

DRUG WAR FACTS

Substance Use and Pregnancy

1. Criminalizing substance abuse during pregnancy discourages substance-using or abusing women from seeking prenatal care, drug treatment, and other social services, and sometimes leads to unnecessary abortions.

Sources: Cole, H.M., "Legal Interventions during Pregnancy: Court-Ordered Medical Treatment and Legal Penalties for Potentially Harmful Behavior by Pregnant Women," Journal of the American Medical Association, 264: 2663-2670 (1990); Polan, M.L., Dombrowski, M.P., Ager, J.W., & Sokol, R.J., "Punishing Pregnant Drug Users: Enhancing the Flight from Care," Drug and Alcohol Dependence, 31: 199-203 (1993); Koren, G., Gladstone, D. Robeson, C. & Robieux, I., "The Perception of Teratogenic Risk of Cocaine," Teratology, 46: 567-571 (1992).

2. "Our study found significant cognitive deficits with cocaine-exposed children twice as likely to have significant delay throughout the first 2 years of life. The 13.7% rate of mental retardation is 4.89 times higher than that expected in the population at large, and the percentage of children with mild or greater delays requiring intervention was 38%, almost double the rate of the high-risk noncocaine- but polydrug-exposed comparison group. Because 2-year Mental Development Index scores are predictive of later cognitive outcomes, it is possible that

these children will continue to have learning difficulties at school age. "Cognitive delays could not be attributed to exposure to other drugs or to a large number of potentially confounding variables. Further, poorer cognitive outcomes were related to higher amounts of cocaine metabolites in infant meconium as well as to maternal self-reported measures of amount and frequency of cocaine use during pregnancy, providing further support for a teratologic model."

...

"Some limitations to this study should be considered. Although examiners were masked to infant drug status, it may have been possible to identify drug exposure through maternal or caregiver characteristics, since all children were assessed with the caregiver present. The sample was also recruited according to hospital screening measures and reflects outcomes only of more heavily exposed infants. Also, the drug assessments were made retrospectively, making reliability of maternal report problematic."

Source: Singer, Lynn T., PhD, Robert Arendt, PhD, Sonia Minnes, PhD, Kathleen Farkas, PhD, Ann Salvator, MS, H. Lester Kirchner, PhD, Robert Kliegman, MD, "Cognitive and Motor Outcomes of Cocaine- Exposed Infants," Journal of the American Medical Association, April 17, 2002, Vol. 287, No. 15, pp. 1957-1959.

3. Research paid for by the National Institute on Drug Abuse (NIDA) and the Albert Einstein Medical Center in Philadelphia states, "Although numerous animal experiments and some human data show potent effects of cocaine on the central nervous system, we were unable to detect any difference in Performance, Verbal or Full Scale IQ scores between cocaine-exposed and control children at age 4 years."

Source: Hallam Hurt, MD; Elsa Malmud, PhD; Laura Betancourt; Leonard E. Braitman, PhD; Nancy L. Brodsky, PhD; Joan Giannetta, "Children with In Utero Cocaine Exposure Do Not Differ from Control Subjects on Intelligence Testing," Archives of

Pediatrics & Adolescent Medicine, Vol. 151: 1237-1241 (1997), American Medical Association.

4. Well-controlled studies find minimal or no increased risk of Sudden Infant Death Syndrome (SIDS) among cocaine-exposed infants.

Sources: Bauchner, H., Zuckerman, B., McClain, M., Frank, D., Fried, L.E., & Kayne, H., "Risk of Sudden Infant Death Syndrome among Infants with In Utero Exposure to Cocaine," Journal of Pediatrics, 113: 831-834 (1988), (Note: Early studies reporting increased risk of SIDS did not control for socioeconomic characteristics and other unhealthy behaviors. See, e.g., Chasnoff, I.J., Hunt, C., & Kletter, R., et al., "Increased Risk of SIDS and Respiratory Pattern Abnormalities in Cocaine-Exposed Infants, "Pediatric Research, 20: 425A (1986); Riley, J.G., Brodsky, N.L. & Porat, R., "Risk for SIDS in Infants with In Utero Cocaine Exposure: a Prospective Study," Pediatric Research, 23: 454A (1988)).

5. Among the general population there has been no detectable increase in birth defects which may be associated with cocaine use during pregnancy.

Source: Martin, M.L., Khoury, M.J., Cordero, J.F. & Waters, G.D., "Trends in Rates of Multiple Vascular Disruption Defects, Atlanta, 1968-1989: Is There Evidence of a Cocaine Teratogenic Epidemic?" Teratology, 45:647-653 (1992).

6. The lack of quality prenatal care is associated with undesirable effects often attributed to cocaine exposure: prematurity, low birth weight, and fetal or infant death.

Sources: Klein, L., & Goldenberg, R.L., "Prenatal Care and its Effect on Pre-Term Birth and Low Birth Weight," in Merkatz, I.R.

& Thompson, J.E. (eds.), New Perspectives on Prenatal Care (New York, NY: Elsevier, 1990), pp. 511-513; MacGregor, S.N., Keith, L.G., Bachicha, J.A. & Chasnoff, I.J., "Cocaine Abuse during Pregnancy: Correlation between Prenatal Care and Perinatal Outcome," Obstetrics and Gynecology, 74:882-885 (1989).

7. Provision of quality prenatal care to heavy cocaine users (with or without drug treatment) has been shown to significantly improve fetal health and development.

Source: Chazotte, C., Youchah, J., & Freda, M.C., "Cocaine Use during Pregnancy and Low Birth Weight: The Impact of Prenatal Care and Drug Treatment," Seminars in Perinatology, 19: 293-300 (1995).

8. Presented with children randomly labeled "prenatally cocaine-exposed" and "normal," childcare professionals ranked the performance of the "prenatally cocaine-exposed" children below that of "normal," despite actual performance.

Source: Thurman, S.K., Brobeil, R.A., Duccette, J.P., & Hurt, H., "Prenatally Exposed to Cocaine: Does the Label Matter?" Journal of Early Intervention, 18: 119-130 (1994).

9. According to a study published by the Journal of the American Medical Association in 2002, "Consistent with previous studies, we found that maternal cigarette smoking was associated with reduced birth weight and an increased risk of LBW, shortened gestation and an increased risk of preterm birth, and intrauterine growth restriction. Our data indicate that maternal cigarette smoking likely affects infant birth weight via both reduced fetal growth and shortened gestation."

Source: Wang, Xiaobin, MD, MPH, ScD, Barry Zuckerman, MD, et al., "Maternal Cigarette Smoking, Metabolic Gene

Polymorphism, and Infant Birth Weight," Journal of the American Medical Association (Chicago, IL: American Medical Association, January 9, 2002), Vol. 287, No. 2, p. 200.

10. According to a study published by the Journal of the American Medical Association in 2002, "For the ever smokers, the mean birth weight was 280 g lower (95% confidence interval [CI], -413 to -147) and the odds ratio (OR) for LBW was higher (OR, 1.8; 95% CI, 1.3-2.7) compared with the never smokers. The mean gestational age for ever smokers was 0.8 weeks shorter (95% CI, -1.3 to -0.2) and the OR of preterm birth was higher (OR, 1.8; 95% CI, 1.3-2.7)."

Source: Wang, Xiaobin, MD, MPH, ScD, Barry Zuckerman, MD, et al., "Maternal Cigarette Smoking, Metabolic Gene Polymorphism, and Infant Birth Weight," Journal of the American Medical Association (Chicago, IL: American Medical Association, January 9, 2002), Vol. 287, No. 2, p. 198.

11. According to a study published by the Journal of the American Medical Association in 2002, "As shown in Table 2, without consideration of genotype, continuous maternal smoking during pregnancy was associated with an OR of 2.1 (95% CI, 1.2-3.7) for LBW and a mean reduction of 377 g (SE, 89 g) in birth weight compared with the never smokers."

Source: Wang, Xiaobin, MD, MPH, ScD, Barry Zuckerman, MD, et al., "Maternal Cigarette Smoking, Metabolic Gene Polymorphism, and Infant Birth Weight," Journal of the American Medical Association (Chicago, IL: American Medical Association, January 9, 2002), Vol. 287, No. 2, p. 198.

12. According to a study published by the British Medical Journal in 2002, "In utero exposures due to smoking during pregnancy may increase the risk of both diabetes and obesity through programming, resulting in

lifelong metabolic dysregulation, possibly due to fetal malnutrition or toxicity. The odds ratios for obesity without type 2 diabetes are more modest than those for diabetes and the scope for confounding may be greater. Smoking during pregnancy may represent another important determinant of metabolic dysregulation and type 2 diabetes in offspring. Smoking during pregnancy should always be strongly discouraged."

Source: Montgomery, Scott M., and Anders Ekborn, "Smoking During Pregnancy and Diabetes Mellitus In a British Longitudinal Birth Cohort," British Medical Journal (London, England: British Medical Association, January 5, 2002), Vol. 321, p. 27.

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