Drug-Exposed Infants: Understanding the Medical Risk

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Abstract

The common, known effects of prenatal drug exposure include intrauterine growth retardation (IUGR) and neurobehavioral dysfunction. A review of the singular effects of cigarettes, alcohol, heroin, narcotics, marijuana, and cocaine are presented, based on findings of previous studies. This paper also summarizes the important research issues, biologic variability determining outcome, and pharmacologic actions and short- and long-term effects of drugs commonly used by pregnant women. The role of the social environment in determining long-term outcome is emphasized.

The thalidomide tragedy, which dramatically brought attention to the possibility that prenatal drug exposure can cause birth defects, gave impetus to the development of the medical field of teratology. Initially, the term teratogenicity, the ability of a drug consumed in pregnancy to cause adverse fetal outcomes, was associated with major birth defects. However, intrauterine growth retardation (IUGR), or poor fetal growth, and neurobehavioral dysfunction are now considered more common effects of prenatal drug exposure. The prenatal effects of cigarettes, alcohol, heroin, and narcotics have been studied extensively. Less information is available regarding effects of marijuana and cocaine, though the increasing prevalence of cocaine use among pregnant women has brought new attention to the effects of drug use during pregnancy.

Unanswered Questions: Research Issues

A rapidly growing body of research literature strongly suggests that prenatal substance exposure is linked to health problems in the newborn as well as to problems in the child’s development. However, much of this literature presents conflicting results regarding the type and magnitude of the impact. An understanding of the problems involved in investigations of prenatal substance exposure helps explain these inconsistent findings and the limitations of our knowledge.

Efforts to isolate the effects of a single drug such as cocaine or marijuana are complicated because it is unusual for humans to use or abuse only one substance.
Mothers who use cocaine while pregnant are statistically more likely to have infants with low birth weight, small head size, and other adverse outcomes, but since mothers who use cocaine while pregnant are also more likely to smoke cigarettes and marijuana, consume alcohol, and have poor nutrition, it is difficult to ascertain how much of the effect is due to cocaine and how much to the other factors, which are also associated with low birth weight, prematurity, and small head circumference. One commonly recommended approach to these confounding variables is the use of multivariate data analyses, statistical techniques that identify the effect of one factor independent of other, interrelated risk factors. However, these analyses are costly and require study of a large number of women.

Another difficult problem of research into prenatal exposure to various chemical substances is the need to rely on subjects’ reports of their own behavior in this emotionally charged area. Naturally, women may underreport their use of illegal or socially stigmatized substances (e.g., crack cocaine, marijuana, and alcohol). Underreporting may, for example, have played a role in some of the inconsistent findings regarding the association between prenatal marijuana exposure and adverse newborn outcomes. The only study to date that actually tested women’s urine for marijuana by-products during pregnancy found that marijuana use was indeed associated with a lower birth weight. The investigators, who wanted to evaluate the role of underreporting, repeated their analysis, ignoring the findings in urine, and categorized marijuana users as only those women who reported their use. The results of this analysis showed no association with marijuana use and lower birth weight. Thus, underreporting, which may vary with the population studied, can lead to conflicting findings regarding the teratogenicity of a drug. Biologic markers such as blood or urine tests for substance use should be used in investigations of the neonatal effects of prenatal substance exposure.

Biologic Variability

Biologic variables, not just the substance taken, significantly influence the outcome for the infant after prenatal substance exposure. A substance’s effect on the fetus depends on the stage of fetal development during which exposure occurs and on the amount of the substance consumed as well as the pharmacologic effects of the particular substance. During the first few weeks of pregnancy, substances that severely affect embryonic development may cause spontaneous abortion. Later, during the first trimester, the organ-forming period, ingestion of substances by the pregnant woman may result in congenital malformations. Each organ has different developmental phases, however, so the type of malformation will be related to the phase of development at the time of exposure. All organs continue to develop after

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Due to genetic or other biologic factors, some newborns may be more vulnerable than others to any given prenatal insult. Thus, one nonidentical twin of an alcoholic mother can be more affected than the other. The probability that the child of an alcoholic woman will be born with fetal alcohol syndrome (FAS) ranges
Principal Risk Factors for Low Birth Weight

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<th>Demographic Risks</th>
<th>Medical Risks in Current Pregnancy</th>
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<td>Age (less than 17; over 34)</td>
<td>Multiple pregnancy</td>
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<td>Race (Black)</td>
<td>Poor weight gain</td>
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<td>Short interpregnancy interval</td>
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<td>Unmarried</td>
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<td><strong>Health Care Risks</strong></td>
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This table lists those factors that when present in an individual woman indicate an increased chance of having a low birth weight infant. These risks have been found to be clearly and consistently linked to low birth weight. One set of risks can be detected before pregnancy; the risks listed under Health Care Risks are not related to characteristics of women themselves. Some of the risks, such as those listed under Demographic Risks, are often interrelated, making it difficult to determine the precise association between any one factor and low birth weight.

An infant described as “low birth weight” weighs less than 2500 grams at birth; a “very low birth weight” infant weighs less than 1500 grams. Low birth weight is a major factor in infant mortality in the U.S. Infants weighing 2500 grams or less are almost 40 times more likely to die during their first 4 weeks of life than the normal birth weight infant. Low birth weight infants are 5 times more likely than normal birth weight infants to die later in the first year and account for 20% of postneonatal deaths. The two major contributors to low birth weight are preterm birth and intrauterine growth retardation (IUGR). Both contribute to inadequate fetal growth. A birth is considered preterm if it has a duration of less than 37 weeks from the last menstrual period. IUGR refers to low weight for a given duration of gestation.

Drug-Exposed Infants: Understanding the Medical Risk

from 2% to 8% in most studies, but the biologic factors that protect some children and make other children vulnerable are unknown. Some fetuses may be less vulnerable than others to the detrimental effects of cocaine because of the variability in the enzyme that metabolizes the drug. This enzyme decreases in many women during pregnancy, but in some it stays the same, and in others it actually increases. Women with high levels of this enzyme will metabolize cocaine quickly, thereby decreasing duration of fetal exposure. Other genetically determined factors, such as differences in the mother's absorption, binding, or excretion of particular drugs, may contribute to variability in the drug's effect on the fetus.

Many of the problems in human studies designed to predict the effect of a substance on a fetus or newborn infant can be overcome by animal studies in which variables, such as dosage, other risk factors, and even the genetic make-up of the subject can be controlled. However, animal studies have important limitations. Almost any substance given in a large amount at a specific time during pregnancy can cause a birth defect, but this type of exposure may not have a counterpart in actual human substance use. In addition, substances sometimes appear to be safe in animals but are not safe in humans. Thalidomide, for example, showed only rare teratogenicity in many different types of animals. On the other hand, animal studies can be very useful in elucidating the biologic mechanisms by which prenatal substance exposure adversely affects the fetus.

With these limitations, how does one draw any firm conclusion regarding the effect of a substance used during pregnancy? There are several criteria that help. The first is consistency. A finding is more likely to be valid if similar findings are produced consistently in different studies. Such consistency has been present in studies regarding the prenatal effects of exposure to cigarette smoking but has not been present, or has been present to a far smaller degree, in regard to the other substances of abuse. The second is biological plausibility. That is, an apparent adverse effect on the fetus should be consistent with the known biologic action of the substance. The third is a dose-response relationship, in which heavier exposure to the substance is associated with worse outcomes.

**Effects of Selected Substances**

Many drugs cross the placenta to some degree. Psychoactive drugs, such as opiates, cocaine, marijuana, and alcohol are generally fat soluble and of small molecular size, so that they also easily cross the blood-brain barrier. These same characteristics allow them to readily cross the placenta, and, therefore, potentially have a direct effect on the fetus. By influencing the mother's physical or emotional function, these substances may also affect the fetus indirectly. The following describes the pharmacology of selected drugs and summarizes current knowledge about their impact. Potential adverse outcomes are numerous; I will emphasize what is known regarding each substance's potential likelihood of causing significant birth defects, poor fetal growth, and neurobehavioral disturbance.

**Cocaine**

Cocaine, the white crystalline powder made from coca leaves, is classified as a stimulant drug. Its principal effects are due to its ability to increase the effect of the body's own neurotransmitters, the chemical messengers that carry impulses from one nerve to the next. Nerves normally reabsorb a portion of the neurotransmitters they have released, but cocaine blocks the ability of these chemicals to be reabsorbed, resulting in excess neurotransmitters between nerves and an exaggerated signal.

The neurotransmitters produced in the body—norepinephrine, epinephrine, and dopamine—have different roles. Epinephrine (sometimes called “adrenalin”) and norepinephrine are produced when one is startled and must respond quickly. These neurotransmitters cause increased heart rate; constriction of many blood vessels, resulting in higher blood pressure; sweating; and raised temperature. Dopamine, on the other hand, is thought to produce feelings of pleasure and euphoria; this action is augmented by certain drugs, creating the “high” experienced by
the cocaine user. One theory proposes that dopamine is depleted with chronic cocaine use, resulting in the depression that is the major symptom of “withdrawal” from cocaine.

Cocaine can be taken in many forms. When absorbed through the mucus membranes of the nose, it reaches peak levels in the bloodstream within 15-20 minutes, with euphoria lasting approximately 60-90 minutes. In the smokable form, called “crack” or “freebase,” cocaine absorbed through the lungs reaches peak levels within 2 minutes, with a duration of euphoria lasting only 20 minutes. The short duration of action and highly rewarding nature of cocaine is thought to contribute to its repetitive use.

Cocaine use can be categorized in many stages, from experimentation to addiction. In one study of cocaine use during pregnancy, one third of the women used less than once a month, while almost 50% used at least once per week. Addiction is characterized by compulsive use, causing dysfunction. The addicted mother spends much time searching for the drug and staying up all night using, so that her ability to hold down a job or care for children or herself is impaired. Whereas addiction to alcohol, heroin, or cocaine in powder form frequently takes many years to develop, crack addiction can occur in months.

Cocaine is metabolized (converted to inactive forms) by the enzyme cholinesterase, which is decreased in many, but not all, pregnant women and in newborns. The inactive metabolites are excreted into the gastrointestinal tract and eliminated by the kidneys. As a result of the latter, evidence of the drug’s presence in the body may be detected in the urine for 2-3 days after use by nonpregnant women and up to 10 days in newborns exposed prior to delivery.

Since prenatal cocaine exposure is a relatively recent problem, many adverse effects attributed to it are not based on systematically conducted, scientific investigations but rather on anecdotal clinical experience with individual patients and on preliminary studies with a small number of subjects. At the present time, good scientific evidence supports an association between prenatal cocaine exposure and impaired fetal growth and smaller head size at birth. However, findings from studies regarding the relationship between prenatal cocaine exposure and birth defects, preterm birth, and neurobehavioral dysfunction are inconsistent, preventing firm conclusions from being drawn. At this time, prenatal cocaine exposure does not appear to be associated with an appreciable risk of sudden infant death syndrome (SIDS).

Infrequently occurring birth defects of the bowel, kidney, heart, and skeletal systems have been described in infants exposed prenatally to cocaine. Because these defects are rare, it has not been possible to establish a causal connection to cocaine by epidemiologic studies. However, the association between these defects and cocaine exposure is plausible since these birth defects might reasonably be expected to result from constriction of fetal blood vessels, which is one of the known effects of cocaine.

Typically, prenatally-exposed newborns are full term (38-42 weeks gestation) but have a lower-than-expected birth weight. In general, the inability of infants to reach their potential size (intrauterine growth retardation) is usually due to decreased nutrient transfer through the placenta because of decreased blood flow and/or a direct effect of a drug or other agent (e.g., infection) on cellular growth. Cocaine, due to its ability to constrict blood vessels, decreases blood flow to the fetus. It is likely that this effect of cocaine is at least partially responsible for the impaired fetal growth experienced by cocaine-exposed newborns.

Not all impaired growth among cocaine-exposed newborns is due solely to cocaine. Confounding variables, such as other substances or risk factors associated with prenatal cocaine exposure, may contribute to impaired fetal growth. An exam-
ple of the role of confounding variables was demonstrated in a study showing a 407-gram deficit (almost 1 pound) in birth weight of infants (2847 g) of women who had a positive urine test for cocaine during pregnancy versus infants (3254 g) of women who did not use cocaine. When the combined effects of cigarettes and marijuana, other substances, poor nutrition, and other potential risks were considered by multivariate analysis, cocaine's contribution to lowering the birth weight was 93 g. Thus, we can conclude that it is the cumulative effect of multiple substances and other risk factors that has the most significant adverse effect on the newborn.

Prenatal cocaine exposure can affect brain development indirectly and directly. The indirect mechanism is by decreasing blood flow from the mother to the fetus, which not only impairs birth weight and length but may also contribute to a smaller head size at birth. A smaller head size, or microcephaly, in newborns is thought to reflect a smaller brain. Infants with impaired fetal growth and microcephaly are more likely to experience developmental and learning problems as they get older, compared to normally grown infants with normal head size.

The direct effect of prenatal exposure to cocaine on the structure or functioning of the brain is unknown. However, the neurotransmitters affected by cocaine play an important role in many aspects of the neurophysiologic development of the brain. Thus, changes in these neurotransmitters due to prenatal cocaine exposure may adversely affect brain development and subsequent behavior and child development.

Prenatal cocaine exposure may affect the structure of the brain by constricting blood vessels supplying the brain, resulting in hemorrhages and cysts. Using a technology called “echoencephalography,” one study has shown that small hemorrhages and cysts form in the brains of cocaine-exposed newborns. (Such hemorrhaging is found to be relatively common in very low birth weight or preterm infants who were not prenatally exposed to drugs.) While cysts in the brains of preterm infants may cause problems in learning and behavior, small hemorrhages usually do not. The type, location, and distribution of these cysts are such that they may not show clinical symptoms during infancy. Studies need to be conducted to determine whether they will cause future problems in learning or behavior.

An indirect method of measuring the newborn central nervous system is to assess neurobehavioral functioning. The most commonly used assessment is the Brazelton Neonatal Behavioral Assessment Scale. This scale includes the assessment of reflexes and behaviors, such as the newborn’s ability to become and remain alert, to follow a face or bright toy with their eyes, to turn towards voices or sounds, to be consolable, and to regulate movements of their arms and legs. Neurobehavioral dysfunction includes lethargy, irritability, poor responsiveness to human faces or voices, inability to follow with their eyes, unconsolability, and abnormal reflexes or muscle tone.

The neurobehavioral functioning of prenatally cocaine-exposed newborns has been inadequately studied; findings to date, tending to indicate an association between prenatal cocaine exposure and neurobehavioral problems in the newborn, are not consistent. Some infants exposed in utero do not have detectable clinical manifestations. A withdrawal syndrome has not been identified and, therefore, at this time it is inaccurate to describe a cocaine-exposed newborn as crack-addicted. Based on the limited studies and on clinical experience, some, but not all, cocaine-exposed newborns tend to be poorly responsive and sleepy. When alert, some are easily overstimulated and therefore become irritable or quickly return to sleep. The cause of some of these cocaine-exposed newborn’s vacillation between sleepiness and overstimulation is unknown; it may be due to withdrawal, to a direct effect of cocaine, or to changes in the brain’s neurotransmitters. Its effects on the child’s later development are also unknown. This behavior probably makes mother-infant interaction difficult and unrewarding, with adverse consequences for parenting and development. (For further discussion, see the article by Kronstadt in this issue.)

Neurobehavioral functioning of infants of mothers who use cocaine while pregnant will most likely vary because other factors, such as other substances and undernutrition associated with cocaine use, also contribute to newborn behavioral
functioning. Transient changes in electroencephalogram (EEG) and visual and auditory evoked potentials (tests that measure electrical activity in the central nervous system) have been shown among cocaine-exposed newborns.\textsuperscript{12,13,14} These findings are not associated with any known clinical problem, and the tests return to normal within a few months. More information from methodologically-sound research studies are needed to determine the neurobehavioral effects of prenatal cocaine exposure and whether these effects reflect a withdrawal syndrome.

Preliminary reports and clinical experience concerning the later development of these infants indicate that outcomes in the first 2 years of life range from rare, significant developmental problems, to subtle behavioral or learning problems, to normal children. How much of subsequent problems result from caretaker dysfunction (including multiple caretakers) and/or prenatal cocaine exposure is unknown. Longer-term implications for the child’s ability to learn in school and function socially are unknown. (See also the discussion by Kronstadt in this issue.)

**Opiates**

Opium is derived from a milky substance produced by the Eurasian poppy. Morphine is one of 20 components of opium and is the basis of heroin as well as of Percodan and Dilaudid; the latter two are both prescribed therapeutically and abused. Methadone is chemically different from opiates but pharmacologically similar. It is used to treat opiate addiction because it supplies the physical need for the drug without providing the psychological “high” of opiates. Opiate drugs are considered depressants, and their main effects are analgesia and drowsiness. This is in contrast to cocaine and its variants, which are stimulants.

Infants born to mothers who use opiates do not have birth defects due to the effects of the opiates, but do have impaired growth, smaller head size, and significant neurobehavioral dysfunction due to withdrawal. Women who use opiates usually use other illegal and legal drugs, are poorly nourished, and frequently have other health problems, all of which contribute to adverse newborn outcomes, especially poor growth and small head size.\textsuperscript{15}

Addicted women use opiates daily because of the severe physical symptoms, including sweating, headaches, abdominal pain, diarrhea, and general discomfort and agitation, they experience if they discontinue use. Similarly, the baby who has become passively addicted or dependent on the drug in utero may suffer a withdrawal syndrome at birth consisting of significant irritability, jitteriness, sweating, stuffy nose, diarrhea, vomiting, and a high-pitched cry. These infants cannot get comfortable and frequently have abrasions on their elbows and knees because of their continual movement. Withdrawal symptoms can start at birth or as late as 7-10 days after birth and usually last from 2-8 weeks. A subacute withdrawal syndrome associated with either heroin or methadone and involving restlessness, poor sleep patterns, and vomiting may continue up to 6 months but does not require drug treatment. Again, some infants exposed in utero do not develop clinical signs and symptoms.

A neonatal abstinence scoring system based on a number of chemical manifestations was developed by Finnegan to identify withdrawal cases and to monitor the progression and effectiveness of the pharmacologic treatment for withdrawal when it is needed.\textsuperscript{16} The most commonly used drugs for treatment of infant withdrawal are paregoric (tincture of opium) and phenobarbital. Some infants have a milder withdrawal and do not need drug therapy. They are hypersensitive to stimulation, however, and are helped by an environment in which stimulation is reduced, lights and noise are kept low, touching and handling are minimized, and the baby is swaddled (wrapped tightly in a blanket).

The outcome of infants born to opiate-addicted mothers includes an increased risk of SIDS. The independent role of prenatal opiate exposure in subsequent growth and developmental problems is difficult to isolate from other factors. In one study, the home environment of children was a more important determinant of the child’s developmental functioning than the amount of prenatal drug exposure.\textsuperscript{17} It is unlikely that narcotics or any one drug solely determines outcome. (For further discussion, see the article by Kronstadt in this issue.)

**Marijuana**

Marijuana is found in the flowering tops of the plant Cannabis sativa. More than half of its psychoactive ingredient, 1,9-tetrahydrocannabinol (9-THC), is absorbed into the blood when smoked. Since 9-THC tends to accumulate in fatty tissues
throughout the body, it takes many days to be excreted in the urine. Commonly, chemicals from smoking a single marijuana cigarette can be identified in the urine for up to 7 days after use.

Marijuana has a small, transient effect on several female reproductive hormones, but these have not yet been shown to have any direct effect on the fetus. Marijuana, like cigarettes, increases carbon monoxide levels in the mother's blood. Marijuana also has a direct effect on the mother's lungs, reducing the amount of oxygen passing into her bloodstream. Both of these mechanisms indirectly decrease oxygen to the fetus.

Since marijuana crosses the placenta, it is reasonable to think that it may have a direct, adverse effect on the fetal brain or on fetal growth, but the small number of studies performed to date have failed to identify major birth defects or a consistent effect on neurobehavioral function. Results regarding the relationship of prenatal marijuana exposure to lowered birth weight are inconsistent. However, the study referred to earlier (under Cocaine), in which the investigators determined that they would not have identified a significant association between marijuana use and smaller size if they had not used a urine assay but relied only on questionnaires, suggests that previous studies, which did not use urine assays, may have missed an association. Thus, there may be a small lowering of birth weight due to marijuana use during pregnancy, which is consistent with marijuana's ability to decrease oxygen to the fetus. This effect is especially important when it is additive, further impairing fetal growth reduced by other drugs and poor nutrition.

No consistent or meaningful newborn neurobehavioral symptoms have been identified with prenatal marijuana exposure. Only one study has assessed long-term outcome of children whose mothers used marijuana. This study shows that at 4 years of age, children exposed to marijuana in utero had somewhat lower developmental scores. This finding had not been seen at earlier ages and needs to be replicated before it can be accepted as proof of an association. (For further discussion, see the article by Kronstadt in this issue.)

Alcohol

Alcohol, like other psychoactive drugs, readily crosses the placenta and rapidly reaches the same concentration in the fetus's blood as in the mother's. The mechanism of its effects on the fetus is unknown. Reports of possible adverse effects of alcohol consumed during pregnancy date back to biblical times. Modern concern about serious adverse effects emerged in the 1970s following several hundred case reports showing that infants of mothers who were chronically alcoholic (typically consuming approximately 14 drinks per day each day during pregnancy) had poor fetal growth, altered facial features, and developmental problems; the phenomenon was named "fetal alcohol syndrome" (FAS). The facial features, though highly publicized, are often not that distinct and cannot be recognized consistently even by experts. Other congenital abnormalities, especially cardiac defects, have also been associated with FAS.

Spurred by the interest in FAS, investigators started to evaluate the possible impact of moderate drinking during pregnancy. Some studies showed that as little as 1 or 2 drinks per day may lower a child's birth weight by approximately 80-120 g, but these studies did not always consider important interrelated factors, such as maternal nutrition, health, and marijuana use. Many studies that did consider these factors did not show any correlation between 2 or fewer drinks per day and lowered birth weight.

No consistent or clinically meaningful newborn neurobehavioral symptoms have been identified with prenatal alcohol exposure. While it is impossible to state that any amount of drinking is absolutely safe, a review of all studies does not appear to indicate a consistently documented risk to infants of well-nourished women who drink 1 or 2 alcoholic drinks per day while pregnant. The role of binge drinking is unknown; theoretically, an average of 14 drinks per week is more harmful if consumed in a short period of time than if taken as one drink at lunch and/or with dinner every day. (Additional discussion can be found in the article by Kronstadt in this issue.)

Cigarettes

Cigarette smoke contains more than 2000 active substances. Nicotine is the most studied, since it is considered to be the
compound primarily responsible for the pharmacologic effects of smoking. Cigarette smoking has not been shown to be associated with birth defects. Although nicotine crosses the placenta, the two primary mechanisms considered responsible for adverse fetal effects are indirect. By constricting the mother’s blood vessels, nicotine reduces the oxygen reaching the fetus. The carbon monoxide in cigarette smoke also diminishes the oxygen obtained by the fetus.

Studies show that cigarette smoking lowers birth weight by an average of 200 g compared with the birth weight of nonsmokers’ babies. Studies show that cigarette smoking lowers birth weight by an average of 200 g compared with the birth weight of nonsmokers’ babies. There also seems to be a dose-response relationship: the more cigarettes smoked, the lower the birth weight. No consistent or meaningful newborn neurobehavioral symptoms have been identified with prenatal exposure to cigarette smoking. Cigarette smoking has also been well documented as causing an increase in the likelihood of spontaneous abortion and SIDS. Long-term evaluation of the development of children who are exposed to cigarette smoking shows that they do less well in a variety of measures of cognitive, language, and academic achievement, including reading and mathematics. (See Kronstadt’s article in this issue for further discussion.)

Long-term Outcome

How does maternal use of psychoactive substances during pregnancy affect the exposed fetus, especially its brain, and what are the long-term implications of the effects? All of the substances described above cross the placenta and might have a direct effect on the developing brain and nervous system. It is very difficult to study the human brain in order to determine the direct effects of these substances. However, new technologies may make this possible in the future.

At least three of these substances have an indirect effect on the developing fetus. Cigarettes, marijuana, and cocaine decrease oxygen to the fetus, and, since most mothers who use cocaine usually smoke cigarettes and marijuana, the effect is cumulative. It is unknown how much oxygen deprivation impairs the developing brain, and over what period of time, but hypoxia, compounded by other indirect or direct adverse effects of substances, certainly creates a potential vulnerability.

Early theories of child development implied that poor developmental outcome and difficulty in learning were the result of a single factor, such as hypoxia, affecting the brain. This approach, called the “main effect model of development,” implies a linear cause-and-effect relationship between risk and outcome. However, better studies have shown the mere presence, or even the degree, of a biologic insult are poor predictors of developmental outcome. Rather, children’s development can best be understood by the dynamic interplay between the environment and the child, so that the child is shaped by the environment and the environment is actively modified by the child.

Studies during the past 20 years highlight the importance of a good social environment to optimize development in the face of risk factors and/or biologic vulnerabilities. For example, in a study of 7-year-olds who were preterm infants, IQ scores were lower among those preterm infants who at 1 month of age were neurologically immature. However, among the neurologically immature preterm infants, responsive caretaking resulted in IQs similar to infants who were not neurologically immature. Thus, responsive caretaking appeared to be a protective factor for those preterm children with biologic vulnerability.

Another study showed that children with equivalent levels of perinatal stress had good outcomes if their families had a high level of stability; it was only the combination of high perinatal stress and low family stability that impaired children’s developmental functioning. The importance of the environment in modifying the effects of prenatal substance exposure has been demonstrated as well. One study of babies exposed to opiates showed that the quality of the home environment, and not the amount of substances taken by the mother while pregnant, was a more important determinant of outcome.

Thus, biologic vulnerability created by exposure to substances in utero can be
highly modified or exacerbated by social factors. Unfortunately, drug- and alcohol-abusing mothers are likely to be dysfunctional caretakers. Thus, their infants may experience a double jeopardy: they suffer biologic vulnerability due to prenatal substance exposure, which may be exacerbated by caretaking dysfunction such as neglect or abuse. Intervention can help children by eliminating or decreasing prenatal substance exposure and/or improving the caretaking environment.


