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Hazardous wastes accumulating at Ville Platte, La., present a problem of safe disposal.

no budget, only one employee and in June was ordered flatly to stop looking for imminent hazards. A June 16 memo told Kaufman's boss to "put a hold on all imminent-hazard efforts" because work on writing hazardous-waste regulations has higher priority "at this time."

Kaufman also claims that when he was able to uncover potentially threatening situations, "trying to get the appropriate EPA regional office to take action against these facilities proved to be an almost insurmountable obstacle." In one such case involving a Deerfield, Ohio, site, the regional office refused to visit — or let headquarters personnel visit — the site. "When I discussed this case with regional officials," Kaufman testified, "I was told that they have in their files other cases that are even worse than [the Deerfield one] and... that they didn't plan to investigate any ... let alone take action against them."

According to the Nov. 17 ENVIRONMENTAL REPORTER, a recent EPA study shows hazardous constituents are entering the groundwater — often in concentrations greater than permitted by drinking-water standards — at 43 of the 50 industrial-waste lagoons and landfills studied. In each case, there had been no prior documented or suspected problem.

The basis of EPA's authority to regulate the management of hazardous wastes comes from the Resource Conservation and Recovery Act of 1976. A set of seven regulations is required; three have been proposed, the rest will be ready within weeks. EPA estimates it will promulgate final standards in January of 1980. But the agency says its authority over abandoned or closed disposal sites rests mainly in enforcing action against the site owner if and when a particular site represents an imminent and substantial public-health hazard. If the owner lacks money to remedy the situation, EPA can't do much, Costle says, because it, too, lacks funds.

EPA hopes a national inventory by the states of open chemical dumps, due to begin next summer, will help at least to define the magnitude of the growing hazardous-waste problem. □

Estrogens and cancer: More questions

During the past three years, studies by five different groups of investigators have found that menopausal women who take estrogens run a considerably greater risk of getting uterine cancer than do women who do not take estrogens. Five separate studies confirming each other constitute scientific proof, right? Wrong! Reproducibility does not necessarily establish validity because the initial studies contained a serious methodological flaw. Or so argue two Yale University School of Medicine researchers in the Nov. 16 NEW ENGLAND JOURNAL OF MEDICINE.

And to further heat the controversy, two Harvard University School of Public Health scientists counter the Yale researchers' conclusions in an accompanying editorial. In fact, they claim that the Yale researchers' findings substantiate, rather than detract from, the initial five studies showing that menopausal estrogens increase the risk of uterine cancer.

Now for the details. All five groups of scientists who found a link between estrogens and uterine cancer used a retrospective case control study. That is, they selected women who had been diagnosed for uterine cancer, matched them against women with other kinds of cancer or no cancer and asked both groups whether they had been using menopausal estrogens. All five groups found that those women with uterine cancer had been using estrogens to a considerably greater degree than had women with other kinds of cancer or no cancer. They concluded, on the basis of their particular findings, that women who use menopausal estrogens are anywhere from three to eight times more at risk for uterine cancer than are women who don't use estrogens.

Ralph I. Horwitz and Alvan R. Feinstein of Yale, however, believed that the methodology used in the above five studies was faulty. Specifically, because uterine bleeding is a well-established side effect from estrogen use, they suspected that far more women using estrogens would come to physicians fearing uterine cancer than would women not using estrogens. As a result, many more cases of uterine cancer would be detected among estrogen users than among nonusers, thus suggesting that estrogens cause uterine cancer. The only unbiased way to determine whether estrogens really cause uterine cancer, they reasoned, would be to match women diagnosed for uterine cancer against women who had come to physicians because of uterine bleeding, but who had not been found to have cancer, and to see whether the women with uterine cancer had used estrogens considerably more than had the women without it.

Horwitz and Feinstein then conducted two studies — one comparable to the five that had established a link between estrogens and uterine cancer and one in which they matched women diagnosed for uterine cancer against women not so diagnosed, but who had come to doctors because of uterine bleeding. The results of the first study, they report, were close to what the five previous studies had found — in fact, even a bit higher; that is, women who use menopausal estrogens run a *twelve* times greater risk of getting uterine cancer than do women who do not use estrogens. The results of the second study, however, found that women with uterine cancer had not used estrogens to any significantly greater degree than had women without uterine cancer. So Horwitz and Feinstein conclude: "The magnitude of the association between estrogens and endometrial [uterine] cancer has been greatly overestimated because of detection bias; when an appropriate compensation for the bias is introduced, the odds ratio approaches a value much closer to one."

In the accompanying editorial, however, George B. Hutchison and Kenneth J. Rothman disagree with Horwitz and Feinstein's conclusions. They contend that women with uterine cancer will eventually have the problem diagnosed whether they had early uterine bleeding from estrogens or not, and thus estrogen use "will have little effect on the total number of uterine cancer cases ultimately found. Therefore estrogen users, especially long-term users, would be very little overrepresented among a series of women with endometrial cancer." On the other hand, Rothman and Hutchison assert, many of the noncancerous conditions detected by estrogen-induced uterine bleeding probably would not be detected if women weren't estrogen takers. Therefore, using women with such conditions as controls tends to "yield falsely high estimates of estrogen use, as compared with what would be obtained from a valid control series. Thus the analysis recommended by Horwitz and Feinstein compares a case series that has minimal selection bias with a control series that has a bias in the direction of exaggerating the frequency of estrogen use. The net effect is to underestimate the estrogen-cancer association."

Horwitz and Feinstein's second study, then, is not valid, but their first study is, Rothman and Hutchison conclude. And the data from their first study "only add to the ... evidence that exogenous estrogens induce endometrial cancer." □