8-1-2004

Speech/Language and Social Behavior Performance as Predictors of Literacy Acquisition in Children with Fetal Alcohol Syndrome/Fetal Alcohol Effects

Kristen Ann Runge

SPEECH/LANGUAGE AND SOCIAL BEHAVIOR PERFORMANCE AS PREDICTORS OF LITERACY ACQUISITION IN CHILDREN WITH FETAL ALCOHOL SYNDROME/FETAL ALCOHOL EFFECTS

by

Kristen Ann Runge
Bachelor of Arts, Bethel College, 1981
Master of Arts, Grace Theological Seminary, 1987

A Dissertation

Submitted to the Graduate Faculty

of the

University of North Dakota

in partial fulfillment of the requirements

for the degree of

Doctor of Philosophy

Grand Forks, North Dakota
August
2004

This dissertation, submitted by Kristen Ann Runge in partial fulfillment of the requirements for the Degree of Doctor of Philosophy from the University of North Dakota, has been read by the Faculty Advisory Committee under whom the work has been done and is hereby approved.

Richard D. Landry
Chairperson

Glenn W. Olson

Ann B. Walker

Jan E. Stubbe

This dissertation meets the standards for appearance, conforms to the style and format requirements of the Graduate School of the University of North Dakota, and is hereby approved.

Joseph P. Benoit
Dean of the Graduate School

July 30 2009
Date

PERMISSION

Title Speech/Language and Social Behavior Performance as Predictors of Literacy Acquisition in Children With Fetal Alcohol Syndrome/Fetal Alcohol Effects

Department Teaching and Learning

Degree Doctor of Philosophy

In presenting this dissertation in partial fulfillment of the requirements for a graduate degree from the University of North Dakota, I agree that the library of this University shall make it freely available for inspection. I further agree that permission for extensive copying for scholarly purposes may be granted by the professor who supervised my dissertation work or, in his absence, by the chairperson of the department or the dean of the Graduate School. It is understood that any copying shall not be allowed without my written permission. It is also understood that due recognition shall be given to me and to the University of North Dakota in any scholarly use which may be made of any material in my dissertation.

Signature Kristin Runge

Date July 15, 2004

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

ACKNOWLEDGMENTS

I would like to thank Dickinson State University for financial support which enabled me to complete this degree program.

Special thanks to Christy Johansen for innumerable acts of friendship which provided invaluable moral and practical support.

This work is gifted to my children, Kevin, Kelly, and Ryan, in the hope that its completion will serve to break ground for the fulfillment of their own unique callings, no matter how intimidating they might appear at the onset.

ABSTRACT

This study was a review of existing case files and focused on the struggles of one particular segment of the special needs student population and the challenges that educators face as they attempt to engage them in meaningful learning experiences. Fetal Alcohol Syndrome/Fetal Alcohol Effects (FAS/FAE) are completely preventable; yet, their presence in classrooms remains high and bears profound implications for educators. This study examined the specific relationships between FAS/FAE and literacy acquisition in children. Specific variables of children with FAS/FAE were considered. They included IQ, speech/language development, and social behavior performance.

Hypotheses guiding this study were the following:

1. Evidence of developmentally delayed speech/language functions and social behavior performance relates highly with literacy acquisition in children with FAS/FAE.
2. Expressive and receptive speech/language functions are expected to be the most critical indicator of literacy performance in children with FAS/FAE.
3. IQ will evidence itself as a factor in literacy acquisition in children with FAS/FAE.

Case files reviewed represented youngsters from the Midwest region, identified through a variety of screening and community services. These subjects have been monitored for several years by a northern plains research university and hospital. Cases represent both genders as well as a variety of racial and socioeconomic backgrounds. Initial referral came from schools, childcare providers, social service personnel, health care workers or physicians, and occasionally from parents themselves. Information gathered included general health and psychological analyses, as well as specific developmental evaluations. Based on the cases reviewed, it generally appeared that social behavior bears slightly more influence on literacy acquisition than does speech/language development.

A variety of conclusions and recommendations were offered. Collaboration between social and health care agencies must be improved. Educational entities must continue to develop effective instructional practices which meet the needs of this particular group of youngsters. Yet, without question, prevention efforts are the only reasonable response and must receive our most concerted efforts.

CHAPTER I

INTRODUCTION

Background of the Study

Literacy instruction is at the core of American elementary schools. Without the ability to read and write effectively, no one can hope to participate fully in society. Federal as well as local initiatives continue to insist that teachers do a better job of reaching and successfully teaching the nation's youth these valuable skills. In fact, some estimates show that of all learners reaching high school, a full 60% are reading at or below a 6th-grade level (Richardson & Morgan, 2000). As a whole, literacy achievement scores in this nation are, indeed, a travesty, given the opportunity and resources with which the country has to work.

Produced by the federal government, No Child Left Behind (NCLB) is a sweeping legislative act that is causing much concern in the teaching and administrative hearts of schools from coast to coast. It is perhaps the most comprehensive and, in fact, intimidating piece of legislation to land on the doorsteps of public schools in recent history. In effect, the federal government is demanding accountability on the part of schools in America, to provide proof of success in their overall program, with particular attention being given to literacy (*No Child Left Behind,* 2001).

Although accountability is not a new issue, the specific mandates of this initiative are unprecedented. As never before, schools are being assessed on quality of output. What has come to be called "high-stakes" testing determines a pass/fail status for schools, resulting in loss or continuance of funding, staff retention or replacement, and even closure, or government takeover of the schools themselves. Staff quality is being redefined by new licensure requirements as well as demands for recertification expectations for existing staff. All children must reach minimum literacy performance standards by third grade, or schools will be forced to demonstrate adequate yearly progress toward that goal to avoid reprimands (*No Child Left Behind,* 2001).

The Problem

Though the federal government has been quite forceful in declaring their expectations, many of the specifics of policy implementation remain uncertain. State legislatures, local school boards, and teacher education programs are scrambling madly to shape a response plan to satisfy expectations, and yet remain viable in their particular contexts. To many, the suggestion that schools across America could hope to produce a standard output, as though they were automobile manufacturing plants, is nothing short of laughable. And yet, it would seem that this is exactly the challenge before them.

One particular outcry that can be heard rising from faculty meetings is concern about assessment of underachieving students due to particular learning difficulties. Many teachers of special needs students are expressing concern, stating that NCLB expectations are unreasonable, given the student population

that they have to work with (National Association of State Directors of Special Education, 2002). Many feel they are already doing all they can to reach slow or disabled learners, students with language differences, or those with particularly difficult home environments.

Statement of Purpose

This study was a review of existing case files and focused on the struggles of one particular segment of the special needs student population, children with Fetal Alcohol Syndrome/Fetal Alcohol Effects (FAS/FAE), and the challenges that these children face as they attempt to engage in learning. Difficulties experienced by this particular group of children are complex and most certainly have their beginning in the home. In fact, the challenges these children experience are initiated before birth in the darkness of their mothers' womb. These difficulties carry over into the children's school experiences, and indeed throughout their lives.

FAS/FAE have been identified as the leading cause of mental retardation in America (Burd, 2001). Despite the fact that their devastating incidence is completely preventable, their presence in classrooms remains high, and bears profound implications for educators. This study examined the specific relationships between FAS/FAE and literacy acquisition in children. Specific variables of children with FAS/FAE were considered. They included IQ, speech/language development, and social behavior performance.

Preliminary Review of Literature

Whether a woman becomes pregnant through the bonds of matrimony in the context of a loving relationship, or in the frenzy of backseat passion with a boyfriend, or, more sadly, as a young girl overpowered in her own bedroom, a new life begins. Both external and internal environments that surround the developing child bear enormous implications on the health and well-being of both immediate and future quality of life.

With relation to this study, healthy central nervous system development is critical if successful outcomes are to be expected with relation to literacy acquisition. Literacy acquisition is dependent upon both cognitive processing and the ability to relate successfully with the environment. Fundamental to preparation for literate activity is one's ability to interact successfully with others, a skill which enables one to value and develop communication skills which can ultimately be transferred into acts of reading and writing.

From birth, and even before, humans are conditioned for communication (Piper, 1998). Language learning initiates long before first words are ever spoken. As infants are surrounded by an environment of communication, they begin to assimilate the constructs of their mother tongue. Syntax, semantics, and pragmatics are internalized, together with early concepts of graphophonemic relationships (Goodman, Watson, & Burke, 1987). From this very practical world of verbal exchange, humans enter the world of abstracts, learning to harness sophisticated nuances of meaning to the printed page.

As a communicative act, literacy requires complex manipulation of information (Adams, 1990). Most basically defined, literacy is the process of assessing text symbols and environmental cues and translating them into meaning. Exchange of ideas must be scaffolded and negotiated, blending prior knowledge with new ideas. Facility with language is fundamental to literate activity.

Social maturity bears implications on literacy acquisition as well. Without a doubt, social maturity is a product of the collected experiences of one's home and peer interactions. However, one's ability to interact socially is also impacted by biological predispositions that influence how one relates to others. For example, Attention Deficit Disorder can interfere with one's ability to interact successfully with others. Also, the ability to attend and participate in communicative acts with others serves to advance comprehension and sophistication of language skills (Piper, 1998).

The communicative acts of reading and writing have their base in the neurological functionings of the brain. When brain development is interrupted by in utero toxin exposure, results are profound. Recent advancements in brain imaging provide new understanding of the interrelationships between neurological structures and behavioral outcomes.

In the case of alcohol exposure, one might describe the experience of a fetus as having been bathed in ethanol (Dorris, 1989). While concentration and frequency of exposure are significant, ethanol is toxic in any amount to a developing embryo. Interestingly, twins have been observed to respond

differently to prenatal alcohol exposure based on genotype (Streissguth, Bookstein, Sampson, & Barr, 1993). Nevertheless, some degree of central nervous system damage is inevitable when exposed to in utero alcohol. Before a woman even discovers she is pregnant, as early as the 11th day of gestation, neural cell formation can be impacted by the presence of alcohol in the bloodstream.

These and other environmental components impact learners long before they enter a classroom, often predisposing individuals to ease or difficulty in learning. Understanding environmental outcomes can serve to guide educators and other stakeholders in effective planning, practice, and assessment.

Research Questions

With these considerations in mind, the present study investigated and answered the following questions:

1. What is the relationship between FAS/FAE and IQ?
2. What is the relationship between FAS/FAE and speech/language development?
3. What is the relationship between FAS/FAE and social behavior?
4. What is the relationship between FAS/FAE and literacy acquisition?
5. What are the levels of literacy of a sample of children diagnosed with FAS/FAE?
6. How does IQ relate to literacy acquisition in children with FAS/FAE?
7. How does speech/language development relate to literacy acquisition in children with FAS/FAE?

8. How does social behavior relate to literacy acquisition in children with FAS/FAE?

Hypotheses

Hypotheses guiding this study were the following:

1. Evidence of developmentally delayed speech/language functions and social behavior performance relate highly with literacy acquisition in children with FAS/FAE.
2. Expressive and receptive speech/language functions are expected to be the most critical indicator of literacy performance in children with FAS/FAE.
3. IQ will evidence itself as a factor in literacy acquisition in children with FAS/FAE.

Significance of the Study/Rationale for the Study

FAS/FAE are detrimental to success in school for a variety of reasons. With regard to literacy, FAS/FAE pose unique challenges to students. Neurodevelopmental damage results in impaired capacity to acquire basic language skills, maintain attention, recall information, and control impulses, to name just a few. This study provides documentation of the relationship between neurologically based speech/language and social behavior patterns and literacy acquisition. Study of such relationships has the potential to suggest exploration of new intervention strategies. As educators gain insights into the particular ways that brain activity is altered through toxin exposure, they can more specifically direct research efforts in an attempt to design teaching strategies that

work cooperatively with remaining neurolinguistic strengths. With this information, realistic academic performance outcomes can be considered, and additional research can be explored that might lead to intervention strategies appropriate to the developmental needs of this particular group of children.

More importantly, this study provides evidence that would suggest that energies and resources should primarily focus on prevention of FAS/FAE through a variety of educational and support strategies. Although it is unrealistic to imagine a world free from alcohol, the abuse of toxic substances might very well be able to be reduced through concerted educational campaigns. Even a small improvement along these lines could result in enormous savings of financial and human resources. Finally, prevention of FAS/FAE would most certainly alleviate stress and heartache in the lives of families. Human suffering which is preventable must receive society's attention.

Operational Definitions

Fetal Alcohol Syndrome (FAS) is the term that has come to describe an array of teratogenic outcomes observable in individuals who have experienced in utero alcohol exposure. FAS is a label coined rather recently, used first by diagnosticians in the early 1970s (Jones, Smith, Ulleland, & Streissguth, 1973).

FAS diagnostic criteria generally include evidence of abnormality in three specific areas:

1. Prenatal and/or postnatal growth retardation (weight, length, and/or head circumference below the 10th percentile when corrected for gestational age).

2. Central nervous system involvement (signs of neurological abnormality, developmental delay, or intellectual impairment).
3. Characteristic facial dysmorphology including at least two of three of the following signs: (a) microcephaly (i.e., unnatural smallness of the head, with head circumference measuring below the 3rd percentile; (b) microphthalmia (i.e., abnormal smallness of the eyes and/or short palpebral fissures, that is, opening between the eyelids; and (c) poorly developed philtrum (i.e., vertical ridges between nose and mouth, thin upper lip, and flattening of the maxillary area, that is, underdevelopment of the mid-facial jawbone region (Clark, Cotton, Hankins, & Phelan, 1999).

Further diagnostic descriptions have been recommended by Burd (2001) and include (a) cardiovascular, renal, orthopedic, dermatologic, and connective tissue abnormalities; (b) respiratory dysfunctions; and (c) neuropsychiatric findings such as hyperactivity or attentional problems, speech/language disorders, learning disabilities, schizophrenia, enuresis and/or encopresis after age 7, tremors or seizures, and echolalia.

Fetal Alcohol Effects (FAE) is another term that surfaces frequently in the literature regarding the impact of alcohol on a developing fetus. FAE is a rather broad net, generally assigned to symptoms of lesser severity than FAS. Despite the temptation to minimize its significance, FAE often includes a range of developmental disorders including, but not limited to, attention deficit disorders, distractibility, and learning disabilities (Spohr & Steinhausen, 1996; Streissguth,

1997). FAS-RD, another catch-all label indicating Fetal Alcohol Syndrome and Related Disorders, is familiar to the literature as well.

Assumptions

Because this study was dependent upon existing data files, it was assumed that the information contained within those files was gathered and maintained by reputable and skilled professionals. Additionally, it was assumed that the data accumulated were a meaningful measure of developmental achievement, and that comparisons of speech/language, social behavior, and literacy performance would be based on common criteria.

Delimitations of the Study

In order to maintain the highest possible number of subjects, independent variables were limited to three categories. There are, in fact, many variables which significantly influence literacy acquisition. This study does not address such factors as language or cultural differences, teaching methodologies, or learning styles.

CHAPTER II

REVIEW OF LITERATURE

This chapter will provide the reader with background information relevant to this study. Topics addressed will include language acquisition, psycholinguistic and neurolinguistic processes, central nervous system structure and development, reading process, and alcohol and its relation to pregnancy. These topics will be discussed using the following outline: Introduction, FAS/FAE, Brain Development, and Literacy Acquisition.

Introduction

People live in a climate of complex educational demands. Society, both in our common neighborhoods as well as in locations far away, wrestles with the reality of the fundamental and essential requirement of literate citizens.

Most children come to school ready to learn. Physical and emotional needs have been looked after by loving families, intellectual curiosity has been encouraged, and a variety of social contexts have become familiar. This all-important "first teaching" provides a powerful foundation for formal instruction in a school setting. Literacy acquisition becomes a natural outgrowth of early home experiences.

Unfortunately, any number of factors can impede children's development, and in some instances, encumber them with significant handicaps which make

school success difficult if not impossible. For instance, early language development is a remarkably powerful predictor of school success. It is well documented that children with specific language impairments are highly at risk for school failure (Badian, McAnulty, Duffy, & Als, 1990; Catts, 1991, 1997) with most developing reading disorders (Lewis, Freebairn, & Taylor, 2000; Stark et al., 1984; Tallal, 1987) despite preschool speech/language interventions (e.g., Padget, 1988).

Although seemingly automatic, language acquisition for infants is hardly a simplistic process. Rather, it is dependent upon complex physiological systems working cooperatively with one another in the context of an environment supplying appropriate stimulation and reinforcement.

From birth, humans express affect, making clear their needs and responses to the environment. Though no one teaches babies to cry and coo, they quickly discover the power to manipulate the response of those around them. Over time, children experiment with a variety of vocalizations, imitating those they hear in their immediate environment. Children born into Chinese speaking families learn to speak Chinese. Circumstances having been different, however, the same child surrounded by French speakers would have just as easily acquired French.

Despite major advancements in scientific research, particularly having to do with brain functions, the exact nature of psycholinguistic and neurolinguistic processes is still an emerging field of study. It is now understood that the first processing stage of visually presented words consists in an analysis of their

physical features such as spatial frequency, orientation, and size in the primary visual area. The recognition of linguistic fonts (i.e., orthographic analysis) apparently occurs about 150 milliseconds post-stimulus in the temporal occipital area (linguae gyrus and fusiform gyrus of left and right visual cortices) as shown, for example, by the magneto-encephalographic (MEG) study of Kuriki, Takeuchi, and Hirata (1998). (See Figures 1 and 2.)

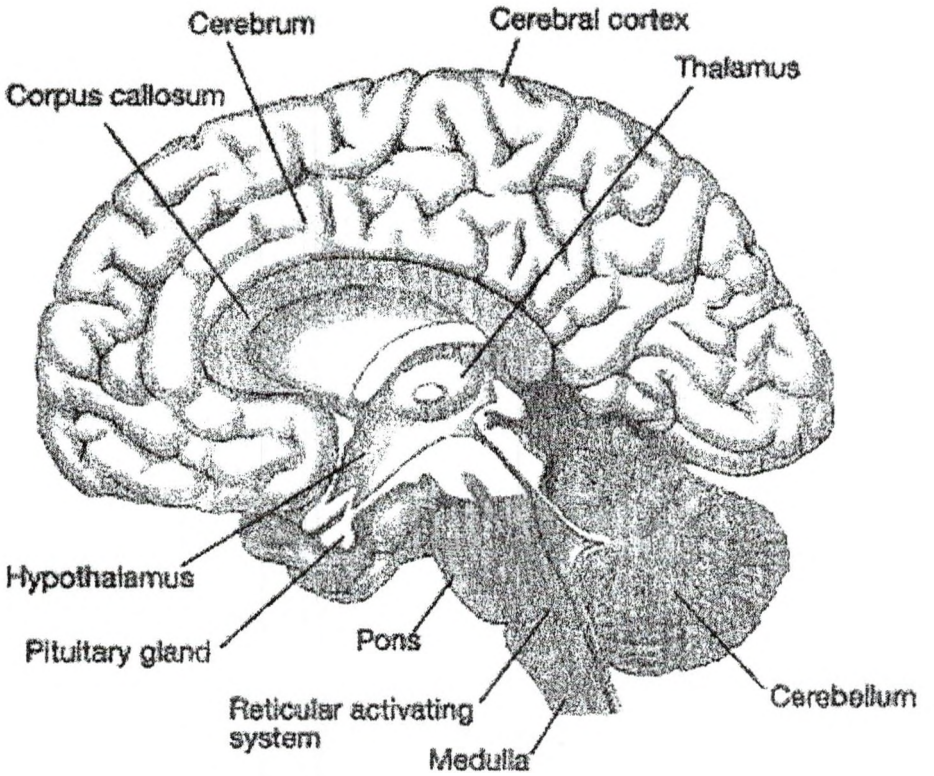

Figure 1. Regions of the Brain.

Note. From http:www.wpscms.pearsoncmg.com/wps/media/objects/271/278463/f04_10.gif

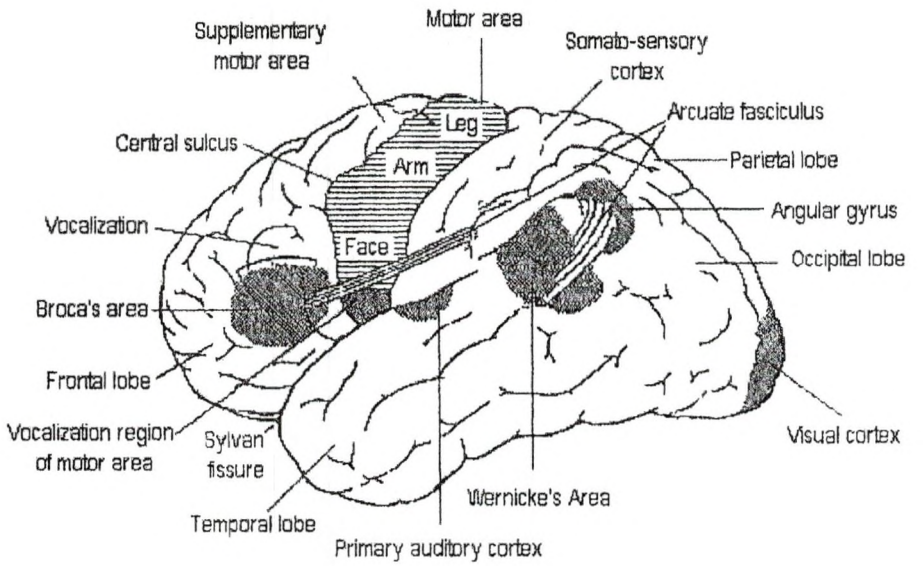

Figure 2. Hemispheric Specialization.

Note. From http:www.facstaff.bucknell.edu/rbeard/workshop/lecnotes.html

How can such remarkable detail be determined? Rather than being limited to examining the brain in an autopsy specimen, or measuring the size of brain regions using static morphometric indices based on CT or MRI, functional imaging offers the possibility of examining brain function during performance of a cognitive task.

Functional magnetic resonance imaging (fMRI) promises to surpass other methods in its ability to map the individual brain's response to specific cognitive stimuli. Because it is noninvasive and safe, it can be used repeatedly, making it ideal for studying humans, especially children.

Most functional imaging studies, whether PET or functional MRI, use a subtraction methodology in attempting to isolate brain/cognitive function relations

(Friston, Frith, Liddle, & Frackowiak, 1993; Petersen & Fiez, 1993; Sergent, 1994). Reading can be considered as involving three component processes: orthographic, phonological, and lexical-semantic processing. Accordingly, the tasks should be able to isolate orthographic, phonological, and lexical-semantic foci. In addition, a variety of subtractions are used in order to converge on a conclusion about the relative function of a given cortical region (Shaywitz et al., 2000). In recent years, several brain-imaging studies have called attention to a small zone in the left fusiform gyrus that is systematically active during processing of printed words (e.g., Beauregard et al., 1997; Cohen et al., 2000; Nobre, Allison, & McCarthy, 1994; Polk & Farah, 2002; Puce, Allison, Asgari, Gore, & McCarthy, 1996; Wagner et al., 1998).

The relationship between overall cerebral size and linguistic functioning has also been assessed, given the research of Leonard et al. (2001) who found overall cerebral volume to be associated with receptive and expressive language functioning and semantically coded measures of verbal short-term memory. In addition, given that gyral variation of the perisylvian region may be related to receptive language functioning (Hiemenz & Hynd, 2000), as well as phonological processing and rapid phonological retrieval (Morgan & Hynd, 1996), it was believed that gyral variation of this region would be related to measures of semantic and phonological short-term memory.

More than one in three children experience significant difficulties in learning to read (Adams, 1990; Shaywitz, Escobar, Shaywitz, Fletcher, & Makuch, 1992), and there is strong continuity between the skills with which

children enter school and their later academic performance. Those children who experience early difficulties in learning to read are unlikely to catch up to their peers (Baydar, Brooks-Gunn, & Furstenberg, 1993; Francis, Shaywitz, Stuebing, Shaywitz, & Fletcher, 1996; Stevenson & Newman, 1986; Torgesen, Wagner, Rashotte, Alexander, & Conroy, 1997; Tramontana, Hooper, & Selzer, 1988).

Verbal deficit explanations of reading have implicated the semantic, phonological, and syntactic-grammatical domains of language; and there is some reason to believe that deficiencies in one or another of these domains may directly or indirectly impair different aspects of the word identification process. Previous studies suggest that proficiency in word identification is contingent upon adequate development of both whole-word/meaning-based and code-oriented approaches to word identification, and that the failure to acquire functional use of one or the other of these approaches may be directly or indirectly related to language deficits and/or to word-processing strategies fostered through limited instructional programs (Vellutino & Denckla, 1996).

As recently as 20 years ago, learning to read was not thought to commence until formal instruction was provided in school. Accordingly, reading disabilities were largely considered to be an educational problem with no known antecedents at earlier ages. It is now clear that reading acquisition is a process that begins early in the preschool periods such that children arrive at school having acquired vastly differing degrees of knowledge and skill pertaining to literacy. Furthermore, virtually every study has confirmed that preschoolers with language impairments are indeed at considerable risk for developing reading

disabilities (as well as for continued oral language difficulties) at older ages. As in research on selected at-risk preschool samples, these studies have found reliable associations between early abilities and later prereading skills and/or reading achievement (Scarborough, 2002).

FAS/FAE

Alcohol is the most frequently abused drug in our society. Alcohol abuse has many long-term effects that result in premature death and an increased propensity for serious illness. Another alcohol-related health issue is ethanol-drug interactions which, in some cases, can be fatal (Davies, 2003).

A pregnant woman who exposes her unborn child to alcohol toxins during periods of cell proliferation and growth creates the potential for a syndrome generally referred to as Fetal Alcohol Syndrome (FAS) or Fetal Alcohol Effects (FAE). In this context, the term syndrome refers to a pattern of similarly occurring anomalies in most patients. In this regard, it is not very different from the way in which the term disease is often understood.

In contrast to the woman who consumes toxic amounts of alcohol throughout pregnancy, a woman who consumes toxic amounts in sporadic binges will expose her child during fewer days of pregnancy, and, therefore, her child is more likely to exhibit individual anomalies than a syndrome. For example, based on the principle of critical periods, it follows that malformations involving the face or bodily organs can arise if toxic exposures occur during the first trimester of pregnancy, when the proliferative stage of cell development occurs. Exposure during the last trimester cannot give rise to malformations

because the proliferative stage is over. However, exposure during this last period could result in decreased overall growth or decreased growth of specific organs. There is no "safe time" for toxic levels of exposure during pregnancy because dynamic changes occur throughout gestation (Abel, 1998).

Alcohol has been called the leading teratogen in the western world because a large literature has accumulated that links it with a wide spectrum of birth defects. Alcohol use during pregnancy is the most common cause of mental retardation in the United States and may be responsible for up to 5% of all congenital anomalies. Throughout gestation, fetal physiology is directly disrupted by alcohol and its primary metabolite, acetaldehyde (Mason, 2000).

Ethanol crosses the placenta freely, and concentrations on either side rapidly equalize after maternal ingestion. The human fetus has less capacity than the adult to eliminate ethanol because of low fetal hepatic alcohol dehydrogenase activity. In the event of acute intoxication, alcohol concentrations may be higher in the fetus than in the mother, as a result of the impaired capacity of the former to detoxify the drug. Acetaldehyde, the main metabolite of ethanol, is potentially more toxic (Folb & Dukes, 1990).

Microcephaly (decreased head circumference below the 3rd percentile) occurs in about 80% of young children with FAS (Loser, 1995; Streissguth et al., 1993) and is a predictable consequence of maternal alcohol abuse, which by definition might suggest regular consumption of five or more drinks per drinking occasion. Microcephaly is also one of the most persistent characteristics of FAS.

Although catch-up growth occurs in adolescence in some instances, microcephaly often remains a visible indication of FAS (Streissguth et al., 1991).

Sensory processing has been relatively ignored in the context of FAS, although there is consistent evidence that sensory functioning is impaired. In a small pilot study, Morse and Cermak (1994) reported that 12 children with FAS had greater problems with visual and auditory stimuli when compared with a control group, although no details were given regarding the control group or the methods used to assess sensory function. Visual impairment is commonly associated with FAS; it is not uncommon for individuals with FAS to require corrective lenses. In many cases, visual impairment is due to the abnormal curvature of the lens. Visual anomalies appear to be so common that a pediatric ophthalmological examination should be routine for anyone receiving a diagnosis of FAS (Stromland, 1985).

Hearing loss is especially common in FAS and is specifically mentioned by the Institute of Medicine (1996), in its diagnostic paradigm as evidence of neurodevelopmental disorder. Hearing problems can be subdivided into three main types: conductive, sensorineural, and central hearing loss. Conductive hearing loss typically results from problems affecting the middle ear and occurs in more than 75% of children with FAS. Recurrent serous otitis media (RSOM) is a leading cause of intermittent conductive hearing loss in children with FAS (Church, Eldis, Blakley, & Bawle, 1997; Church & Gerkin, 1988; Rossig, Wasser, & Oppermann, 1994). The impediment in sound transmission associated with RSOM results from the buildup of fluid in the middle ear.

Hearing losses of 15 decibel magnitude commonly associated with serous otitis media are clinically significant for young children because this amount of hearing loss can adversely affect school performance and development of speech and language skills (Church, Abel, Kaltenbach, & Overbeck, 1996; Rossig et al., 1994).

Sensorineural hearing loss is the result of auditory receptor cell ("hair cell") and/or auditory nerve damage. While it has been found in 1% to 3% of all children, it has been found in as many as 27% to 29% of children with FAS (Church et al., 1997; Church & Gerkin, 1988).

It is estimated that about 60% of American women drink at least occasionally. Most women are light to moderate drinkers who have few problems related to their drinking. In a national study of women conducted in 1991, it was found that 44% were considered light drinkers, 12% were moderate drinkers, and 3% were classified as heavy drinkers. In the same study, 73% of the women in their 20s and 69% in their 30s used alcohol in the past year. Drinking rates are the highest among young women and tend to decline with age (Leonardson, 2003).

According to the National Natality Survey, 39% of women admitted to alcohol use during their pregnancies. The prevalence of heavy or problem drinkers has been estimated to be 6% to 11% (Andres & Jones, 1994). Worldwide, full-blown FAS can be identified in 1 in 300 to 1 in 1,000 live births. Lesser degrees of damage, called Fetal Alcohol Effects (FAE), may occur in as

many as 1 in 100 live births. However, there is no known threshold for FAE (Mason, 2000).

In North Dakota, from a cohort of 7,600 to 8,000 live births per year, the prevalence rate of FAS ranges from 1.3 to 2.0 cases per 1,000 live births or about 12-18 new cases each year. This yields a population estimate for FAS in the state of North Dakota of between 180 and 320 children, birth through 18 years of age, and a total FAS population (children and adults) that could be as high as 1,100 (Burd, Martsolf, Klug, & Kerbeshian, 2003).

The number of individuals exposed to alcohol in utero and affected with physical anomalies and/or brain alterations, but without the characteristic and recognizable pattern of Fetal Alcohol Syndrome, is not known. Many authors have stated that the frequency is at least the same as Fetal Alcohol Syndrome, but it is potentially far greater (Clarren & Astley, 2001). Clearly, a relatively large number of otherwise biologically normal infants may be irreversibly damaged by maternal alcohol abuse during pregnancy (Little, VanBeveren, & Gilstrap, 1998).

Maternal drug use impacts multiple social systems, including the health care system, the criminal justice system, the educational system, and child protective agencies. The health consequences of maternal substance use are exceedingly costly and far-reaching.

Brain Development

Behavioral and cognitive dysfunction are among the most common consequences of exposure to substances such as alcohol because the actions of these substances are not confined to any critical period. The brain continues to

fine tune itself throughout gestation and beyond. When finally developed, the brain's 1,400 cubic centimeters contain several hundred different cell types, each with their own distinctive morphological, physiological and biochemical characteristics, about 15 billion nerve cells in all. Because all of these cells come into existence during the first 12 weeks of life, the developing brain must produce about 150,000 of them a minute. If this first "growth spurt" is interfered with, it may result in malformations, microcephaly, or in a normal-sized brain with decreased cells in a particular area, such as the corpus callosum or cerebellum. A second growth spurt begins in the final two months before birth. Because all of the brain's neurons have been formed during organogenesis, this latter growth period reflects increases in the size of individual neurons and changes in their shapes, for example, sprouting of axons and dendrites or myelination of axons. If exposure to toxic levels of alcohol occurs during this second growth spurt, children may develop behavioral and cognitive disorders even though their brains are normal in size. Final maturation does not end until the second year of life. These latter cellular refinements underlie the brain's unique functional abilities (Abel, 1998).

In vertebrates, the first morphological sign of emergence of the nervous system is the appearance of the neural plate, the medio-dorsal line of which is in intimate contact with a mesodermal structure, the notochord. Laterally, the neural epithelium is separated from the presumptive superficial ectoderm by a transitional zone, the neural fold. The next important step in neurogenesis is the transformation of the initially flat neural anlage into a tubular structure. The

dorso-medial part of the neural plate thus becomes medio-ventral and its lateral ridges reach the medio-dorsal line where they fuse to form the neural crest and the roof plate. During these morphogenetic events, the floor plate acquires important inductive properties under the influence of the notochord (Yamada, Placzek, Tanaka, Dodd, & Jessell, 1991) and the neural tube becomes divided into six compartments along the dorsoventral axis: ventrally the floor plate in contact with the notochord, the roof plate dorsally, and laterally the alar and basal plates corresponding respectively to the dorsal and ventral quarters of the neural tube. Although these territories have long been recognized and named, it is only recently that their developmental significance is being really investigated (Le Douarin, Hallonet, & Pourquié, 1994).

Neural tube malformations can result in leaving the central nervous system exposed. These malformations include holoprosencephaly, myelomeningocele, and spina bifida and are collectively referred to as neural tube defects (NTD). In general, NTD teratogenesis is caused by very high maternal alcohol ingestion. Because they are rare and often fatal, the nature and incidence of minor failures of neural tube development prior to birth are unknown. It is suspected that a wide range of minor neural tube abnormalities may occur with milder doses of alcohol, which would not normally render grossly evident NTD or facial dysmorphogenesis at birth but may contribute to more subtle neurodevelopmental deficits seen in Alcohol Related Neurodevelopmental Disorder (ARND). The ARND, which includes neuropsychiatric and intellectual deficit, accounts for a greater incidence of FAE than full-blown FAS with facial

dysmorphogenesis and mental retardation (Zhou, Sari, Powrozek, Goodlett, & Li, 2003).

As the neural tube develops, the midline tissue including the roof and floor of future ventricular areas remains thin and vulnerable. The neural tube midline is highly vulnerable to alcohol during development and can result in a compromised neural tube midline (cNTM). The cNTM refers to any deviation from proper neural tissue formation and maturation in the midline. Any defect at developmentally sensitive sites such as the floor and roof plates or other midline structures may impose critical consequences. Prenatal exposure from low to moderate doses of alcohol has been observed to cause an underdevelopment of serotonin neurons. Non-genetic, causal effects of abnormal neural tube development may be due to in utero alcohol exposure that depends on the amount, pattern, and timing of alcohol consumption (Zhou et al., 2003).

Exposure to alcohol produces improper neural tube development: The neural tube advances in stages, but the brain mass (at this stage mostly indicative of number of brain cells) lags behind, resulting in a significantly reduced framework to support the neural tube. The enlargement of ventricles occurs as an increase in the neural tube lumen size without a proportional increase in the neural tissue mass. When brain mass (i.e., number of cells) is insufficient in the large neural tube frame, it is plausible that breakage occurs at the thinnest point, the midline roof and floor (Zhou et al., 2003).

Even a moderate blood alcohol level causes a high frequency of neural tube midline defects during early development. With moderate alcohol exposure,

the compromised neural tube midline can be mended without traces of gross physical malformation near or after birth. However, fewer serotonin neurons are left behind. This development is similar to ARND; although physical facial or gross brain features appear normal, individuals are plagued with abnormal neural development (Zhou et al., 2003).

Ethanol can be neurotoxic and lead to decreased numbers of neuroblasts and/or glioblasts and general reductions in white matter. Animal models have demonstrated that virtually every neurotransmitter can be decreased or increased utilizing some methodological approach. Recent reports of magnetic resonance images in patients with Fetal Alcohol Syndrome reveal common specific abnormalities including thinning or absence of the corpus callosum and cerebellar vermis hypoplasia. Experimental studies in humans and animals, when taken as a whole, suggest that ethanol does not produce any specific focal pattern of brain lesions but rather causes diffuse lesions that vary in severity within and among affected persons (Roebuck, Mattson, & Riley, 1998).

Evaluation of the brain for evidence of organic brain damage is the most important part of the diagnostic process because it leads to understanding of the problems faced by the patient and is necessary for treatment planning. Organic brain damage can be discussed structurally, neurologically, or psychometrically. The firmest evidence of organic brain damage is through physical findings such as microcephaly. A head circumference more than 2-3 standard deviations below the mean is clearly beyond the normal growth range and, in Fetal Alcohol Syndrome, is due to decreased brain growth. Imaging studies that identify

anomalies produced during brain formation, such as absent corpus callosum, small cerebellar verbs, cerebral heteropias, etc., are other clear indicators of brain malformation (Clarren & Astley, 2001).

Development of the nervous system depends on patterned neuronal migration and axonal growth, which are regulated by cell-cell and cell-substrate interactions. These interactions are mediated through cell adhesion molecules (Greenberg, 2003).

Experimental evidence demonstrates that alcohol interferes with many molecular, neurochemical, and cellular events occurring during the normal development of the brain. Some brain areas are more affected than others and, even within a given region, some cell populations are more vulnerable than others. The neocortex, hippocampus, and cerebellum are especially susceptible to alcohol and have been associated with behavioral deficits. For example, alcohol exposure during the development of neocortex increases natural apoptosis and induces cell necrosis. Impairment of several neurotransmitter systems and/or their receptors, as well as changes in the endocrine environment during brain development, are also important factors involved in the neurodevelopmental liabilities observed after in utero alcohol exposure (Guerri, 2002).

The cerebellum frequently undergoes damage in connection with FAS. Comparable neuroanatomical anomalies have been found in nonhuman primates exposed to regular bingelike episodes of alcohol exposure (Astley, Weinberger, Shaw, Richards, & Clarren, 1995). Postmortem examination of 16 children with

FAS found 4 with a dysgenesis of the cerebellum (Clarren, 1986). Magnetic resonance imaging (MRI) of children with FAS has likewise found instances of gross neuroanatomical peculiarities in the cerebellum (Mattson et al., 1992, 1994; Sowell, Mattson, Riley, & Jernigan, 1996). A positron emission tomography (PET) study of four children with FAS, ages 4 to 6, found this area of the brain had a very low glucose utilization rate (Hannigan, Martier, Chugani, & Sokol, 1995), suggesting decreased functional activity, possibly related to disturbances in fine motor control (Jones et al., 1973), ataxia (Kyllerman et al., 1985; Marcus, 1987), and gait problems (Marcus, 1987).

The cerebellum has received a disproportionate amount of attention from neuroanatomists not only because of its susceptibility to alcohol's neuropathological effects, but also because of a distinctive cytoarchitecture, which enables researchers to observe fine distinctions in the types of cells damaged by alcohol. The two main types of cerebellar cells are the relatively large Purkinje cells and the smaller granule cells. Those undergoing dendritic development earliest are affected the most, as reflected by a greater cellular loss, compared with those that develop later. In the rat, Purkinje cells are generated prenatally; granule cells, postnatally. Vulnerability to alcohol's teratogenic effects is greatest if exposure occurs after the cells have passed the formative stage and are in the differentiation stage. In the case of the rat cerebellum, if alcohol exposure occurs prenatally, when Purkinje cells are in their formative stage of neurogenesis, the impact on cell density is minimal. On the other hand, if exposure occurs postnatally, during the brain's growth spurt

(corresponding to the third trimester in humans), when the Purkinje cells are undergoing differentiation, dendritic development, and synaptogenesis, they are affected to a much greater extent than the granule cells, which are in their neurogenerative and proliferative stage (Phillips & Cragg, 1982; Pierce, Goodlett, & West, 1989; West, Goodlett, Bonthius, Hamre, & Marcussen, 1990).

The optic nerve and the organ of Corti are two additional areas of the brain arising from the ectoderm that undergo damage from alcohol. Although relatively common, these anomalies have received much less attention than those previously mentioned, possibly because sensory function in general has been relatively ignored in fetal alcohol abuse studies (Abel, 1998).

In addition to gross anomalies affecting major structures, ultrastructural anomalies due to high levels of alcohol exposure in utero occur throughout the brain. An autopsy of a 4-year-old child with FAS (Ferrer & Galofre, 1987), for example, found that in addition to extensive gross neuroanatomical damage, there were also major decreases in the numbers of dendritic spines and increases in morphologically abnormal spines.

Neurocognitive effects of alcohol exposure tend to be widespread and generalized. Attention and executive functions do not seem to be selectively affected. Further, the relationship between duration of prenatal alcohol exposure and neurocognitive development continues to be significant in early adolescence (Korkman, Kettunen, & Autti-Ramo, 2003).

People with FAS also have high rates of comorbid conditions including attention deficit hyperactivity disorder (40%), mental retardation (15-20%),

learning disorders (25%), speech and language disorders (30%), sensory impairment (30%), cerebral palsy (4%), and epilepsy (8-10%). Birth defects are common (Burd et al., 2003).

As noted previously, ethanol leads to structural alterations of the brain at intracellular, intercellular, microcellular, and/or macrocellular levels. There are additional neurochemical impacts. The types and degrees of resulting cognitive and behavioral dysfunction are protean in diversity and severity, and they are often compounded by other teratogenic or genetic factors of prenatal origin as well as difficult or destructive early postnatal environments. Frequently, in children older than age 6-8 years, there are problems of emotional response to chronic poor performance and lack of encouragement as well as emotional response to early environmental problems like abuse, neglect, and multiple moves and caretakers (Streissguth, 1997). Children often show signs of depression and/or anxiety and may meet the clinical criteria for reactive attachment disorder, oppositional defiant disorder, or posttraumatic stress disorder. Brain problems can best be summarized as "complex" (Clarren & Astley, 2001).

Literacy Acquisition

Given the complex disturbances to development experienced by children with FAS or FAE, it is not surprising to discover that challenges these children experience in the home carry over into school environments. School environments place many demands on students. Activities are structured and scheduled. Group participation, independent work, and cooperation with

authorities are expected. Classroom instruction is generally highly dependent upon attentiveness to verbal explanations, and an ability to follow through with independent practice on skill lessons taught. The focus of early elementary instruction is on learning to read and write, and on basic numeracy. In the upper elementary grades and beyond, emphasis shifts to using those literacy skills to obtain understanding of more complex materials in a variety of content areas.

Mental retardation has long been considered the most serious persistent consequence of FAS/FAE (Dehaene et al., 1977; Iosub, Fuchs, Bingol, & Gromisch, 1981; Lemoine, Harousseau, Borteryu, & Menuet, 1968; Majewski, 1981; Olegard et al., 1979; Robinson, Conry, & Conry, 1987; Steinhausen, Nestler, & Spohr, 1982: Streissguth et al., 1978). FAS/FAE is now recognized as one of the leading known causes of mental retardation in the industrialized world (Abel & Sokol, 1987). However, mental retardation is not an invariable outcome of FAS/FAE. The prevalence for mental retardation, as defined by IQ scores below 70, is about 50% in FAS/FAE (Abel, 1990). Streissguth et al. (1978) found IQs ranging from 16 to 105 in a group of 20 children with FAS/FAE. Children with the most severe dysmorphia and growth retardation have the lowest IQ scores (Dehaene et al., 1977; Majewski et al., 1976; Steinhausen et al., 1982; Streissguth et al., 1978).

In another study, Streissguth, Clarren, and Jones (1985) reported on the mental and intellectual development of children exposed to alcohol prenatally. None of the children were mentally normal and more than 75% were found to be profoundly retarded. Caruso and ten Bensel (1993) confirmed these findings in

their study on the effects of maternal alcohol abuse on fetal development. They found that the average IQ of a group of FAS children was under 70. In addition, the children were hyperactive and had attention deficit disorders. As adolescents and adults, the characteristic craniofacial malformations of FAS become less distinctive (Spohr, Willms, & Steinhausen, 1993), but the individuals were consistently smaller, lighter, and less intelligent than national standards (Streissguth et al., 1991). Importantly, the mean IQ of the FAS subjects in the study was 68, with a wide range of scores, and the average academic functioning was found to be consistently below normal (Little et al., 1998).

Reading skills, which have been shown to be highly dependent upon other verbal abilities (Clarren & Astley, 2001), are achieved with varying degrees of success. FAS-related speech pathology is due, in many cases, to articulation problems (Becker, Warr-Leeper, & Leeper, 1990; Church et al., 1997; Iosub et al., 1981), arising from physical anomalies in the structure of the jaw, teeth, palate, and gums. Alternatively, fine motor dysfunction involving coordination of muscles in the tongue or larynx may be responsible for the inability to articulate vowels and consonants. Because speech is also a function of hearing, articulation problems may also be related to alcohol's effects on the auditory system (Abel, 1998).

Linguistic ability was impaired in 82% of the 63 children with FAS studied by Iosub et al. (1981) and 86% of the 12 children with FAS examined by Church et al. (1977) (Becker et al., 1990; Sparks, 1984; Steinhausen et al., 1982). Although these studies involved children from predominantly low socioeconomic

backgrounds, when children with FAS were compared with children of the same age, sex, and socioeconomic background, the same linguistic difficulties were apparent; children with FAS were less advanced in their use of complex grammatical structures, and their short-term memory was not as good as controls (Becker et al., 1990; Hamilton, 1981).

Communication skills are likewise impaired in FAS. In general, these communication disorders and related problems appear to be due to difficulties in cognitive processing rather than speech reception. Morse (1993) has metaphorically described the information-processing problem of the child with FAS as a dysfunctional word processor: Full sentences are typed in, but only some of it is saved or stored in an inaccessible area, so only partial sentences can be retrieved. Sometimes the lost material may reappear if a random key is inadvertently hit, or some special cue related to the missing information is accidentally accessed. As a result, the conversation of the child with FAS is often not connected to what is being discussed (Abel, 1998).

In one study group, children showed deficits in intellectual functioning (short-term memory and encoding) and displayed problems in preacademic skills. Becker et al. (1990) found abnormalities in speech production and speech comprehension in FAS subjects aged 4.5 to 9.5 years of age as did Autti-Ramo and Granstrom (1991) in 18- and 19-month-old children who had been continuously exposed to alcohol during gestation (Little et al., 1998). Articulation, linguistic, and communication disorders resulting from prenatal alcohol exposure are only encountered in conjunction with FAS/FAE. At levels that do not cause

FAS/FAE, these skills are not compromised (Fried & Watkinson, 1988; Greene et al., 1991; Morrow-Tlucuk & Ernhart, 1987; Streissguth et al., 1993; Russell, Czarneci, Cowan, McPherson, & Mudar, 1991).

Disturbances in information processing may also be responsible for conduct problems in the classroom. A teacher may believe that these children understand what they have just been told, but when they walk away as if oblivious, the teacher may interpret their behavior as disrespectful rather than as a lack of understanding (Morse, 1993).

Clinical features of attention deficit hyperactivity disorder are frequently found. Psychometric testing often demonstrates wide variations on subtest scales, irregular patterns of academic achievement, language processing difficulties, and problems with attention. These abnormalities lead learning problems at school and at home and poor intersocial relationships. Parents are often at a loss to guide, discipline, and otherwise manage these children (Clarren & Astley, 2001).

Adolescents with FAS have been described as irresponsible, learning impaired, impulsive, having poor judgment, lacking in inhibition and remorse, and unable to appreciate the consequences of their actions. They are also said to be prone to lying, cheating, and stealing. They continue to be garrulous, socially winsome, and often convincing, insisting that they can control their own lives. However, their conversation is often unresponsive, their winsome behavior may make them easily manipulated and victimized, and they may require constant supervision (Abel, 1998).

Adolescents with FAS/ARBE are also described as being gullible and having poor problem-solving abilities. Abstract reasoning is difficult for them. Consequently, their mathematical skills are poorly developed. Related problems involve management of time and money. Keeping appointments is difficult because they do not equate clock time with the need to do something or be somewhere. Saving money for a future purchase is too abstract a concept for them to understand it well. Normal functioning is said to be possible for them only in highly structured situations (Morse & Weiner, 1996).

In summary, along with the characteristic sequelae of alcohol exposure, children exposed to alcohol during the prenatal period exhibit behavioral anomalies that include hyperactivity, decreased attention, lack of social inhibition, and poor psychosocial functioning. These behavioral anomalies are accompanied by deficits in measures of intelligence, verbal fluency, and spatial memory (Tran & Kelly, 2003). To the degree that these characteristics are evident, children with FAS/FAE inevitably struggle with literacy acquisition and the typical school experience as a whole.

This chapter provided the reader with background information relevant to this study. Topics addressed included language acquisition, psycholinguistic and neurolinguistic processes, central nervous system structure and development, reading process, and alcohol and its relation to pregnancy. From this review of literature, it is abundantly evident that children subjected to in utero alcohol exposure are indeed at high risk for interrupted development and subsequent difficulties related to learning. This study provided review of speech/language

and social behavior development in children with FAS/FAE and subsequent achievements related to literacy acquisition.

CHAPTER III

METHODOLOGY

In this chapter, a description of the study will be provided. Participants will be described, measures will be defined, study design will be outlined, and procedures used in the study will be detailed.

Participants

Youngsters from the Midwest region, identified through a variety of screening and community services, have been monitored for several years by a northern plains research university and hospital. Subjects represent both genders as well as a variety of racial and socioeconomic backgrounds. Initial referral comes from schools, childcare providers, social service personnel, health care workers or physicians, and occasionally from parents themselves. Information gathered includes general health and psychological analyses, as well as specific developmental evaluations. Permission was granted by the University of North Dakota Institutional Review Board (IRB) and the IRB of the cooperating health care facility to have access to these records under secure conditions. Approximately 150 files were reviewed to determine usefulness for this study. Of that number, 51 cumulative files of children ages 14 and under were selected because descriptive data indicated identification of FAS/FAE. Thirty-five of the examined files were found to contain sufficient data relevant to this study,

including speech/language assessments, developmental social behavior scores, and literacy assessment scores. Table 1 reflects whether data available were numerical test scores or narrative description. Some cases were missing specific categories of data.

Data were collected only from existing files. Contact or interaction with actual subjects was not required. File subjects maintain anonymity throughout the study and reporting process. No identifying descriptions such as name or home community were cited. Because human subjects were not directly involved, an exempt review was approved by the Institutional Review Board on December 23, 2003. In addition, an exempt review was approved by the cooperating health care facility on March 7, 2004.

Measures

School and medical screening procedures served to identify children with FAS/FAE. First, specific identifiers of FAS/FAE included such things as evidence of a characteristic pattern of facial anomalies that include features such as short palpebral fissures and abnormalities in the premaxillary zone (e.g., flat upper lip, flattened philtrum, and flat midface). Secondly, evidence of growth retardation had to be observed, as in the case of low birth weight for gestational age, decelerating weight over time not due to nutrition, or disproportional low weight to height. Finally, evidence of central nervous system (CNS) neurodevelopmental abnormalities had to be apparent, as in decreased cranial size at birth, structural brain abnormalities (e.g., microcephaly, partial or complete agenesis of the corpus callosum, or cerebellar hypoplasia), or neurological hard or soft signs (as

Table 1. Categorical Data Available for 35 Cases of Children With FAS/FAE.

Case #	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q
1	✓	✓	✓				✓	✓	✓	×	×	×	×		✓	✓	×
2	✓	✓	×	✓	✓	✓	✓	×	×						✓	✓	
3	✓	✓	✓					✓	✓	×	×	×	×	×			
4	✓	✓	✓					×	×			×					
5	✓	✓	×	×	×	×	×	×	×	×	×	×	×	×	×	×	×
6	✓	✓	✓	✓	✓	✓	×			×	×	×		×			
7	✓	✓	×	✓	✓	✓		×							✓	✓	✓
8	✓	✓	×	✓	✓	✓						×		×			
9	✓	✓	×	✓	✓	✓	×	×	×	✓	✓	✓	✓	✓	✓	✓	✓
10	✓	✓	✓	✓	✓	✓	×	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
11	✓	✓	✓						×			×		×	✓	✓	✓
12	✓	✓	✓					×		×	×	×	×	×			
13		×	×					×	×	×							
14	✓		×	✓	✓	✓	×	×	×	✓	✓	✓	✓	✓			
15		×	✓	✓	✓	✓				✓	✓	✓	×	✓	✓	✓	×
16	×	×	×	✓	✓	✓	×	✓	✓	✓	✓	✓		✓	✓	✓	×
17	×	×	×				×	✓	✓	✓	✓	✓	✓	✓			
18		×	×					✓	✓	✓	✓	✓		✓	✓	✓	
19		×	×	✓	✓	✓	×	×	×			×		×			
20	×	×	×	✓	✓	✓	×	×	×	✓	✓	✓	✓	✓	✓	✓	×
21	✓	×		✓	✓	✓	×	×	×						×	×	×
22	✓	✓	×	✓	✓	✓	×	×	×			×		×	✓	✓	×
23	✓	✓	×	✓	✓	✓		×		×		×			✓	✓	✓
24	✓	✓	×	✓	✓	✓	×	×	×					×			
25	✓	✓	×	✓	✓	✓	×	×	×	×	×	×		✓	×	✓	×
26	✓	✓	×	✓	✓	✓	×	×	×	×	×	×		×	✓	✓	×
27	✓	✓	×	✓	✓	✓	×	×	×	×		×		×	✓	✓	×
28	✓	✓	✓				×	×	×			×	×	×			
29	×	×	✓	✓	✓	✓	×	×	×	×		×		×			
30	✓	✓	×	✓	✓	✓	×	×	×	✓	✓	✓	×	✓	×	×	×
31		×	✓	✓	✓	✓		×	×	✓	✓	✓		✓	✓	✓	×
32		×	×	×	×	×	×	×	×	×		×	×	×			
33	×	×	×	✓	✓	✓	×		×	✓	✓	✓	✓	✓	×	×	×
34	✓	✓	×	✓	✓	✓	×	×	×	×	×	×	×	×			
35	×	×	×				×	×	×	×		×		×			

A=Minor Criteria, B=Exposure, C=IQ/Developmental Level, D=Verbal, E=Performance, F=Full Scale, G=Phonology/Articulation, H=Expressive Vocabulary, I=Receptive Vocabulary, J=Adaptive Behavior: Communication, K=Adaptive Behavior: Daily Living, L=Adaptive Behavior: Socialization, M=Adaptive Behavior: Motor Skill, N=Adaptive Behavior: Composite, O=Reading, P=Spelling, Q=Reading Comprehension

✓ Numeric Assessment

× Narrative Assessment

age appropriate), such as impaired fine motor skills, neurosensory hearing loss, poor tandem gait, or poor eye-hand coordination. In some instances, the child's observed or documented behavior contributed to the identification. Together, these traits are generally referred to as minor diagnostic criteria. Confirmed maternal alcohol exposure was important but not essential for diagnosis of FAS/FAE (Burd, 2001). Documented Minor Criteria and Exposure are reflected on Table 1 as categories A and B, respectively. In cases 13, 15, 18, 19, 31, and 32, minor criteria were not specifically noted in the subjects' hospital files. In these instances, however, narrative description of exposure clearly suggested a diagnosis of FAS/FAE. In case 14, exposure level was not available in the subject's file. In this instance, description of minor criteria was sufficient to suggest FAS/FAE.

IQ scores or estimates were chosen to be included in this study. Because FAS/FAE impacts neurocognitive development, it would be reasonable to assume that compromised central nervous system functioning would likely impact overall measures of intelligence. These, in turn, would likely contribute to speech/language and social behavior development, as well as literacy acquisition.

A variety of assessment techniques were used to determine IQ or developmental level. File data included scores obtained from such instruments as Bayley Scales of Infant Development, a tool which measures the mental and motor development and test behavior of infants from 1 to 42 months of age for the purpose of identifying developmental delays or disabilities; the Leiter

International Performance Scale, a nonverbal intelligence test which contains norms for ages 2 through 18; the Developmental Activities Screening Inventory, which provides early detection of developmental disabilities in children functioning between 1 and 60 months; the WISC-III, an assessment of verbal comprehension, perceptual organization, freedom from distractibility, and processing speed for ages 6-16; the Kaufman Brief IQ Test, which screens verbal and nonverbal IQ and is useful for nonreaders and the learning disabled; the McCarthy Scales, which test cognitive and motor ability in ages 2.5 through 8.5; the Stanford Binet (4th edition), which measures verbal reasoning, abstract and visual reasoning, quantitative reasoning, and short-term memory in ages 2 through adult; the Denver Developmental Screening Test, an instrument which determines developmental progress in the categories of personal/social, fine motor adaptive, language, and gross motor skills; or the Wechsler Intelligence Scale-Revised, which evaluates verbal abilities related to information, comprehension, arithmetic, digit span, similarities, and vocabulary, as well as performance abilities related to picture arrangement, picture completion, block design, object assembly, and digit symbol. Table 1 reflects available case data related to overall IQ or Developmental Level and is labeled category C. Subscores related to Verbal, Performance, and Full Scale categories of the WISC-III were most frequently noted in file data and are included in Table 1 as categories D, E, and F, respectively.

Speech/language assessments included measurements of both receptive and expressive language skills. In some instances, phonology and articulation

were also evaluated. Skill levels were compared to standard developmental milestones. Assessments were generally completed by using such instruments as the Arizona Articulation Proficiency Scale, a clinical measure of articulatory competence in children ages 1 to 18 years, or the Clinical Evaluation of Language Fundamentals (3rd edition), which is used to diagnose language disorders in children, adolescents, and adults, ages 6 to 21 years. Available data related to speech/language development are noted in Table 1 under categories G, H, and I, and reference Phonology/Articulation, Expressive Vocabulary, and Receptive Vocabulary, respectively.

Social behavior scores were determined, in most instances, by the Vineland Adaptive Behavior Scales. This instrument measures observable social behavior from infancy through adolescence and compares it to standard developmental milestones. Domains of assessment include communication, daily living skills, socialization, motor skills, and maladaptive behavior. Alternate assessment tools included such instruments as the Child Behavior Checklist, a tool designed to assess social competence and behavior problems in children ages 4 to 18, or the Normative Adaptive Behavior Test, an instrument which assesses how well a child compares to peers of his/her age (birth through 21 years) in performing skills needed for independent living. Data related to social behavior are reflected on Table 1 and categorized as Adaptive Behavior: Communication (J), Adaptive Behavior: Daily Living (K), Adaptive Behavior: Socialization (L), Adaptive Behavior: Motor Skill (M), and Adaptive Behavior: Composite (N).

Literacy proficiency was determined through examination of emergent prereading skills as well as early reading performance. Word recognition as well as reading comprehension were considered. Evidence of writing skill included assessments of motor skills, spelling, and effectiveness of communication. Common test instruments used to gather file data were the Wide Range Achievement Test-Revised, an instrument used to measure reading recognition, spelling, and arithmetic computation in children ages 5 and up; the Sucher-Allred Reading Placement Inventory, a tool for determining appropriate grade level placement related to reading, and Timed Tests of Basic Skills, designed to measure ability in reading and math through timed evaluation; the Kaufman Test of Educational Achievement, designed to measure school achievement of children, grades 1 through 12, in the areas of reading, math, and spelling; or the Woodcock Johnson, an instrument intended to measure both cognitive and achievement domains including academic aptitude and possible disabilities. Available data were noted on Table 1 and categorized as Reading (word recognition) (O), Spelling (P), and Reading Comprehension (Q). Although many case files reflected math achievement levels, these scores were not included in Table 1 because they did not bear relevance to this particular study.

Procedure

To initiate the study, permission was secured from a northern plains hospital and cooperating university research institute to access existing patient files for the purpose of identifying cases where children had been identified as

evidencing Fetal Alcohol Syndrome (FAS) or its milder form known as Fetal Alcohol Effects (FAE).

Approximately 150 files were reviewed. Each of these files bore some reference to in utero alcohol exposure, or physical or behavioral symptoms common to FAS/FAE, though not all cases appeared equally conclusive in identifying the condition. Fifty-one files were eventually selected for close study due to satisfaction of study criteria. Finally, a set of 35 was identified for evaluation and summary; because of the total number of files reviewed, this group had the most complete set of assessment scores and narrative available. All 35 cases included for analysis have been identified as evidencing FAS or FAE. For this study, they are grouped together as one and labeled FAS/FAE.

A charting form was designed to be used in recording data from the files. Information recorded included test scores and instruments, as well as anecdotal notes of interest which provided additional description or explanation of test results or case background. Data were then charted onto categorical grids to allow analysis of files. Categories examined were speech/language development, social behavior development, and literacy acquisition. IQ was also noted due to its relationship to academic potential, and to assist understanding of any puzzling relationships that might be observed in the other three categories.

IQ was broken down into six categories and are reflected on Table 2 as Severity. They include normal, borderline mental retardation (MR), mild MR, moderate MR, severe MR, and profound MR. The definitions of these subcategories follow commonly accepted psychological criteria.

Table 2. Available IQ Assessments of 35 Cases of Children With FAS/FAE.

Severity	Number of Cases	Percent
Normal	8	23
Borderline mental retardation	16	46
Mild mental retardation	4	11
Moderate mental retardation	3	9
Severe mental retardation	0	0
Profound mental retardation	1	3

Mental retardation (MR) refers to (a) significant limitations in general intellectual functioning; (b) significant limitations in adaptive functioning, which exist concurrently; and (c) onset of intellectual and adaptive limitations before the age of 22 years.

Significant limitations in intellectual functioning are determined from the findings of assessment by using a valid and comprehensive individual measure of intelligence that is administered in a standardized format and interpreted by a qualified practitioner. The criterion of significance is an IQ or comparable normed score that is two or more standard deviations below the population mean for the measure (Editorial Board, 1996).

Significant limitations in adaptive functioning are determined from the findings of assessment by using a comprehensive, individual measure of adaptive behavior. For adaptive behavior measures, the criterion of significance

is a summary index score that is two or more standard deviations below the mean for the appropriate norming sample or that is within the range of adaptive behavior associated with the obtained IQ range sample in instrument norms. The latter would be used, for example, if the adaptive behavior instrument does not present information on the means and standard deviations of the norming sample. For adaptive behavior measures that provide factor or summary scores, the criterion of significance is multidimensional; that is, two or more of these scores lie two or more standard deviations below the mean for the appropriate norming sample or lie within the range of adaptive behavior associated with the intellectual level consistent with the obtained intelligence quotient, as indicated by the instrument norms. For adaptive behavior measures that permit assessment of both adaptive and maladaptive behavior, presence of clinically significant maladaptive behaviors in the absence of significant limitations in adaptive behavior, as defined here, does not meet the criterion of significant limitations in adaptive functioning. The concurrent limitations in intellectual functioning and adaptive functioning must originate before the age of 22 years (Editorial Board, 1996).

The degree of speech/language and social behavior performance manifested with each level of mental development provides some correlation to successful literacy acquisition. In this study, normal IQ classification refers to individuals evidencing intellectual and adaptive behaviors generally expected of a particular developmental stage. Borderline MR refers to individuals evidencing intellectual and adaptive behaviors on the lowest end of normal classification in

which selected or occasional behaviors are marked by slight developmental delay.

People classified with mild MR evidence small delays in the preschool years but often are not identified until after school entry, when assessment is undertaken following academic failure or emergence of behavior problems. Modest expressive language delays are evident during early elementary school years, with the use of 2- to 3-word sentences common. During the later elementary school years, these children develop considerable expressive speaking skills, engage with peers in spontaneous interactive play, and can be guided into play with larger groups. During middle school, they develop complex sentence structure, and their speech is clearly intelligible. The ability to use simple number concepts is also present, but practical understanding of the use of money may be limited. By adolescence, normal language fluency may be evident. Reading and number skills will range from 1st- to 6th-grade level, and social interests, community activities, and self-direction will be typical of peers, albeit as affected by pragmatic academic skill attainments (Editorial Board, 1996).

People classified with moderate MR, because of more evident and consistent delays in their attainment of early developmental milestones, particularly in language facility and social play, are likely to be identified during the preschool years. At entry to elementary school, these children may communicate through a combination of single words and gestures and evidence self-care and motor skills similar to those of average 2- to 3-year-olds. During elementary school, they will develop the use of 2- and 3-word phrases to

communicate and by age 12 may evidence useful, pragmatic communication skills. Skill development, both academically and adaptively, is delayed during middle school, compared to peers with mild MR. By age 14, they may develop basic self-care skills, undertake simple conversations, begin to read, and manifest cooperative social interaction skills (Editorial Board, 1996).

People classified with severe MR are typically identified during infancy (i.e., ages birth to 2 years) because of substantial delays in development and their increased tendency to display biological anomalies that can be readily detected (Reschly, 1988). Milestones such as standing, walking, and toilet training may be markedly delayed by several years. Compared to their peers, these children are at greatly increased risk for motor disorders (e.g., cerebral palsy) and the epilepsies. At school entry, basic self-care skills may not be evident, although between ages 6 to 9 year, elements of self-feeding, self-dressing, and self-toileting typically are acquired. Elementary school-age children may communicate with single words and gestures and still engage in parallel play. By age 12 years, some 2- to 3-word phrases may be used, and some children may engage in interactive play. Between the ages of 13 and 15 years, academic and adaptive performance similar to that of an average 4- to 6-year-old can be attained by many of these children (Editorial Board, 1996).

People classified with profound MR also are typically identified as infants because of marked delays in development and biological anomalies. The responsiveness of the older preschooler (i.e., 4 years) is similar to that of a 1-year-old, and the child may sit alone, imitate simple sounds, understand simple

words (e.g., "No"), and recognize familiar people. These children have a markedly high early mortality rate compared to peers classified with severe MR (Switzky, Haywood, & Rotatori, 1982). Switzky et al. (1982) have identified two distinct subgroups: (a) people who totally lack all adaptive behavior skills and exist in a fragile medical state, and (b) older, less organically involved people with some ambulation, language, communication, and self-help skills. By age 10, some of these children will walk, acquire elements of basic self-care skills (but require supervision in performing self-care), communicate primarily through gesture, respond to selected requests, recognize familiar people, and engage in parallel play or activities (Editorial Board, 1996).

Tables 3 and 4 reflect available speech/language, social behavior, and literacy acquisition data. Categories were each subdivided into normal, moderately delayed, and severely delayed. Normal development was defined as being within the expected range of developmental milestones for a given age. Moderately delayed development was defined as being somewhat below the expected range of developmental milestones for a given age. Generally, for example, subjects would be receiving speech/language services, or ADD or ADHD would have been identified. Severely delayed development was defined as being significantly below the expected range of developmental milestones for a given age. For example, academic assessments might have revealed an 11-year-old student still placed in a 2nd-grade classroom, or a child may have been identified as being out of control, resistant to discipline, exhibiting aggressive or defiant behaviors. Generally, to be classified as severely delayed,

multiple challenges were evident. Anecdotal file notes frequently used the terms moderate or severe when describing conditions. These specific references also assisted in categorizing the findings.

Table 3. Frequency of Occurrence of Severity Observed in 35 Cases of Children With FAS/FAE.

Severity	Speech/Language	Social Behavior	Literacy Acquisition
Normal	4	1	2
Moderately delayed	22	20	20
Severely delayed	6	12	6

Table 4. Frequency of Occurrence of Severity of Social Behavior Performance in 35 Cases of Children With FAS/FAE.

Severity	Number of Cases	Percent
Normal	1	3
Moderately delayed	20	57
Severely delayed	12	34

Frequency distributions were noted and compared in various ways, looking for patterns. To help answer the study's specific questions, Tables 5, 6, 7, and 8 reflect the relationship between scores of speech/language development, social behavior development, and literacy acquisition in children identified with FAS/FAE. Further, Tables 5, 6, 7, and 8 provide information which

suggests whether scores of speech/language and social behavior could be used to predict literacy achievement.

Table 5. Summary of Severity in Four Categories by Individual Case of Children With FAS/FAE.

Case #	IQ	Speech / Language	Literacy Acquisition	Social Behavior
1	1	3	1	2
2	2	3	3	
3	4	2		3
4	4	3		3
5	6	3	3	3
6	2	2	2	3
7	2	2	3	
8	2		2	3
9	2	2	2	3
10	1	2	2	3
11	1		2	3
12	2	2		2
13		2	2	2
14	1	1	2	2
15	2		2	2
16	1	2	2	2
17		2		2
18	4	2	3	3
19	1	1	2	2
20	2	2	2	2
21	2	2	2	2
22	1	2	1	2
23	2	1	2	1
24	1	2		2
25	3	2	2	3
26	2	2	2	3
27	2	2	2	2
28	3	2	2	2
29	2	1	2	3
30	2	2	2	2
31	2	2	3	2
32		2		2
33	3	3	3	2
34	3	3	2	2
35	2	2		2

IQ Legend	
1	Normal
2	Borderline MR
3	Mild MR
4	Moderate MR
5	Severe MR
6	Profound MR

Speech/Language, Literacy Acquisition, Social Behavior Legend	
1	Normal
2	Moderately Delayed
3	Severely Delayed

Table 6. Literacy Levels Compared to Levels of IQ in 35 Cases of Children With FAS/FAE.

Literacy Acquisition Level	IQ Level						
	1	2	3	4	5	6	nd
nd	1	2	0	2	0	0	2
1	2	0	0	0	0	0	0
2	5	11	3	0	0	0	1
3	0	3	1	1	0	1	0

nd=Not Determined, 1=Normal, 2=Moderately Delayed, 3=Severely Delayed.

Table 7. Literacy Levels Compared to Levels of Speech/Language Development in 35 Cases of Children With FAS/FAE.

Literacy Acquisition Level	Speech/Language Development Level			
	1	2	3	nd
nd	0	6	1	0
1	0	1	1	0
2	4	12	1	3
3	0	3	3	0

nd=Not Determined, 1=Normal, 2=Moderately Delayed, 3=Severely Delayed.

Table 8. Literacy Levels Compared to Levels of Social Behavior in 35 Cases of Children With FAS/FAE.

Literacy Acquisition Level	Social Behavior Performance Level			
	1	2	3	nd
nd	0	5	2	0
1	0	2	0	0
2	1	11	8	0
3	0	2	2	2

nd=Not Determined, 1=Normal, 2=Moderately Delayed, 3=Severely Delayed.

Analysis

Frequency distributions of relevant variables were evaluated. Particularly, speech/language development and social behavior development were examined as predictors of literacy acquisition in children with FAS/FAE. Since all variables to be considered in this study were quantitative, degree of relationship was the primary research purpose.

CHAPTER IV

CASE DATA

This chapter provides a summary of data gathered from subject files. Thirty-five files of accumulated records representing children with FAS/FAE were evaluated. Each of these files provided extensive descriptions of physical, psychological, and social development. Both male and female subjects are represented in this summary. Files examined revealed considerable variation in the timing of screenings and data accumulation, however, subjects were generally evaluated shortly after birth, particularly in the case of high-risk pregnancies, prior to entering school, and some time during the course of the early elementary school years. In some instances, data continued to be recorded into the upper elementary school and middle school years. References to normal, moderately delayed, or severely delayed development or performance are generalized descriptors. Specific definitions of those descriptions were impossible due to variations of assessment instruments and narrative contained within the files. Findings are summarized here by individual case.

Case 1: IQ: average

> Speech/language performance: language disorder, significantly below average
>
> Social behavior performance: hyperactivity, adaptive scores suggesting performance somewhat below grade level

Academic performance: reading at grade level, spelling help needed

Other: general developmental delay, low or very low range in auditory memory, difficulty in duplicating designs

Case 2: IQ: borderline level of intellectual functioning

Speech/language performance: substantial language acquisition delays, articulation difficulties

Social behavior performance: no data available

Academic performance: attention difficulties, functioning several grade levels behind

Other: no information available

Case 3: IQ: moderate MR

Speech/language performance: several months delayed

Social behavior performance: a year or more delayed

Academic performance: no data available because subject was too young

Other: no information available

Case 4: IQ: moderate MR

Speech/language performance: significant delay

Social behavior performance: significant delay, history of social deprivation

Academic performance: no data available

Other: general developmental delays; at age 2, history of bones broken on multiple occasions; dysgenesis of corpus collosum; cerebral atrophy; premature birth

Case 5: IQ: profound MR

Speech/language performance: severely delayed

Social behavior performance: severely delayed

Academic performance: severely delayed

Other: paralytic scoliosis, cerebral palsy, profound developmental delays generally, mild microcephaly with roving gaze, not interactive or responsive, mother alcoholic, baby born at home

Case 6: IQ: low average range of intellectual functioning

Speech/language performance: early language delays, verbal, argumentative

Social behavior performance: emotionally disturbed, conduct disorder, ADHD

Academic performance: moderate delay

Other: being served in an emotionally disturbed classroom

Case 7: IQ: borderline intellectual functioning

Speech/language performance: adequate expressive vocabulary, concern about strengthening overall verbal abilities to improve academic potential and performance on language based tasks

Social behavior performance: no information available

Academic performance: reading is especially difficult, overall functioning below grade level

Other: no information available

Case 8: IQ: borderline intellectual functioning

Speech/language performance: no data available

Social behavior performance: behavior problems, aggressive, impulsive

Academic performance: general difficulty

Other: inhalant abuse, sexual abuse, satanic ritual abuse, multiple mental symptoms, mother alcoholic and drug abuser

Case 9: IQ: borderline intellectual functioning

Speech/language performance: moderate impairment

Social behavior performance: severe delay with particular difficulty with daily living, communication, and socialization

Academic performance: moderate delay

Other: no information available

Case 10: IQ: normal

Speech/language performance: 2 years delayed with particular difficulty in expressive vocabulary

Social behavior performance: significantly below average; 12-year, 7-month-old subject manifesting adaptive behavior of a beginning 4-year-old

Academic performance: moderate delay with particular difficulty in reading comprehension

Other: two languages spoken at home

Case 11: IQ: normal

Speech/language performance: delayed receptive vocabulary

Social behavior performance: ADD, long-standing behavioral difficulties, noncompliance

Academic performance: performing slightly below grade level, learning disability in reading comprehension

Other: substance abuse, running away

Case 12: IQ: borderline intellectual functioning

Speech/language performance: general difficulties

Social behavior performance: 20-month delay

Academic performance: no data available, subject too young for evaluation

Other: no information available

Case 13: IQ: no information available

Speech/language performance: direct and indirect speech therapy needed to foster expressive and receptive language, speaks with simple sentences

Social behavior performance: developmental delay

Academic performance: cognitive delays

Other: receiving special education services, premature birth, hydrocephaly with shunt

Case 14: IQ: normal

Speech/language performance: age appropriate to above average

Social behavior performance: moderate delay, particular difficulty with motor skills

Academic performance: 6 to 8 months delayed on developmental testing

Other: no information available

Case 15: IQ: borderline

Speech/language performance: no data available

Social behavior performance: moderately low to adequate, particular difficulty with daily living skills, ADHD

Academic performance: low, particularly in spelling and word recognition

Other: no information available

Case 16: IQ: average

Speech/language performance: moderately low expressive and receptive vocabulary, articulation ok

Social behavior performance: moderate delays in all areas

Academic performance: below grade level, particularly in reading comprehension

Other: no information available

Case 17: IQ: no information available

Speech/language performance: some difficulty with articulation, expressive vocabulary delayed, receptive normal

Social behavior performance: moderate delay

Academic performance: cognitive delays

Other: central nervous system abnormalities, mom used alcohol and methamphetamine, birth weight 1 lb. 4 oz.

Case 18: IQ: moderate MR

Speech/language performance: moderately delayed, particularly in expressive and receptive vocabulary

Social behavior performance: significantly delayed, particularly related to communication, daily living, and socialization

Academic performance: significantly below grade level with particular difficulty in spelling

Other: no information available

Case 19: IQ: normal

Speech/language performance: difficulty with articulation, vocabulary in normal range

Social behavior performance: special needs education recommended due to behavior, ADHD

Academic performance: mild educational delays

Other: mother binged on weekends, 18 cans to a case during last 5 months of pregnancy, FAS determined by mother's history and primary behavioral manifestations, physical manifestations were minor

Case 20: IQ: borderline

Speech/language performance: articulation average, expressive and receptive vocabulary 1 ½ to 2 years delayed, borderline to below average functioning

Social behavior performance: low-level functioning, particularly related to socialization and communication, ADHD

Academic performance: at grade level in most areas, yet significant problems related to reading comprehension

Other: lack of physical manifestations

Case 21: IQ: borderline

Speech/language performance: articulation intelligible, expressive vocabulary normal, receptive vocabulary 3 years delayed

Social behavior performance: attention deficit

Academic performance: LD placement in all core areas

Other: mother reportedly smoked 2 packs of cigarettes per day, drank 3-4 beers per day, and 1 glass of hard liquor a day; lacking physical stigmata; whole language approach recommended for academics

Case 22: IQ: normal

Speech/language performance: speech/language disorders

Social behavior performance: ADD, behavioral problems

Academic performance: generally normal, slight difficulty with writing

Other: mother alcoholic, prenatal alcohol exposure with no significant effect

Case 23: IQ: borderline

Speech/language performance: expressive vocabulary lower, perhaps due to limited exposure or cultural influence, generally no difficulty communicating

Social behavior performance: ADD, good social skills

Academic performance: moderately delayed, low grade level performance

Other: microcephaly, mother heavy to moderate drinking, neuropsychiatric evaluation evidenced no abnormalities

Case 24: IQ: normal

Speech/language performance: speech/language disorders

Social behavior performance: ADD

Academic performance: no data available, too young to evaluate

Other: special education

Case 25: IQ: mild MR

Speech/language performance: developmental disorder; weaknesses in long-term verbal memory, verbal and word knowledge, word fluency, and word usage

Social behavior performance: autistic-like behaviors, hyperactivity, and attentional problems

Academic performance: placed in special education due to academic underachievement

Other: no data available

Case 26: IQ: borderline

Speech/language performance: mild deviation in articulation, moderate deficit related to expressive and receptive vocabulary

Social behavior performance: Oppositional Defiant Disorder, behavior difficulties, hyperactive attention problems

Academic performance: several years behind with particular difficulty in reading comprehension

Other: mother went on and off drugs while breastfeeding; heavy alcohol, 2 packs of cigarettes/day, and illicit drugs including cocaine during pregnancy

Case 27: IQ: borderline

Speech/language performance: intervention needed, articulation disorder

Social behavior performance: ADHD or oppositionality and emotional maturity

Academic performance: below grade level, receiving LD services related to word recognition and reading comprehension

Other: mother used alcohol and cocaine during and after pregnancy

Case 28: IQ: mild MR

Speech/language performance: speech/language disorder

Social behavior performance: ADD, hyperactivity

Academic performance: special education placement

Other: neurological health concerns, microcephaly evaluation done, poor fine motor coordination, mother moderate to heavy alcohol consumption

Case 29: IQ: borderline

Speech/language performance: good articulation, high average expressive vocabulary, average receptive vocabulary

Social behavior performance: verbal impulsivity, hyperactive, short attention span, needs reminders to accomplish a task, demands continuous attention, resistant to discipline, aggression, swearing, restlessness, distractibility, delayed motor development

Academic performance: slow thought processes

Other: neurological exam normal, both parents heavy alcohol and cocaine use

Case 30: IQ: borderline

Speech/language performance: developmental language disorder related to expressive and receptive vocabulary, prolonged pauses, fillers, starters, and perseverative repetitions and phrases, strong grammar and syntax, weak vocabulary, language comprehension, word finding and retrieval, auditory retention

Social behavior performance: socialization and motor skills scores significantly below normal

Academic performance: significantly below grade level

Other: no information available

Case 31: IQ: borderline

Speech/language performance: difficulties related to expressive and receptive vocabulary

Social behavior performance: severely delayed with particular difficulties related to communication, ADHD

Academic performance: severely delayed

Other: history of victimization, LD in reading, vocabulary deficits, mother alcoholic

Case 32: IQ: general developmental delay

Speech/language performance: moderate delay

Social behavior performance: moderate delay

Academic performance: no data available, subject too young for evaluation

Other: failure to thrive, premature birth, ventricular septal defect, mother MR and alcoholic

Case 33: IQ: mild MR

Speech/language performance: articulation ok, receptive vocabulary severely delayed

Social behavior performance: moderate deficit, ADD, conduct disturbance, impulsive, inattentive, overall emotional behavior function is several years delayed

Academic performance: difficult in all areas

Other: age 11 in 1st grade

Case 34: IQ: mild MR

Speech/language performance: severe articulation disorder, expressive and receptive vocabulary disorder

Social behavior performance: severely delayed, ADHD, poor fine motor skills

Academic performance: serviced by an educable handicapped classroom

Other: hearing problems, mother used alcohol and smoked 1-2 packs of cigarettes/day

Case 35: IQ: borderline

Speech/language performance: speech/language delay

Social behavior performance: ADHD, emotional behavior consistent with abused or neglected children, high activity level, short attention span, distractibility

Academic performance: no data available, subject too young for evaluation

Other: same mother as case 34 who used alcohol and smoked 1-2 packs of cigarettes/day

Data from 35 cases of children identified with FAS/FAE have been described. Categories of relevant data have included IQ, speech/language performance, social behavior performance, and academic performance with special emphasis being given to literacy skills. A miscellaneous category entitled "other" was also included to provide a place for additional descriptive data which might lend additional understanding to case circumstances. Analysis of group patterns will be discussed in the next chapter.

CHAPTER V

RESULTS

Upon evaluation of accumulated data and summary tables, the following patterns were observed:

Evidence of developmentally delayed speech/language functions and social behavior performance generally relate highly with literacy acquisition in children with FAS/FAE, as suggested by the study's first hypothesis. Descriptions contained within case files limited the study's ability to note specific aspects of the literacy acquisition process such as phonemic awareness and other subskills.

Based on the cases reviewed, it generally appears that social behavior bears slightly more significant influence on literacy acquisition than does speech/language development. This finding calls into question the study's second hypothesis which stated that speech/language development would prove to be the critical indicator of literacy acquisition. Instead, a child's ability to attend to instruction and interact positively in the environment of the classroom suggested likelihood of academic success. In all cases where speech/language development was normal, literacy skills were still low. In each of these instances, social behavior showed moderate to severe delay. IQ may have been a

contributing factor as well. In the worst cases of speech/language delay, literacy skills measured at roughly the same level of delay.

Categories of data evaluated included IQ, speech/language development, social behavior performance, and literacy acquisition. Every category contained instances of missing data. Where practical, predictions of missing values were made based on narrative descriptions found in case files. In some instances, information was too sketchy to justify value description. Consequently, summary totals do not always reflect 100% case representation. It should be noted that 7 out of 35 cases were missing literacy values, generally due to the age of the subject evaluated. This is approximately double the missing values from other categories. Therefore, it is likely that if these values had been available, summary findings could have been different. For example, speech/language delays may have shown to be more critical to literacy acquisition than presently apparent.

Each category was subdivided to offer more specific definition or better description of severity. Numerical values were then assigned to these subdivisions to enable comparison.

Out of all the cases reviewed, only 2 cases demonstrated normal literacy acquisition when speech/language and social behavior were delayed. Cases studied generally demonstrated moderate literacy delay. In both of those cases of normal literacy, IQ was normal. Generally, normal IQ did not correlate with normal speech/language or social behavior. Most cases revealed normal or borderline IQ.

This chapter has provided summarizing description of the subject files reviewed for this study. Group patterns have been described. Initial research questions pertaining to this study have been answered as follows:

- What is the relationship between FAS/FAE and IQ?

  In 24 out of 32 cases, IQ measures were observed to be below normal measures, with the majority of cases falling into the borderline MR category. (See Table 2.)

- What is the relationship between FAS/FAE and speech/language development?

  In 28 out of 32 cases, speech/language measures were observed to be below normal, with the majority of cases falling into the moderately delayed category. (See Table 3.)

- What is the relationship between FAS/FAE and social behavior?

  In 32 out of 33 cases, social behavior measures were observed to be below normal, with the majority of cases falling into the moderately delayed category. (See Table 3.)

- What is the relationship between FAS/FAE and literacy acquisition?

  In 26 out of 28 cases, literacy acquisition measures were observed to be below normal, with the majority of cases falling into the moderately delayed category. (See Table 3.)

- What are the levels of literacy of a sample of children diagnosed with FAS/FAE?

In 2 out of 28 cases, literacy acquisition levels appeared normal.

In 20 out of 28 cases, literacy acquisition levels appeared to be moderately delayed.

In 6 out of 28 cases, literacy acquisition levels appeared to be severely delayed. (See Table 3.)

- How does IQ relate to literacy acquisition in children with FAS/FAE?

Thirty-two out of 35 cases reported IQ scores. In 8 out of those 32 cases, IQ scores were normal. Of that number, literacy acquisition appeared normal in 2 cases, while literacy acquisition appeared moderately delayed in 5 cases. One case did not report literacy scores.

In 16 out of 32 cases reporting IQ scores, scores revealed borderline MR. Of that number, literacy acquisition appeared moderately delayed in 11 instances, and severely delayed in 3 cases. One case did not report literacy scores.

In 4 cases out of 32, IQ scores showed mild MR. Of that number, literacy acquisition appeared moderately delayed in 3 cases, and severely delayed in 1 case. One case did not report literacy scores.

In 3 cases out of 32, IQ scores showed moderate MR. Of that number, literacy acquisition appeared severely delayed in 1 case. Two cases did not report literacy scores.

No cases out of 32 showed severe MR.

In 1 case out of 32, IQ scores showed profound MR. In that case, literacy acquisition was severely delayed.

- How does speech/language development relate to literacy acquisition in children with FAS/FAE?

Thirty-two out of 35 cases reported speech/language scores. Of that number, 4 showed normal speech/language development, and all 4 demonstrated moderately delayed literacy acquisition.

In 22 out of 32 cases, moderately delayed speech/language scores were noted. Of that number, 1 reported normal literacy acquisition, 12 reported normal literacy, and 3 reported severely delayed. Six did not report literacy scores.

In 6 out of 32 cases, severely delayed speech/language scores were reported. Of that number, 1 reported normal literacy acquisition, 1 reported moderately delayed literacy, and 3 reported severely delayed literacy. One did not report a literacy score.

- How does social behavior relate to literacy acquisition in children with FAS/FAE?

Thirty-three out of 35 cases reported social behavior scores. Of that number, 1 reported normal social behavior, and that case showed moderate literacy delay.

In 20 out of 33 cases, moderate delay was evident in social behavior. Of that number, 2 showed normal literacy, 11 showed moderate literacy delay, and 2 showed severe literacy delay. Five did not report literacy scores.

In 12 out of 33 cases, severe delay was evident in social behavior. Of that number, 0 cases showed normal literacy, 8 showed moderate literacy delay, and 2 showed severe literacy delay. Two did not report literacy scores.

In summary, cases of children identified with FAS/FAE generally demonstrated moderate literacy delay. IQ in children identified with FAS/FAE ranged from normal to profound MR. Speech/language and social behavior performance in the cases studied ranged from normal to severely delayed.

CHAPTER VI

CONCLUSIONS AND RECOMMENDATIONS

This chapter provides a synthesis of study findings, draws conclusions, and provides recommendations for further study and consideration.

Ethanol is clearly a teratogen that can cross the placenta and interrupt development at any time in pregnancy (Clarren & Astley, 2001). Timing and extent of exposure often determine manifestations of the condition, though variations certainly exist. The United States Surgeon General's conclusion that zero exposure to alcohol equals zero risk remains the only defensibly true statement (U.S. Public Health Service, 1981).

In utero alcohol exposure carries with it a range of developmental risks potentially impacting every system of the human body, most typically the central nervous system. There is no known treatment to undo its effects. Medication and rehabilitation services can subdue damage in some cases, though these interventions are generally costly and of limited help.

Fetal Alcohol Syndrome (FAS) and its more broadly defined condition, Fetal Alcohol Effects (FAE), impact scores of individuals and their families every day, yet costs are staggering to the whole of society. In addition to emotionally straining already dysfunctional families and demanding endless resources from

social welfare entities, these conditions carry the additional stigma of being completely preventable.

It is important to realize that most women have no knowledge of FAS/FAE. For example, in a statewide survey in Texas, it was found that more than 60% of women had never heard of FAS, nor were they aware of the adverse effects of drinking in pregnancy (Little et al., 1998).

Certainly, a prudent response to this vulnerability would be pervasive educational programs targeting sexually active women of childbearing age. Beginning at the middle school level, young girls should begin to hear about the potential risks of alcohol during pregnancy. This campaign should continue throughout high school and crescendo on university campuses where partying and sexual activity are at their peak. Every woman should routinely receive information from health care providers. Social service agencies serving women in poverty or other stressful circumstances should certainly offer counseling.

Schools have a responsibility to offer appropriate services for children with FAS/FAE who are struggling in the classroom. Cooperative efforts must be coordinated with health and social service entities. Research-derived guidelines must be provided for parents and school personnel so that learning activities, environments, and expectations can be appropriately designed.

At the federal and state levels, common sense legislation is imperative. Rather than penalizing individuals for not keeping pace with their academic peers, reasonable expectations must be formulated which respond flexibly to the unique circumstances of individual children and their families. Funding must be

allocated to back aggressive public education regarding the risks of alcohol during pregnancy.

Children who have a hearing or language impairment or who lack age-appropriate skills in literacy related cognitive processing must be identified as early as possible and given intervention to support language and literacy development. This recommendation implies the need for all adults responsible for the child's well-being to understand and use, in a regular and systematic way, multiple identification measures. Guidance about how to identify signs that children are having difficulties achieving early language and literacy skills needs to be provided to parents and other significant adults in the child's life: pediatricians, nurses, social workers, speech and language professionals, and daycare and preschool professionals. All children, especially those at risk for reading difficulties, should have access to early childhood environments that provide language and literacy growth and that address reading risk factors in an integrated rather than isolated fashion (Committee on the Prevention of Reading Difficulties in Young Children, 1998).

Schools must do their best to provide appropriate services for these children. Health care must do its best to meet physical needs and conduct ongoing research. But most significantly, society must respond to the disintegration of social structures which creates an environment where such misery can spawn. Cultural acceptance of intoxication and binge drinking must certainly be addressed. Even moderate social drinking must come under reevaluation.

Stable family structures must be fostered. Parents who are incapable of making wise health and behavioral choices before and during pregnancy are rarely able to abruptly change their lifestyles in any significant way so that they are ready to care for a high-needs infant. Abuse and neglect are all too common in the lives of many of these children. Again, education and social support structures must be accessible.

Foster care placements for these children are common. Yet, even the best care providers suffer burnout if adequate respite care is unavailable. Multiple placements for already troubled children serve only to complicate matters. Stable environments must be provided which will foster healthy emotional and social development.

In addition to numerous social implications, this study suggests a number of considerations related to future research.

It must be noted that many variables impact literacy acquisition. It would, therefore, be inappropriate to state that any one or two variables account for success or failure in literacy acquisition. Certainly, speech/language development cannot be denied its place. Social behavior, as this study suggests, is at least of equal importance. IQ, as reviewed by this study, can be noticeably impacted by FAS/FAE and in turn brought to bear in the equation as well. The list of contributing factors in the process of literacy acquisition, however, is extensive. Anything from learning styles to teaching methods, to name only two potential variables, could be considered.

Future studies might consider the impact of FAS/FAE severity levels on literacy acquisition. Identification of minor criteria and prenatal alcohol exposure levels would need to become more precise, a difficult task where mothers are often hesitant to be straightforward with information or where they do not follow through with consistent prenatal or postnatal care.

Significance tests of individual variables might also yield insights into the development of appropriate response strategies. A host of special service providers could benefit from additional understanding of the variables that contribute to not only literacy acquisition but the entire range of life skills.

A battery of specific language tests might be offered to children with FAS/FAE to determine any patterns of specific developmental delays. Patterns detected might serve to suggest research topics related to intervention strategies.

Additional research needs to be done to determine the extent to which a father's alcohol use compromises the health of the sperm, and if, in fact, any genetic predisposition to FAS/FAE is established. Very little can be found in the literature which addresses the role of the father. Certainly, both parents bear responsibility for the health of their child.

Diagnostic screenings making use of common assessment tools should be standard procedure between health care workers and their clients. Efforts must be made to build trust relationships with women so that they can feel safe and subsequently more open to health care suggestions. This requires time, commitment, and follow-through by nonjudgmental staff who can build rapport

with women and help guide them to healthier choices both for themselves and for their babies.

Information storage and retrieval procedures need improvement as well. Records must be computerized in standard formats if they are to be accessible in an efficient way to researchers and others. The sheer time it takes to wade through hand-scrawled notations regarding patients is enough to discourage further study. Electronic databases would go a long way toward swift manipulation and reflection upon individual or mass cases.

In summary, efficient data gathering and storage must be developed. Continued brain research will no doubt yield new and amazing insights into the workings of the human body. Collaboration between social and health care agencies must be improved. Educational entities must continue to develop effective instructional practices which meet the needs of this particular group of youngsters. Yet, without question, prevention efforts are the only reasonable response and must receive our most concerted efforts. Anything else is too little, too late. For the researcher, the most significant thing that has come out of this study has been the opportunity to get a glimpse into the lives of precious little ones who, through no fault of their own, endure great pain and hardship, as a result of choices their parents have made. Certainly, literacy acquisition is but one area needlessly impacted by FAS/FAE.

REFERENCES

Abel, E. L. (1990). *Fetal Alcohol Syndrome.* Oradell, NJ: Medical Economics.

Abel, E. L. (1998). *Fetal alcohol abuse syndrome.* New York: Plenum Press.

Abel, E. L., & Sokol, R. J. (1987). Incidence of Fetal Alcohol Syndrome and economic impact of FAS-related anomalies. *Drug and Alcohol Dependence, 19,* 51-70.

Adams, M. J. (1990). *Beginning to read: Thinking and learning about print.* Cambridge: The MIT Press.

Andres, R. L., & Jones, K. L. (1994). Social and elicit drug use in pregnancy. In R. K. Creasy & R. Resnick (Eds.), *Maternal-fetal medicine: Principles and practice* (3rd ed., pp. 182-198). Philadelphia: W. B. Saunders.

Astley, S. J., Weinberger, E., Shaw, D. W. W., Richards, T. L., & Clarren, S. K. (1995). Magnetic resonance imaging and spectroscopy in fetal-ethanol-exposed Macaca nemestrina. *Neurotoxicology and Teratology, 17,* 523-530.

Autti-Ramo, I., & Granstrom, M-L. (1991). The effect of intrauterine alcohol exposition in various durations on early cognitive development. *Neuropediatrics, 22,* 203-210.

Badian, N. A., McAnulty, G. B., Duffy, F. H., & Als, H. (1990). Prediction of dyslexia in kindergarten boys. *Annals of Dyslexia, 40,* 152-169.

Baydar, N., Brooks-Gunn, J., & Furstenberg, F. F. (1993). Early warning signs of functional illiteracy: Predictors in childhood and adolescence. *Child Development, 64,* 815-829.

Beauregard, M., Chertkow, H., Bub, D., Murtha, S., Dixon, R., & Evans, A. (1997). The neural substrate of concrete, abstract, and emotional word lexical: A positron emission tomography study. *Journal of Cognitive Neuroscience, 9,* 441-461.

Becker, M., Warr-Leeper, G. A., & Leeper, H. A. (1990). Fetal Alcohol Syndrome: A description of oral motor, articularity, short-term memory, grammatical, and semantic disabilities. *Journal of Communication Disorders, 23,* 97-124.

Burd, L. (2001). *Children with Fetal Alcohol Syndrome: A handbook for parents and teachers.* Grand Forks: North Dakota Fetal Alcohol Syndrome Center.

Burd, L., Martsolf, J. T., Klug, M. G., & Kerbeshian, J. (2003). Diagnosis of FAS: A comparison of the Fetal Alcohol Syndrome diagnostic checklist and the Institute of Medicine criteria for Fetal Alcohol Syndrome. *Neurotoxicology and Teratology, 25*(6), 719-724.

Caruso, K., & ten Bensel, R. (1993). Fetal Alcohol Syndrome and Fetal Alcohol Effects. *Minn Med, 76,* 25.

Catts, H. W. (1991). Early identification of dyslexia: Evidence from a follow-up study of speech-language impaired children. *Annals of Dyslexia, 41,* 163-177.

Catts, H. W. (1997). The early identification of language-based reading disabilities. *Language, Speech, and Hearing Services in the Schools, 28,* 86-89.

Church, M. W., Abel, E. L., Kaltenbach, J. A., & Overbeck, G. W. (1996). Effects of prenatal alcohol exposure and aging on auditory function in the rat: Preliminary results. *Alcoholism: Clinical and Experimental Research, 20,* 172-179.

Church, M. W., Eldis, F., Blakley, B. W., & Bawle, E. W. (1997). Hearing, language, speech, vestibular, and dentofacial disorders in the Fetal Alcohol Syndrome (FAS). *Alcoholism: Clinical and Experimental Research, 21,* 227-237.

Church, M. W., & Gerkin, K. P. (1988). Hearing disorders in children with Fetal Alcohol Syndrome: Findings for case reports. *Pediatrics, 82,* 147-154.

Clark, S. L., Cotton, D. B., Hankins, G. D. V., & Phelan, J. P. (Eds.). (1999). *Critical care obstetrics* (3rd ed.). Malden, MA: Blackwell Science.

Clarren, S. K. (1986). Neuropathology in Fetal Alcohol Syndrome. In J. R. West (Ed.), *Alcohol and brain development* (pp. 158-166). New York: Oxford University Press.

Clarren, S. K., & Astley, S. J. (2001). Fetal Alcohol Syndrome. In S. B. Cassidy & J. E. Allanson (Eds.), *Management of genetic syndromes* (pp. 151-164). New York: Wiley-Liss.

Cohen, L., Dehaene, S., Naccache, L., Lehericy, S., Dehaene-Lambertz, G., Henaff, M. A., & Michel, F. (2000). The visual word form area: Spatial and temporal characterization of an initial stage of reading in normal Subjects and posterior split-brain patients. *Brain, 123,* 291-307.

Committee on the Prevention of Reading Difficulties in Young Children. (1998). *Preventing reading difficulties in young children.* Washington, DC: Natinal Academy Press.

Davies, M. (2003). The role of $GABA_A$ receptors in mediating the effects of alcohol in the central nervous system. *Journal of Psychiatry Neuroscience, 28*(4), 263-274.

Dehaene, P. H., Samaille-Villette, C., Samaille, P. P., Crepin, G., Waldbaum, R., et al. (1977). Le syndrome d'alcoolisme, foetal dans le nord de la France. *Revere de l'Alcoolisme, 23,* 145-158.

Dorris, M. (1989). *The broken cord.* New York: Harper & Row.

Editorial Board. (1996). Definition of mental retardation. In J. W. Jacobson & J. A. Mulick (Eds.), *Manual of diagnosis and professional practice in mental retardation* (pp. 11-54). Washington, DC: American Psychological Association.

Ferrer, I., & Galofre, E. (1987). Dendritic spine anomalies in Fetal Alcohol Syndrome. *Neuropediatrics, 18,* 161-163.

Folb, P. I., & Dukes, M. N. G. (1990). *Drug safety in pregnancy.* Amsterdam, The Netherlands: Elsevier.

Francis, D. J., Shaywitz, S. E., Stuebing, K. K., Shaywitz, B. A., & Fletcher, J. M. (1996). Developmental lag versus deficit model of reading disability: A longitudinal, individual growth curve analysis. *Journal of Educational Psychology, 88,* 3-17.

Fried, P. A., & Watkinson, B. (1988). 12- and 24-month neurobehavioural follow-up of children prenatally exposed to marijuana, cigarettes, and alcohol. *Neurotoxicology and Teratology, 10,* 305-313.

Friston, K. J., Frith, C. D., Liddle, P. F., & Frackowiak, R. S. J. (1993). Functional connectivity: The principal-component analysis of large (PET) data sets. *Journal of Cerebral Blood Flow and Metabolism, 13,* 5-14.

Goodman, Y. M., Watson, D. J., & Burke, C. L. (1987). *Reading Miscue Inventory: Alternative procedures.* Katonah, NY: Richard C. Owen Publishers.

Greenberg, D. A. (2003, July 8). Linking acquired neurodevelopmental disorders to defects in cell adhesion. *Proceedings of the National Academy of Sciences, USA, 100*(14), 8043-8044.

Greene, T., Ernhart, C. B., Ager, J., Sokol, R., Martier, S., et al. (1991). Prenatal alcohol exposure and cognitive development in the preschool years. *Neurotoxicology and Teratology, 13,* 57-68.

Guerri, C. (2002). Mechanisms involved in central nervous system dysfunctions induced by prenatal ethanol exposure. *Neurotox Res, 4*(4), 327-335.

Hamilton, M. A. (1981). *Linguistic abilities of children with Fetal Alcohol Syndrome.* Ann Arbor, MI: University Microfilms International.

Hannigan, J. H., Martier, S. S., Chugani, H. T., & Sokol, R. J. (1995). Brain metabolism in children with Fetal Alcohol Syndrome (FAS): A positron emission tomography study. *Alcoholism: Clinical and Experimental Research, 19,* 53A.

Hiemenz, J. R., & Hynd, G. W. (2000). Sulcal/gyral pattern morphology of the perisylvian language region in developmental dyslexia. *Brain and Language, 74,* 113-133.

Institute of Medicine. (1996). *Fetal Alcohol Syndrome.* Washington, DC: National Academy Press.

Iosub, S., Fuchs, M., Bingol, N., & Gromisch, D. S. (1981). Fetal Alcohol Syndrome revisited. *Pediatrics, 68,* 475-479.

Jones, K. L., Smith, D. W., Ulleland, C. N., & Streissguth, A. P. (1973). Pattern of malformation in offspring of chronic alcoholic women. *Lancet, 1,* 1267-1271.

Korkman, M., Kettunen, S., & Autti-Ramo, I. (2003). Neurocognitive impairment on early adolescence following prenatal alcohol exposure of varying duration. *Neuropyschol Dev Cogn Sect C Child Neuropyschol, 9*(2), 117-128.

Kuriki, S., Takeuchi, F., & Hirata, Y. (1998). Neural processing of words in the human extrastriate visual cortex. *Cognitive Brain Research, 6,* 193-203.

Kyllerman, M., Aronson, M., Sabel, K. G., Karlberg, E., Sandin, B., et al. (1985). Children of alcoholic mothers: Growth and motor performance compared to matched controls. *Acta Paediatrica Scandinavica, 74,* 10-26.

Le Douarin, N. M., Hallonet, M. E. R., & Pourquié, O. (1994). Cell migrations and establishment of neuronal connections in the developing brain: A study using the quail-chick chimera system. In F. E. Bloom (Ed.), *Neuroscience: From the molecular to the cognitive* (pp. 3-17). Amsterdam, The Netherlands: Elsevier.

Lemoine, P., Harousseau, H., Borteryu, J. P., & Menuet, J. C. (1968). Les enfants de parents alcooliques: Anomalies observées à propos de 127 cas [The children of alcoholic parents: Anomalies observed in 127 cases]. *Quest Medicale, 21*, 476-482.

Leonard, C. M., Eckert, M. A., Lombardino, L. J., Oakland, T., Kranzler, J., Mohr, C. M., et al. (2001). Anatomical risk factors for phonological dyslexia. *Cerebral Cortex, 11*, 148-157.

Leonardson, G. R. (2003). Risk factors for alcohol use during pregnancy in a multistate area. *Neurotoxicology and Teratology, 25*(6), 651-658.

Lewis, B. A., Freebairn, L. A., & Taylor, H. G. (2000). Academic outcomes in children with histories of speech sound disorders. *Journal of Communication Disorders, 33*, 11-30.

Little, B. B., VanBeveren, T. T., & Gilstrap, L. C., III. (1998). Alcohol use during pregnancy and maternal alcoholism. In L. C. Gilstrap & B. B. Little (Eds.), *Drugs and pregnancy* (2nd ed., pp. 395-404). New York: Chapman & Hall.

Loser, H. (1995). *Alkohol embryopathie and alkoholeffekte* [Alcohol embryopathy and alcohol effects]. Stuttgart, Germany: Gustav Fischer Verlag.

Majewski, F. (1981). Alcohol embryopathy: Some facts and speculations about pathogenesis. *Neurobehavioral Toxicology and Teratology, 3,* 129-144.

Majewski, F., Bierich, J. R., Loser, H., Michaelis, R., Leiber, B., et al. (1976). Zur klinik und pathogenese der alkohol-embryopathie; berich uber 68 falle [Clinical aspects and pathogenesis of alcohol embryopathy: A report of 68 cases]. *Munchen Medizinisché Wochenschrift, 118,* 1635-1642.

Marcus, J. D. (1987). Neurological findings in the Fetal Alcohol Syndrome. *Neuropediatrics, 18,* 158-160.

Mason, E. (2000). Drug abuse. In W. M. Barron & M. D. Lindheimer (Eds.), *Medical disorders during pregnancy* (pp. 557-585). St. Louis, MO:Mosby.

Mattson, S. N., Riley, E. P., Jernigan, T. L., Ehlers, C. L., Delis, D. C., et al. (1992). Fetal Alcohol Syndrome: A case report of neuropsychological, MRI, and EEG assessment of two children. *Alcoholism: Clinical and Experimental Research, 16,* 1001-1003.

Mattson, S. N., Riley, E. P., Jernigan, T. L., Garcia, A., Kaneko, W. M., et al. (1994). A decrease in the size of the basal ganglia following prenatal alcohol exposure: A preliminary report. *Neurotoxicology and Teratology, 16,* 283-289.

Morgan, A. E., & Hynd, G. W. (1996). *Anatomical variation of the planum temporale: Implications for dyslexia and linguistic ability.* Unpublished manuscript.

Morrow-Tlucuk, M., & Ernhart, C. B. (1987). Maternal prenatal substance use and behavior at age 3 years. *Alcoholism: Clinical and Experimental Research, 11,* 213.

Morse, B. A. (1993). Information processing. In J. Kleinfeld & S. Wescott (Eds.), *Fantastic Antoine succeeds!: Experiences in educating children with Fetal Alcohol Syndrome* (pp. 223-236). Anchorage: University of Alaska Press.

Morse, B. A., & Cermak, S. (1994). Sensory processing in children with FAS. *Alcoholism: Clinical and Experimental Research, 18,* 503.

Morse, B. A., & Weiner, L. (1996). Rehabilitation approaches for Fetal Alcohol Syndrome. In H. L. Spohr & H. C. Steinhausen (Eds.), *Alcohol, pregnancy, and the developing child* (pp. 249-268). Cambridge, UK: Cambridge University Press.

National Association of State Directors of Special Education. (2002, June). *Implementing the No Child Left Behind Act: What it means for IDEA.* Alexandria, VA: Author.

No Child Left Behind (executive summary). (2001, January 29). Retrieved February 12, 2001, from http://ed.gov/inits/nclb/part2.html

Nobre, A. C., Allison, T., & McCarthy, G. (1994). Word recognition in the human inferior temporal lobe. *Nature, 372,* 260-263.

Olegard, R., Sabel, K. G., Aronsson, M., Sandin, B., Johansson, P., et al. (1979). Effects on the child of alcohol abuse during pregnancy: Retrospective and prospective studies. *Acta Paediatrica Scandinavica, 275,* 112-121.

Padget, S. Y. (1988). Speech- and language-impaired three and four year olds: A five year follow-up study. In R. L. Masland & M. W. Masland (Eds.), *Preschool prevention of reading failure* (pp. 52-77). Parkton, MD: Yorkton Press.

Petersen, S. E., & Fiez, J. A. (1993). The processing of single words studied with positron emission tomography. *Annual Review of Neuroscience, 16,* 509-530.

Phillips, S. C., & Cragg, B. G. (1982). A change in susceptibility of rat Purkinje cells to damage by alcohol during fetal, neonatal, and adult life. *Neuropathology and Applied Neurobiology, 8,* 441-454.

Pierce, D. R., Goodlett, C. R., & West, J. R. (1989). Differential neuronal loss following early postnatal alcohol exposure. *Teratology, 40,* 113-126.

Piper, T. (1998). *Language and learning: The home and school years* (2nd ed.). Upper Saddle River, NJ: Merrill.

Polk, T. A., & Farah, M. J. (2002). Functional MRI evidence for an abstract, not perceptual, word-form area. *Journal of Experimental Psychology: General, 131,* 65-72.

Puce, A., Allison, T., Asgari, M., Gore, J. C., & McCarthy, G. (1996). Differential sensitivity of human visual cortex to faces, letter strings, and textures: A functional magnetic resonance imaging study. *Journal of Cognitive Neuroscience, 16,* 5205-5215.

Reschly, D. J. (1988). Assessment issues, placement litigation, and the future of mild mental retardation classification and programming. *Education and Training in Mental Retardation, 23,* 285-301.

Richardson, J. S., & Morgan, R. F. (2000). *Reading to learn in the content areas* (4th ed.). Belmont, CA: Wadsworth.

Robinson, G. C., Conry, J. L., & Conry, R F. (1987). Clinical profile and prevalence of Fetal Alcohol Syndrome in an isolated community in British Columbia. *Canadian Medical Association Journal, 137,* 203-207.

Roebuck, T. M., Mattson, S. N., & Riley, E. P. (1998). A review of the neuroanatomical findings in children with Fetal Alcohol Syndrome or prenatal exposure to alcohol. *Alcoholism: Clinical and Experimental Research, 22,* 339-344.

Rossig, C., Wasser, S. T., & Oppermann, P. (1994). Audiologic manifestations in Fetal Alcohol Syndrome assessed by brainstem auditory-evoked potentials. *Neuropediatrics, 25,* 245-249.

Russell, M., Czarneci, D. M., Cowan, R., McPherson, E., & Mudar, P. J. (1991). Measures of maternal alcohol use as predictors of development in early childhood. *Alcoholism: Clinical and Experimental Research, 15,* 991-1000.

Scarborough, H. S. (2002). Connecting early language and literacy to later reading (dis)abilities: Evidence, theory, and practice. In S. B. Newman & D. K. Dickinson (Eds.), *Handbook of early literacy research* (pp. 97-110). New York: The Guilford Press.

Sergent, J. (1994). Brain-imaging studies of cognitive function. *Trends in Neuroscience, 17,* 221-227.

Shaywitz, B. A., Pugh, K. R., Jenner, A. R., Fulbright, R. K., Fletcher, J. M., Gore, J. C., & Shaywitz, S E. (2000). The neurobiology of reading and reading disability (dyslexia). In M. L. Kamil, P. B. Mosenthal, P. D. Pearson, & R. Barr (Eds.), *Handbook of reading research* (Vol. 3, pp. 229-249). Mahwah, NJ: Lawrence Erlbaum Associates.

Shaywitz, S. E., Escobar, M. D., Shaywitz, B. A., Fletcher, J. M., & Makuch, R. (1992). Evidence that dyslexia may represent the lower tail of the normal distribution of reading ability. *New England Journal of Medicine, 326,* 145-150.

Sowell, E. R., Mattson, S. N., Riley, E. P., & Jernigan, T. L. (1996). Abnormal development of the cerebellar vermis in children prenatally exposed to alcohol: Size reductions in lobules I-V. *Alcoholism: Clinical and Experimental Research, 20,* 31-34.

Sparks, S. (1984). Speech and language in Fetal Alcohol Syndrome. *Journal of the American Speech and Hearing Association, 26,* 27-31.

Spohr, H. L., & Steinhausen, H. C. (Eds.). (1996). *Alcohol, pregnancy and the developing child.* Cambridge, Great Britain: University Press.

Spohr, H. L., Willms, J., & Steinhausen, H. C. (1993). Prenatal alcohol exposure and long-term developmental consequences. *Lancet, 341,* 907-910.

Stark, R. E., Bernstein, L. E., Condino, R., Bender, M., Tallal, P., & Catts, H. (1984). Four-year follow-up study of language impaired children. *Annals of Dyslexia, 34,* 49-68.

Steinhausen, H. C., Nestler, V., & Spohr, H. L. (1982). Development and psychopathology of children with the Fetal Alcohol Syndrome. *Developmental and Behavioral Pediatrics, 3,* 49-54.

Stevenson, H. W., & Newman, R. S. (1986). Long-term prediction of achievement and attitudes in mathematics and reading. *Child Development, 57,* 646-659.

Streissguth, A. (1997). *Fetal Alcohol Syndrome: A guide for families and communities.* Baltimore: Paul H. Brookes Publishing Co.

Streissguth, A. P., Aase, J. M., Clarren, S. K., Randels, S. P., LaDue, R. A., et al. (1991). Fetal Alcohol Syndrome in adolescents and adults. *Journal of American Medical Association, 265,* 1961-1967.

Streissguth, A. P., Bookstein, F. L., Sampson, P. D., & Barr, H. M. (1993). *The enduring effects of prenatal alcohol exposure on child development: Birth through seven years, a partial least squares solution* (International Academy for Research in Learning Disabilities Monograph Series, No. 10). Ann Arbor: The University of Michigan Press.

Streissguth, A. P., Clarren, S. K., & Jones, K. L. (1985). Natural history of the Fetal Alcohol Syndrome: A 10-year follow-up of eleven patients. *Lancet, 1,* 85-91.

Streissguth, A. P., Herman, C. S., & Smith, D. W. (1978). Intelligence, behavior, and dysmorphogenesis in the Fetal Alcohol Syndrome: A report on 20 patients. *Journal of Pediatrics, 92,* 363-367.

Stromland, K. (1985). Ocular abnormalities in the Fetal Alcohol Syndrome. *Acta Ophthalmologica, 171,* 63.

Switzky, H. N., Haywood, H. C., & Rotatori, A. F. (1982). Who are the severely and profoundly mentally retarded? *Education and Training of the Mentally Retarded, 17,* 268-272.

Tallal, P. (1987). *Developing language disorders* (Report to U.S. Congress). Bethesda, MD: Interagency Committee on Learning Disabilities.

Torgesen, J. K., Wagner, R. K., Rashotte, C. A., Alexander, A. W., & Conroy, T. (1997). Preventative and remedial interventions for children with severe reading disabilities. *Learning Disabilities, 8,* 51-62.

Tramontana, M. G., Hooper, S., & Selzer, S. C. (1988). Research on preschool prediction of later academic achievement: A review. *Developmental Review, 8,* 89-146.

Tran, T. D., & Kelly, S. J. (2003). Critical periods for ethanol-induced cell loss in the hippocampal formation. *Neurotoxicology and Teratology, 25*(5), 519-528.

U.S. Public Health Service. (1981). Surgeon general's advisory on alcohol and pregnancy. *FDA Bulletin, 11,* 9-10.

Vellutino, F. R., & Denckla, M. B. (1996). Cognitive and neuropsychological foundations of word identification in poor and normally developing readers. In R. Barr, M. L. Kamil, P. B. Mosenthal, & P. D. Pearson (Eds.), *Handbook of reading research* (Vol. 2, pp. 571-608). Mahwah, NJ: Lawrence Erlbaum Associates.

Wagner, A. D., Schacter, D. L., Rotte, M., Koutstaal, W., Maril, A., Dale, A. M., et al. (1998). Building memories: Remembering and forgetting of verbal experiences as predicted by brain activity. *Science, 281,* 1188-1191.

West, J. R., Goodlett, C. R., Bonthius, D. J., Hamre, K. M., & Marcussen, B. L. (1990). Cell population depletion associated with fetal alcohol brain damage: Mechanisms of BAC-dependent cell loss. *Alcoholism: Clinical and Experimental Research, 14,* 813-818.

Yamada, T., Placzek, M., Tanaka, H., Dodd, J., & Jessell, T. M. (1991). Control of cell pattern in the developing nervous system: Polarizing activity of the floor plate and notochord. *Cell, 64,* 635-647.

Zhou, F. C., Sari, Y., Powrozek, T., Goodlett, C. R., & Li, T. K. (2003). Moderate alcohol exposure compromises neurol tube midline development in prenatal brain. *Developmental Brain Research, 144*(1), 43-55.