

Ray Peat's Newsletter

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"Cocaine babies," criminal responsibility, and medicine

In the 1950s and later, the US medical establishment was still arguing for the safety of prenatal x-rays.

Through the 1950s and 1960s the U.S Atomic Energy Commission and the medical profession collaborated in keeping the public from knowing the dangers of prenatal exposures to harmful influences, including drugs, diet, and radiation. Childhood cancer and brain defects were produced in millions of children as a result. Fetotoxic drugs are still in use by physicians.

The problem is, partly, that medicine is not a biological science. It is a profession, meaning that whatever it professes to know has the legal status of truth.

Problem-centered ecological health education would be a step toward solution of the problems of public health.

Many pregnant women have been prosecuted for "delivering an illegal drug to their fetus" when they use cocaine.

If a person chooses to believe something that's contrary to the best evidence, when they are aware of the evidence, that can be called faith. But when their behavior harms others, their willful ignorance becomes a moral issue.

When the medical profession starts to get something right, a century after researchers had clarified the facts, the general euphoria should be tempered by what is actually happening. The medical profession is now concerned about the harmful effects of using cocaine and alcohol in pregnancy. Exactly what does this new seemingly realistic attitude mean? What could motivate this great new interest in the well-being of the fetus?

Why are these substances the focus of such concern? I'm afraid it is purely because these are all self-administered substances, and the blame can be placed on the user, rather than on any powerful institutions.

Just 30 years ago, the conventional wisdom was that the fetus is isolated and insulated from

"The legal responses to the "crack baby scare" clearly did much more harm than good to both the mothers and the children. Making substance abuse during pregnancy a crime kept mothers from prenatal medical care, thereby endangering the fetus far more than would be the case with drug use, and discouraged them from seeking drug treatment."

". . . the fact remains that recent studies on cocaine use by pregnant women clearly demonstrate that the pharmacological impact of cocaine has been greatly exaggerated, that many other factors impact on fetal and newborn development, and that legal responses to maternal cocaine use have made every aspect of the problem worse."

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the mother's metabolism. The thalidomide episode was already forcing doctors to acknowledge that *some* drugs might get through the placenta, though the drug industry was reluctant to lose the pregnant-woman market.

The idea that fetal damage could result from the mother's malnutrition was rejected as an impossibility. The food industry didn't want to have to test their GRAS (generally recognized as safe) additives for fetal safety, and it didn't want women to get the idea that they should avoid junk foods, such as pasta and bread, during pregnancy. People at the Harvard School of Public Health said that it was nonsense to think that foods could

be nutrient-deficient--"if the soil doesn't have the chemicals plants need, the plants won't grow; therefore, all crops are fully nutritious." The chemical composition of plants was supposedly determined strictly by their genes, so the soil couldn't determine their nutrient content. The fetus, like a plant, was supposed to be genetically determined, but unlike a plant, it was said to live like a parasite on the mother, taking everything it needs from the mother's tissues, rather than from the mother's dietetic intake. If the mother was alive, her nutritional condition couldn't have anything to do with the fetus.

Probably in reaction against the biological evidence that maternal malnutrition produced defective offspring, the American medical establishment adopted the concept that "fetal malnutrition" existed, and was caused by a defective uterus or placenta, not by anything the mother might have eaten or failed to eat. In Italy and Germany in the 1970s, a slightly saner concept was emerging, in which it was proposed that a malnourished fetus could be fed by infusing amino acids into the amniotic fluid, or that the mother and fetus might benefit from intravenous glucose and amino acids. The use of progestins to stimulate placental development was also suggested in Europe. (Farad and Picas, 1976.)

Poor, illiterate, drug using women are now being charged with "giving cocaine to their fetus" when they use the drug. What this means is that there are elements in our judicial system which are saying that illiterates and derelicts must be held to a higher standard than doctors of medicine were when these women were children--and to a higher standard than that of the present state licensing boards which have nothing to say to physicians who prescribe reducing diets and diuretics and teratogenic drugs, and even x-rays, to pregnant women.

Several articles on cocaine babies have emphasized the huge cost resulting from their health care. I have never seen a discussion of the economics of the damage caused by medical and governmental aggressions against the public. If the cost to the public of caring for cocaine babies is the important issue, then we should consider the cost of preventing the damage in the most direct

way, but that requires knowing what the real causes of the damage are.

The evidence that these babies' defects are caused by cocaine is fairly odd. Being scientifically careful about evaluating the evidence in this situation is important, considering the history and the context.

In the late 1960s, most of the publications on the dangers of LSD and marihuana were fraudulent. While typical LSD users took about 100 micrograms of the drug, experiments using doses that were the human equivalent of tens of thousands of micrograms were used to argue for the drug's dangers. Those doses would have killed people, in a short time.

In humans, as little as 20 milligrams of cocaine injected intravenously is said to have been fatal, and the average lethal *oral* dose is 500 milligrams. Injected cocaine is much more powerful than oral doses. **In animal studies, doses equivalent to thousands of milligrams per day in humans are injected into rats before harmful effects are seen in the fetuses.** Drugs injected intravenously or injected into the peritoneal cavity are very rapidly and completely absorbed, and the only doses that caused measurable fetal harm in rats were doses that, on a weight basis, almost certainly would have caused nearly instantaneous death in humans.

Cocaine suppresses appetite. When pregnant animals are fed the same limited amount of food consumed by cocaine-treated animals, their undernourished offspring show most of the same effects seen in the litters that were exposed to cocaine.

The human placenta passes a smaller percentage of cocaine to the fetus than the rat placenta does, and the liver of the human fetus is more effective at detoxifying cocaine, so any effects of cocaine on rat fetuses are probably going to be larger than any effect on the human fetus.

Behavioral disturbances in the offspring are considered to be a very sensitive indicator of a chemical's teratogenicity, but some researchers have concluded that cocaine might not be a behavioral teratogen. (Riley and LaFiette, 1996) Others have described it as "not a potent neuroteratogen." (Chen, et al., 1996)

The effects of cocaine that are supposedly seen in humans seem not to be produced by cocaine in lab animals. Intrauterine growth retardation, microencephaly, abruptio placentae, spontaneous abortion, sudden infant death syndrome, and mental retardation are commonly said to be the consequences of prenatal exposure to cocaine. Many things can contribute to those problems, but malnutrition is the most common cause, and is the one that American medicine goes to great lengths to avoid mentioning. Prenatal x-rays, diuretics, and pregnancy weight-control diets are other important causes.

How is it that cocaine and starvation can produce the same symptoms? I think the evidence indicates that cocaine isn't the cause of the pregnancy problems that are blamed on it. But, since cocaine suppresses the appetite, it does have some role in the problems of gestation caused by malnutrition.

Most crack smokers are poor, very few eat well, and nearly all of them smoke. Smoking causes damage to the fetus that is much easier to demonstrate than the theoretical damage caused by cocaine. The media are filled with the cocaine-baby issue. "Have you ever seen a cocaine baby?" But do we hear them asking "Have you ever seen a baby born to a smoker?" Or, "have you seen the babies of malnourished women?"

To pretend that any effects produced in pregnant rats, by a dose of cocaine which is many times larger on a weight basis than the supposedly lethal human dose, are relevant to public health, is crude propaganda, and presumably has the purpose of supporting political, judicial or military actions that are completely unrelated to public health or morality.

Putting the medico-legal propaganda in its best light, it is scientifically atrocious. But the actual moral issue is that it is not used for any constructive purpose. One nutritious high protein meal every day would probably solve the "cocaine baby problem," but it appears that neither the government nor the medical establishment is interested in solving the problem.

Novocaine (procaine) was introduced as a substitute for cocaine, but it has been found to have protective effects on the organism, as an

adaptogen. Lidocaine and other local anesthetics have many important protective effects. In this context, some experiments that hint at a fetus-*protecting* effect of cocaine deserve further investigation.

Cocaine was displaced from medical use, first by the proprietary synthetic local anesthetics, and then, in the 1930s, by the proprietary amphetamines.

As a stimulant, cocaine increases the metabolic rate, which in turn can lead to the wastage of protein, to provide energy. Conventional first aid for a cocaine overdose includes providing glucose and thiamine, to provide energy. The same nutritional support, but preferably with additional protein, is obviously appropriate for protection of the fetus. The presence of increased amounts of ammonia in the urine indicates malnutrition in both adults and newborns, and might be a useful indicator for nutritionally stressed pregnancies. Supplementing both dietary protein and carbohydrate might be the simplest treatment for many types of gestational stress, including preeclampsia and "recreational" cocaine use.

The Andean people who use coca regularly appear to suffer no harmful effects. Coca chewing prevents the hypoglycemia that can be produced by extremely high altitude. It is common to keep a wad of the leaf in the mouth all day. The dental health of coca-chewers is so good that a Bolivian company has marketed a toothpaste containing coca leaf. (But it is probably the high altitude itself that is mainly responsible for their good teeth.) The coca leaf has been used medicinally in South America for improved fertility, prolongation of virility into old age, prevention of altitude sickness, and various other purposes. The association of coca chewing in these cultures with magic and gods with erect phalluses (*El Tio*, *Supay*) probably affected the herb's treatment by the anti-erotic cultures.

At very high altitude, fertility is limited by the availability of oxygen. Altitude itself tends to increase the ratio of progesterone to estrogen, as a protective adaptation involving increased carbon dioxide in the tissues. Cocaine increases the production of the pituitary gonadotrophin, LH

(luteinizing hormone), which increases ovarian progesterone production. In men, LH increases testosterone production. Chewing coca leaves has been found to sharply increase the amount of progesterone in saliva. Increased progesterone would increase the ability to maintain pregnancy under stressful conditions.

Used moderately, for health purposes, there would seem to be no problem with cocaine, and there seems to be a physiological rationale even for its use during pregnancy under some conditions. The use of progesterone, thyroid, and nutrition would obviously be better ways to protect fetal development.

Smoking during pregnancy is a much bigger public health issue than cocaine use. Ranking with smoking, in terms of damage done to developing babies, are prescribed diuretics, inappropriate drugs for epilepsy during pregnancy, weight-control diets for pregnant women, x-rays of the developing fetus, and--probably the biggest problem of all--poverty and malnutrition.

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unchanged in midluteal females. Male testosterone increased by 50% above average base-line levels 50 min after the LH peak (80 min postcocaine). These data are consistent with our previous findings that cocaine increased LH and enhanced luteinizing hormone-releasing hormone-stimulated LH in early follicular females."

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hemorrhages. Despite suppression of maternal weight gain, **there was preservation of fetal weights at cocaine doses up to and including 80 mg/kg/day, suggesting some protection of fetal growth.**"

Am J Perinatol 1989 Jan;6(1):4-7. **Increased neonatal urinary ammonia: a marker for in utero caloric deprivation?** Wolfe HM, Sokol RJ, Dombrowski MP, Bottoms SF, Norman GS. **The decline in the urinary urea to ammonia ratio represents a simple measure of nutritional status in the adult.** We examined the relationship of this ratio to nutrient-related fetal growth retardation. Levels of ammonia and urea nitrogen were measured in the first voided urine and cord blood from 15 term infants exhibiting a wide range of growth. Analysis by multiple regression with neonatal ponderal index as the primary dependent variable revealed a significant correlation between lowered ponderal index and decreased urinary urea and ammonia. **The correlation was primarily a function of increasing ammonia levels, with no relationship between fetal leanness and urinary urea.** Comparable cord artery and vein ammonia suggest that **placental ammoniogenesis was not a major determinant** of observed elevations in urinary ammonia. **Confirmation of the striking correlation between increased urinary ammonia and lowered neonatal ponderal index may afford a simple test for the identification of nutrient-related growth retardation.**

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