between exposure to THM's and a resultant adverse effect. When Tuthill took this potential lag time into account by comparing the current cancer rates and chlorination procedures 20 years ago in the Massachusetts communities, he found no relationship.

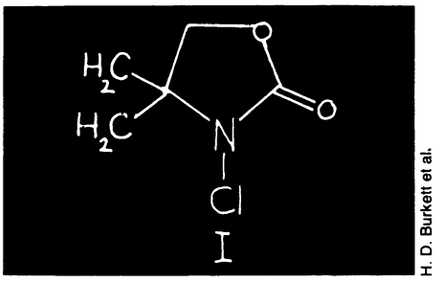
Still, Tuthill admits that his study has its limitations — the examination of only a small number of communities and the study's ecologic approach (experimental and control groups are geographic areas rather than individuals). "The results from ecological studies are not nearly as definitive as those conducted on the individual level," Tuthill says. "The question [of chlorination safety] isn't answered yet."

Despite the uncertainties surrounding it, "The practice of chlorination," Tuthill and Moore conclude, "should not be phased out in preference to another method of disinfectant until there is stronger epidemiological and clinical evidence to support the charge that it produces serious health risks and until the potential health effects of substitute disinfectants are carefully explored."

One of those potential substitutes now under scrutiny is 3-chloro-4,4-dimethyl-2-oxazolidinone, or Agent I. At the recent American Chemical Society regional meeting in New Orleans, H. D. Burkett of Auburn University in Alabama reported that in addition to being as efficient as chlorine in eradicating bacteria, Agent I has certain advantages over the present method of chlorination:

- The current method involves storing chlorine as a gas before it is converted to its acidic disinfectant form. Agent I, says Burkett, is a nontoxic solid, much less hazardous to work with than toxic chlorine gas.
- Agent I is a "slow-releasing agent," liberating free chlorine as needed.
- Agent I is less likely than the conventional chlorine form to form THM's.

While preliminary toxicity tests on white rats and chickens show no adverse effects of Agent I or its metabolites, its effect on humans is unknown. □



H. D. Burkett et al.

The novel disinfectant compound Agent I.

Attacking cancer after it has spread

Immune system stimulators, until now, have been a second string team in the cancer fight. "Immunotherapy" is very attractive in that it has no known side effects, but traditionally it has been used only after other therapies in attempts to fight residual cancer. At the recent meeting in Toronto of the American Association for the Advancement of Science, Robert S. Kerbel reported results from animal studies that suggest that in some vicious cancers the immune system, if cleverly manipulated, could be the best first line of defense.

The most difficult problem in treating many cancers is stopping or reversing their spread. Kerbel estimates that dispersed cancer cells, which initiate new tumors, are responsible for 85 percent of human cancer deaths. Kerbel and James W. Dennis have concentrated their work at Queen's University in Kingston, Ontario, on the most potent metastatic, or spreading, cancers.

One characteristic that distinguishes the cancers that spread most rampantly through the body is their ability to evade the immune system. But Kerbel and Dennis have developed a method to stimulate a reaction to such cancer cells. They take cells from the original tumor of a mouse cancer that spreads rapidly and widely and, in the laboratory, select cells with genetic mutations that result in altered membrane characteristics. A few of these variants have lost their disguise, the researchers find. The variants still cause cancer in mice with deficient immune systems, but in mice with normal immune response, the variant cells are killed in a style resembling transplant rejection.

When the variant cancer cells were used in the laboratory to stimulate immune system cells called cytotoxic T-lymphocytes, Kerbel was surprised to discover that these "educated" cells are as effective at killing cells from the original tumor as at killing the variants. When injected into mice that had widespread metastases from the original tumors, the educated cells reduced dispersed tumors, although eventually the animals still died.

In a more effective procedure, the investigators surgically removed the original tumors in mice with widespread disease. They then irradiated the animals and injected killer cells previously stimulated by the selected cancer cell variants. After seven weeks almost all the control animals had died, but the six receiving the educated lymphocytes appeared healthy. Autopsies indicated that five of these mice were cured of their cancers.

Kerbel calls the results "interesting and encouraging." But the difficulty in providing therapy for patients with established, widespread disease will be in working out a reasonable protocol to select variants and stimulate a patient's own lymphocytes. Kerbel concludes that such therapy is now "still impractical, but not impossible." □

Cholesterol: You can't win

One of the toughest health questions facing Americans last year was how much cholesterol they should consume — authorities disagreed over whether lowering dietary intake of cholesterol protects against heart attacks and there was evidence that lowering blood levels of cholesterol, at least with drugs, increases a person's susceptibility to death from causes other than heart attacks (SN: 6/7/80, p. 357; 9/13/80, p. 165). And now it looks as if the question of how much cholesterol is good for you is going to be even more difficult to answer. Two new studies suggest that while a low-cholesterol diet *can* protect against heart attacks, it may increase vulnerability to cancer.

The first of the studies, reported in the Jan. 8 NEW ENGLAND JOURNAL OF MEDICINE by Richard B. Shekelle of Rush-Presbyterian-St. Luke's Medical Center in Chicago and his colleagues, started some 20 years ago. The investigators evaluated dietary cholesterol and blood levels of cholesterol in 1,900 middle-aged men working for the Western Electric Co. in Chicago. A positive association was found between the subjects' dietary intake of cholesterol and blood levels of cholesterol. The scientists then checked 20 years later to see which subjects had had fatal heart attacks and found that dietary intake of cholesterol appears to influence blood levels of cholesterol and in turn a person's susceptibility to heart attacks—as several previous studies have suggested. Other investigations, however, have failed to make these associations — hence the still-unresolved question of whether dietary cholesterol really influences blood cholesterol and in turn the risk of a heart attack.

The second of the new studies, reported in the Jan. 16 JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION by Roger R. Williams and colleagues at the National Heart, Lung and Blood Institute in Bethesda, Md., is part of the NHLBI's ongoing Framingham, Mass., heart study, which over the years has shed, and continues to shed, light on heart attack risk factors. Williams and his co-workers documented 691 cases of cancer in some 5,000 Framingham subjects and found that a low blood level of cholesterol was significantly associated with later susceptibility to cancer, particularly colon cancer, but only in men. Williams and his team admit that they do not know how a low blood level of cholesterol might predispose to cancer, especially since some studies have shown a link between high fat consumption (purportedly related to blood cholesterol) and colon cancer. Nor do they know why blood cholesterol was related to cancer in their male but not in their female subjects. □