Letters to the Editor

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Underestimating Cocaine Use during Pregnancy

Paradoxically, the article by McCalla et al.[1] showing a decrease in the percentage of women with cocaine in their urine at the time of delivery from 1988 through 1989 to 1991 through 1992 sheds light on the need to expand drug treatment for pregnant and other women.

Although the epidemic of cocaine use among pregnant women may have peaked several years ago, further decreases seem to have plateaued. New York City birth certificates show a slowing in cocaine use during pregnancy: 2.11% in 1988, peaking at 2.32% (1989), then decreasing from 1.76% (1990) to 1.63% (1991) to 1.31% (1992) to 1.21% (1993). However, the Substance Abuse and Mental Health Services Administration's Drug Abuse Warning Network found a 21.2% increase in cocaine-associated emergency room visits from 1988 through 1989 to 1991 through 1992 among New York City women aged 15 through 44 years (personal communication from Janet Greenblatt, February 17, 1994).

Why the discrepancy? One possible reason is that women who use cocaine during pregnancy have become harder to identify at delivery. During the past decade the New York City Health and Hospitals Corporation has gradually tightened its policy regarding maternal and newborn drug testing; it now prohibits maternal urinary drug surveillance during pregnancy and delivery without informed consent. The New York City Department of Health collaborated from 1990 through 1994 in developing, administering, and evaluating PACE (Parent and Child Enrichment), a drug treatment program for cocaine-using pregnant and postpartum women. Unfortunately, most women entered the program in order to be drug free at the time of delivery so that they could retain custody of the infant, and many women left the program prematurely after they were awarded custody.[2] While abstinence from cocaine use during the third trimester of pregnancy is beneficial for mother and child, women who leave treatment prematurely must be considered at high risk for relapse.

We must not become complacent. Cocaine use among pregnant women will not go away. Overestimating a decrease may lead policymakers to decrease vital funding for drug treatment for pregnant and other women using cocaine. □

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Asbestos-Related Cancer and the Amphibole Hypothesis

1. The First Documentation of the Association

An annotation[1] and a paper[2] published in a recent issue of your journal must have confused readers on the subject of the association between exposure to asbestos dust and diffuse mesotheliomas of the pleura and peritoneum. It is important that this situation be clarified, as misrepresentation can affect the use and control of these materials in many countries. As the original discoverer of the development of mesotheliomas in people exposed to asbestos dust,[3] I would appreciate an opportunity to try to clarify the situation.

In 1957, in the Cape area of South Africa, we established that mesotheliomas of the pleura were occurring in people living in the vicinity of mills processing blue (crocidolite) asbestos and in people milling and mining the material. The main paper was published in 1960, and in 1963 we confirmed that the tumors were occurring in the crocidolite mining area, but not in the amosite or chrysotile areas of southern Africa. Southern Africa was

the ideal area for these investigations, as the three main types of asbestos were produced in the same amounts by a similar labor force. In an international study, we set out to see whether the role of crocidolite could be confirmed in other countries. Over the years, our initial hypothesis has held. The vast majority of mesotheliomas are associated with exposure to crocidolite asbestos. A small number of cases have been recorded following exposure to other forms of amphibole asbestos: amosite, tremolite, and anthophylite. No mesotheliomas have been shown to have occurred in chrysotile-exposed workers, unless the exposure has been intense and for more than 20 years. In addition, there must be tremolite contamination of the chrysotile.

Two other facts are of great importance:

1. The majority of these tumors occurred following prolonged exposure to large quantities of fiber. This situation rarely exists today.

2. There is a "natural" incidence of these diffuse mesotheliomas. At least 10% of these diffuse mesotheliomas occur without exposure to asbestos dust, and sporadic cases of these tumors were reported before the widespread use of asbestos. □

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2. *Stayner and Colleagues Respond*

We thank Dr Wagner for his comments regarding our paper.[1] We do not believe our article misrepresented the evidence for an association between mesothelioma and asbestos exposure. We agree with Dr Wagner's main point, which is that the incidence of mesothelioma is greater in epidemiologic studies of workers exposed to crocidolite than either chrysotile or amosite, and recognized this fact in our paper. Interpretation of these epidemiologic findings is hampered by the lack of control for potential differences in exposure levels and fiber dimensions.

Nonetheless, ample evidence suggests that exposure to chrysotile is a risk factor for mesothelioma. As we reviewed in our paper, numerous cases of mesothelioma have been reported in several studies of workers exposed to chrysotile. In fact, Dr Wagner recognized in his early papers[2,3] that there were cases of mesothelioma in South Africa and Britain whose only known exposure was to chrysotile asbestos. Toxicologic studies, some of which were conducted by Dr Wagner,[4] also demonstrate an increase in mesotheliomas among animals exposed to chrysotile.

Dr Wagner suggests mesotheliomas occur in chrysotile-exposed workers only when there is tremolite contamination. While this statement is technically correct, it is virtually uninformative. Contamination by small percentages (<1%) of tremolite has been present in all of the reported epidemiologic studies of chrysotile-exposed workers. Unfortunately, studies of workers exposed to pure chrysotile have yet to be reported. In addition, this issue may be viewed as academic since workers are exposed to a mixture of fiber types and to commercial chrysotile containing tremolite.

A key point of our paper is that irrespective of mesothelioma, exposure to chrysotile asbestos should be viewed as a significant carcinogenic hazard. There is no serious disagreement in the scientific community that chrysotile asbestos exposure is causally associated with lung cancer and appears to be as potent a lung carcinogen as crocidolite or other forms of asbestos. The excess of lung cancer is generally far larger than the excess of mesothelioma in most epidemiologic studies of asbestos-exposed workers, a fact recognized in a recent review by Dr Wagner.[5]

Dr Wagner suggested that our paper and the accompanying editorial may have "confused readers." We hope that this letter will help to clarify any remaining confusion about exposure to chrysotile asbestos. Such exposure should be regarded as a serious potential public health hazard. □

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3. The Amphibole Hypothesis: Neither Gone nor Forgotten

Stayner et al.[1] and Cullen[2] failed to present the amphibole hypothesis in a developmental context that lent credence to their assertions that the hypothesis lacked scientific merit. Both indicated that chrysotile may be less potent than some amphibole asbestos minerals in causing mesothelioma. For crocidolite, the evidence is well beyond maybe. In 1964, Wagner[3] reported on a series of 120 mesotheliomas in the Republic of South Africa, (where the three major commercial asbestos fiber types—crocidolite, amosite, and chrysotile—were mined and milled). One hundred cases occurred in individuals exposed to the crocidolite mined and milled in the Cape Province; 10 cases occurred in industrial workers (9 exposed to crocidolite); 1 mesothelioma followed chrysotile exposure in Swaziland; and none followed exposure to amosite in the Transvaal. No exposure data are available for the remaining cases.