, E. P. (1999). Executive functioning in children with heavy prenatal alcohol exposure. *Alcoholism: Clinical and Experimental Research, 23*, 1808–1815.
- Mayer, M. (1969). *Frog, where are you?* New York: Dial Books for Children.
- McCauley, E. (2001). *Assessment of language disorders in children*. Mahwah, NJ: Erlbaum.
- McCauley, E., & Swisher, L. (1984). Psychometric review of language and articulation tests for preschool children. *Journal of Speech and Hearing Research, 49*, 34–42.
- McFadyen, R., & Kitson, W. (1996). Language comprehension and expression among adolescents who have experienced childhood physical abuse. *Journal of Child Psychology, Psychiatry & Allied Disciplines, 37*, 551–562.
- Morriset, C., Barnard, K., Greenberg, M., Booth, K., & Spieker, S. (1990). Toddlers' language development: Sex differences within social risk. *Developmental Psychology, 31*, 851–865.
- Newcomer, P., & Hammill, D. (1988). *Test of Language Development–P*. Austin, TX: Pro-Ed.
- Newcomer, P., & Hammill, D. (1997). *Test of Language Development–P:3*. Austin, TX: Pro-Ed.
- Norbury, C., & Bishop, D. (2003). Narrative skills of children with communication impairments. *International Journal of Language and Communication Disorders, 38*, 287–313.
- Olswang, L., Coggins, T., & Timler, G. (2001). Outcome measures for school-age children with social communication problems. *Topics in Language Disorders, 22*, 50–73.
- Olswang, L., Svensson, L., Coggins, T., Beilinson, J., & Donaldson, A. (2006). Reliability issues and solutions for coding social communication events in classroom settings. *Journal of Speech, Language, and Hearing Research, 49*, 1058–1071.
- Owens, R. (1999). *Language disorders: A functional approach to assessment and intervention* (3rd ed.). Boston: Allyn & Bacon.
- Paul, R. (1995). *Language disorders from infancy through adolescence: Assessment and intervention*. St. Louis, MO: Mosby Yearbook.
- Perner, J. (1991). *Understanding the representational mind*. Cambridge, MA: MIT Press.
- Plumridge, D., Bennett, R., Dinno, N., & Branson, C. (1993). *The student with a genetic disorder: Educational implications for special education teachers and for physical therapists, occupational therapists and speech pathologists*. Springfield, IL: Charles C. Thomas.
- Reilly, J., Losh, M., Bellugi, U., & Wulfeck, B. (2004). "Frog, where are you?" Narratives in children with specific language impairment, early focal brain injury, and Williams syndrome. *Brain and Language, 88*(2), 163–257.
- Renfrew, C. (1991). *The bus story* (2nd ed.). Oxford, England: Author.
- Semel, E., Wiig, E., & Secord, W. (1987). *Clinical Evaluation of Language Fundamentals–R*. San Antonio, TX: The Psychological Corporation.
- Semel, E., Wiig, E., & Secord, W. (1995). *Clinical Evaluation of Language Fundamentals–3*. San Antonio, TX: The Psychological Corporation.
- Silliman, E., Diehl, S., Bahr, R., Hnath-Chisolm, T., Zenko, C., & Friedman, S. (2003). A new look at performance on theory-of-mind tasks by adolescents with autism spectrum disorder. *Language, Speech, and Hearing Services in Schools, 34*, 236–252.
- Singer, B., & Bashir, A. (1999). What are executive functions and self-regulation and what do they have to do with language-learning disorders? *Language, Speech, and Hearing Services in Schools, 30*, 265–273.
- Streissguth, A. (1997). *Fetal alcohol syndrome: A guideline for families and communities*. Baltimore: Paul H. Brookes.
- Streissguth, A., Barr, H., Kogan, J., & Bookstein, F. (1996). *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 on Grant No. R04/CCR008515* (Tech. Report No. 96060). Seattle: University of Washington Fetal Alcohol and Drug Unit.
- Streissguth, A., & Kanter, J. (1997). *The challenge of fetal alcohol syndrome: Overcoming secondary disabilities*. Seattle: University of Washington Press.
- Tager-Flusberg, H. (1993). What language reveals about the understanding of minds in children with autism. In S. Baron-Cohen, H. Tager-Flusberg, & D. Cohen (Eds.), *Understanding other minds: Perspectives from children with autism* (pp. 138–157). Oxford, England: Oxford University Press.
- Thomas, S., Kelly, S., Mattson, S., & Riley, E. (1998). Comparison of social abilities of children with fetal alcohol syndrome to those of children with similar IQ scores and normal controls. *Alcoholism: Clinical and Experimental Research, 22*, 528–533.
- Thorne, J., Coggins, T., Carmichael Olson, H., & Astley, S. (in press). Exploring the utility of narrative analysis in diagnostic decision-making: Elaboration, reference strategy, and fetal alcohol spectrum disorder. *Journal of Speech, Language, and Hearing Research*.
- Timler, G., & Olswang, L. (2001). Variable structure/variable performance: Caregiver and teacher perspectives of a school-age child with fetal alcohol syndrome. *Journal of Positive Behavior Intervention, 3*, 48–56.
- Timler, G., Olswang, L., & Coggins, T. (2005). "Do I know what I need to do?": A social communication intervention for children with complex clinical profiles. *Language, Speech, and Hearing Services in Schools, 36*, 73–84.
- Trabasso, T., & Rodkin, P. (1994). Knowledge of goal/plans: A conceptual basis for narrating Frog, where are you? In R. Berman & D. Slobin (Eds.), *Relating events in narrative: A cross-linguistic developmental study* (pp. 85–108). Hillsdale, NJ: Erlbaum.
- Wellman, H. (1990). *The child's theory of mind*. Cambridge, MA: MIT Press.
- Weinberg, N. (1997). Cognitive and behavioral deficits associated with prenatal alcohol use. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*, 1177–1186.
- Wiig, E., & Secord, W. (1989). *Test of Language Competence*. San Antonio, TX: The Psychological Corporation.
- Wiig, E., & Secord, W. (1992). *Test of Word Knowledge*. San Antonio, TX: The Psychological Corporation.
- Willis, D., & Silovsky, J. (1998). Prevention of violence at the societal level. In P. Trickett & C. Schellenach (Eds.), *Violence against children in the family and the community* (pp. 401–416). Washington, DC: American Psychological Association.

Received September 30, 2005

Accepted May 25, 2006

DOI: 10.1044/0161-1461(2007/012)

Contact author: Truman E. Coggins, 1417 N.E. 42<sup>nd</sup> Street, Seattle, WA 98105. E-mail: tec@u.washington.edu