
The pill and heart attacks: Exaggerated?

Ever since oral contraceptives hit the market in the early 1960s, they have been linked with an increasing number of side effects — breast cancer, uterine cancer, high blood pressure, strokes, heart attacks, blood clots, gall bladder disease and even the disruption of sex pheromones. The scientific evidence for these supposed side effects, however, is far from solid.

In 1977, a research analyst at the Population Council in New York City reported that women ages 40 through 44 who both smoke and use the pill have a heart attack death rate nine times that of women of the same age who neither smoke nor use the pill (SN: 4/9/77, p. 233). The same year, British researchers reported that pill users have 4.3 times more fatal circulatory illnesses, including heart attacks, than do nonusers, and that women smokers older than age 35 are especially susceptible (SN: 10/29/77, p. 280). Now three new studies call such findings into question.

Two of the studies, which are published in the March-April *FAMILY PLANNING PERSPECTIVES* by Christopher Tietze, a biostatistician at the Population Council in New York City, and Mark Belsey and colleagues at the World Health Organization in Geneva, suggest that the risk of heart attack deaths to oral contraceptive users may have been greatly exaggerated by previous studies. Tietze calculated that if oral contraceptives have increased the risk of heart attack deaths four times among

women ages 15 through 44, then 2,200 of 7,000 heart attack deaths among this group of women each year would be attributed to the pills. But this is not the case, Tietze found. What's more, he points out, heart attack deaths among young women in the United States have been dropping during the years that oral contraceptives have been on the market. Thus, if the pills were triggering a lot of these deaths, then the rest of the deaths, among nonpill users, would have had to have declined astronomically to create an overall death rate decline — an unlikely occurrence. Belsey, on the other hand, re-analyzed data used in a British study that concluded that oral contraceptive users in 21 countries have a risk of dying from a heart attack that is three times greater than that of nonusers. His analysis, which took into account differences in population size and ages, showed no consistent relationships between pill use and heart attack deaths in those countries.

The third study, to be published in the April 7 *LANCET* by Samuel Shapiro of Boston University School of Medicine and colleagues, agrees with previous findings that pill users have a four times greater risk of getting a heart attack than do nonpill users, and that older pill users who are heavy smokers are particularly vulnerable. It suggests, however, that cigarette smoking, not oral contraceptives, is the major culprit. □

may be the absence of uranium-ore dust (a possibly compounding factor) in the Swedish studies or the fact that large-scale uranium mining only began in the 1950s, Axelson says, leaving insufficient time for development of the cancer. Average exposure prior to the onset of cancer in Axelson's study was 37 years for smokers, 49 years for nonsmokers.

Why is it that smoking might give miners protection against cancer? Axelson suggests that a buildup of smoking-associated mucus in the respiratory tract may shield cells of the lung from the effects of inhaled particles emitting alpha radiation. Increasing the distance from the radiation-emitting particles to the affected tissue from 36 microns to 46 microns would reduce the radiation dose by half, he claims.

The lower the dose, the longer the period prior to onset of cancer, Axelson says. If so, why do smokers with an extra protective sheath develop cancer quicker than do nonsmokers? Axelson says that tobacco may serve as a cancer promoter (in contrast to or in addition to being a cause of cancer). He says once lung-cancer induction begins, the disease may develop more quickly in smokers. Or, he adds, it might be that radioactive species in cigarette smoke augment the radiation dose delivered by radon and its daughters. □

An Irish polar bear?

Did you ever see a polar bear in a green coat? If you visited the San Diego Zoo last summer that's just what you would have seen. The usually creamy-white fur of three of the zoo's otherwise healthy polar bears had turned green — particularly on the flanks, on the outer fur of the legs and in a band across the rump. And unlike the pink elephant, the green polar bear was not a figment of anyone's imagination.

Because the swimming pool that the polar bears use contains nitrogenous wastes that promote the growth of certain algae, researchers guessed that the green bear hair was caused by the presence of algae on the surface of the hair. Microscopic examination proved this hypothesis to be only partially correct. Ralph A. Lewin of the Scripps Institution of Oceanography in San Diego and Phillip T. Robinson of the Jennings Center for Zoological Medicine at the San Diego Zoo report in the March 29 *NATURE* that the surface of the hair was clean, but that a blue-green alga (possibly *Aphanocapsa montana* Cramer) had taken up residence in the hollow centers of many of the wider, stiffer guard hairs of the bears' outer coats. Zoo researchers are currently investigating the circumstances under which the greening occurs (there and at other zoos) in the hope of devising a method to keep the bears the color visitors expect them to be. □

Reevaluated risk for radon-cancer link

A link between smoking and an elevated risk of lung cancer among underground miners has been firmly established. But studies of three different mining populations in Sweden now suggest that "smoking actually seems to offer some (relative) protection against lung cancer," according to Olav Axelson, a physician with the Department of Occupational Medicine at the University Hospital in Linköping, Sweden.

At an environmental health conference in Park City, Utah, last week, he described studies which show that although smoking miners face a greatly increased risk of developing lung cancer, underground miners who never smoked appear to face an even higher one. This is a striking departure from what had become accepted theory — that the lung cancer risk to nonsmoking miners is low, perhaps near zero.

The cancer risk is attributed in great part to the presence of radon and its daughters — fission isotopes it spawns during radioactive decay. Radon, itself a decay product of radium, is present in all uranium and hardrock mines. Studies indicate that smoking has a synergistic effect in the presence of radon; it appears to greatly increase a miner's risk of developing lung cancer.

Citing a study he and Lennart Sundell (of Regional Hospital in Örebro, Sweden) reported in the *SCANDINAVIAN JOURNAL OF WORK, ENVIRONMENT AND HEALTH* last year, Axelson said they found the lung-cancer rate at two connected lead-zinc mines in Hammar, Sweden, to be 16 times the national average. Although afflicted smokers developed their cancers an average of 12 years earlier than never-smoking counterparts, the lifetime risk to nonsmokers proved much higher — the crude rate was 58.1 percent for nonsmokers versus 14.9 percent for smokers. The retrospective lifetime study involved 44 miners.

A preliminary study of iron-ore miners, and another of copper-arsenic miners, both in northern Sweden, provided collaborative data. Both showed that smokers developed lung cancer at an earlier age than nonsmokers, but that the lifetime risk to nonsmokers was greater. Axelson said rough exposure estimates indicate the dose to miners probably was on the order of "one working level" (1.3×10^5 million electron volts of alpha radiation per liter of air).

The reason a similarly high frequency of nonsmoking uranium miners in the United States have not developed lung cancer