"Crack Kids": Not Broken

Reports in the lay press based on anecdotal evidence have resulted in a rush to judgment about the impact of in utero exposure to illicit drugs, particularly cocaine, on the health, behavior, and development of America's children.¹ Children with a history of prenatal cocaine exposure, labeled "crack kids," are portrayed in the media as inevitably and permanently damaged. An article on Rolling Stone Magazine (October 18, 1990) stated that these babies are "like no others, brain damaged in ways yet unknown, oblivious to any affection." An article in the New York Times reported that "the parents and researchers say a vast majority of children exposed to significant amounts of drugs in the womb appeared to have suffered brain damage that cuts into their ability to make friends, know right from wrong, control their impulses, gain insight, concentrate on tasks, and feel and return love." Public response to these reports has crystallized into an outcry for the punishment of substance-using mothers and the disenfranchisement of their children as an unsalvageable, almost demonic, "biologic underclass" (Baltimore Sun. January 4, 1990). A university president recently quoted in the Boston Globe (April 30, 1991) suggested that health care resources were being squandered by "spending immense amounts on crack babies who won't ever achieve the intellectual development to have consciousness of God." Some clamor for substantially segregated educational programs to be established for children exposed prenatally to illicit drugs (New York Times. August 19, 1990).

These drastic public policy proposals have evolved in the absence of any credible scientific data regarding the sequelae of prenatal exposure to cocaine beyond the newborn period.¹ Moreover, this furor over prenatal exposure to cocaine obscures in the public mind any debate regarding society's responsibility for other conditions, such as lack of access to prenatal or pediatric care, malnutrition, measles, or lead poisoning, which jeopardize the development of many impoverished American children, whether substance-exposed or not.

To date evidence from the newborn period has been too inadequate and inconsistent to allow any clear predictions about the effects of prenatal exposure to cocaine on children's development and behavior.¹⁻³ The work of Chasnoff and colleagues⁴ in this issue of *Pediatrics* represents a useful preliminary effort to move the issue of long-term outcome of children exposed to cocaine, but not opiate, beyond the arena of anecdotal reports and unfounded speculation. This study constitutes an important advance over the only other published work describing outcome of children exposed to cocaine by Rodning and colleagues,⁵ where only 18 infants with polydrug exposure, including opiates and phencyclidine, with or without cocaine, were assessed by nonblind ex-

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aminers using different assessment tools than those used for the control infants. The Chasnoff study excludes opiate-exposed infants, considers marijuana, alcohol, and cigarette effects, includes an appropriate control group of the same social class, and uses a consistent well-standardized assessment tool administered by blind examiners. With this study design and in the context of comprehensive clinical interventions for mothers and children, the study shows no mean differences on the Bayley Scales of Infant Development at 2 years of age when cocaine-exposed children are compared with social class-matched controls. There is, however, a higher rate of scores more than 1 SD below the test mean for children exposed prenatally to cocaine.

Still, the results in this month's *Pediatrics* warrant cautious methodologic evaluation. As may be unavoidable in a substance-abusing population, there is a high attrition rate, higher in the substance-using than the nonsubstance-using groups. Only 27% of the 106 children recruited at birth remain in the cocaine-exposed sample at 24 months, compared with 62% of the 81 children in the nonexposed sample. It is also unclear how children were selected for developmental assessment, because growth outcomes are reported for 141 children at 2 years of age, but developmental outcomes for only 93. Such attrition and selective assessment may bias outcomes in either a positive or a negative direction. As the authors correctly point out, the most dysfunctional families may fail to return for follow-up, leading to bias toward retaining children with more favorable outcomes. On the other hand, families with obviously impaired children may cooperate more readily with developmental assessment, leading to an overestimation of the rates of impairment. Table 4 in Chasnoff et al suggests that the apparent increase in the rates of cocaine-exposed children with Mental Development Index scores 1 SD below the mean at 24 months of age may be an artifact of parents of impaired children being more readily compliant with follow-up developmental assessments. The absolute number of delayed children (3) is the same at 12, 18, and 24 months, but the denominator of all children assessed has decreased from 57 at 12 months to 29 at 24 months.

In spite of these limitations, these data support cautious optimism regarding the early developmental competence of infants with prenatal cocaine exposure. The real importance of this study is a demonstration of the potential value of intervention for the infants whose mothers remained in the program.

The newborn brain has a significant capacity for adaptation.^{6,7} The prenatal effects of drugs on the central nervous system may create biologic vulnerability. Potentially associated developmental dysfunction may be compensated partially or completely by the brain itself and/or by competent caretaking. However such biologic vulnerability may render a child more vulnerable to the effects of poor caretaking. In studies of other populations of children with potential central nervous system vulnerability, high family stability (*Scientific American.* 1989;106:111) and responsive caretaking⁸ protects against develop-

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mental impairment. Even among infants exposed to opiates in utero, the quality of the postnatal environment and not the amount of maternal opiate use appears to be a more important determinant of developmental outcome.⁹

Chasnoff's findings⁴ have a number of scientific and public policy implications. From a scientific point of view, it will be critical to replicate this study in larger samples, with intensive efforts to minimize attrition. Due to methodologic problems and overgeneralization from small samples, many of the initial observations about prenatal exposure to cocaine including associations with a high rate of Sudden Infant Death Syndrome, with congenital abnormalities, and with neonatal neurobehavioral dysfunction have not been confirmed consistently in subsequent studies.^{2,3} In addition, as the authors themselves emphasize, children at developmental risk should be assessed in a number of domains which may not be measured accurately by traditional psychometric testing. Due to cocaine's action of altering neurotransmitter levels and the preliminary evidence of early perturbation of neurotransmitters following in utero cocaine exposure,^{10,11} there is at least theoretical reason to be concerned about an exposed child's capacity for attention and self-regulation. Since these functions cannot be evaluated accurately in infants and young children, there is a potential to underestimate the possible effect of prenatal cocaine exposure unless children are observed to school age. However, given the traumatic life experiences and other high-risk health conditions of low-income children in general and of children of substance-abusing parents of any social class,^{2,12,13} it may be difficult to ascertain the relative contribution of prenatal exposure to cocaine and the postnatal environment to attentional or behavioral differences if any are found.

From a public policy point of view, this study should help place prenatal exposure to cocaine in perspective as a potential but not inevitable developmental insult. Prematurity provides a useful analogy. Prematurity, although a risk factor for impairment, in general does not result in the stigmatization of the exposed child in the eyes of the public or their teachers. Studies of premature or ill newborns fail to support biologic determinism. Supportive environments contribute significantly to the outcome of infants with biologic vulnerabilities at birth (Scientific American. 1989;106:111)^{8,14,15} In the study published in this issue of Pediatrics, even decreased head circumference, a plausible marker for biologic insult, shows a decreasing correlation with prenatal exposure as the children become older. Although decreased head circumference correlates with less optimal developmental outcome (Tables 8 and 9), Table 10 shows the effect of prenatal substance exposure on head circumference attenuates by 18 months suggesting that intervening environmental factors, such as nutrition, may play an important role in the relationship between head circumference and developmental outcome.

The relatively positive outcome of the children whose mothers remained in the research/intervention program underscores the importance of multifaceted interventions that are not limited to drug treatment and include support services that all families need. Availability of these multiple services is critical, but the coordination of service delivery is just as important. Consistent with the recommendation of the National Commission on Infant Mortality and with our own experience, the provision of services in a model of "one-stop shopping" may be most effective because keeping multiple appointments in different sites is difficult for all parents with young babies and especially for parents who use drugs. Providing key services such as pediatric health care, drug treatment, child development, and family planning at one location with one appointment system and the same staff has the potential to facilitate compliance with these services and improvement of the health and well being of both parents and children. We need programs for the whole child and the whole family.

Those who question the cost of these interventions only need to look at the cost of not providing them. A recent study shows that neonatal hospital costs until medically cleared for discharge were \$5200 more for cocaine-exposed than for unexposed infants. The costs of infants remaining in the nursery due to social evaluation or foster care placement further increased the cost by \$3500.¹⁶ If some of these dollars were spent for prenatal care coordinated with drug treatment and postnatally for other needed services, children and parents would benefit and money would be saved. What better investment?

Prenatal drug exposure is only one of multiple treatable or preventable biologic and social stressors experienced by children living in poverty. By focusing on cocaine and not on lack of adequate nutrition, health care, and education, we conveniently can blame mothers and not the conditions of poverty. Although the present study⁴ shows that with intervention children prenatally exposed to cocaine can do as well in their global development scores at age 2 years as their social class-matched peers, both groups function below national norms, reflecting the double jeopardy afflicting children living in poverty.¹² Poor children experience higher rates of prenatal exposure to cocaine compared with their nonpoor peers.^{17,18} However, whether substance-exposed or not, children living in poverty are more likely than their more advantaged peers to suffer from low birthweight, prematurity, malnutrition, anemia, pre- and postnatal lead poisoning, and congenital infections. For any given biologic risk factor such as lead exposure or congenital cytomegalovirus, low-income children experience more serious developmental consequences than children of higher socioeconomic status, underscoring the critical effect of environment in potentiating or ameliorating the impact of health insults.¹² It is a national disgrace that children living in poverty deteriorate developmentally by age 2 years, largely from our inability to provide a materially and psychologically supportive environment for all children.

Early health and education problems associated with poverty, whether or not children are exposed to drugs prenatally, are often chronic. Treatment needs to be continuous. There is no magic bullet for these

problems. Immunizations exist for diphtheria, tetanus, pertussis, measles, or rubella where a few shots will last a lifetime. We do not know how to inoculate against the effects of poverty. Diabetes is a better analogy; as long as children get their insulin daily, they will continue to do well. Children need nutrition, health care, safe environments, family support, and early childhood education daily. When you stop effective treatment or only provide partial treatment and the problem does not go away, it is incorrect to conclude that the treatment is not effective. Children who receive either educational benefits from Head Start or nutritional supplements from The Special Supplemental Food Program for Women, Infants, and Children (WIC) may be better off than children who receive neither, but certainly will not be as well off as children who receive both. Similarly, children who have medical care to treat lead poisoning or nutritional supplementation to treat malnutrition will not do as well as children who receive developmental enrichment in conjunction with these medical and nutritional benefits. If there are three problems, all of them need to be treated. If we only spend money to treat two problems, it should not be surprising that children are still ill or impaired. At the present time, even basic effective nutritional and early educational services such as WIC and Head Start are rationed to children and the majority of children who are eligible cannot receive them because of inadequate funding. In addition, eleven million American children do not have health insurance which exposes them to many preventable diseases. If we want to achieve the President's goal that all children enter school ready to learn by the year 2000, we have to provide the basic cost-effective services that support children's health and development and prevent impairment of the child's brain and soul. All children, whether drugexposed or not, deserve no less.

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ERRATUM

In the November issue of *Pediatrics*, a typographical error was overlooked in the Letter to the Editor, "Response to Pneumococcal Vaccine," by Gerald Schiffman (*Pediatrics* 1991;88:1074–1075). The last paragraph of the Letter should begin with: "I *do* agree with the authors that a level of 200 ng of antibody N/mL after immunization is hyporesponsive if, by that term, they mean not producing sufficient antibody for protection."