tive or accurate enough, that animal tests have not indicated what level of DES does not cause cancer, and that birth control pills have not been in use long enough to indicate whether estrogen intake will increase cancer incidence after a typical lag of 20 to 30 years.

The CAST report also hits the current food additive regulations because there is no consideration of possible benefits. It describes a 10 to 12 percent saving in feed due to DES use and a higher proportion of lean meat in animals receiving the hormone. "The uses of these substances in livestock production are of great value to the American public in terms of food costs and nutrition," it says. If benefits are to be weighed against risk of cancer, however, it would be a major change in regulatory policy that would probably have to come from Congress rather than the FDA. □

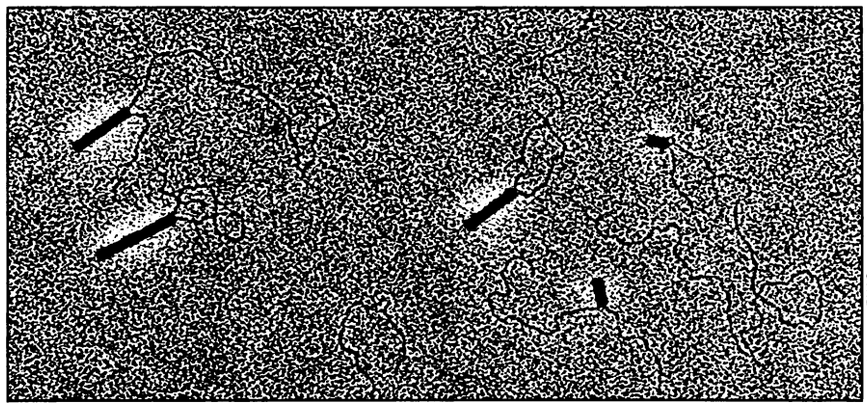
Cambridge resumes genetic research

The Cambridge, Mass., city council has finally approved an ordinance regulating research on recombinant DNA, the splicing of separate strands of genetic material. The ordinance is essentially the same bill that passed the council's preliminary vote two weeks ago permitting moderate-risk research (SN: 1/29/76, p. 70), although some clarifying amendments were added during the council vote Feb. 7.

One of the most significant alterations appearing in the final ordinance was the expressed restriction of any P4 (high-risk) research, an area only implied in the earlier version. Neither MIT nor Harvard, the two research centers affected by the law, has any present facilities or future plans to conduct P4 research. Another amendment specifically set a $200-a-day fine on any violation of the ordinance, answering some critics who charged the law had no enforcement powers. A third amendment expressly authorized the public health officials to close down any violating laboratory, a power already inherent in existing city ordinances. The final ordinance, then, permits all P3 research in Cambridge, provided the facilities use the EK2 organisms, genetically deficient bacteria and viruses that cannot survive outside the laboratory environment. Cambridge mayor Alfred Vellucci, the research's most persistent critic, attempted at the last minute to add another amendment restricting all but P1 research, but the amendment failed.

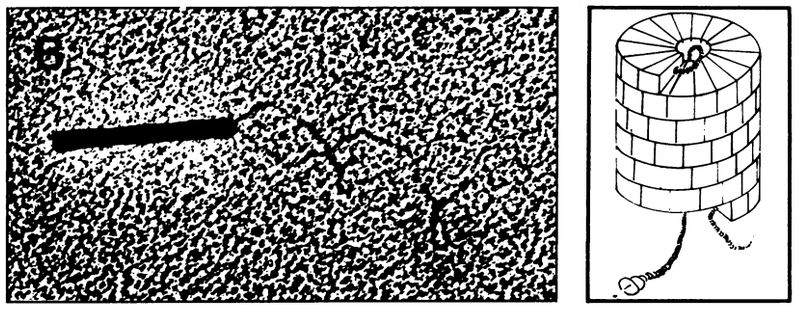
Although Cambridge now becomes the first city in the United States to regulate research on recombinant DNA, other cities are now considering similar proposals. Public hearings and study committees continue to debate the problem in San Diego, Madison and Bloomington and in New York State. □

How the virus builds itself



Lebeurier et al., PNAS

The growing tobacco mosaic viruses have two RNA tails, here magnified 68,000 ×.



Left, virus at 136,000 ×. Right, model of the RNA looped inside the protein helix.

The agent that causes mosaic blight in tobacco plants was recognized in 1899 to be a new type of organism, a subcellular form of life. Since then scientists have been repeatedly amazed by this virus's extreme simplicity but perfect competence to do what all forms of life must do—reproduce itself.

When the genetic material, RNA, of the virus enters a tobacco cell, it subverts the cell's machinery to copy viral RNA and to make viral coat protein. New RNA and protein then spontaneously aggregate into mature virus particles. Each complete virus consists of about 2,000 identical protein molecules arranged in a helix. The single-stranded RNA molecule fits into a helical groove that winds around the long axis of the rod-shaped particle.

Laboratories looking at the way the virus assembles were recently surprised by evidence that the virus builds from the inside out. Previously, researchers assumed the protein subunits added to the immature virus as if they were beads and the RNA was a string. The RNA molecule, they predicted, would extend from the growing end of the virus. Now researchers in two laboratories report evidence that in the partially assembled virus the RNA chain forms a loop at the growing end and returns down the virus's hollow core. The extended RNA tail would be pulled up into the cylinder as RNA from the loop is inserted into the protein helix.

Electron microscopic pictures provide the main evidence for this model. G. Lebeurier, A. Nicolaieff and K. E. Richards of Université Louis Pasteur found that almost all the incomplete virus particles they observed when they mixed viral protein and RNA had two RNA tails protruding from the same end of the particle. One tail was approximately the same length on all the particles, whereas the length of the other, usually longer, tail was inversely related to the length of the incomplete rod, the researchers report in the January PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES.

Other researchers at the Medical Research Council in Cambridge, England, arrived at the same model from experiments with partially disassembled, as well as growing, rods. P. G. J. Butler, J. T. Finch and D. Zimmern used a chemical treatment to strip part of the protein from completed viruses. In these disassembled particles, the long RNA tail did extend from the growing end, in contrast to the observations of partially assembled rods. When more protein was added to the suspensions of incomplete viruses, partially assembled rods seemed to elongate more than ten times as fast as the partially stripped rods. This result supports the idea that the looping back of the RNA is important to the virus's growth. The researchers suggest in the Jan. 20 NATURE that a loop inside the elongating virus would allow RNA greatest access to binding sites on incoming protein, while holding the bulk of the RNA out of the way of the assembly activity. □