vice president for research and development at the General Electric Co., "We are losing position to major foreign competitors in terms of R&D as a percent of GNP [gross national product]." What's more, he said, "Figures don't tell the entire story, because half of the U.S. federal R&D funds are for defense. In Japan and West Germany, by contrast, R&D budgets for defense are minimal; virtually all of their R&D funds are aimed at economic development."

He said that Japan, with less than half the population of the United States, is estimated to have as many engineers and scientists working in that type of activity as we do. "And while our effort appears not to have grown between 1970 and 1976, Japan's effort increased 50 percent" — from around 160,000 people in 1970 to about 240,000 in 1976.

Research and development is linked with increasing employment and productivity, several speakers explained. Bueche cited a recent study by Data Resources, Inc., showing that during the past 10 years U.S. high-technology companies increased employment nine times faster than low-technology companies, "and these same high-technology companies increased overall productivity by about four percent per year, or twice the national rate of two percent."

But high technology is dependent on innovation, Bueche said, and there are signs that the rate of innovation may be slowing in the United States. "Our performance in patented inventions is perhaps one of the best single measures of what's happening in terms of R&D output," he said. "The number of patents granted U.S. residents declined 21 percent between 1971 and 1976," he said, "while patents to foreign residents grew by 16 percent to total 37 percent of all U.S. patents granted in 1976."

The Commerce Department will be conducting at least two studies on the R&D problem within the next year. The first, scheduled at the behest of the President, will examine factors that affect and may have discouraged industry investment in research. The second will look for ways in which the government might influence the rate and direction of industrial innovation. □

Viruses prepare to plug into chromosome

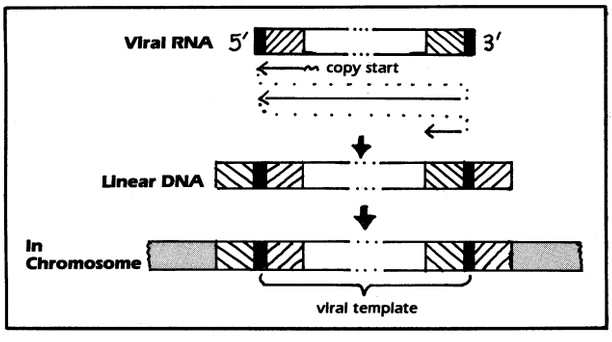
Recent focus on genes that hop in and out of chromosomes has stolen some of the spotlight from the previously recognized genetic transients. Yet there is continuing progress on understanding the mechanics of viruses, which can drop their essential genetic baggage into cellular chromosomes and have it maintained and reproduced there briefly or for millions of years.

At the recent Cold Spring Harbor Symposium on DNA: Replication and Recombination, researchers described analyses of an intriguing set of viruses. The RNA tumor viruses, which produce animal cancers, use RNA instead of DNA to encode their genetic information. They are often called "retroviruses" because they reverse a step in one central dogma of biology: DNA makes RNA makes protein. Viral RNA, once in a cell, must create double-stranded DNA molecules. Then the viral genes can slip into an animal chromosome.

The DNA copy deposited in the cell's chromosome must contain all the information of the virus. Yet copying the RNA requires priming. An RNA molecule (transfer RNA) from the cell's cytoplasm binds to about 16 nucleotides of the viral RNA and the DNA copy grows at the end of that chain. The transfer RNA is later clipped off. Therefore, if DNA were simply produced from one end to the other of the linear viral RNA template, the stretch bound to the primer would be lost.

The virus's way out of the dilemma involves at least one jump of the replicating enzyme from one end of the RNA molecule to the other. Results from the laboratories of Harold E. Varmus at the University of California in San Francisco, David Baltimore at the Massachusetts Institute of Technology and John Taylor of the Institute for Cancer Research in Philadelphia indicate that the replicating machinery actually makes that jump twice.

Synthesis of DNA from the viral RNA template begins near one end (called the 5′ end) of the RNA molecule. Test-tube experiments with isolated components show distances of 100 to 150 nucleotides, depending on the virus, from that start to the end of the molecule, reports William A. Haseltine of Harvard Medical School. In the June 1 NATURE Haseltine and Dennis G. Kleid of Stanford Research Institute propose that retro-viruses of various animals can be classified on the basis of the length of that initial copy, running from the primer to the 5′ end.



Viral RNA serves as a template for the DNA that slips viral genes into an animal cell chromosome. Tricky maneuvering produces a DNA segment padded at each end with an extra copy of the opposite end of the viral genetic message.

Varmus and colleagues have looked at DNA copies of viral RNA made in infected animal cells. The linear DNA molecule has the same sequence at both ends and is longer than the RNA molecule. The repeated stretch includes one copy of the sequences at each end of the RNA molecules. Haseltine proposes that the DNA forms from the start on the RNA to the 5′ end and then that DNA reanneals with the short repeat at the RNA 3′ end and continues copying. Near the 5′ end, it again jumps, either to an RNA or DNA template, in order to finish.

In his test-tube experiments with disrupted viruses, Baltimore and co-workers find mouse leukemia virus gives two major DNA products. The longer product seems to include the repeated sequences, as if the copying apparatus successfully made the second jump.

In the nuclei of cells, linear DNA is converted into circles. Varmus finds two major circles, one equal in length to the linear DNA molecules and with two copies of the end region side-by-side, and the other circle missing one copy of the repeated regions (300 base pairs in avian sarcoma virus and 1,200 base pairs in mouse mammary tumor virus.)

The researchers are not yet certain which form of the DNA joins the cell's chromosome. But the inserted DNA contains the repeated end regions. Unlike SV40, a DNA-containing animal tumor virus, the RNA tumor viruses always connect to a host chromosome at the same viral site. Thus the central stretch that will be the template for new viral RNA is bordered by virus-specified segments, the repeats of the viral RNA ends. Taylor and Varmus propose that the extra segments may be important in controlling viral genes.

Although there is no direct evidence that the viral stretches of DNA hop around in chromosomes, Varmus suggests that the genetic inserts by RNA tumor viruses do resemble the movable genetic elements, transposons (SN: 6/17/78, p. 390). The viral genes in the chromosome, like the genes of a transposon, are flanked by a natural repeat. Varmus and Robert A. Weinberg of the Massachusetts Institute of Technology showed that the viral genes can insert into cellular chromosomes in many sites, although researchers do not know yet whether the genes prefer specific regions. The investigators now plan to determine exact sequences of the repeated end regions for comparison with the insertion sequences that flank transposon genes. □