PREFACE

During the mid-1980s, the media warned that "crack babies" would be a lost generation. The media hype surrounding these children evoked images of underweight infants who trembled or were rigid and who were unable to form social attachments. It was predicted that children who were exposed to cocaine *in utero* would not be able to function adequately at school or in society. In contrast to these dire predictions, the first generation of clinical research suggested no significant developmental differences, and many argued that these children were no different from other children growing up in similar environments. The second generation of clinical research, however, has suggested that the picture is far from simple. Although prenatal cocaine exposure does not result in severely retarded cognitive or emotional development, it may have more subtle linguistic, cognitive, and social effects in at least a percentage of cocaine-exposed children. Whether or not such effects are manifest in any particular child and if they are, what the particular nature and severity of the deficits are, is dependent on a complex interaction of a range of biological and environmental risk factors. For example, critical variables appear to be how much cocaine the mother abused during pregnancy as well as the type of environment in which the child is raised.

The study of the effects of prenatal cocaine exposure on child development is complicated by the many methodological challenges that face researchers in this area. To attribute developmental effects to fetal cocaine exposure, a number of confounding variables need to be controlled. These include the use of drugs other than cocaine during pregnancy, such as tobacco, alcohol, and other illicit drugs; differences in the social environment of cocaine abusers such as poverty, prenatal care, housing, and domestic violence; and differences in the physical and emotional health of cocaine abusers in terms of nutrition, psychiatric illness, sexually transmitted diseases, and stress. Another methodological challenge concerns the specific abilities tested and the methods used for evaluating behavior. Some of the original findings of no developmental differences in cocaine-exposed and non-exposed children may be attributable to the use of tests that did not sample affected areas or were too insensitive to identify differences. If areas vulnerable to disruption are not evaluated, differences will not be found. For example, in studies of language development, many studies have found no developmental differences in expressive language when evaluated on formal standardized tests or developmental scales, but have found differences in language abilities when evaluated in spontaneous language through language sampling analysis. A further example is that while children with prenatal cocaine-exposure may have no difficulty with certain tasks under simple testing conditions, they may have difficulty under more complex conditions. An important question is not just how these children do in quiet controlled testing situations, but also how they do in the real world where distractions abound and the social, linguistic, and academic demands made on the child may be high.

In this issue of *Seminars in Speech and Language,* some of the latest research on the effects of *in utero* exposure to cocaine and associated risk factors is discussed. The articles provide an overview of the effects of fetal cocaine and other drug exposure

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on neurological, neurobehavioral, language, and play development. In the first article, Kosofsky provides an overview of cocaine-induced alterations in neurological development. He discusses the effects of cocaine on fetal brain development and offers an explanation of clinical findings in terms of an altered aminergic system. A selective review of the clinical data is provided which covers such topics as prenatal effects, perinatal behavior, neurologic development, neurophysiologic studies, language development, cognition, and behavior. He discusses future directions for research in terms of the clinical questions that can be asked at the pharmacologic/physiologic, developmental, systems, and behavioral/cognitive levels.

In the second article, Lester, La Gasse, and Bigsby provide an insightful, comprehensive, and critical evaluation of current views on the effects of prenatal cocaine use and child outcome, issues that still need to be investigated, and specific treatment implications. In the first section of the article, the authors describe a database of the characteristics and findings of published studies and use this as the basis for discussing such topics as methodological issues, neurobehavioral outcome, and themes that reflect the increasing sophistication of the field. In the next section, they discuss how what we have learned from the study of preterm infants can be applied to the study of cocaine-exposed children and emphasize the importance of not jumping to premature conclusions, the heterogeneity of the population and subtlety of effects, and the value of the use of multiple risk models. In their discussion of the clinical implications of the problem, they demonstrate that small mean differences between groups can translate into large and meaningful differences in the number of children affected and referred for clinical services. They also show that the effects of drug-exposure are best identified through assessments of specific function, such as language, rather than through the use of global measures. In their discussion of assessment and intervention implications they point out that what distinguishes this group of children from other at risk children is the issue of parental addiction and describe several models for and approaches to intervention that take this and the child's social environment into account.

In the third and fourth articles, the effects of prenatal cocaine exposure on language development and clinical implications for children with high risk profiles are discussed. In the third article, Mentis provides a critique and general overview of the literature on early communicative and language development. The reasons why language may be compromised, including the impact of biological and environmental risk factors and the relationship between language deficits and other neurobehavioral developmental deficits, are discussed. Studies which have evaluated language development are reviewed and results vary from findings of no differences, to findings of problems in receptive language, expressive language, and specific expressive domains such as pragmatics. The results are also discussed from a clinical perspective in terms of specific assessment and treatment implications. In the fourth article, Bland-Stewart, Seymour, Beeghly, and Frank, provide a detailed examination of differences in the expressive semantic development of a group of African-American children prenatally exposed to cocaine. Methodologically, this study has many strengths compared to other studies in the area including the longitudinal design of the larger study of which this was a part, the methods used for subject selection, and the use of blinded testing procedures. General language and cognitive data and detailed semantic analysis data of 22 children are provided. The results are discussed in relation to the clinical significance of the delayed semantic development of the cocaine-exposed children and similarities and differences between their semantic profiles and those that characterize "normal" development. Diagnostic and treatment strategies that target delayed semantic development are presented. This article highlights the importance of the use of appropriately sensitive and focused as-

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sessment measures in the evaluation of cocaine-exposed children.

In the fifth article, Lundgren discusses the effects of fetal cocaine exposure on the development of play. Delayed and deviant developmental play profiles are described and the effects of both biological and environmental factors on the development of play are explored. Clinical implications are discussed in terms of the need to use sensitive and focused assessment measures that target specific play behaviors that are at risk for impairment and the importance of play in relation to a child's social, cognitive, and language development.

It is too soon to draw definitive conclusions regarding the effects of prenatal cocaine-exposure on child development. Clearly, the frightening predictions of the 1980's have not come to pass. It has become evident that the problem is a complicated and multifactorial one and that outcome is the result of a dynamic interplay among a wide range of risk and protective factors. It is not possible to talk about the effects of cocaine alone on development, but only about the effects of cocaine in combination with other drugs and associated risk factors. It appears that a subset rather than all exposed children may demonstrate effects, that specific areas of function may be more affected than others, and that the effects are subtle rather than gross. There are still a number of questions that need to be answered. One set of questions concerns the particular role that biological and environmental risk and protective variables play in determining outcome. Another set concerns whether and to what extent the effects that have been identified are clinically significant and penalize the child in real world social and academic contexts.

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Guest Editor

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