# PRENATAL EXPOSURE TO DRUGS/ALCOHOL

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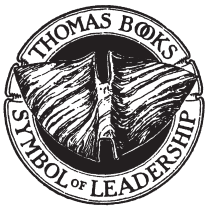
Second Edition

# PRENATAL EXPOSURE TO DRUGS/ALCOHOL

## Characteristics and Educational Implications of Fetal Alcohol Syndrome and Cocaine/Polydrug Effects

*By*

**JEANETTE M. SOBY**

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# PREFACE

This book describes the characteristics of youngsters effected by prenatal drug/alcohol exposure and explores strategies to circumvent this damage, maximizing the individual's remaining strengths. Information and suggestions are primarily for the professionals in education who can provide supportive coordination for caregivers, mental health, and medical service providers; in terms of relaying information and pinpointing techniques for learning that are the most successful for each youngster.

Medical literature on the physical, cognitive, and behavioral characteristics of this population is described for readers without a medical background. Terminology commonly used by various disciplines, outside of education, is included generally as background to the continued investigations that this text hopefully inspires. Research related to aspects of learning, particularly relevant to deficiencies seen in this population, is included to provide the background necessary for the development of individual instructional strategies that cover the needs of both severely effected and moderately effected individuals. Discoveries about the organization and development of normal memory/learning have been brought together from the disciplines of education, biology, sociology, speech, cognitive psychology, and the neurosciences. The combined strength of various disciplines help us take into consideration the child's total environment, prenatal and postnatal.

Scientific knowledge is advancing rapidly with technology adding to the velocity, new findings add to and integrate with older research or inspire new ways for us to understand. For example, early research found prenatal cocaine more damaging, based on studies that often did not rule out other drugs that can cause birth defects, teratogens, such as tobacco and alcohol.

A sampling of relevant studies are referenced as support and information detail. The combination of risk factors in the substance-abusing population have made it difficult for researchers to determine specific independent effects of prenatal exposure to a single drug, or a single event.

Advances in technology are opening windows previously unavailable, allowing researchers to see mental operations of the brain as it learns and remembers. Neuroimaging that locates which areas of the brain are damaged can more accurately influence our expectations for a youngster, while pointing to instructional strategies and adaptations. Neurophysiology, neurochemistry, and neuropsychology inform the field of behavioral teratology. Understanding the biology of learning provides us a foundation to inform current and future education. While neurologists are studying the location and functioning of the learning brain–educators are looking at what activities produce the fastest, most stable, long-term learning.

However, we must look at research as tentative knowledge that points to ways of looking at, of understanding; a beginning step toward remediation design. Yet, the definitive statements we want from research are, only directions–qualified directions.

Children damaged from prenatal exposure to drugs/alcohol have been in the classroom all along, with teachers providing them an education. This text looks to educators who have found successful instructional techniques for use with students exhibiting many of the same physical, intellectual, and behavioral characteristics as students with effects from prenatal exposure to alcohol or cocaine/polydrugs. Educational needs, successful learning environments, and instructional techniques are addressed. Yet, despite medical, educational, and social support, most children with brain damage caused by prenatal alcohol exposure retain their handicaps throughout life. Thus, the environment must be adapted to optimize the experiences of these youngsters, because brain damage has removed much of their flexibility to use cognitive strategies.

I've included theoretical positions regarding cognitive processes that relate to the practical demands of instruction and successful skill acquisition. Unavoidably, the theoretical interpretations and topics presented dealing with learning are biased by my experience. I have made an effort to keep some reasonably concise parameters on the aspects of learning and instructional strategies that fit the range of

needs presented by youngsters damaged from prenatal drug exposure.

Research and experience have provided such an extensive base of information I'm sure my indebtedness to some sources will go unacknowledged. Articles I have read and conversations that became so much a part of me, I no longer recognize an idea is not my own. I weave together medical research on maternal drug use and subsequent child health, with cognitive research focused on learning to inform remedial instructional possibilities. Areas of concern are touched on: attachment, infant-stimulation, communication, the cognitive processes involved in learning, instructional techniques, learning environments. Questions brought up during my lectures are included in this edition.

Some information is included as a reference point to where inquiry is headed. Whereas, many studies have been done on prenatal exposure to alcohol, after doctors David Smith and Kenneth Jones published in 1973, studies of other prenatal drug exposures, such as marijuana, are not as abundant. Why? Maybe funding has not been available, maybe there is limited interest in the answer, or damage is not considered significant enough to study.

Youngsters born with drug effects resulting from maternal use of alcohol have a medical diagnostic label. At a summit hosted by the National Organization of Fetal Alcohol Syndrome (NOFAS), April 2004, a consensus statement regarding diagnostic terminology was made "Fetal Alcohol Spectrum Disorders (FASD) is an umbrella term describing the range of effects that can occur in an individual whose mother drank alcohol during pregnancy. These effects may include physical, mental, behavioral, and/or learning disabilities with possible lifelong implications for problems in many areas of life: work, school, and social relations. The term FASD is not intended for use as a clinical diagnosis" (www.nofas.org). The umbrella term FASD will be used to represent Fetal Alcohol Syndrome (FAS), Partial FAS, Fetal Alcohol Effects (FAE), prenatal alcohol effects (PAE), alcohol-related birth defects (ARBD), alcohol-related neurodevelopmental disorder (ARND), and alcohol exposed static encephalopathy, throughout this book.

Diagnostic labels describing specific characteristics of impairments have not been attached to other drugs such as cocaine, heroine, marijuana, or other newer "designer drugs." The primary focus of the medical research included here is on alcohol and cocaine. Addictive mater-

nal behavior for drugs other than alcohol often involves polysubstance abuse, use of a variety of drugs. Although not a medical diagnosis, the term *Fetal Drug Effects* (FDE) will be used to describe youngsters manifesting possible drug effects from prenatal cocaine and polydrug exposure, with cocaine the primary drug used, except when references to effects from a specific drug need to be delineated.

Looking at prenatal fetal cocaine/polydrug exposure, the consistent theme emerging is a correlation with subtle decrements in measures of cognitive development; sustained attention, arousal, and regulation of responses to stress. The popular media predictions of catastrophic life outcomes and effects on offspring in the late 1980s failed to evaluate the physiologic research results in light of maternal-fetal health problems and psychosocial risks that can accompany severe addictions to alcohol, cocaine, tobacco, and other drugs. The cumulative risk of disadvantaged social and environmental circumstances, compound biological frailties. This confluence of events contribute to an infant's poorer functioning; inadequate parenting, social isolation, maltreatment, domestic violence, and poverty.

The book falls into three sections. Part one presents the characteristics of youngsters prenatally drug exposed, giving the reader an understanding of possible damage. Part two presents background on the cognitive processes involved in learning. The primary focus of this section is on normal learning processes. Understanding normal cognitive processes allows the reader to extrapolate based on how a specific youngster is functioning. Part three describes instructional strategies, for the learning and everyday life experiences youngsters with disabilities find challenging.

In addition to medical and education research, information came from my work with families, community services, the judicial system, and education services. Experiences were derived from my work in the field of special education, from my service on the Citizen Review Board for the Oregon Justice Department, and from interviews with medical foster moms, teachers, social workers, nurses, other service and care providers, together with parents. I have also used experiences from professionals in the field who attended my course on prenatal exposure to drugs and alcohol. Working with the Juvenile Justice System and the Children Service Division, I reviewed placement and services for youngsters removed from their homes. Paternal substance abuse is frequently involved when children are removed from their

homes due to neglect, physical abuse, and sexual abuse.

Interviews with Chris Amos, Joan Marguis, Robin Lindsley, Billie McKenzie, and Beth Caruso provide examples of successful instructional and management techniques. Interviewees included a social worker, a nurse, a school psychologist, and teachers employed in the Portland Public Schools, a small city community with an urban population enrollment of fifty-six thousand students.

Additional descriptions of hands-on experiences came from interviews with medical foster moms working with the Children's Services Division in Portland, Oregon. Some of the most down-to-earth heartfelt information came from interviews with medical foster moms, the moms that take medically high-risk drug effected newborns home from the hospital. Many of the moms' I talked to had histories devoted to child care, their own children, adopted children, and foster care children.

All the adults I interviewed report recognizing the need for them to consistently present a calm demeanor, and to make a conscious effort not to take the difficult behavior of these youngsters personally. Behavior that from a nonneurologically damaged youngster would mean malicious intent. Keeping a calm atmosphere was found to be a successful instruction/behavior management technique. All of the people interviewed had to continually work at accepting the youngsters' lack of social judgment.

Parents and educators need to recognize deficits primarily so that strategies can be found to circumvent these deficits. Instructional and management recommendations are made with this in mind.

J.M.S.

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# PRENATAL EXPOSURE TO DRUGS/ALCOHOL

# PART I

# Chapter 1

# INTRODUCTION: WHAT IS THE PROBLEM?

*Polydrug* use, taking more than one drug, is typical for chemically addicted mothers. Commonly abused drugs during pregnancy include: alcohol, a depressant; marijuana, that overstimulates the sensory nerves of touch, taste, sight, and hearing; amphetamine, that stimulates; heroin that produces euphoria; morphine that produces euphoria; and cocaine that produces euphoria. A problem that is significant to both the offspring and society.

This chapter discusses risk and causal factors; the need for identification and multilevel intervention. Research cautions are presented, giving the reader a broader perspective to evaluate the research this book is based on, and the additional research sparked by interest.

## INCIDENTS

The most widely used prevalence estimate of FAS, in the general United States population, is 1 to 1.5 cases per 100 live births. Also, mortality is about two thousand infant deaths in the U.S. from FAS and related disorders (Burd, Cotsonas-Hassler, Martsolf, and Kerbeshian 2003). In addition, the Centers of Disease Control (CDC) (2000), found about thirty percent of the women who knew they were pregnant, reported alcohol consumption. Youngsters who have not been diagnosed have not been counted. Maternal substance abuse crosses all social levels, however, doctors frequently do not look for subtle signs of alcohol or cocaine exposure in babies born full term that appear healthy. Thus, rates may be low. Incidences of FASD for

different ethnic groups derived from the Birth Defects Monitoring Program of the CDC show Native Americans are at the highest risk with 29.9 per 10,000 births, varying with different tribes (Chavez, Cordero, and Becerra 1989). These figures reflect American Indians may be physiologically predisposed to alcoholism because of deficits in the ability to metabolize acetaldehyde, a product of alcohol degradation. This may add to the misperception that American Indians drink more alcohol (only 42% of adult Navajo Indians drink alcohol) than other ethnic groups (Carney and Chermak 1991).

Medically needy babies require longer hospital stays with increased overall hospital costs. The additional medical costs for drug/alcohol exposed newborns, at the national level have been estimated in the billions. These cost estimates do not include the lifespan support resources needed. For example, the medical foster parents and extended families who care for these children need respite care and parent training. The financial and profound social costs of this problem demand public health involvement in prevention, drug treatment, prenatal care, and educational services.

A recurrent theme stated by medical, judicial, and educational professionals providing services to youngsters is that "kids are different now than they were ten years ago." Drug use changes people, changes society. Today's problems are different; thus, solutions must be different. No matter how dedicated teachers are, how good schools are, a great education *is not as good as a bad family.* The families' impact is paramount. During a discussion of these concerns with medical and education providers, a prekindergarten teacher's sincere remark captured the fears, compassion, and hopelessness this social problem evokes, "At the end of the school day when I have two or three children who do not want to leave, it's scary to me." What kind of home are these youngsters avoiding? The life experiences some of these youngsters are exposed to suggest that a safe home is an anomaly rather than the norm. Teachers work hard to provide safety, structure, and control during school hours; then many students go home to chaotic environments. Children prenatally exposed to drugs/alcohol may have had lots of adults coming in and out of their lives: parents, relatives, foster care, and a variety of service providers. They may not ever have had a stable adult in their life.

> At the end of the school day when I have two or three children who do not want to leave; it's scary to me (pre-kindergarten teacher).

## ADDICTION LIFESTYLE–RISK FACTORS

These children can be impacted by a group of risk factors: chaotic lifestyle, violence, abuse, neglect, being raised by brothers and sisters who are children themselves, and multiple placements with relatives or foster care. The parent-child relationships of youngsters living with parents expose them to the maternal personality disorders that flourish with drug use. Disruption and chaos describe the households of chemically addicted parents who have a commitment to chemicals, not to their children. As addiction worsens, the procurement of drugs becomes consuming; substances of abuse take precedent over all other considerations, including maternal and fetal health, nothing but the drug has any significance (Gawin 1991; MacGregor, Do, Keith, Bachicha, and Chasnoff 1989). ". . . Addictive drugs not only modify behavior but the brain itself . . ." (Restak 1988). Brain reward systems involved in the reinforcing effects of drugs of abuse promote drug use behavior, cocaine addicts report that all thoughts center on cocaine during binges. Disregard of the child's needs, neglect and abuse, follow parental addiction. These addicted, hopeless, scared mothers under the influence of mind-altering drugs, need care themselves.

Adult lifestyle is intimately tied to child development and familial relationships. How will the alcohol or cocaine addicted mother be able to take care of an addicted baby that is likely to have ongoing medical and educational needs? Can the alcoholic mother provide a safe and nurturing home for the child with FASD? When getting high is of prime importance, can a drug addicted mother, living in a chaotic drug environment care for and cope with the frustrations of an inconsolable infant with shakes and a sharp piercing cry? Will this mother be alert to the medical needs of a fragile infant? Inconsistent and intermittent nurturing may come from parents or caregivers who are emotionally needy themselves.

Youngsters may be exposed to unpredictable environments with parents coming in and out of their lives; at risk for multiple placements and multiple caregivers. Many of the mothers with chronic addictions do not live long enough to raise their children. Foster care placement, often multiple, provides the family environment for many of these fragile youngsters who require multiple educational and health care services. Prenatal drug/alcohol exposure can cause a wide range of impairments which are mitigated or exacerbated by the child's early environment. Not one of the eight children in the Los Angeles School District pilot project kindergarten class, for youngsters damaged by prenatal drug exposure, lives with his or her biological mother. Some children have been in as many as eight different homes. Most of the children are being reared by foster parents or grandparents (Trost 1989).

Maternal drug use may spread for generations resulting in a multigenerational cycle of drug addition. The risks of drug use compound rather than remain limited to just the specific days used taking drugs. A medical foster care mom wonders "were their parents alcoholic and their grandparents also? How many generations?"

## PREVENTION

Prospective mothers might not recognize that their life style, especially drug/alcohol use and abuse, can have unintended harmful consequences on the outcome of their newborn. Consequences that can impact the child's whole life, putting intellectual and social opportunities at peril. Women in their childbearing years and pregnant women might choose not to use alcohol or other drugs if they were informed about the detrimental consequences that drug/alcohol use can have on a fetus: consequences that can persist into adulthood.

The mother most likely to jeopardize her pregnancy with drug use and give birth to an infant with FASD/FDE may be a victim of FASD herself. Because of her limited abstract thinking, she cannot understand the serious effects drug/alcohol use during pregnancy can have on her child. Without multilevel intervention involving a partnership of services, prenatally drug/alcohol effected newborns could be in the next generation of parents with effected offspring (Dorris 1989; Clarren 2005). In addition to the poor judgment risk of mothers with FASD, a

genetic bias toward alcoholism may exist.

Educators found drug use prevention activities, including teaching parents-to-be about the influence of drug/alcohol abuse on the developing fetus, most effective with students nongenetically susceptible to addiction. Teachers in both middle and high school, report using a strategy based on "others" problems rather than the student's own, to be the most successful of all the prevention curriculum activities. Students' write a paper or make a list of problems people they know, who are using drugs/alcohol, are having. Based on this activity, genuine "real-life" class discussions have taken place. Remnants of old antidrug strategies, exaggerating the effects of drugs, which led to a loss of credibility, remain today.

Doctor Lemoine's early observations of children of alcoholic mothers in 1967, found that 5 alcoholic mothers, who after giving up drinking gave birth to one or more normal babies (Lemoine 2003; Koren, Nulman, Chudley, and Loocke 2003). During a workshop at a private school, the teachers discussed a mother who had given birth to eight children with FASD, she stopped drinking and gave birth to a child who did not have FASD. In agreement with this, Korkman, Kettunen, and Autti-Ramo (2003) found "If pregnant mothers are able to stop drinking even as late as in trimester II, the risk of negative cognitive sequelae is considerably reduced."

## IDENTIFICATION

Parents may not be aware their newborn has unique needs. Early identification alerts parents and service providers, so appropriate care for an infant's development and health care needs can be given. A contrasting opinion regarding drug exposure, was expressed in the January, 1992 issue of *Journal of the American Medical Association*, commentary section. Doctors were concerned that labeling youngsters prenatally exposed to cocaine, as having irremediable damage, would result in fewer services provided for these children because expectations would be so low (Mayes, Granger, Bornstein, and Zuckerman 1992). Although labeling has the potential for negative consequences, accurate expectations based on early identification are likely to have a positive affect on the child's life (Streissguth et al. 2004). Identification of FASD is complicated, youngsters are usually seen by a pediatrician,

then referred to a dysmorphologist for a thorough diagnosis. Stoler and Holmes (2000) recommend obstetricians provide medical records to pediatricians, to help alert early identification. The ability to intervene early in the child's life is based on diagnosis. Doctors' Streissguth and Kanter (1999) use the term "secondary disabilities" to describe FASD problems, that are a consequence of "primary" deficits in cognition, communication, and learning; which can impact social competence. This complex nature-nurture relationship between biology and environmental factors on development, is the reasoning for early intervention.

Early diagnosis ensures youngsters will not miss the benefits of early intervention and access to multidisciplinary child development teams. Poitra and colleagues (2003) found a community/school-based screening program had a 95 percent accuracy rate, and screening took less than 15 minutes per child. Ashley, Stachowiak, Clarren, and Clausen (2002) looked at the incidence of FASD in the foster care population using the *FAS DPN Facial Photographic Screening Tool* which was developed for assessment of photographs on a computer monitor. Screenings took approximately 10 minutes per child. They found the prevalence of FASD was 10 to 15 times greater than in the general population (Astley et al. 2002). Other maternal report screening tools, one using three to four questions about maternal alcohol use was able to identify offspring at highest risk for FASD; hence, directing the youngsters to a complete medical evaluation (Barr and Streissguth 2001).

An infant care specialist, a school nurse, and others interviewed, who are concerned with identifying youngsters with FASD/FDE, find parents to be very poor historians. Identification of youngsters, by means of self-reported maternal histories suffer from parental reluctance to admit to either drug/alcohol use or the amount and frequency of use. A nurse for a head start program has found a way around parents who understate their youngsters prenatal drug exposure. With nonjudgmental assertiveness, when taking the child's history, she asks if the child demonstrated any of the symptoms of withdrawal, specifically asking if as a newborn the child, was startled easily, had a high-pitched cry, poor suck, seizures, tight muscles, interest in the human face, and so on. She has found that parents are more likely to answer these questions honestly. She walks the parents' through examples of a variety of day-to-day routines and early milestones. As the child's

past history is reviewed, patterns may be discovered that help explain current behaviors, areas of strength, needs, and perceptual uniqueness.

**Table 1. Basic Functioning History.**

| |
|---|
| Medical problems/history |
| Bedtime, sleeping awakening |
| Temperament |
| Independent coping behaviors |
| Self-care skills |
| Chores/responsibilities |
| Skills/talents |
| Play skills/behaviors |
| Adult relationships |
| Peer relationships |
| Unusual behaviors |

## ALCOHOL METABOLISM: FETAL RISK

Alcohol metabolism takes place in the liver, where alcohol is initially oxidized to acetaldehyde, by alcohol dehydrogenase (ADH), this toxic acetaldehyde is then metabolized, by mitochondrial aldehyde dehydrogenase (ALDH2), to acetate. Genes involved in alcohol metabolism: genes that effect functional ADH and ALDH activities may influence the risk of alcoholism. Genes that effect alcohol drinking behavior: the quantity and frequency of alcohol consumption.

Maternal consumption of the teratogen alcohol during pregnancy is the cause of FASD, the most common nongenetic cause of mental retardation. Yet, not all women who use alcohol during pregnancy have offspring with FASD. Genetic differences may be predictors. Researchers are looking at alleles (variations of a gene) of the alcohol metabolizing enzyme gene ADH2, as predictors of FASD. Studies of different alleles of the ADH2 gene show differing results (Chambers and Jones 2002). The ADH2*3 allele, found primarily in Africans, is associated with rapid metabolism of alcohol, possibly enzymes more efficiently metabolize alcohol at high blood alcohol concentrations (Eriksson et al. 2001). The ADH2*2 allele was found to be more common in mothers who did not have offspring with FASD, suggesting the ADH2*2 may contribute protection against FASD (Viljoen et al. 2001).

The implication is that there is a genetic potential of a person to metabolize alcohol which may reduce or promote excessive alcohol use; thus, increasing the risk of FASD for offspring or of having a protective effect (Jones 2003). Continued research may help us predict, which maternal genotype, which women who drink alcohol, are at the highest risk of having an effected child.

Francis Collins and Craig Venter, two principle genome sequencers for the Human Genome Project; published in 2001, a sequence of the human genome. Personality trait variations related to alcoholism, are found to be from combinations of many genes rather than one single gene. For example, a high interest in novelty-seeking impulsive behaviors, along with a low interest in harm avoidance uninhibited behaviors (Cloninger, Sigvardsson, and Bohman 1996). Scientists are in the early stages of understanding the genetic contribution to susceptibility for alcoholism, looking at how and when different genes work together.

## PATERNAL CONTRIBUTION

FASD is caused by maternal drinking, not fathers. Researchers have found conflicting results concerning lower birth weight on the offspring of fathers who consumed alcohol before conception. In general, the newborns' risk of behavior and cognitive deficits increase as the birth weight decreases. Low birth weight is commonly accepted as $5^1/_2$ pounds; very low birth weight as $2^1/_4$ pounds. Little and Sing (1986) found that infants of fathers who drank two drinks a day had offspring that weighed about a half a pound less than offspring of fathers who did not. Conflicting findings were produced by other researchers who did not find that paternal drinking before conception and during their partners pregnancy resulted in lower birth weight for offspring (Savitz, Zhang, Schwingl, and John 1992; Passaro, Little, Savitz, and Noss 1998). Damaged sperm may cause spontaneous abortion, Mother Nature's first defense, often before the mother is aware of her pregnancy. Future research may find a paternal genetic contribution; possibly influencing if or how much the fetus is protected or vulnerable to maternal alcohol consumption.

## RESEARCH EVALUATION CAUTION

Particular disciplines investigate different aspects of a subject: a social scientist sees the world through different eyes than a neurologist. Cautions are many, the title and the discussion section of a published study may suggest a more encompassing attribute than the results support or that the methods used measure. The researcher may have a personal or discipline-related emotional investment that can lead to an overemphasis of the importance of their findings. Hence, Montori and colleagues (2004) recommend reading the methods and results sections only. They also caution graphs can be misleading, for example, comparing different time frames to harms and benefits. Studies flawed by uncontrolled or unexamined potential variables: differences in sample characteristics and size, faulty comparisons, small treatment effects, examination of moderating characteristics, relevancy of subgroup analyses, as well as the degree of statistical control, can lead to differing conclusions. Different agendas = different conclusions. These methodologic limitations fuel the fire of research disagreements.

Subjects recruited from a population referred to clinics are often used in the studies cited in this book. Clinic subjects are likely to be in a low socioeconomic risk factor group and also may be associated with high developmental risk without drug/alcohol exposure. Studies may exclude offspring of mothers who are very seriously addicted and mothers who's doctors haven't questioned them regarding drug use. People with congenital malformations or other more disabling conditions, or individuals with milder symptoms who can cover or compensate, may not be represented in the studies used. Control groups may come from families with multiple life stressors, such as brief homelessness and changes in the primary caregiver. Subjects for each study possesses a unique constellation of variables, and the degree of statistical control varies widely from study to study.

Many investigators have turned to animal models; both to reduce the conflicting findings of and to inform human studies, and to determine exactly what are the causal factors. Is there a connection between prenatal drug exposure and the birth defects seen in humans? Animal studies provide models of outcome based on control of exposure and timing. Patterns of dosage levels can be isolated to the prenatal period and isolated to a specific drug exposure. Drug administration protocols offer control of dosage level, other drugs used, and

nutrition. No measure of drug/alcohol exposure is completely accurate, extrapolation from animal study data to effects on children has limitations, yet provides direction.

Mayes (2003) offers key principles to evaluate research questions on the relationship between a prenatal toxin exposure and later developmental impairment (1) what are the possible mechanisms of effect, (2) what is the specific teratogenic agent or event, (3) the timing of exposure, (4) what are the possible dose-response relations, (5) what are the most likely outcomes related to the mechanisms of action of the exposure, (6) when are outcomes most likely to be apparent, and (7) what conditions ameliorate or exacerbate exposure-related functional outcomes. How does the teratogenic exposure, disruption of process x affect other maturational processes?

Studies may not be designed in ways that fully reveal children's capabilities. Tasks used to measure a skill may not isolate that skill. For example, results of a test using pictures rather than words may provide a result too narrow in scope, a result that statistical controls do not adjust for. Although I advise the reader of research to be skeptical, I, too, may have accepted as factual only possibilities.

# Chapter 2

# PRENATAL DRUG/ALCOHOL EXPOSURE

The primary drugs of abuse with the possibility of a teratogenic effect producing anomalies in offspring are alcohol and cocaine. A description of the intellectual, physical, and behavioral characteristics of youngsters with a diagnosis of FASD and youngsters prenatally exposed to other drugs, based on medical research, is presented in this chapter. There is no typical profile, the continuum of impairments range from mild to severe.

## THE FETAL ENVIRONMENT

Any drug that crosses the blood-brain barrier and has an effect on the central nervous system of the mother also crosses the placenta, affecting both maternal and fetal circulation. The placenta separates the circulatory systems of the fetus and the mother, transferring substances from the mother to the fetus. For example, as a result of the placenta's metabolic activity, which is separate from the mother's, cocaine is changed into a less active metabolite; hence, providing a moderate degree of protection for the fetus (Beaconsfield, Birdwood, and Beaconsfield 1980; Roe, Little, Bawdon, and Gilstrap 1990).

Many drugs involved in maternal substance abuse are *teratogens*, drugs that in certain dosages can cause birth defects. The word teratogen is derived from the Greek word "Terato" which means literally, "to make monsters." Teratogens have a long lag period; it may take ten years after birth for all the effects of the drug to show up. For example, the academic demands and social expectations for an adolescent

may expose deficits not previously identified.

Variable fetal effects can be partially accounted for by individual differences in drug metabolism, and that drugs are slowly metabolized by the immature fetus. The original drug and the metabolites of that drug remain in the amniotic fluid much longer than in the mother. "Moreover, generally speaking the fetus is exposed to the same drugs, food additives and environmental pollutants that the pregnant mother is exposed to" (Beaconsfield et al. 1980). Cocaine metabolites were found in newborns' urine 96 hours after birth, when maternal use was one to two days before delivery (Van de Bor, Walther, and Ebrahimi 1990). Cocaine may be present in the mother from 24 to 48 hours after use, while metabolites remain in the neonate from four to six days (Burkett, Yasin, and Palow 1990; Johanson and Fischman 1989; Peters and Theorell 1991). Both mother and fetus may have increased and prolonged exposure and toxicity to cocaine due to altered metabolism during pregnancy (Gingras, Weese-Mayer, Hume, and O'Donnell 1992).

The main intoxicant in alcoholic beverages is ethyl alcohol, a relatively simple organic chemical made up of carbon, oxygen, and hydrogen, that is soluble in both water and fat. The fetus lacks an enzyme, known as *alcohol dehydrogenase*, which is responsible for metabolizing alcohol. Because the fetal liver and kidneys are immature, drugs are slow to be broken down and excreted. Thus, the level of alcohol can build up in the fetus, particularly in the brain.

Is there a safe amount of alcohol for a pregnant woman to consume? No. But not all women who drink alcohol during their pregnancy have offspring with FASD, the individual health of the mother and the gestational stage of alcohol consumption play an important part. Although researchers do not agree on a safe amount, 1.5 drinks per day have been found to be associated with a high incidence of FAS (Autti-Ramo 2002). Olson and colleagues (1997) found subtle alcohol related neuropsychological deficits in offspring, exposed to even maternal 'social drinking' levels, that were consistent with behavioral dysfunction seen in youngsters prenatally exposed to higher doses of alcohol: deficits in sustained attention, response inhibition, spatial memory, and variability in task performance. Willford, Richardson, Leech, and Day (2004) also found memory processing deficits in both recall and recognition memory for verbal memory of word-pairs, in offspring of mothers who engaged in moderate drinking. In addition,