2-1997

Prenatal Cocaine Exposure and Mother–Infant Interaction: Implications for Occupational Therapy Intervention

Heather Miller-Kuhaneck
Sacred Heart University

Recommended Citation

Miller-Kuhaneck, H. (1997). Prenatal cocaine exposure and mother–infant interaction: Implications for occupational therapy intervention. *American Journal of Occupational Therapy, 51*(2), 119-131. doi: 10.5014/ajot.51.2.119

Prenatal Cocaine Exposure and Mother–Infant Interaction: Implications for Occupational Therapy Intervention

Heather Miller

Key Words: occupational therapy (treatment) • pediatrics • substance dependence

The literature from multiple disciplines on in utero cocaine exposure and mother–infant interaction and attachment was examined for possible relationships and implications for occupational therapists. Maternal cocaine use and neurobehavioral deficits in neonates prenatally exposed to cocaine may result in interactional difficulties between mother and infant. Knowledge of child development, sensory regulation, and infant cues will enable therapists to assist the mother in creating positive interactive experiences between herself and her child.

Heather Miller, MS, OTR/L, is Staff Therapist—Preschool Programs, Madison County Educational Service Center, Main Street, London, Ohio 43140, and Author/Publisher, *OT/PT Update,* a newsletter for pediatric therapists.

This article was accepted for publication February 22, 1996.

The recent increase in cocaine use by pregnant women in the United States has led to frequent media reports of a new group of children doomed to failure, the "crack babies." Although scientific research has been controversial, there is enough evidence to prove that the crack baby is a media myth. Presently, we are without convincing evidence that fetal exposure to cocaine is a predetermination of ultimate overall deficiency. However, research has shown that there are potential long-term detrimental effects of cocaine use during pregnancy. These effects on the neonate, along with the characteristics of both infants prenatally exposed to cocaine and mothers who use cocaine, will be explored. The ultimate concern is the relationship between prenatal cocaine exposure and mother–infant attachment and how occupational therapists may intervene to promote the attachment process.

Historical Perspectives of Cocaine Use

The recent cocaine "epidemic" is not the first. In the late 1800s, cocaine was readily available for use as a stimulant and digestive aid. It was introduced to the public in Coca-Wine (which later became Coca-Cola®) and enjoyed a brief period of success (Atkins, 1988; Hutchings, 1993). Later, when the addictive properties were recognized, the public became suspicious of cocaine, and it was removed from Coca-Cola and replaced with caffeine. By 1914, legislation was passed that restricted cocaine to prescription use only (Atkins, 1988; Hutchings, 1993). Use of cocaine remained limited until the 1970s and 1980s when it was rediscovered by the wealthy, and rates of use increased dramatically (Plessinger & Woods, 1993). The high cost of cocaine limited its use to the middle and upper class initially; however, patterns changed drastically with the introduction of "crack" cocaine in the mid 1980s. Crack is a smokable form of cocaine that is extremely addicting, readily available, and fairly inexpensive. This combination of characteristics led crack to become widely used in the poor, inner city, and minority populations. Although there has recently been a decline of cocaine use in the general population, use of crack has risen in minority groups, particularly in the inner city. It has been estimated that between 10% and 15% of pregnant women in the United States use cocaine in some form (Glantz & Woods, 1993); however, in some cities, up to 50% of women delivering babies report or test positive for cocaine use (Amaro, Fried, Cabral, & Zuckerman, 1990).

Physiological Effects of Cocaine

Cocaine is a stimulant. In the form of crack, it is 10 to

20 times more powerful, has immediate onset of action once smoked, and reaches peak action within 10 to 15 minutes. The "high" only lasts 20 minutes or so and is followed by a "crash" or craving that often leads to immediate readministration. Smoking crack cocaine is extremely addicting, and lifestyle changes caused by repeated use can occur within weeks or months (Atkins, 1988; Breshnahan, Brooks, & Zuckerman, 1991; Zuckerman, 1991).

In the user, cocaine and crack work neurochemically by blocking the reuptake of neurotransmitters—particularly dopamine, norepinephrine, and serotonin—leading to accumulation in the synapse. This results in overstimulation of the receptors for these neurotransmitters, which can eventually lead to cell death (Lenn, 1991). In the dopamine system, the accumulation exaggerates the pleasure response and feelings of increased esteem. It also enhances alertness and heightens energy and productivity (Bandstra & Burkett, 1991). Chronic use leads to depletion of dopamine, which is thought to be the cause of depression, anxiety, and withdrawal symptoms in addicts (Frank, Breshnahan, & Zuckerman, 1993; Zuckerman, 1991). In the norepinephrine system, the overstimulation leads to increased heart rate, vasoconstriction, increased blood pressure, and mild tremors (Bandstra & Burkett, 1991). Through the serotonin system, mood, affect, and sleep–wakefulness cycles may be disrupted (Bandstra & Burkett, 1991).

Pregnancy may slow the metabolism of cocaine in both the mother and the fetus, leading to increased sensitivity to cocaine and longer exposure from a single administration (Plessinger & Woods, 1993). Cocaine directly enters the fetus and increases sympathetic nervous system effects, leading to vasoconstriction, increased blood pressure, tachycardia, and the possibility of seizures. Further, cocaine indirectly affects the fetus via the mother's central nervous system. Vasoconstriction in her body, particularly the placenta, reduces oxygen and nutrient availability. Reduced blood supply to the fetus at critical times may cause deformation of growing structures. Hypoxia due to vasoconstriction may be the mechanism for reduced size and weight (Lester et al., 1991). In the fetus, cocaine may alter neurotransmitter concentrations, receptor site development, synaptogenesis, and neuronal migration and differentiation (Frank et al., 1993; Lauder, 1988).

Effects of Fetal Cocaine Exposure

Neonatal and Infant Studies

In reviewing the literature regarding the effects of cocaine use in human pregnancy, one finds many controversial reports. Many studies have been completed to determine the extent of negative neonatal outcomes after intrauterine cocaine exposure. The risk of Sudden Infant Death Syndrome is slightly increased in neonates prenatally exposed to cocaine, but much less so than for those of mothers who used other drugs (Zuckerman, 1991). Abruption of the placenta occurs infrequently, particularly with cocaine use in the last trimester of pregnancy (Frank et al., 1993). Prematurity is reported by some (Chasnoff, Lewis, Griffith, & Wiley, 1989; Cherukuri, Minkoff, Feldman, Parekh, & Glass, 1988; Frank et al., 1993; Neerhof, MacGregor, Retsky, & Sullivan, 1989; Plessinger & Woods, 1993) but not others (Hadeed & Siegel, 1989) and may be due to lack of prenatal care. Cardiac difficulties (Frassica, Orav, Walsh, & Lipshultz, 1994) and congenital, visual, brainstem, and cerebral abnormalities, which are thought to be related to vasoconstriction during important times of structural formations in the fetal brain, are also reported (Dominguez, Villa-Coro, Slopis, & Bohan, 1991; Good, Ferriero, Golabi, & Kobori, 1992; Hoyme et al., 1990). Intrauterine growth retardation, small head circumference, and low birth weight are the most frequently and consistently reported effects of exposure to cocaine in utero (Chasnoff, Griffith, Freier, & Murray, 1992; Chasnoff, Lewis, Griffith, et al., 1989; Cherukuri et al., 1988; Chiriboga, Bateman, Brust, & Hauser, 1993; Frank et al., 1993; Hadeed & Siegel, 1989; Hurt et al., 1995; Neerhof et al., 1989; Nulman et al., 1994; Sallee et al., 1995). These effects have been shown to be diminished if the mother ceases cocaine use during the first trimester (Chasnoff, Griffith, MacGregor, Dirkes, & Burns, 1989).

Some studies have found neurological deficits and seizures (Dixon & Bejar, 1989; Dusick et al., 1993; Frank et al., 1993), but they may resolve over time. Other studies have found no neurological abnormalities. Magnetic resonance imaging was done on eight infants exposed to cocaine in utero and showed no evidence of cranial infarction or other abnormalities (Link, Weese-Mayer, & Byrd, 1991). Of 39 neonates who were exposed to cocaine in utero and given electroencephalographs (EEGs) after birth, 17 were found to have abnormal EEGs (Doberczak, Shanzer, Senie, & Kandali, 1988). The abnormalities reflected the type of pattern present in cerebral irritation and may be related to the neurobehavioral effects, such as tremors, irritability, and poor state regulation, often seen in neonates exposed to cocaine. However, by 3 to 12 months of age, all the EEGs were normal. Legido, Clancy, Spitzer, & Finnegan (1992) also studied EEGs of 35 neonates prenatally exposed to cocaine but found no significant abnormalities. Cohen, Sloves, Laugani, Glass, and DeMarinas (1994) used neurosonography to examine the brains of 18 full-term neonates with prenatal exposure and 19 infants without prenatal exposure. They noted no infarctions or hemorrhages in either group, but in the cocaine-exposed group, 8 neonates showed evidence of cyst formation in the brain. Dixon

and Bejar (1989) found sonographic abnormalities in 35.1% of 74 infants studied who were exposed prenatally to multiple drugs. Of the infants who were exposed to cocaine, 32 (41%) had abnormal echoencephalographic lesions. However, another study using neurosonography found no differences between neonates who were exposed prenatally and those who were not (King et al., 1995). Caudate nucleus abnormalities were found in 48% of infants of heavy cocaine users studied compared with 22% to 23% infants of light or nonusers (Frank, McCarten, Cabral, Levenson, & Zuckerman, 1994).

Additional research has been done to determine neurobehavioral effects of prenatal exposure to cocaine. Many of these studies have used the Brazelton Neonatal Behavioral Assessment Scale (NBAS) to investigate the outcomes of prenatal exposure on infants' behavior. The early studies demonstrated alterations in the infants' abilities to orient to stimuli, habituate to repeated stimuli, and maintain state regulation (Chasnoff, Griffith, MacGregor, et al., 1989; Chasnoff, Lewis, Griffith, et al., 1989; Eisen et al., 1991; Griffith, 1989; Hume et al., 1989). Mayes, Granger, Frank, Schottenfield, & Bornstein (1993), found significantly lower performance on the habituation items of the NBAS in neonates who were exposed to cocaine. This effect remained even when controlling for the low birth weight of these infants. There was also a relationship between low birth weight and poor state regulation, which may have been why earlier studies not controlling for weight had found state regulation difficulties. Mayes et al.'s study controlled for multiple variables, and across all examinations, habituation scores were lower for the infants who were exposed to cocaine. Another study, which used the Bayley Scales of Infant Development (BSID) to test two groups of 3-month-old infants, also noted habituation difficulties (Mayes, Bornstein, Chawarska, & Granger, 1995). One group consisted of infants who were prenatally exposed to cocaine, whereas the control group of infants were not exposed. The infants prenatally exposed to cocaine were more likely to react with irritability and less likely to habituate to novel stimuli than those in the control group. However, scores on the mental scales of the BSID were similar in the two groups.

Another study also found cognitive development to be similar in 6-month-old infants who either were or were not prenatally exposed to drugs, although temperament differences were noted (Edmondson & Smith, 1994). The infants who had been exposed were reported to be harder to manage. A recent study examined learning and emotional reactivity in 72 4-month-old to 8-month-old infants, half of whom had been prenatally exposed to cocaine (Bendersky, Alessandri, Sullivan, & Lewis, 1995). The researchers found the infants who had been exposed to drugs to be less engaged in a learning task, demonstrate lower levels of arousal, and have fewer positive responses to learning. Overall, the researchers believed that there was a lack of motivation for learning, especially when challenged, and a lack of emotional responses, which they proposed could alter relations with the caregiver.

Motor delays and tonal abnormalities are sometimes reported as well. One study demonstrated a 40 times greater risk for motor delays in infants who were prenatally exposed versus controls at 4 months of age (Schneider, 1988). Another study found that infants who had been exposed to cocaine were more likely to exhibit abnormal tone and movement patterns, particularly hypertonia and tremors (Chiroboga, Bateman, Brust, & Hauser, 1993). A study of only very low-birth-weight infants who either were or were not prenatally exposed to cocaine found that the infants who had been exposed were more likely to have depressed scores on cognitive and motor assessments (Singer et al., 1994). However, motor delays are not consistently reported and may not persist as infants age (Hurt et al., 1995).

Lester et al. (1991) proposed that variations in neurobehavioral findings of some studies are due to cocaine's dual effects when used during pregnancy (direct effects on the fetus vs. indirect effects from the mother). By examining infants' cries, they found that the infants tended to fall into one of two groups: depressed behavior or excitable behavior. The researchers proposed, and their data supported, that the indirect or growth effects were responsible for the depressed activity, whereas the direct nervous system effects were responsible for the excitability. Other researchers explain the dual effects of cocaine exposure by cocaine's metabolites, or by-products (Konkol, Murphey, Ferriero, Dempsey, & Olsen, 1994). Certain metabolites seem to be in higher concentrations in excitable infants, whereas others are more prevalent in lethargic infants.

Some researchers have reported minimal to no difference between neonates who had been exposed to cocaine and neonates who had not (Coles, Platzman, Smith, James, & Falek, 1992; Hutchings, 1993; Neuspiel, Hamel, Hochberg, Green, & Campbell, 1991). Inconsistent findings are not surprising when one considers the many confounding variables present in this type of research. Cocaine often is not the only drug being used; many of the mothers also smoke cigarettes and drink alcohol. Maternal report of the frequency and dosages of cocaine used may be inaccurate, the amount of prenatal care received may vary greatly, and differing postnatal environments are an issue (Neuspiel, 1995; Zuckerman & Frank, 1992). Because of these difficulties in human research, animal models that control for most variables are extremely

important. Some researchers believe that despite inconclusive evidence in humans, animal models provide enough information about cocaine's harmful effects to warrant continued concern about its use during human pregnancy (Henderson & McMillan, 1990; Plessinger & Woods, 1993; Smith, Mattran, Kurkjian, & Kurtz, 1989; Spear et al., 1989; Spear & Heyser, 1992).

Animal Research

Rat pups exposed to cocaine in utero were underweight at birth and had delayed maturation of the righting reflex (Henderson & McMillan, 1990). Considerable alterations in metabolism in the brains of adult rats exposed to cocaine prenatally have also been noted (Dow-Edwards, Freed, & Fico, 1990). These changes occurred in subcortical, limbic, hypothalamic, and two cortical regions—the primary somatosensory and motor cortices. Another study of cocaine exposure in rats looked at brainstem development and found prolonged auditory brainstem responses possibly due to delayed myelinization (Salamy, Dark, Salfi, Shah, & Peeke, 1992). Cocaine-exposed rat offspring were also shown to have tactile hypersensitivity and aversion to tactile stimuli (Chen, Anderson, & West, 1993), have difficulties learning (Spear et al., 1989; Vorhees et al., 1995), respond abnormally to stressful situations (Molina, Wagner, & Spear, 1994), and demonstrate poorly developed social play (Wood, Bannoura, & Johnson, 1994).

When cocaine-exposed rat offspring reach adulthood, they may have reduced neurotransmitter function. In particular, Minabe, Ashby, Heyser, Spear, & Wang (1992) showed dopaminergic function to be affected and found a smaller number of spontaneously active midbrain dopaminergic cells than is typical. These researchers believed that the increased irritability, decreased interactive behavior, and learning deficits found in human offspring prenatally exposed to cocaine might partially be accounted for by these results if the same reduction in dopaminergic function could be found in humans. However, dopamine system changes may not persist into adulthood in all cases (Keller, Maisonneuve, Nuccio, Carlson, & Glick, 1994). The serotonergic system may also be affected, with changes lasting into adulthood. Neuroendocrine responses, which are end-products of serotonergic function, have been shown to be altered in female rats exposed to cocaine prenatally (Cabrera, Levy, Li, Van de Kar, & Battaglia, 1994).

Prenatal cocaine exposure may increase the vulnerability of the dopamine system, particularly to hypoxic events (Weese-Mayer et al., 1994). When cocaine is present in the neonatal rat brain at the same time a hypoxic event occurs, cell damage has been shown to increase (Spraggins, Seidler, & Slotkin, 1994). A study of brain growth in rats exposed to cocaine determined that actual brain weight and brain structures are unaffected by cocaine exposure during the brain growth spurt (Chen et al., 1993), lending support for the theory that changes in neurotransmitters and receptors are the basis for the neurobehavioral effects being reported.

Studies of rat offspring prenatally exposed to cocaine have shown both immediate and long-term effects, suggesting a change in cognitive processing and overall functioning rather than a delay in cognitive development (Smith et al., 1989; Spear & Heyser, 1992). The animal models provide reason for concern regarding cocaine's long-term effects in humans. Most researchers point out, however, that models that use human subjects are needed to determine whether long-term difficulties with learning, attention, and self-control result from intrauterine cocaine exposure.

Long-Term Studies in Humans

Long-term effects on the child, ranging from subtle learning problems to considerable delays, are possible because of cocaine's ability to cross into the fetal brain. Cocaine also impairs fetal oxygenation, which may make the child vulnerable to later difficulties (Bandstra & Burkett, 1991; Lester et al., 1991; Zuckerman, 1991). The effects on fetal neurotransmitters may lead to attention difficulties, hyperactivity, and potential sensory processing problems (Mayes et al., 1993; Needlman, Zuckerman, Anderson, Mirochnick, & Cohen, 1993; Stallings-Sahler, 1993; Zuckerman & Frank, 1992). However, studies that examine the long-term effects of cocaine exposure on the child are fraught with design problems. Without control of the environment in which the child is raised, one cannot determine which deficits are directly the result of cocaine exposure and which are caused by the environment. Often, the postnatal environment is a more important determinant of outcome than the drug use during pregnancy (Breshnahan et al., 1991; Zuckerman, 1991). Few studies have attempted to control for postnatal environment, although this appears to be the trend recently. Usually, such controls include studying only children from one socioeconomic status (SES) group, studying children who were prenatally exposed but were then adopted into a new home, or using other infants and children at high risk as a control group. It is unknown how well these methods truly control for environmental variables, but it is known that the environment is very important in a child's development. The difficulty in controlling for all confounding variables common in this type of research should be kept in mind while reading the results of the following studies.

Language

Language delays are frequently reported in children exposed to cocaine prenatally (Davis et al., 1992; Griffith, Azuma, & Chasnoff, 1994; Nulman et al., 1994; van Baar & de Graaff, 1994), and children with language delays are more likely to have been prenatally exposed to cocaine than children without language delays (Angelilli et al., 1994). Fulks and Harris (1995) found that children who had been exposed to drugs score low on the verbal items of the Miller Assessment for Preschoolers. Nulman et al. (1994) found scores on language-based assessments to be significantly lower in children exposed to cocaine prenatally compared with children who were not exposed, even when intelligence quotient scores were similar. A 3-year follow-up study of children prenatally exposed to cocaine used an intelligence quotient test rather than the BSID and found that cocaine exposure was associated with decreased verbal reasoning (Griffith et al., 1994). A study of children ages 4 to 6 years found that the children who had been prenatally exposed to crack performed poorly on receptive language measures but were comparable to children who had not been exposed in terms of expressive language (Bender et al., 1995). These findings are supported by another study that reported finding greater impairments in receptive versus expressive language abilities in a group of preschoolers prenatally exposed to cocaine (Malakoff, Mayes, & Schottenfield, 1994).

Cognition

Most studies that examined cognitive functioning found normal or near-normal abilities when children who had been exposed to cocaine were compared with those who had not. One study followed children who had been exposed to cocaine and other drugs, children who had been exposed to alcohol and marijuana, and controls through the first 2 years of life and found relatively few significant differences among the groups when tested with the BSID (Chasnoff et al., 1992). Additionally, measures of intelligence quotient were found to be similar between children who had been exposed and those who had not (Nulman et al., 1994). Hurt et al. (1995) tested 219 infants of low SES mothers at 6-month intervals for 30 months and found no significant differences on BSID scores between the group of infants who had been exposed and the controls. Another study compared toddlers who had been exposed to drugs with toddlers at high risk raised in environments matched for ethnicity and social class in order to control for confounding variables such as poor prenatal care, poverty, and adverse postnatal environments (Rodning, Beckwith, & Howard, 1989). The researchers observed the toddlers in a play situation and gave them the Gesell Developmental Schedules and BSID. The toddlers who had been exposed to drugs performed at a lower level on the developmental assessments than the toddlers at high risk but were still within a normal range. Preschoolers prenatally exposed to cocaine performed within the borderline range on the cognitive portions of the McCarthy Scales of Children's Abilities (Beckwith, Crawford, Moore, & Howard, 1995). These same children had been tested earlier with the BSID and were found to be within an average range on the mental scales.

Play Skills, Temperament, Sensory Processing, and Behavior

In the Rodning et al. (1989) study, although the cognitive measures were within a normal range, the play skills of the toddler's who had been exposed to drugs were delayed. They had fewer representational play events and decreased spontaneous play. Their play consisted primarily of banging, throwing, scattering, and dropping. In a study of the play of 2-year-olds, Beckwith et al. (1995) found that children exposed prenatally to cocaine and phencyclidine (PCP) demonstrated immature play skills, poor attention, deviant behaviors, and fewer instances of positive social interaction with the caregiver. However, a subgroup of this group played normally. The differences found between the children who played normally and those who did not appeared to be that children who played normally had a greater level of attachment to their primary caregiver, a higher level of developmental abilities, and a more responsive type of mothering during their first year of life.

Children who had been exposed to drugs have been rated as more aggressive than controls (Griffith et al., 1994). This was also noted in Beckwith et al.'s (1995) study of preschool play with peers. They found the preschoolers exposed prenatally, although comparable in many play skills with their peers who were not prenatally exposed, to be more aggressive and inappropriate with peers, more insensitive, and less compliant in general. The preschoolers prenatally exposed to either cocaine or PCP were found to be no different in their ability to delay responses than children who had not been exposed, but they were found to have difficulty remaining in their seat and were more likely to need coaxing to transition between tasks. Beckwith et al. also rated them as more irritable and needing more adult assistance to perform.

One study reported that both biological and foster mothers of children who had been exposed to drugs perceived the children as being overactive, restless, inattentive, perseverative, hypersensitive to sensory stimulation, and difficult to calm (Kelley, 1992). Other therapists and

researchers have observed poor organization of behavior as well, with low stress tolerance, distractibility and poor concentration, impulsiveness, auditory processing difficulties, and problems with spatial motor tasks. In addition, sensory hypersensitivity and poor motor planning have been observed (Stallings-Sahler, 1993). With the Miller Assessment for Preschoolers, Fulks and Harris (1995) found that preschoolers who had been exposed to drugs have difficulties on sensory processing items. Finally, a recent study found no difference in behaviors such as hyperactivity, distractibility, or following directions; however, children with prenatal cocaine exposure were found to perform more poorly on the Developmental Test of Visual-Motor Integration and the draw-a-person test (Bender et al., 1995).

A retrospective study found major developmental delays in 70 children with prenatal cocaine exposure who were referred for a developmental evaluation at ages from 1 month to 5 years (Davis et al., 1992). In addition, 11% of these children met the criteria for a diagnosis of autism. Autism is thought to occur normally in 2 to 21 per 1,000 live births. This study also found hyperactivity, poor play skills, explosive behavior, and fine motor and visuomotor delays in the majority of these children.

However, not all studies find these types of differences. In one study of behavior in the classroom, no significant differences were found between eight children exposed to cocaine and eight controls (Rotholz, Snyder, & Peters, 1995). Hurt et al. (1995) proposed that it was environmental variables such as SES rather than prenatal cocaine exposure that leads to reported low scores on tests such as the BSID in children who had been exposed to cocaine. These authors found that children from low SES backgrounds, with or without prenatal exposure to cocaine, had lower BSID scores than children from higher SES backgrounds.

Whether it is the prenatal exposure to cocaine or the subsequent poor environment that leads to these difficulties, there is evidence to suggest the potential for long-term problems in these children. The importance of positive mother and infant interactions and improved postnatal environment becomes all the more critical in light of this evidence and in order to afford the child optimal opportunities for development.

Mother–Infant Relationships in Dyads Not Exposed to Drugs

Attachment

Attachment is defined as an *affectionate tie* between the infant and the caregiver (Swanwick, 1984) that grows over time and is the outcome of the combined behaviors of each (Egeland & Farber, 1984). Infant behavior normally is a biologically prescribed repertoire of actions designed to encourage and promote interactions with the mother (Swanwick, 1984). These infant behaviors include smiling, vocalizing, and gazing, with infant gazing being crucial for continuing face-to-face interaction (Blehar, Lieberman, & Ainsworth, 1977). The maternal behavior most important to the attachment process is responsiveness. Responsiveness to positive infant behaviors is thought to increase their frequency, whereas unresponsiveness is thought to lead to fussiness and gaze aversion (Blehar et al., 1977). Over time, the infants learn either to expect their caregivers to be accessible and available or to expect that the caregivers will be unresponsive to their needs (Ainsworth, 1979).

Multiple studies have shown that responsive mothering leads to securely attached infants (Belsky, Bovine, & Taylor, 1984; Egeland & Farber, 1984; Isabella, Belsky, & von Eye, 1989). Mothers of securely attached infants notice their babies' cues and use them to guide their responses. They also are positive about their babies and seem to get pleasure from being with them and holding them (Pederson et al., 1990). Not only do mothers of securely attached infants tend to hold their babies more often, but they also have greater face-to-face contact and, in general, are more sensitive during feeding (Crockenburg, 1981). Positive affection and comforting close-body contact has been found to be important in developing attachments (Isabella et al., 1989). Positive interactions are influenced by mothers' sensitivity to infant cues and respect for the infants' present state. Pacing or timing of interaction is also important (Blehar et al., 1977). Van den Boom (1994) found that mothers trained in methods of appropriate responsiveness and in accurately monitoring infants' signals have better attachments with their babies at 12 months than do mothers who were not trained.

Characteristics that hinder attachment are numerous. Crockenburg (1981) found that mothers with low social support, when combined with an irritable infant, led to avoidant and anxious attachments of the infants. In addition, the combination of an irritable infant and poor support systems leads to less responsive mothering in general (Crockenburg, 1981). Denham and Moser (1994) also showed that fussy babies have less attached and less responsive mothers.

Other authors have reported that overstimulation by the mother leads to avoidant behaviors in the infant (Isabella et al., 1989). Anxiously attached babies have mothers who are relatively insensitive and unresponsive and have had early experiences of only brief face-to face interactions (Blehar et al., 1977). Their mothers also tend to avoid close bodily contact and were likely to exhibit

affective expression (Ainsworth, 1979). One study reported that mothers of anxious, avoidant infants lack confidence, show poor caregiving skills, react negatively to motherhood, limit the handling of their infants, and respond less to their infants in general (Egeland & Farber, 1984).

Maternal actions with an infant can have a considerable effect on later attachments in either a positive or negative direction. Egeland and Farber (1984) found that over time, if the mother became more skilled in parenting and responding appropriately and more positive in general about motherhood, the attachment improved. Another study found maternal variables to be more important in predicting later attachment than did the child variables (van Ijzendoorn, Goldberg, Kroonenberg, & Frenkel, 1992). The authors hypothesized that although a responsive mother can compensate somewhat for problem behaviors in the infant, the infant is unable to do so when the mother's interactive skills are lacking.

Long-Term Effects of Mother–Infant Interactions

The mother–infant relationship is not only critical to the emotional health of the infant, but also has been shown to have long-term effects on development. Ainsworth (1979) stated that securely attached infants become more competent and more sympathetic with their peers. She also stated that securely attached children demonstrate better play skills, have more developed problem-solving ability, are more self-directed, and tend to perform better on developmental and language tests. Early mother–infant interaction with positive physical contact and frequent verbal stimulation has been associated with cognitive competence at age 2 years (Olson, Bates, & Bayles, 1984). One study evaluated quality of attachment and later adaptation in preschool and found that mother–infant attachment was a powerful predictor of children's abilities to adapt to the difficulties and challenges facing them in preschool (Suess, Grossman, & Stroufe, 1992). The authors reported that these children were more competent and showed fewer behavioral problems. The poorly attached children tended to be more hostile, to view others as unavailable, and to have shorter attentions spans. Early attachment problems also predict preschool aggression and hostility (Lyons, Alpern, & Repacholi, 1993). In general, it appears that the initial relationship with the mother affects the way the infant organizes his or her behavior in later relationships and in other environmental contexts (Ainsworth, 1979).

Mother–Infant Interaction in Dyads Exposed to Drugs

Attachment flourishes when both mother and infant participate in the development of the relationship. The mother's primary requirement is to be responsive to the infant and the infant's is to encourage the interaction with the mother by smiling, vocalizing, and maintaining eye contact. In dyads who have been exposed to drugs, both mother and baby may face difficulties in fulfilling their roles appropriately.

Maternal Variables

The mother's ability to understand her infant's cues and respond to them adequately may be limited depending on her own life history, personality, and level of drug use (Freier, Griffith, & Chasnoff, 1991). One study found that mothers with positive urine screens for cocaine were less sensitive to their infant's cues than mothers with negative screens (Barabach, Glazer, & Norris, 1992). These mothers often come from dysfunctional families themselves, may be in a primary relationship with their drug of choice and not with their child, and may have a tendency toward depression or irritability due to drug withdrawal (Zuckerman & Frank, 1992). The mother's dependence on the drug may result in her having few social supports and lowered self-esteem. Money for the child's care may be used instead for drugs; therefore, the child may be at risk for malnutrition, anemia, and overall poor health (Burns & Burns, 1988; Griffith, 1988). This poor care of her child may cause the mother's feelings of guilt and lowered self-esteem. Feelings of inadequacy, guilt, and depression may leave the mother emotionally unavailable to form a secure attachment with the infant. The combination of negative past experiences and dysfunctional families of origin, when combined with possible emotional instability, lack of knowledge of parenting, a vulnerable or difficult infant, and limited social supports, can result in poor parental responsiveness, including withdrawal, frustration or rejection, and overt hostility (Burns & Burns, 1988; Griffith, 1988).

One study that examined the stress of parenting children who had been exposed to drugs found that mothers who use drugs are more likely to feel considerable stress (Kelley, 1992). In turn, child maltreatment was higher when compared with control groups of foster mothers of children who had been exposed to drugs and mothers who were no longer using drugs. Mothers with addictions may be egocentric, seeing the child as fulfilling her needs but at the same time resenting the child for being demanding. The mother may reject or criticize her child and show limited sensitivity to her infant's needs (Burns & Burns, 1988). Even if abstinent, the mother may be struggling with remaining so and may be dealing with many other issues of addiction surrounding her own recovery. This again may leave her unavailable for her infant (Frank et al., 1993).

Infant Variables

"In order to build an optimally functioning maternal–infant relationship which is fulfilling to both partners, mother and infant must each have the behavioral repertoire and the adaptability to respond appropriately to the stimulation provided by the other" (Griffith, 1988, p. 105). The effects of cocaine use that have the most impact on the infant's ability to be a responsive partner in attachment are neurobehavioral changes. These vary depending on whether the cocaine effects are direct or indirect. Direct effects of cocaine on the infant's neurological system tend to cause excitability, irritability, and hypersensitivity. The indirect effects from vasoconstriction and hypoxia tend to depress the system, causing withdrawal, sleepiness, and lethargy (Lester et al., 1991). Other neurobehavioral difficulties include an inability to regulate state and poor habituation and orientation (Griffith, 1988). These infants can be tremulous and startle easily (Frank et al., 1993) or be poorly responsive and overly sleepy. The sleepiness may be the infants' mechanism for withdrawing from stimulation (Griffith, 1989) because they tend to be easily overstimulated by the sensory environment when they are awake (Hyde & Trautman, 1989). These infants can abruptly switch from state to state and require considerable environmental structuring to attain a quiet alert state that allows for interaction with another (Griffith, 1988). Again, this interaction must be sensitive to the infant's cues, or overstimulation is likely to occur (Griffith, 1988, 1989). One study found that infants who had been exposed to cocaine were more likely to send cues that were unclear and that they were less responsive to the parent in general (Barabach et al., 1992). Another study found these infants to be asleep or distressed for longer periods than were control infants (Gottwald & Thurman, 1994).

Because infants who had been exposed to cocaine are unable to appropriately respond, interactions may be unrewarding for the mother (Freier et al., 1991). Mother–child interaction may be strained, with later consequences on child development and parenting (Zuckerman, 1991). Often, mothers perceive the infant's behaviors as rejection, and in trying to engage, they may overstimulate the infant, show hostility, and eventually detach emotionally (Griffith, 1989).

There has been little research that specifically examines attachment in this population and none where cocaine was the only drug being used. However, in one study of mothers who either used cocaine and PCP or were drug free and who were matched for SES and other variables, 64% of the infants in the drug-free group were rated as securely attached to their mother (Beckwith et al., 1995). Only 18% of the infants of mothers who used drugs were rated as securely attached. In this group, 68% were rated as disorganized in their attachment. Attachment ratings were not found to be affected by the number of changes of the primary caregiver or who the primary caregiver was. An important variable was whether the mother had stopped using drugs after delivery. Only those infants whose mothers had stopped using drugs were considered to be securely attached.

Discussion: Implications for Occupational Therapists

Occupational therapists may more and more frequently encounter infants and children who were exposed to cocaine prenatally. Although working with these children and their families can be challenging, appropriate intervention may positively affect long-term outcomes. Early negative reports from the media, which led to the belief that crack babies were doomed from birth, have not proven true. Therapists must be aware of the existing controversy surrounding research in this area. Most of the human research studies about prenatal cocaine use and outcomes have problems with their design, including confounding variables that are not always sufficiently controlled for. However, there is now enough evidence both to refute the earlier media contention that children who had been exposed to cocaine were destined to be below average in all areas and to support the belief that there are potential long-term difficulties facing children with prenatal exposure. It is important that therapists be familiar with the literature and the variables most influential to outcomes in order to provide the most effective interventions.

It is believed that crack cocaine affects neurotransmitters and brain processes but appears not to affect brain structures, and children with prenatal drug exposure tend to have average or low–average cognitive skills. Human research suggests that such neurological problems may be related to prenatal cocaine exposure. This is supported by animal research, which has found that changes in neurotransmitter functioning might lead to behavioral difficulties, sensory abnormalities, and changes in cognitive processing, with cognitive function remaining intact. Although the literature from human studies supports the belief that there is a potential for difficulties in certain areas, including neurobehavioral regulation, play skills, sensory processing, temperament, and receptive language abilities, it also suggests that factors other than the prenatal exposure itself may be extremely important. Most encouraging is research supporting the postnatal environment as a more important determinant of outcome than the drug exposure per se. Environments can be changed, whereas prenatal events cannot. Additionally, literature on attachment in dyads without drug exposure have proposed that attach-

ment is important for later competence, play skills, problem solving, language skills, cognitive abilities, control of aggression and hostility, and attention span. All of these are areas in which problems have been reported in children with cocaine exposure. Perhaps prenatal cocaine exposure leads to poor attachments due to poor mother and infant responsiveness, which then hinders positive development in those areas. We can only speculate on the differential effects of poor attachment and the cocaine exposure itself. However, attachment, like the postnatal environment, can be improved.

Occupational therapists with knowledge of sensory integration theory, regulatory disorders, emotional development, nervous system functioning, and mother–infant interaction may provide both mother and infant with appropriate intervention. Intervention might target mother–infant attachment specifically to potentially improve childhood outcomes. Attachment will be positively affected by enhancing maternal responsiveness and knowledge and improving infant responsiveness and cues. Specific techniques might include education, direct clinical intervention, and environmental modification.

Occupational therapists can educate the mother about her infant's needs and cues. The ability to respond appropriately to those cues may be improved with proper information, modeling, and careful cuing from the therapist. Occupational therapists can provide both a role model for interactions with the infant and assistance in recognizing and responding to infant cues. Mothers can be taught to alter their infant's environment in response to the infant's needs. At the Perinatal Center for Chemical Dependence in Chicago, researchers found that education programs aimed at increasing responsiveness were beneficial for both the mother and the infant (Griffith, 1989). As the mother's responsiveness improved, infant state regulation also improved.

In addition to learning to respond appropriately to infant cues, mothers' understanding of their infants' sensory toleration is critical. Understanding their infants' altered needs for sensory stimulation as the reason for positive environmental modifications may help the mother begin to make these changes independently. Additionally, avoidance behaviors should be explained in terms of an escape from excessive sensory stimulation so that the mother does not misinterpret the behaviors as rejection. Providing information and promoting both maternal confidence and responsiveness in order to enhance attachment are the primary goals of occupational therapy intervention with the mother.

Occupational therapists can also offer direct treatment for the infant on the basis of sensory integration principles. A primary goal of treatment is to help the infant become a receptive partner in interactions with the mother and other caregivers. Receptive behaviors include the ability to regulate state, self-console when upset, and tolerate sensory stimulation from others and the environment. Treatment could consist of calming sensory input, such as deep pressure and slow, gentle linear motion, to assist the infant in becoming receptive to interaction. Initial interactions may consist of brief periods of gazing alone, with no vocalizations or movements. Typically, only one sensory system should be stimulated at a time, and early signs of overstimulation, such as gaze aversion, yawns, hiccoughs, spitting up, changes in skin color, and changes in respiration, should be heeded. Maintaining a calm alert state is of utmost importance in continuing interaction, and positive interactions are necessary for improved attachment.

Appropriate environmental modifications should also be initiated while the infant is still in the hospital. The infant can be provided with ways to feel more secure in the crib, including swaddling and positioning with its hands near its face for self-soothing. The crib can also be covered to reduce light and noise. Basic tasks such as feeding and dressing can be adapted and modeled to increase the use of the types of sensory input that the infant can tolerate and decrease excessive stimulation. Typically, this includes firm, deep touch; swaddling; changing positioning so that the infant is held snugly in either a vertical or horizontal position with contact along as much of the baby's body surface as possible; and very slow movements. Additionally, it might be necessary to dim lights, reduce environmental noise, and reduce the demands for interaction during these tasks. Adaptations such as these may make it easier for the infant to tolerate the tasks without becoming overstimulated, thus allowing the mother to become more confident in handling her infant. Perhaps more normal mother–infant interactions during feeding and dressing will support the attachment process.

One specific technique that could prove beneficial to both mother and infant is pediatric massage. For therapists experienced in pediatric massage, it can become a vehicle for promoting more normal interaction and enhancing attachment in the dyads. For the infant, massage can provide calming deep pressure and joint input, improved circulation, maintenance of muscles in an optimum state, and reflex stimulation in the skin (Drehobl & Fuhr, 1991). Massage may assist with reducing pain and sensory hypersensitivity as well as with promoting a normal touch experience. Massage has also been shown to be beneficial in modulating state (White-Traut & Pate, 1987). Neonates with cocaine exposure who received 10 days of massages showed fewer stress behaviors and demonstrated more mature behaviors when tested with the NBAS than

did those in the control group (Wheeden et al., 1993). The mother who learns to massage her baby may gain confidence in both handling her infant and responding to the infant's body and cues. She may also be given a powerful method of calming her baby. Infants are more likely to attain a quiet alert state after massage (White-Traut & Nelson, 1988), thereby allowing the mother a chance for greater interaction. Thus, massage may be an initial way for mother and infant to experience pleasure with each other. Massage techniques, when combined with additional education for the mother and appropriate sensory experiences for the infant, have the potential for positively affecting mother–infant interaction and long-term outcomes for both.

Conclusion

Although much of the information currently available on prenatal cocaine exposure is at times contradictory or inconclusive, it does appear that there is potential for long-term effects of prenatal cocaine exposure, including a negative impact on the relationship formed between the mother and infant. Direct effects of cocaine exposure on the infant, such as neurological alterations and intrauterine growth retardation, may have lasting implications for the infant's development as well as the infant's ability to provide positive interactive experiences for the mother. Appropriate sensory experiences and techniques, such as pediatric massage, may assist the infant in gaining improved self-regulation, orientation, and interactive skills. However, the indirect effects of maternal cocaine use, such as poor postnatal environment and poor maternal responsiveness, may be more detrimental to the infant's outcome than the exposure itself. Therapists need to be aware of the possible difficulties faced by these dyads in order to observe interactions and intervene to promote a more positive relationship and outcome. Knowledge of normal infant development and state regulation as well as sensory processing abilities and play provide the therapist with the tools needed to assist mothers in interacting appropriately with their infants. ▲

Acknowledgment

I thank Jane Case-Smith, EdD, OTR, BCP, for assistance with this article.

References

Ainsworth, M. D. S. (1979). Infant mother attachment. *American Psychologist, 34,* 932–937.

Amaro, H., Fried, L. E., Cabral, H., & Zuckerman, B. (1990). Violence during pregnancy and substance use. *American Journal of Public Health, 80,* 575–579.

Angelilli, M. L., Fischer, H., Delaney-Black, V., Rubinstein, M., Ager, J. W., & Sokol, R. J. (1994). History of in utero cocaine exposure in language delayed children. *Clinical Pediatrics, 33,* 514–516.

Atkins, W. T. (1988). Cocaine: The drug of choice. In I. J. Chasnoff (Ed.), *Drugs, alcohol, pregnancy and parenting* (pp. 91–96). Dordrecht, Netherlands: Kluwer Academic.

Bandstra, E. S., & Burkett, G. (1991). Maternal–fetal and neonatal effects of in utero cocaine exposure. *Seminars in Perinatology, 15,* 288–301.

Barabach, L. M., Glazer, G., & Norris, S. C. (1992). Maternal perception and parent–infant interaction of vulnerable cocaine-exposed couplets. *Journal of Perinatal and Neonatal Nursing, 6*(3), 76–84.

Beckwith, L., Crawford, S., Moore, J. A., & Howard, J. (1995). Attentional and social functioning of preschool age children exposed to PCP and cocaine in utero. In M. Lewis & M. Bendersky (Eds.), *Mothers, babies, and cocaine: The role of toxins in development* (pp. 287–304). Hillsdale, NJ: Erlbaum.

Belsky, J., Bovine, M., & Taylor, D. G. (1984). The Pennsylvania Infant and Family Development Project III. The origins of individual differences in infant mother attachment: Maternal and infant contributions. *Journal of Child Development, 55,* 718–728.

Bender, S. L., Word, C. Q., DiClemente, R. J., Crittendon, M. R., Persaud, N. A., & Ponton, L. E. (1995). The developmental implications of prenatal and or postnatal crack cocaine exposure in preschool children: A preliminary report. *Developmental and Behavioral Pediatrics, 16,* 418–424.

Bendersky, M., Alessandri, S. M., Sullivan, M. W., & Lewis, M. (1995). Measuring the effects of prenatal cocaine exposure. In M. Lewis & M. Bendersky (Eds.), *Mothers, babies, and cocaine: The role of toxins in development* (pp. 163–178). Hillsdale, NJ: Erlbaum.

Blehar, M. C., Lieberman, A. F., & Ainsworth, M. D. (1977). Early face to face interaction and its relation to later infant–mother attachment. *Child Development, 48,* 182–194.

Breshnahan, K., Brooks, C., & Zuckerman, B. (1991). Prenatal cocaine use: Impact on infants and mothers. *Pediatric Nursing, 7,* 123–129.

Burns, W. J., & Burns, K. A. (1988). Parenting dysfunction in chemically dependent women. In I. J. Chasnoff (Ed.), *Drugs, alcohol, pregnancy and parenting* (pp. 159–171). Dordrecht, Netherlands: Kluwer Academic.

Cabrera, T. M., Levy, A. D., Li, Q., Van de Kar, L. D., & Battaglia, G. (1994). Cocaine induced deficits in ACTH and corticosterone responses in female rat progeny. *Brain Research Bulletin, 34*(2), 93–97.

Chasnoff, I. J., Griffith, D. R., Freier, C., & Murray, J. (1992). Cocaine/polydrug use in pregnancy: Two year follow up. *Pediatrics, 89*(2), 284–289.

Chasnoff, I. J., Griffith, D. R., MacGregor, S., Dirkes, K., & Burns, K. A. (1989). Temporal patterns of cocaine use in pregnancy. *Journal of the American Medical Association, 261,* 1741–1744.

Chasnoff, I. J., Lewis, D. E., Griffith, D. R., & Wiley, S. (1989). Cocaine and pregnancy: Clinical and toxicological implications for the neonate. *Clinical Chemistry, 35,* 1276–1278.

Chen, W. A., Anderson, K. H., & West, J. R. (1993). Cocaine exposure during the brain growth spurt: Studies of neonatal survival, somatic growth, and brain development. *Neurotoxicology and Teratology, 15,* 267–273.

Cherukuri, R., Minkoff, H., Feldman, J., Parekh, A., & Glass, L. (1988). A cohort study of alkaloidal cocaine ("crack") in pregnancy. *Obstetrics and Gynecology, 72*(2), 147–151.

Chiroboga, C. A., Bateman, D. A., Brust, J. C. M., & Hauser, W. A. (1993). Neurologic findings in neonates with intrauterine cocaine exposure. *Pediatric Neurology, 9*(2), 115–119.

Cohen, H. L., Sloves, J. H., Laugani, S., Glass, L., & DeMarinas, P. (1994). Neurosonographic findings in full term infants born to maternal cocaine abusers: Visualization of subependymal and periventricular cysts. *Journal of Clinical Ultrasound, 22,* 327–333.

Coles, C. D., Platzman, K. A., Smith, I., James, M., & Falek, A. (1992). Effects of cocaine and alcohol use in pregnancy on neonatal growth and neurobehavioral status. *Neurotoxicology and Teratology, 14,* 23–33.

Crockenburg, S. (1981). Infant irritability, mother responsiveness, and social support influences on infant–mother attachment. *Child Development, 52,* 857–865.

Davis, E., Fennoy, I., Laraque, D., Kanem, N., Brown, G., & Mitchell, J. (1992). Autism and developmental abnormalities in children with perinatal cocaine exposure. *Journal of the National Medical Association, 84,* 315–319.

Denham, S. A., & Moser, M. H. (1994). Mother's attachment to infants: Relations with infant temperament, stress, and responsive maternal behavior. *Early Child Development and Care, 98,* 1–6.

Dixon, S., & Bejar, K. (1989). Echoencephalographic findings in neonates associated with maternal cocaine and methamphetamine use: Incidence and clinical correlates. *Journal of Pediatrics, 115,* 770– 778.

Doberczak, T. M., Shanzer, S., Senie, R. T., & Kandali, S. R. (1988). Neonatal neurologic and electroencephalographic effects of intrauterine cocaine exposure. *Journal of Pediatrics, 113,* 354–358.

Dominguez, R., Villa-Coro, A. A., Slopis, J. M., & Bohan, T. P. (1991). Brain and ocular abnormalities in infants with in utero exposure to cocaine and other street drugs. *American Journal of Diseases in Children, 145,* 688–695.

Dow-Edwards, D. L., Freed, L. A., & Fico, T. A. (1990). Structural and functional effects of prenatal cocaine exposure in adult rat brain. *Developmental Brain Research, 57,* 263–268.

Drehobl, K. F., & Fuhr, M. G. (1991). *Pediatric massage.* Tucson, AZ: Therapy Skill Builders.

Dusick, A. M., Covert, R. F., Schreiber, M. D., Yee, G. T., Browne, S. P., Moore, C. M., & Tebbett, I. R. (1993). Risk of intracranial hemorrhage and other adverse outcomes after cocaine exposure in a cohort of 323 very low birth weight infants. *Journal of Pediatrics, 122,* 438–445.

Edmondson, R., & Smith, T. M. (1994). Temperament and behavior of infants prenarally exposed to drugs: Clinical implications for the mother–infant dyads. *Infant Mental Health Journal, 15,* 368–379.

Egeland, B., & Farber, E. (1984). Infant–morher attachment: Factors related to its development and changes over rime. *Child Development, 55,* 753–771.

Eisen, L. N., Field, T. M., Bandstra, E. S., Roberts, J. P., Morrow, C., Larson, S. K., & Steele, B. M. (1991). Perinatal cocaine effects on neonatal stress behavior and performance on the Brazelton scale. *Pediatrics, 88*(3), 477–479.

Frank, D. A., Breshnahan, K., & Zuckerman, B. (1993). Maternal cocaine use: Impact on child health and development. *Advances in Pediatrics, 40,* 65–99.

Frank, D. A., McCarten, K., Cabral, H., Levenson, S., & Zuckerman, B. (1994). Association of heavy in utero cocaine exposure with caudate hemorrhage in term newborns. *Pediatric Research, 35,* 269a.

Frassica, J. J., Orav, E. J., Walsh, E. P., & Lipshultz, S. E. (1994). Arrhythmias in children prenatally exposed to cocaine. *Archives of Pediatric and Adolescent Medicine, 148,* 1163–1169.

Freier, M. C., Griffirh, D. R., & Chasnoff, I. J. (1991). In utero drug exposure: Developmental follow up and maternal–infant interaction. *Seminars in Perinatology, 15,* 310–316.

Fulks, M. L., & Harris, S. R. (1995). Children exposed to drugs in utero: Their scores on the Miller Assessment for Preschoolers. *Canadian Journal of Occupational Therapy, 62,* 7–15.

Glantz, J. G., & Woods, J. R. (1993). Cocaine, heroine, and phencyclidine: Obsterric perspectives. *Clinical Obstetrics and Gynecology, 36,* 279–301.

Good, W., Ferriero, D. M., Golabi, M., & Kobori, J. A. (1992). Abnormalities of the visual system in infants exposed to cocaine. *Ophthalmology, 99,* 341–346.

Gottwald, S. R., & Thurman, S. K. (1994). The effects of prenatal cocaine exposure on mother–infant interaction and infant arousal in the newborn period. *Topics in Early Childhood Special Education, 14,* 217–231.

Griffith, D. R. (1988). The effects of perinatal cocaine exposure on infant neurobehavior and early maternal infant interaction. In I. J. Chasnoff (Ed.), *Drugs, alcohol, pregnancy and parenting* (pp. 105–113). Dordrecht, Netherlands: Kluwer Academic.

Griffith, D. R. (1989, February). Neurobehavioral effects of intrauterine cocaine exposure. *Ab Initio: An International Newsletter for Professionals Working With Infants and Their Families, 1*(1), 1–5.

Griffith, D. R., Azuma, S. D., & Chasnoff, I. J. (1994). Three year outcome of children exposed prenatally to drugs. *Journal of the American Academy of Child and Adolescent Psychiatry, 33*(1), 20–27.

Hadeed, A. J., & Siegel, S. R. (1989). Maternal cocaine use during pregnancy: Effect on the newborn infant. *Pediatrics, 84*(2), 205–210.

Henderson, M. E., & McMillan, B. A. (1990). Effects of prenatal exposure to cocaine or related drugs on rat developmental and neurological indices. *Brain Research Bulletin, 24*(2), 207–212.

Hoyme, H. E., Jones, K. L., Dixon, S. D., Jewitt, T., Hanson, J. W., Robinson, L. K., Msall, M. E., & Alanson, J. E. (1990). Prenatal cocaine exposure and fetal vascular disruption. *Pediatrics, 85*(5), 743–747.

Hume, R. F., Maj, M., O'Donnell, K. J., Stanger, C. L., Kilam, A. P., & Gingras, J. L. (1989). In utero cocaine exposure: Observations of fetal behavioral state may predict neonatal outcome. *American Journal of Obstetrics and Gynecology, 161,* 685–690.

Hurt, H., Brodsky, N. L., Betancourt, L., Braitman, L. E., Malmud, E., & Gianetta, J. (1995). Cocaine exposed children: Follow up through 30 months. *Journal of Developmental and Behavioral Pediatrics, 16,* 29–35.

Hutchings, D. E. (1993). The puzzle of cocaine's effects following maternal use during pregnancy: Are there reconcilable differences? *Neurotoxicology and Teratology, 15,* 281–286.

Hyde, A. S., & Trautman, S. E. (1989, December). Drug-exposed infants and sensory integration: Is there a connecrion? *Sensory Integration Special Interest Section Newsletter, 12*(4), 1–2, 6.

Isabella, R. A., Belsky, J., & von Eye, A. (1989). Origins of infant–mother attachment: An examination of interactional synchrony during the infant's first year. *Developmental Psychology, 25*(1), 12–19.

Keller, R. W., Maisonneuve, I. M., Nuccio, D. M., Carlson, J. N., & Glick, S. D. (1994). Effects of prenaral cocaine exposure on the nigrostriatal dopamine system: An in vivo microdialysis study in the rat. *Brain Research, 634,* 266–274.

Kelley, S. J. (1992). Parenting stress and child maltreatment in drug-exposed children. *Child Abuse and Neglect, 16,* 317–328.

King, T. A., Perlman, J. M., Laprook, A. R., Rollins, N., Jackson, G., & Little, B. (1995). Neurologic manifestations of in utero cocaine exposure in near term and rerm infants. *Pediatrics, 96*(2), 259–264.

Konkol, R. J., Murphey, L. J., Ferriero, D. M., Dempsey, D. A., & Olsen, G. D. (1994). Cocaine metabolites in the neonate: Potential for toxicity. *Journal of Child Neurology, 9,* 242–248.

Lauder, J. M. (1988). Neurotransmitters as morphogens. *Progress in Brain Research, 73,* 365–387.

Legido, A., Clancy, R. R., Spitzer, A. R., & Finnegan, L.P. (1992). Electroencephalographic and behavioral state studies in infants of cocaine-addicted mothers. *American Journal of Diseases of Children, 146,* 748–752.

Lenn, N. J. (1991). Neuroplasticity: The basis for brain development, learning, and recovery from injury. *Infants and Young Children, 3,* 39–48.

Lester, B. M., Corwin, M. J., Sepkoski, C., Seifer, R., Peucker, M., McLaughlin, S., & Golob, H. L. (1991). Neurobehavioral syndromes in cocaine-exposed newborn infants. *Child Development, 62,* 694–705.

Link, E. A., Weese-Mayer, D. W., & Byrd, S. E. (1991). Magnetic resonance imaging in infants exposed to cocaine prenatally: A preliminary report. *Clinical Pediatrics, 30,* 506-507.

Lyons, R., Alpern, L., & Repacholi, B. (1993). Disorganized infant attachment classification and maternal psychosocial problems as predictors of hostile–aggressive behavior in the preschool classroom. *Child Development, 64,* 572–585.

Malakoff, N. E., Mayes, L. C., & Schottenfield, R. S. (1994). Language abilities of preschool age children living with cocaine using mothers. *American Journal on Addictions, 3,* 346–354.

Mayes, L. C., Bornstein, M. H., Chawarska, K., & Granger, R. H. (1995). Information processing and developmental assessments in 3 month old infants exposed prenatally to cocaine. *Pediatrics, 95*(4), 539–545.

Mayes, L. C., Granger, R. H., Frank, M. A., Schottenfield, R., & Bornstein, M. H. (1993). Neurobehavioral profiles of neonates exposed to cocaine prenatally. *Pediatrics, 91*(4), 778–783.

Minabe, Y., Ashby, C. R., Heyser, C., Spear, L. P., & Wang, R. Y. (1992). The effects of prenatal cocaine exposure on spontaneously active midbrain dopamine neurons in adult male offspring: An electrophysiological study. *Brain Research, 586,* 152–155.

Molina, V. A., Wagner, J. M., & Spear, L. P. (1994). The behavioral response to stress is altered in adult rats exposed prenatally to cocaine. *Physiology and Behavior, 55,* 941–945.

Needlman, R., Zuckerman, B., Anderson, G., Mirochnick M., & Cohen, D. J. (1993). Cerebrospinal fluid monoamines precursors and metabolites in human neonates following in utero cocaine exposure: A preliminary study. *Pediatrics, 92*(1), 55–60.

Neerhof, M. G., MacGregor, S. N., Retsky, S. S., & Sullivan, T. P. (1989). Cocaine abuse during pregnancy: Peripartum prevalence and perinatal outcome. *American Journal of Obstetrics and Gynecology, 161,* 633–645.

Neuspiel, D. R. (1995). The problem of confounding in research in prenatal cocaine effects on behavior and development. In M. Lewis & M. Bendersky (Eds.), *Mothers, babies, and cocaine: The role of toxins in development* (pp. 95–110). Hillsdale, NJ: Erlbaum.

Neuspiel, D. R., Hamel, S. C., Hochberg, E., Green, J., & Campbell, D. (1991). Maternal cocaine use and infant behavior. *Neurotoxicology and Teratology, 13,* 229-233.

Nulman, I., Rovet, J., Altmann, D., Bradley, C., Einarson, T., & Koren, G. (1994). Neurodevelopment of adopted children exposed in utero to cocaine. *Canadian Medical Association Journal, 151,* 1591–1597.

Olson, S. L., Bates, J. E., & Bayles, K. (1984). Mother–infant interaction and the development of individual differences in children's cognitive competence. *Developmental Psychology, 20,* 166–179.

Pederson, D. R., Moran, G., Sitko, C., Campbell, K., Ghesquire, K., & Acton, H. (1990). Maternal sensitivity and the security of infant–mother attachment: A Q-sort study. *Child Development, 61,* 1974–1983.

Plessinger, M. A., & Woods, J. R. (1993). Maternal, placental, and fetal pathophysiology of cocaine exposure during pregnancy. *Clinical Obstetrics and Gynecology, 36,* 267–278.

Rodning, C., Beckwith, L., & Howard, J. (1989). Prenatal exposure to drugs: Behavioral distortions reflecting CNS impairment? *Neurotoxicology, 10,* 629–634.

Rotholz, D. A., Snyder, P., & Peters, G. (1995). A behavioral comparison of preschool children at high and low risk from prenatal drug exposure. *Education and Treatment of Children, 18*(1), 1–18.

Salamy, A., Dark, K., Salfi, M., Shah, S., & Peeke, H .V. S. (1992). Perinatal cocaine exposure and functional brainstem development in the rat. *Brain Research, 598,* 307–310.

Sallee, F. R., Katikaneni, L. P., McArthur, P. D., Ibrahim, H. M., Nesbitt, L., & Sethuraman, Q. (1995). Head growth in cocaine-exposed infants: Relationship to neonate hair level. *Journal of Developmental and Behavioral Pediatrics, 16,* 77–81.

Schneider, J. W. (1988). Motor assessment and parents education beyond the newborn period. In I. J. Chasnoff (Ed.), *Drugs, alcohol, pregnancy and parenting* (pp. 115–125). Dordrecht, Netherlands: Kluwer Academic.

Singer, L. T., Yamashita, T. S., Hawkins, S., Cairns, D., Baley, J., & Kliegman, R. (1994). Increased evidence of intraventricular hemorrhage and developmental delay in cocaine exposed, very low birth weight infants. *Journal of Pediatrics, 124,* 765–771.

Smith, R. F., Mattran, K. M., Kurkjian, M. F., & Kurtz, S. L. (1989). Alterations in offspring behavior induced by chronic prenatal cocaine dosing. *Neurotoxicology and Teratology, 11,* 35–38.

Spear, L. P., & Heyser, C. W. (1992). Cocaine and the developing nervous system: Laboratory findings. In I. S. Zagon & T. A. Slotkin (Eds.), *Maternal substance abuse and the developing nervous system* (pp. 155–175). San Diego, CA: Academic.

Spear, L. P., Kirtsein, C. L., Bell, J., Yootansumun, V., Greenbaum, R., O'Shea, J., Hoffman, H., & Spear, N. E. (1989). Effects of prenatal cocaine exposure on behavior during the early postnatal period. *Neurotoxicology and Teratology, 11,* 57–63.

Spraggins, Y. R., Seidler, F. J., & Slotkin, T. A. (1994). Cocaine exacerbates hypoxia induced cell damage in the developing brain: Effects of ornithine decarboxylase activity and protein synthesis. *Biology of the Neonate, 66,* 254–266.

Stallings-Sahler, S. (1993, September). Prenatal cocaine exposure and infant behavioral disorganization. *Sensory Integration Special Interest Section Newsletter, 16*(3), 1–4.

Suess, G. J., Grossman, K. E., & Stroufe, L. A. (1992). Effects of infant attachment to mother and father on quality of adaptation in preschool: From dyadic to individual organization of self. *International Journal of Behavioral Development, 15*(1), 43–65.

Swanwick, M. (1984). Attachment and social behavior. *Nursing (Oxford), 2,* 651–653.

van Baar, A., & de Graaff, B. M. (1994). Cognitive development at preschool age of infants of drug dependent mothers. *Developmental Medicine and Child Neurology, 36,* 1063–1075.

van den Boom, D. C. (1994). The influence of temperament and mothering on attachment and exploration: An experimental manipulation of sensitive responsiveness among lower-class mothers with irritable infants. *Child Development, 65,* 1457–1477.

van Ijzendoorn, M. H., Goldberg, S., Kroonenberg, P. M., & Frenkel, O. J. (1992). The relative effects of maternal and child problems on the quality of attachment: A meta analysis of attachment in clinical samples. *Child Development, 63,* 840-858.

Vorhees, C. V., Reed, T. M., Acuff-Smith, K. D., Schilling, M. A., Cappon, G. D., Fisher, J. E., & Pu, C. (1995). Long term learning deficits and changes in unlearned behaviors following in utero exposure to multiple daily doses of cocaine during different exposure periods and maternal plasma cocaine concentrations. *Neurotoxicology and Teratology, 17,* 253–264.

Weese-Mayer, D. E., Silvestri, J. M., Lin, D., Buhrfiend, C. M., Ptak, L. R., Lo, E. S., & Carvey, P. M. (1994). Hypoxia after prenatal cocaine exposure attenuates striatal dopamine and neurotrophic activity. *Neurotoxicology and Teratology, 16,* 177–181.

Wheeden, A., Scafidi, F. A., Field, T., Ironson, G., Valdeon, C., & Bandstra, E. (1993). Massage effects on cocaine-exposed preterm neonates. *Journal of Developmental and Behavioral Pediatrics, 14,* 318–322.

White-Traut, R. C., & Nelson, M. W. (1988). Maternally administered tactile, auditory, visual, and vestibular stimulation: Relationship to later interactions between mothers and premature infants. *Research in Nursing and Health, 11*(1), 31–39.

White-Traut, R. C., & Pate, C. M. H. (1987). Modulating infant state in premature infants. *Journal of Pediatric Nursing, 2*(2), 96–101.

Wood, R. D., Bannoura, M. D., & Johnson, I. B. (1994). Prenatal cocaine exposure: Effects on play behaviors in the juvenile rat. *Neurotoxicology and Teratology, 16,* 139–144.

Zuckerman, B. (1991, Spring). Drug-exposed infants: Understanding the medical risk. *The Future of Children,* 26–35.

Zuckerman, B., & Frank, D. (1992). Prenaral cocaine and marijuana exposure. In I. S. Zagon & T. A. Slotkin (Eds.), *Maternal substance abuse and the developing nervous system* (pp. 125–153). San Diego, CA: Academic.