Crack in America

Demon Drugs and Social Justice

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As a consequence, researchers have been unable to determine the magnitude of cocaine's impact on pregnancy—or, indeed, whether cocaine has an independent impact at all.

Had this research been published at any other time, it might have gone unnoticed outside the scientific community. However, its appearance in the late 1980s—at the height of the crack scare—practically guaranteed the attention of the popular press. In fact, although the studies themselves generally made no mention of the route through which pregnant women had consumed cocaine, journalists almost uniformly identified crack as the drug causing extensive fetal harm. By ignoring the methodological limitations in the scientific research, they presented preliminary data as fact.

THE CRACK BABY: A MEDIA-CREATED CRISIS

Virtually every adverse outcome found in every fetal study involving cocaine—whether the subjects were humans or rats—was reported in the mass media as evidence that crack causes damage in babies. Journalists described “crack babies” as permanently impaired—physically, intellectually, and emotionally. Some of these babies, it was claimed, so lacked “normal human feelings” and “impulse control” that, as they matured, they were certain to pose a danger to others. Continually, Americans were told about the financial cost to taxpayers of the growing number of crack babies—many of whom would need extensive medical treatment, special education, and long-term institutional care. Estimates of the magnitude of the “crisis” varied, but the media often quoted a Department of Health and Human Services report predicting one hundred thousand crack-damaged babies per year, at an annual cost to society of about $20 billion (Kusserow, 1990).

Journalists also continually portrayed crack babies as having been born “addicted” to cocaine. For example, one television news broadcast depicted a tiny African-American baby in an incubator waving his arms in apparently futile gestures as a voice-over described the horror of watching such babies “craving cocaine.” In numerous magazine and newspaper articles as well, journalists described “tiny addicts” who were “poisoned in the womb” and then forced, at birth, into a “world of nightmarish withdrawal.”

Of all the drug horror stories ever told, perhaps none has provoked as much public concern as that of the crack baby. In response, various remedial programs were implemented, particularly in the public schools, with the goal of helping crack babies compensate for their handicaps (Chira, 1990; Toufexis, 1991), but more commonly, a punitive approach has been taken. For example, hospitals now regularly test the urine of babies whose mothers they suspect of having used drugs, and babies are often taken away on the basis of a positive drug test alone (Siegel, 1991). In some parts of the country, women are prosecuted and imprisoned for using drugs
during pregnancy (see Chapter 12 of this volume; Paltrow, 1992; Siegel, 1991), and state legislatures are searching for new ways to control pregnant drug users—for example, laws that would force them, once detected, to choose between drug treatment and sterilization (Berrien, 1990; Chavkin, 1991). A recent survey of college students found widespread support for such policies—particularly when the drug being used by pregnant women was cocaine (Vener et al., 1992)—and probably most Americans would agree. Indeed, among defenders of drug prohibition, the goal of “saving crack babies” is now often offered as the primary justification for escalating the entire War on Drugs.  

The “crack baby” on which drug policy is increasingly based does not exist. Crack babies are like Max Headroom and reincarnations of Elvis—a media creation. Cocaine does not produce physical dependence, and babies exposed to it prenatally do not exhibit symptoms of drug withdrawal. Other symptoms of drug dependence—such as “craving” and “compulsion”—cannot be detected in babies. In fact, without knowing that cocaine was used by their mothers, clinicians cannot distinguish so-called crack-addicted babies from babies born to comparable mothers who had never used cocaine or crack (Hadeed and Siegel, 1989; Parker et al., 1990).

In the scientific literature itself, the issue of fetal damage related to cocaine is more complicated, but journalists have blatantly misrepresented that literature by reporting only studies that found evidence of harm and then minimizing, if not ignoring, the limitations in their research design. The mass media have consistently portrayed crack as a direct cause of adverse pregnancy outcomes even though no study has convincingly shown that to be so. In fact, there is now evidence that cocaine actually contributes little to the abnormalities detected in the babies of women who use cocaine during pregnancy.

A number of people have criticized the cocaine and pregnancy studies, pointing out how biased sample selection and the lack of control over other variables prevent their being used as evidence that cocaine causes fetal harm (Alexander, 1990; Kandall, 1991; Mayes, 1992; Mayes et al., 1992; Neuspiel and Hamel, 1991). In addition, the few studies that have monitored cocaine-exposed babies during the first few years of life have found that the differences detected at birth almost disappear by age two (Chasnoff et al., 1992; Graham et al., 1992). However, the study we find most persuasive was done by a group of Canadian researchers who combined data from the twenty best-designed studies published prior to 1989 and performed a “meta-analysis” that challenges most of their findings (Lutiger et al., 1991). A meta-analysis is particularly useful when the results of similarly designed studies are inconsistent, as they are in this case. It also reduces the impact of selection bias, increases control over potentially
confounding variables, and eliminates some of the problems of small sample size—thus permitting the use of more sophisticated statistical measures.

After combining the data from both drug users and controls, Lutiger et al. compared the reproductive risks associated with (1) polydrug use, including cocaine; (2) polydrug use, excluding cocaine; (3) cocaine use only; and (4) no drug use. Analyzing the data as a whole, they discovered that most of the fetal effects associated with cocaine disappeared. They did find significant differences between the offspring of women who had used drugs during pregnancy and those who had not—but both the type and rate of fetal abnormalities were similar regardless of the drugs consumed.

This latter finding is important because it calls into question the alleged harmful consequences of cocaine’s vasoconstrictive impact on the umbilical cord and placenta. In sufficient doses, cocaine probably does restrict the flow of blood from mother to fetus, but because infants exposed prenatally to cocaine tend to be indistinguishable from those exposed to drugs that do not cause vasoconstriction, we cannot conclude that cocaine’s slowing of the blood flow compromises fetal development. In fact, there is evidence that, in response to cocaine’s presence, receptors in the placenta “down-regulate” fairly quickly, reducing vasoconstriction even before serum levels decline substantially—thus shortening the period of time in which blood from the mother is restricted (Wang and Schnoll, 1987).

We still know almost nothing about cocaine’s interaction with the fetal brain, although the incidence of cardiovascular and central nervous system damage seems to be quite low (Neuspiel and Hamel, 1991). It has been suggested that the fetal neural system is more sensitive than that of adults and therefore more easily damaged by cocaine. But it is just as possible that the opposite is true. We know that fetal anatomy and function differ from those of adults—so much so that inferences about a drug’s fetal effects can never be made on the basis of detected effects in adults (Miller and Kellogg, 1985; Rudolph, 1985; Wang et al., 1985). Some drugs are less harmful to fetuses than adults and some are more harmful; however, overall, human fetuses have proven to be remarkably resistant to the drugs consumed by their mothers (Alexander et al., 1985).

Given the recent increases in cocaine use and our failure to persuade some pregnant women not to take it, it is fortunate that the evidence to date does not suggest that cocaine is among the drugs that are particularly damaging to the fetus. This does not mean that cocaine use by pregnant women poses no risk. However, it is now clear that the high rate of abnormalities found in babies exposed prenatally to cocaine has less to do with the pharmacological effects of the drug than with other factors of
high-risk pregnancy that “cluster” in drug users—particularly impoverished drug users who more often have poor diets and no prenatal care and who are more frequent victims of violence against women and other crimes.\textsuperscript{44}

The route through which cocaine is administered probably makes little difference,\textsuperscript{45} although the greater use of crack by the inner-city poor means that crack users are more likely than powder cocaine users to have unhealthy babies. In addition, impoverished drug users are more likely than their wealthier counterparts to be enmeshed in a deviant lifestyle that carries with it many additional pregnancy risks. This association between crack use and adverse pregnancy outcomes will continue to exist as long as poor women are overrepresented among crack users and as long as socioeconomic status remains a critical determinant of many non-drug-related pregnancy risks. Again, there is no evidence that crack is a direct cause of fetal harm, so reductions in crack use will not lead automatically to a reduction in the number of unhealthy babies being born.

CONCLUSION

Popular beliefs and attitudes about cocaine and crack have been shaped by journalists. Because the media are businesses seeking ever-larger markets of readers and audiences, they generally frame stories in ways that resonate with the sympathies and antipathies that make up conventional wisdom regarding drugs. In this sense, the crack story is simply the most recent installment in a series of morality tales that simultaneously construct and confirm Americans’ belief in the power of drugs to disinhibit and harm users. However, there is something new—or at least refined—in crack journalism: the emergence of a group of “drug experts” who use pharmacological language and concepts to support existing drug myths while ignoring pharmacological principles and evidence that challenge those myths. Some of the articles published in drug abuse and medical journals appear scientific but are not because the taken-for-granted premise of their authors—like that of most journalists—is simply that any crack use is highly destructive.

Our review of the available literature indicates that most of the claims that have been made about crack’s hazards are either exaggerated or unfounded. In both powder and crack form, cocaine can be toxic, especially when consumed in large doses, and even small doses may produce harm in some users. However, most users experience no serious adverse health consequences related to their use. Cocaine also appears to be weak as a fetal toxin, and no physical or developmental abnormalities in infants can be attributed causally and specifically to maternal use of cocaine or crack. In both fetuses and adults, the relatively large safety margin associated with
cocaine is probably linked to humans' extensive homeostatic responses to stimulant drugs—protective mechanisms confirmed by pharmacological science but rarely even mentioned by those interested in publicizing cocaine's harms.

Cocaine does not produce physical dependence, and babies are not born addicted to this drug. Numerous studies have shown that laboratory animals can be manipulated to self-administer cocaine repeatedly, but such studies provide very little insight into cocaine's addictive potential in humans. Among humans, cocaine addiction is relatively rare as a proportion of the total number of people who have tried it, regardless of the form in which the drug is employed. Early claims that smokeable cocaine caused instant addiction were clearly wrong. In fact, there is no evidence that the rapid onset/rapid decline of effect associated with smoking makes addiction or even escalated use inevitable. As Reinerman et al. suggest in Chapter 4, smoking may increase the likelihood that cocaine users will engage in bingeing. But it may also turn out that the problems associated with such bingeing may move crack users—"drug dependent" or not—more quickly toward quitting or curtailing their use. Because the excessive use of a drug over a short period of time is likely to cause more individual and social dysfunction than moderate use over a long period of time, the tendency of crack users to binge means that crack can be viewed as more risky than powder cocaine. However, it is important keep in mind that many crack users take the drug occasionally, do not engage in prolonged binges, and do not become dysfunctional.

We have argued that the route of cocaine administration matters less than the public has been led to believe—a conclusion based on comparing smoking and sniffing, the two modes of ingestion most prevalent in American society. The practice of swallowing cocaine, although not free from abuse potential, almost certainly provides users with a substantially wider safety margin. Of course, swallowing is also a more "inefficient" way to consume a drug, and under a system of drug prohibition, such milder (and more "expensive") modes of ingestion tend to disappear. In this sense, the emergence of crack is part of a general trend that has been operating since cocaine prohibition was put into place early in the twentieth century. Fortunately, this more efficient mode of ingesting cocaine has not dramatically increased the risks associated with its use. Although there are risks involved in using crack, they have been consistently exaggerated. As the other chapters in Part I of this book demonstrate, most of the problems associated with crack are products of the social context in which it arose and is used, not its pharmacological powers or "efficient" route of administration.