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Childhood Medical and Behavioral Consequences of Maternal Cocaine Use¹

Lynn Singer²,

Case Western Reserve University School of Medicine

Kathleen Farkas, and

Case Western Reserve University

Robert Kliegman

Case Western Reserve University School of Medicine

Abstract

Reviewed available studies of the impact of fetal cocaine exposure on child medical and developmental outcome, as well as the current status of clinical psychological interventions and research strategies. Current studies are inconclusive but suggest that prenatal exposure to crack-cocaine can have significant effects on the growth and neurological development of the infant, with the potential of later learning and behavioral disabilities. Social-environmental correlates of maternal cocaine use are confounding factors with known negative effects on child outcome. Large, population-based studies using multivariate analyses are needed to determine the independent effects of cocaine on child outcome relative to other confounding variables.

Keywords

cocaine; child development; pediatric psychology

Psychotropic drug use during pregnancy may have adverse effects on the mother, her pregnancy, the developing fetus, newborn, and growing child. Some drugs, such as heroin, adversely affect reproductive outcome and fetal growth, while others, such as alcohol, are fetal teratogens, producing congenital anomalies of the heart and central nervous system with subsequent mental retardation (Finnegan, Chappel, & Kreek, 1982). The greatest increases in drug use popularized in the late 1970s and accelerating into the 1990s have been among young adults of child-bearing age using cocaine (Dixon, 1989). As a result, new casualties of the recent “crack” cocaine epidemic have been primarily pregnant women and their children. Maternal use of cocaine during pregnancy has risen to affect 10–15% of the populations of urban neonatal nurseries and intensive care units in major cities in the United

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²All correspondence should be addressed to Lynn Singer, Rainbow Babies and Childrens Hospital, 2101 Adelbert Road, Room 373, Cleveland, Ohio 44106.

States (Chasnoff, Landress, & Barrett, 1990; Dixon, 1989; Gillogley, Evans, Hansen, Samuels, & Batra, 1990).

The number of infants born exposed in utero to cocaine has increased over the last 5 years. The National Hospital Discharge Survey identified 13,765 infants with indications of drug use in 1988, increasing from 9,202 in 1986 (U.S. General Accounting Office [GAO], 1990a). Because of lack of rigorous nationwide detection procedures, and the wide variance in estimates of incidence among hospitals, those numbers are considered a significant underestimation. The 1989 National Drug Control Strategy report estimated that, of the 3.5 million to 4 million annual live births, there are at least 100,000 infants who are affected by their mothers' cocaine use during pregnancy (U.S. GAO, 1990a).

Maternal cocaine use during pregnancy can impact on child health and behavior in numerous ways. First, maternal cocaine use during pregnancy has the potential to directly and permanently damage the developing central nervous system of the fetus, resulting in later behavioral and learning disabilities. Second, associated health and life-style characteristics of the cocaine-using mother, such as poor nutrition, polydrug use, and inadequate prenatal care, also negatively affect fetal development (Frank et al., 1988). Third, chronic maternal cocaine use can alter the caregiving environment of the infant, since it affects maternal psychological status and behavior, and thus can have long-term effects on child cognitive and emotional development (Cregler & Mark, 1986). Fourth, by adversely affecting pregnancy, infants born to cocaine-using mothers may undergo fetal distress, asphyxia, or be born with low birth weight, and/or prematurity (Dixon, 1989; Zuckerman, Frank, et al., 1989). These medical problems have significant consequences for neurobehavioral outcome independent of exposure to cocaine.

Because maternal cocaine use is most often identified by health practitioners late in the pregnancy, or at the infant's birth, pediatricians and pediatric psychologists are on the front lines of dealing with the neurodevelopmental consequences of the cocaine-exposed infant. Pediatric psychologists need to be informed about the research and clinical literature that reports on the impact of maternal cocaine use on child health and behavior, as well as the confounding medical and social problems of these families. They will encounter increasing numbers of affected children in pediatric clinics, hospitals, and school settings in the coming decade. Psychologists will also be involved in designing intervention and research programs for these children. This paper reviews the literature on maternal cocaine use and subsequent child medical and psychological health, focusing on the following general areas: (a) pharmacologic and medical effects of cocaine; (b) epidemiologic studies; (c) behavioral and developmental outcome of cocaine-exposed infants; (d) research methodology; and (e) clinical interventions.

COCAINE—PHARMACOLOGIC AND MEDICAL EFFECTS

In terms of fetal effects, animal studies indicate that cocaine readily crosses the placenta and enters the fetal circulation. Cocaine also induces constriction of uterine blood vessels, producing acidosis and possible asphyxia which is further complicated by contraction of the uterus (Woods, Plessinger, & Clark, 1987). This reduction in the supply of oxygen to the

fetus has implications for later neurological and cognitive-behavioral development. Abrupt uterine contractions are considered one reason for the higher rate of spontaneous abortions and uterine/placental separation (*abruptio placentae*) in cocaine-using women (Cregler & Mark, 1986; Frank et al., 1988). These cocaine-initiated events may cause premature birth and birth asphyxia. Cocaine is also passed readily to the developing infant through maternal breast milk, with at least one report of acute intoxication of an infant through that mechanism (Chasnoff, Lewis, & Squires, 1987). Neuspiel and Hamel (1991) have outlined five potential mechanisms for cocaine's effects on infant behavior. Down-regulation of neurotransmitter receptors may result in defective synaptic development. Cerebral hypoxia, as noted above, can relate to deficits in development. Vascular disruption prenatally has been related in some cocaine studies to malformations in offspring. Fetal malnutrition, mediated by prematurity or maternal malnutrition, may result in growth retardation or microcephaly. Specific areas of the fetal brain may be affected by cocaine exposure, instigating alterations in cerebral activity or structure that negatively affect development.

Increased rates of spontaneous abortions, preterm birth, and lowered gestational age have been noted (Chasnoff, Burns, Schnoll, & Burns, 1985; Hadeed & Siegel, 1989; MacGregor, Keith, Bachicha, & Chasnoff, 1989; Singer, Song, Warshawsky, & Kliegman, 1991; Zuckerman, Frank, et al., 1989), placing infants at risk for the multiple developmental hazards of prematurity due to immature organ systems. Intrauterine growth retardation, also known to be associated with later child developmental problems, has been found in almost all studies comparing cocaine-exposed to cocaine-free infants (Chasnoff & Griffith, 1990; Hadeed & Siegel, 1989; MacGregor et al., 1989; Zuckerman, Frank, et al., 1989). Brain growth appears to be similarly affected as determined by small head circumferences. In fact, the most compelling finding to date of the adverse effect of fetal cocaine exposure on infant development is that of intrauterine growth retardation. Several recent studies using adequate sample size, multivariate analyses, careful matching or stratification methods, and control for confounding variables of maternal age, ethnicity, prematurity, and polydrug use, have indicated an independent effect of cocaine on growth retardation (Coles, Platzman, Smith, James, & Falek, 1991; Eisen et al., 1991; Frank et al., 1990; Neuspiel, Hamel, Hochberg, Green, & Campbell, 1991; Zuckerman, Frank, et al., 1989).

Most available studies of the development of cocaine-exposed infants have focused on full-term infants. As prematurity and low birth weight appear to be obstetric effects of maternal cocaine use, the follow-up of only full-term cohorts may be misleading and overly optimistic by eliminating the most affected infants. The developmental risks of prematurity may be exacerbated in cocaine-exposed preterms, or alternatively, may be reduced because of lowered fetal exposure. A follow-up study of a cohort of very low birth weight infants with chronic lung disease has indicated that 25% of a cohort born in 1990, in an urban, tertiary-care hospital were cocaine-exposed, more than double the rate of the hospital's general obstetric sample (Singer, 1990).

Neonatal neurologic abnormalities, including cerebral infarction, EEG, BAER, and ultrasonographic, or pneumographic abnormalities, have been noted in several studies of cocaine-exposed infants (Chasnoff, Hevet, Kletter, & Kaplan, 1989; Dixon & Bejar, 1989; Doberczak, Shanzer, Seine, & Kandall 1989). These neurological abnormalities may be

factors affecting the early developmental problems seen in follow-up studies and have implications for long-term academic and learning disabilities. Thus, in utero cocaine exposure has been related to a variety of significant medical and neurologic risks for the infant.

EPIDEMIOLOGIC STUDIES

From 10 to 20% of pregnant women use some illegal drugs during their pregnancies, with a similar incidence found in both minority, low income, and middle class, private care populations (Chasnoff et al., 1990; Hagopian et al., 1990; Zuckerman, Frank, et al., 1989). The most commonly used method of detection of cocaine use in pregnant women in studies to date has been urine screening at delivery. Cocaine's metabolite, benzoylecgonine, is detectable in urine for 3 to 6 days after use. Thus, studies relying on urine screening alone are likely to underidentify maternal cocaine use; as are studies dependent on only self-report and clinical interview (Zuckerman, Frank, et al., 1989). Newborn meconium can also be screened through radioimmunoassay and is a more sensitive measure of benzoylecgonine than urine assays (Ostrea, Brady, Parks, Asensio, & Naluz, 1989). Although not yet well validated, meconium assays, which can potentially identify maternal cocaine use throughout the entire gestation period, are likely to replace urine assays in research programs due to their advantages. Analysis of hair specimens, both in infants and adults, has also been proposed as a potentially accurate and easy method of estimation of duration, degree, and even time of drug exposure (Graham, Korero, & Klein, 1989). However, hair analysis remains a controversial method, requiring further study and validation.

Racial and social class differences have been noted as to preferred drugs assessed through anonymous urine toxicology screens. The few available studies suggest that cocaine use is more prevalent among poor minority women and that marijuana use predominates among white, middle-class patients (Chasnoff et al., 1990; Hagopian et al., 1990). The social and legal consequences of detecting drug use during pregnancy may be racially biased. Despite similar rates of illegal drug use detectable in urine screens at their infants' birth, minority women were found to be ten times more likely to be referred to the Department of Human Services for drug use than white private-care patients (Chasnoff et al., 1990).

Multiple associated social and health risks detrimental to fetal and infant development have been uniformly documented in all studies examining the characteristics of cocaine-using pregnant women. Cocaine-use during pregnancy is associated with heavy and chronic use of additional legal and illegal drugs. Marijuana, alcohol, and cigarette use are increased at almost three times the rate found in non-cocaine-using samples from similar racial and social class groups (Frank et al., 1988; Gillogley et al., 1990; Singer, Song, et al., 1991). Other commonly used substances include methadone, heroin, amphetamines, PCP, barbiturates, LSD, and diazepam. These confounding drugs may also adversely effect the outcome of the pregnancy or the neurobehavioral development of the infant. Poor prenatal care is characteristic, with some studies reporting that from 50 to 60% of their sample received no prenatal care at all (Cherukiri, Minkoff, Feldman, Parekh, & Glass, 1988; Singer Song, et al., 1991).

Cocaine-using women weigh less at the birth of their infant, and may gain less weight during their pregnancies, suggesting that anorexia and poor maternal nutrition, known side effects of chronic cocaine usage, may exacerbate any direct effect of cocaine on fetal growth (Frank et al., 1988; Singer, Song, et al., 1990). Rates of infection are also high in cocaine-using women, particularly of sexually transmitted diseases, and in some areas of the country, of HIV and syphilis, both of which adversely affect perinatal outcome. Cocaine-using women report that their infants' fathers also use cocaine and other drugs (Frank et al., 1988). Paternal use of drugs is an important, as yet unquantified, issue in assessing the sequelae of cocaine exposure in infants, since animal studies have implicated paternal use as a significant factor in evaluating the effects of other teratogens on offspring development. Variations in prenatal timing, dosage, frequency, and patterns of cocaine and other drug use among pregnant women which may affect perinatal and infant outcome may be significant and need to be studied. Social class, cultural attitudes, and regional differences in drug availability, purity, potency and mode of use, and associated nutritional, medical care, and life-style differences also await further epidemiologic study as these factors may worsen or mitigate cocaine's effects on fetal development.

BEHAVIORAL AND DEVELOPMENTAL OUTCOMES

The general public has been familiarized with child developmental problems associated with the cocaine epidemic through the popular media. Alarming but unsystematic reports on "crack babies" and "coke kids" have pervaded the public consciousness. These reports have characterized cocaine-exposed children in such terms as "joyless, inconsolable, unmotivated, unable to learn, incapable of normal human attachment, aggressive, and hyperactive" (Blakeslee, 1990; Chira, 1990). Currently, there are no available studies of the development or behavior of cocaine-exposed infants beyond 3 years of age. Thus, no firm conclusions can be drawn regarding the possible severity of, or types of, abnormal developmental outcomes associated with fetal cocaine exposure. These dramatic clinical examples may be the exception rather than the rule. Such severe patients may be highlighted by the media but may not be representative of the characteristics of the entire population of cocaine-exposed infants.

The clinical literature does have serious concerns about the social behaviors and educational needs of cocaine-exposed children. In areas of the country where the crack-cocaine epidemic has become entrenched, child welfare departments have documented significant increases in the need for infant and child foster care. Although it is generally accepted that parental abandonment, abuse, and neglect of children are associated with increases in maternal drug use (Dixon, 1989; U.S. GAO, 1990a), there is variation among states on current legislative practices in child abuse and neglect (Marshall, 1991). The recent explosive increase in maternal drug use has prompted innovative alternatives for protective custody including in-home intensive programming, home visiting, and residential drug treatment for mothers and children (U.S. GAO, 1990b; Macro Systems, Inc., 1991; LRP, 1991). Many of these programs have begun only recently so information about outcomes is not complete.

In addition, an emerging infant developmental literature has begun to document specific neurobehavioral effects of fetal cocaine exposure on maternal and infant behavior. These

studies have focused on infant behavior, infant cognitive development, and maternal–child interaction.

NEONATAL BEHAVIOR

In their pioneering studies, Chasnoff and colleagues described cocaine-exposed neonates as different from non-drug-exposed infants (Chasnoff et al., 1985; Chasnoff, Griffith, MacGregor, Dirkies, & Burns, 1989). Lethargy, poor responsivity, irritability, tremulousness, hypertonicity, and disorganization of feeding and sleeping patterns were characteristics described by workers caring for these infants in neonatal nurseries. Subsequently, several standardized scales have been used to describe objectively and to quantify these behavioral characteristics in the fetal and neonatal periods.

To determine whether or not cocaine-exposed infants experienced a withdrawal syndrome after birth similar to that seen in heroin- or methadone-addicted infants, some investigators have used the Neonatal Abstinence Scale (Finnegan, 1984) with varying results. This scale measures 21 signs of drug withdrawal, including tone, sleeping and feeding irregularities, tremulousness, and tachypnea. An infant showing signs of withdrawal, for example, may cry incessantly, as well as sleep or eat poorly. While some studies (Dixon & Bejar, 1989; Oro & Dixon, 1987) have noted mild withdrawal symptoms in cocaine infants, other reports found no withdrawal symptoms in cocaine infants in controlled studies (Coles et al., 1991; Finnegan, Kaltenbach, Weiner, & Haney, 1990). Since withdrawal scales designed for narcotic drugs may not be relevant to cocaine, other measures may be more appropriate for describing the spectrum of behaviors seen in cocaine-exposed neonates.

All but one study (Woods, Eyler, Behnke, & Conlon, in press) have found sensory and behavioral deficits in cocaine-exposed neonates in comparison to social class and racially matched controls using the Neonatal Behavioral Assessment Scale (NBAS, Brazelton, 1973). The NBAS was developed to assess the behavioral repertoire of the full-term infant from birth through the first month of life. Items assess visual and auditory orientation, habituation, reflex behavior, physiological stability, and interactive behavior with the examiner, including ability to alert and attend. These items are typically expressed through cluster scores. Abnormalities in comparison to non-cocaine-exposed infants have been found across several studies which examined cocaine-exposed infants with the NBAS in the neonatal and early infant periods (Chasnoff, Griffith, et al., 1989; Coles et al., 1991; Eisen et al., 1991; Neuspiel et al., 1991).

Several of these studies have had adequate sample sizes so that some control could be made for potentially confounding variables. Eisen et al. (1991) assessed 26 primarily black, lower social class, full-term cocaine-exposed neonates within 1 week of urine toxicology screen and, at a standardized test time (i.e., approximately 1 hour after feeding). Control infants were matched based on a random stratification procedure on sex, ethnicity, gestational age, and birth weight. Groups were equivalent on maternal age, gravidity, abortion history, and presence of hepatitis. While mothers who used opiates were excluded, cocaine-using women were found to have higher use of cigarettes and alcohol. Marijuana use was reported by 39% of the cocaine group, but was an exclusionary factor for controls. Depressed habituation

scores on the NBAS were noted in cocaine-exposed newborns, indicating that cocaine-exposed neonates were less adept at screening out aversive stimulation than comparison infants. Results from a stepwise regression analysis indicated maternal cocaine use to be the only significant variable entering the equation, predicting a significant amount of variance in infant habituation score. Since habituation is an early form of learning, which allows the infant to screen out aversive or redundant stimuli, decreased habituation skills may be a factor in the cocaine-exposed infant's reported irritability and inconsolable behaviors.

A longitudinal investigation at 2, 14, and 28 days postnatal age was undertaken by Coles et al. (1991), also using only full-term, healthy infants. This study controlled experimentally for gestational age, other drug use, associated maternal illness, and duration of drug use. Maternal drug use was assessed via self-report and urine screening. Infants of women who used cocaine or alcohol, and no other drugs except cigarettes and marijuana, were recruited. Comparison infants were mothers of similar age, race, and social class. Full-term healthy infants were examined at 2, 14, and 28 days, and grouped as follows: no cocaine or other drugs, cocaine-positive postpartum, cocaine-negative postpartum, and alcohol only. Behavioral outcome was measured through Lester, Als, and Brazelton's (1982) NBAS subscales, except for habituation, for which there were inadequate data. While no group differences were found at 2 days, cocaine-exposed infants who were positive postpartum showed poorer autonomic regulation at 14 and 30 days, and had more abnormal reflexes at 30 days than the no-cocaine group. Using a simultaneous regression model to assess independent and interactive effects of cocaine with other drugs on infant behavioral outcome, this study found duration and/or frequency of cocaine use to contribute significant variance in NBAS cluster scores at all ages. The effects were most pronounced by 28 days, when cocaine independently predicted abnormalities in motor and reflex behavior and state regulation. The importance of assessment of polydrug use was also demonstrated in this study, as marijuana, alcohol, and cigarette use, either alone or in interaction with cocaine, also predicted NBAS differences.

Less optimal infant behavior on NBAS assessment was noted in another study which followed term, cocaine-exposed and non-exposed infants at birth and at 1 month of age (Neuspiel et al., 1991). No differences were found on NBAS clusters at birth; however, cocaine-exposed infants demonstrated reliably poorer motor responses at follow-up. Cocaine exposure accounted for a significant portion of the motor cluster score variance, but not when potentially confounding variables, including other drugs, perinatal variables, and demographic and examination factors, were controlled. The high rate of attrition (60%) in this study limits these findings, particularly as sample size in the cocaine group for the regression analysis was inadequate for the large number of independent variables considered.

Using a matched design to control for confounding variables of race, parity, tobacco use, intrauterine growth, and delivery type, Woods et al., in press) assessed cocaine-exposed and non-exposed infants on the NBAS immediately after birth and at 1 month. They found no differences between groups; at either time. However, as infants were assessed at less than 3 days of age, the effects of other medications used in delivery for both groups may have obscured differences. As with Coles's study, sample size for the habituation cluster was

much smaller than the overall sample size. At 1 month follow-up, sample size was reduced such that power to detect differences may have been inadequate.

Thus, of the few currently available studies, there have been no consistent findings related to specific newborn behavioral abnormalities in term, healthy cocaine-exposed infants, perhaps due to methodologic differences in sample selection, variation in control for various confounds, timing and nature of assessment instruments, and attrition rates. Only one study (Coles et al., 1991) had adequate sample size beyond the immediate newborn period to assess the effects of cocaine, relative to other drugs and related to duration and frequency of use, on infant behavior. This study suggests both independent and interactive effects of cocaine on infant behavior that warrant further study.

COGNITIVE DEVELOPMENT

Beyond the newborn period, cocaine-exposed infants have been reported to score similarly to non-cocaine-exposed infants on standard IQ tests through 3 years of age, the length of time on which some follow-up is available (Griffith, Chasnoff, Gillogley, & Frier, 1990). Only children of mothers who had entered a prenatal drug treatment program and who may not be representative of general sample of cocaine-exposed infants were studied, however. The level of play behavior, also a correlate of cognitive development, may also be affected. Using play behavior, one study (Rodning, Beckwith, & Howard, 1989a) found preterm toddlers exposed to cocaine and other drugs to show poorly developed play behaviors, and a lack of interest and motivation in unstructured situations, in comparison to a group of high risk preterm children. Although this study was inconclusive due to methodologic problems, the use of a standardized free play assessment during which levels of manipulative, functional, and symbolic play were rated may be useful in providing information about cognitive processing deficits not apparent in typical IQ testing.

Although current findings are preliminary, they are consistent with clinical and research reports of early behavioral abnormalities in the neonatal period, and with human and animal studies that document fetal problems of hypoxia and growth retardation known to be related to neurological development and functioning. Thus, cocaine-exposed infants appear to be at risk for later learning and behavioral disabilities from a biological perspective.

CARETAKING AND MATERNAL-INFANT INTERACTION

As environmental and caretaking factors also exert considerable effects on the long-term outcome of children, these factors must be considered in evaluating risk in cocaine-exposed infants. In a 10-hospital national study, the U.S. General Accounting Office (1990a) found that approximately 1,200 of the 4,000 drug-exposed infants born in 1989 surveyed were placed in foster care, suggesting a serious level of neglect and abuse requiring out-of-home placement. While foster placement may be considered a better alternative than the often chaotic and dangerous environment associated with maternal drug use, the poor quality of many foster homes and the lack of supportive health and social services to foster care families also may be detrimental. Because the estimated demand for foster care has increased nationwide by 29% from 1986 to 1989, it is questionable whether an already

beleaguered social welfare system can adequately meet the increased needs of drug-exposed children (U.S. GAO, 1990a).

For those infants who return home from the hospital with their mothers, the effects of maternal drug life-style must be considered. For example, the impact of an impoverished home environment in which available resources are funneled into drug procurement can only be negative. Domestic violence, child neglect and abuse, and poor health care directly and negatively affect child development. The drug life-style may also have direct toxic effects on neurological development as infants and children passively inhale smoke in the crack-cocaine user's environment (Bateman & Heagarty, 1989). Regan, Ehrlich, and Finnegan (1987) wrote that the overlap of drug use and social problems for this target population warrant comprehensive monitoring of maternal depression and confusion, as well as interventions aimed at the restoration of self-esteem, promotion of better parenting skills, and addressing episodes of spouse abuse as an alternative to a sole referral to child welfare services.

Little is known about the interactional and caretaking behaviors of cocaine-using women. Davis (1990) reviewed the effects of women's alcohol and drug use on parenting skills. Women who abuse drugs often have negative self-concepts and poor tolerance for frustration which leads to ineffective parenting and ambivalence about motherhood. Maternal psychological status and interaction with her infant may be impaired through drug intoxication or withdrawal (Chethick, Burns, Burns, & Clark, 1990). Several preliminary studies are examining the caretaking interactions of cocaine-using mothers with conflicting findings (Fitzgerald, Kaltenbach, & Finnegan, 1990; Neuspiel et al., 1991). Fathers of drug-exposed infants, 50% of whom are reported to also use drugs (Frank et al., 1988), also have not been studied. Some reports suggest they are often involved in a criminal or violent life-style which would impact on child emotional and behavioral outcomes (Howard, Beckwith, Rodning, & Kropenske, 1989).

Maternal depressive symptoms, either as a precipitating factor or as a consequence of cocaine use or drug life-style, may also mediate infant developmental outcome in cocaine-exposed populations. Cocaine use has been linked to depressive symptoms in pregnant women (Zuckerman, Amaro, Bauchner, & Cabral, 1989), and depression has been noted as a correlate of withdrawal or "crash" stages of chronic cocaine use (Gawin, 1988). Infants of depressed mothers have been considered at risk for later emotional problems due to alterations in maternal and infant behaviors shown in infants of healthy mothers simulating depression (Cohn & Tronick, 1983), and postpartum depressed mothers (Cohn, Campbell, Matias, & Hopkins, 1990; Field, 1984). Assessment of depressive, as well as other psychological symptoms that might affect maternal behavior and care of her infant appear to be an important research focus.

Using the short version of the Beck Depression Inventory, one study (Woods et al., in press) compared cocaine-using women to non-cocaine-using women immediately postpartum and 1 month later. Higher levels of depressive symptomatology were reported immediately after birth in the cocaine group, but were diminished by 1 month. However, significant sample attrition (45%) by the follow-up assessment attenuated the validity of these findings. Further

studies of the depressive and/or other psychological symptoms of cocaine-using mothers are needed to provide greater information about maternal personality and emotional status and their impact on child outcome.

One aspect of early infant development thought to be a correlate of later emotional competence has been the consolidation of a secure attachment to a primary caregiver in the first year of life. Through the experimental “strange situation” paradigm (Ainsworth, Blehar, Waters, & Walls, 1978), secure and insecure attachments have been examined in normal and at-risk populations, including failure-to-thrive (Valenzuela, 1990), chronically ill (Fisher-Fay, Goldberg, & Simmons, 1988), and abused infants (Crittenden, 1985). Using this paradigm, one study (Rodning, Beckwith, & Howard, 1989b) found drug-exposed toddlers, some of whom had been exposed to cocaine in utero, to be at greater risk for insecure and anxious caregiver attachments. Although this study was limited by a small sample and lack of control for many confounding factors, such as foster care, multiple drug use, and prematurity, the issue of attachment in cocaine-exposed infants warrants further investigation as an early indication of potential developmental and emotional problems.

In summary, in addition to constitutional factors, which place the cocaine-exposed infant at high risk for long-term developmental problems, chronic poverty, altered caregiver interaction, modeling of a drug life-style, and the high potential for exposure to violence, abuse, and neglect sustain and exacerbate the developmental risk to these infants.

RESEARCH METHODOLOGY

Research on the prevalence of maternal cocaine use during pregnancy and subsequent developmental sequelae for the cocaine-exposed infant can be strengthened by addressing the possible sources of bias in current research, which include issues of sampling, choice of measures, timing of measures, and response rates. To date, the majority of epidemiologic studies have focused on low-income, minority urban women. Studies that use randomized or stratified random techniques to select a sample from a frame that includes all income groups, as well as urban and rural settings, will increase knowledge about middle-class white or rural women and how their health care, obstetric risk, and later caregiving behaviors are affected by cocaine use during pregnancy. Obtaining accurate information about cocaine use during pregnancy is problematic. Many drug users tend to deny their addictions; additionally, the detection of use of an illegal drug may impose penalties on the pregnant woman, including incarceration and referral of the infant to child welfare services (English, 1990). Policies that safeguard the respondent’s confidentiality and a clear protocol for explaining those safeguards must be part of the design of studies to minimize refusals and/or a tendency to underreport use because of fear. Cocaine use is not routinely assessed in prenatal care, and may be missed in women who do not seek prenatal care. Routinely, including multiple indicators (e.g., urine toxicology screens and/or meconium screening, and questions about cocaine and other drug use) for all obstetrical patients regardless of income level, and regardless of the amount of prenatal care received will aid in understanding the differences and similarities of drug use among various racial groups and social classes.

As cocaine-using women also show increased use of other legal and illegal drugs also known to affect infant outcome, the impact of other substances should be considered in studies of cocaine exposure. Urine toxicology screens can identify only relatively recent users of the drug, leaving women whose use may have been confined to earlier trimesters unnoticed. Some studies have enrolled only women engaged in addiction treatment programs prenatally. However, only approximately 50% of cocaine-using women enter drug treatment programs during pregnancy and up to 60% of cocaine-using women may receive no prenatal care at all (Singer, Song, et al., 1991). Follow-up studies should include children whose mothers were not in treatment prenatally, as well as those whose mothers did receive treatment to capture the spectrum of effects produced by maternal cocaine use and to increase generalizability to the majority of cocaine-exposed infants. Studies of preterm cocaine-exposed infants are needed as well, to follow-up on the fact that prematurity has been frequently noted as one of the sequelae of maternal cocaine usage during pregnancy.

Research into the effects of fetal cocaine exposure is relatively new. Building upon the information initially gleaned from studies with small samples, future researchers should obtain samples that minimize the bias tied to race and social class, and should employ designs that include appropriate comparison groups. Follow-up studies should extend through the school age years when learning and behavioral deficits usually become evident, and should incorporate instruments that have adequate standardization as well as known reliability. The numerous confounding factors related to maternal cocaine use (i.e., poor maternal nutrition and lack of prenatal care, polydrug use, infections, and poverty) make it difficult to disentangle the effects of fetal cocaine exposure per se from the effects of other deleterious obstetric and environmental factors on child development and behavior. However, initial studies documenting intrauterine growth retardation, and the known independent negative effects of these confounding factors, suggests significant clinical risk to the cocaine-exposed infant (Singer, Garber, & Kliegman, 1991).

CLINICAL INTERVENTIONS

The expertise of pediatric psychologists is needed to design and provide clinical services to the growing number of infants and children exposed to crack-cocaine in utero. One necessary component to the provision of appropriate interventions is an understanding of the neurobehavioral and social-emotional effects of fetal drug exposure. This data base is currently being hastily compiled because of the pressing need for accurate information regarding the development of drug-exposed infants and children. A second critical component involves educating pediatric psychologists about the special needs of the cocaine-using parent.

Even though the literature pertaining to cocaine use among pregnant women and mothers is only now emerging, there is a relatively large body of literature on the impact and treatment of drug use among women that can be useful in the development of treatment plans and service delivery systems for women who abuse cocaine (Beckman, 1975; Gomberg, 1986; Lief, 1985; Mondanaro, 1989; Reed, Beschner, & Mondanaro, 1982; Tamerin, 1987).

Tittle and St. Claire (1989) have outlined the specialized needs of the crack-cocaine-using mother, her infant, and the dyad. Special needs of the mother include drug treatment, emotional support, and help in securing basic needs. Special infant needs may include prematurity, possible withdrawal symptoms, and neurobehavioral problems. The pediatric psychologist must also be aware of and assess potential neurobehavioral deficits of the crack-exposed infant which may make parenting difficult. This high risk maternal-infant dyad may be in need of social supports, organization, and in some cases, intensive infant-parent psychotherapy. Some programs have developed special protocols for positioning, carrying, and handling cocaine-exposed infants. Techniques such as swaddling and hydrotherapy to calm the baby, and structured activities and education to facilitate maternal-infant attachment have been recommended (Schneider & Chasnoff, 1987). Cocaine-exposed infants have been characterized as both “overstimulated” and in need of calming, and as “underaroused” and in need of external sources of motivation. Some support for both characteristics was found in a study (Lester et al., 1991) that found both excitable and depressed neurobehavioral syndromes in cocaine-exposed newborns.

Other programs have used a case-management approach offering medical and support services as well as early identification of infant developmental delays. Within a program context of a nonjudgmental, caring environment, practitioners have noted improvement in drug-using mothers’ confidence and ability to parent (Tittle & St. Claire, 1989). The pediatric psychologist working in settings with cocaine-exposed infants needs specialized knowledge related to crisis intervention for failure-to-thrive, child neglect and abuse, maternal dual-diagnosis, AIDS, and domestic violence, all of which are more likely to occur in families in which parents use drugs (Bays, 1990). Basic health care, safety, housing, and nutritional needs of the infants are threatened by living with a caretaker preoccupied with drug procurement.

Preliminary clinical reports of toddlers and older preschoolers have suggested that cocaine-exposed children exhibit patterns of behavioral problems similar to those seen in emotionally disturbed children. Poor motivation to learn, difficulty organizing responses, sudden mood swings, and slow language acquisition are symptoms noted in crack-exposed children which require special educational efforts (Blakeslee, 1990; Chira, 1990). Pilot programs have used intensive one-to-one teaching and environmental structure along with multimodal cues to enhance learning, emotional control, and organization in cocaine-exposed children (Blakeslee, 1990; Chira, 1990). It remains an open question as to how effective these programs will prove, and to what extent in utero cocaine exposure will result in significant later behavioral and learning disabilities. There is no descriptive data base on school-age or older preschool children exposed to crack-cocaine prenatally, partly because identified cohorts of children are only now reaching school age. Of note is the fact that many descriptions of cocaine-exposed toddlers and preschoolers are similar to those of other at-risk children. As yet, no specific crack-cocaine behavioral syndrome has been identified, and current intervention available to treat other high-risk children may prove fruitful for drug-exposed children.

SUMMARY

Available studies suggest that prenatal exposure to crack-cocaine can have significant medical effects on the fetal growth and neurological development of the infant, potentially resulting in long-term behavioral and intellectual disabilities. Maternal and environmental correlates of crack-cocaine use, such as poverty, domestic violence, foster care, and neglect, also negatively affect cognitive and emotional competence in the over 100,000 infants born annually with exposure to cocaine. Large, population-based studies using multivariate analyses are necessary to determine the independent effects of cocaine on child developmental outcome relative to confounding medical and environmental variables. Pediatric psychologists are in a position to take leadership roles in designing and participating in research and intervention programs to address the negative consequences of fetal cocaine exposure in this growing, at-risk group of children.

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