mechanisms of action of the RNA tumour viruses. Also in this issue of *Nature New Biology* (page 229), Green and his colleagues report the detection, in both the cytoplasm and nuclei of cells transformed by mouse sarcoma virus, of RNA which hybridizes with the DNA which is transcribed *in vitro* by reverse transcriptase programmed with mouse sarcoma virus RNA. These RNA species seem to be intermediates in the replication of mouse sarcoma virus ; very probably, some of them are destined to become the genomes of progeny virus particles. Until today they have escaped detection, but can now be identified first because reverse transcriptase is available to synthesize very radioactive complementary DNA, and secondly, because the techniques of nucleic acid hybridization have been developed and perfected in experiments with bacteria and their phages.

These experiments, and almost all other research concerned with the biology of tumour viruses, depend either on ideas or techniques, or both, derived from classical molecular biology. The chances of understanding malignant transformation, to name but one example, will be slender indeed if the flow from this productive source is cut back. That should not be forgotten when it comes to allocating the enormous sums of money which the United States Government seems determined to appropriate for cancer research. For a few years, scientific research can no doubt live off existing concepts and technologies ; but unless investment in the molecular biology of bacteria and other simple organisms is maintained at effective levels, research into more complicated organisms—including, of course, man—will gradually decline, so that within perhaps a decade, scientific advance of any significant magnitude will become very much more difficult, if not impossible.

Enigma of Satellite DNA Variations

THE function of the highly reiterated fraction of the DNA of eukaryotic cells, which in some species has a base composition sufficiently different from that of the bulk of the genome for it to be resolved as a satellite band in caesium chloride density gradients, remains a great enigma. Apparently, satellite DNAs are not transcribed, and even if they were, it is hard to imagine that they could code for any useful protein, for their base sequences are extremely restricted. The basic repeating sequences of guinea-pig and mouse satellite DNAs, for example (see *Nature*, **227**, 775 ; 1970), seem to be only six and eight to thirteen base pairs long, respectively. (The extreme example of such repetition is a satellite of a crab species which is almost pure poly dAT.)

Satellite fractions, in at least some species, are localized in the nucleoli at interphase and in the vicinity of the centromeres of mitotic chromosomes ; and so it has been suggested that the repetitive DNA may play some role in maintaining the structural integrity of chromosomes—what has been termed housekeeping for want of a more precise description. Of course, other equally unsatisfactory suggestions have been discussed, including roles in chromosome pairing and recombination or the provision of a pool of "neutral" DNA which evolution could mould.

But any explanation of the function of satellite DNA will have to account for the fact that in at least some cells it is dispensable. For in the *Journal of Molecular Biology* (**56**, 579 ; 1971), Maio reports that BSC1 cells—a stable cell line derived from African green monkey kidney cells —lack a satellite fraction (ρ) which constitutes some five to seven per cent of the total nucleolar DNA which is to be found both in cells of primary explants from this species and in CV1 cells. These are another stable line derived from an African green monkey.

Apparently, during the process of its selection, the particular BSC1 line with which Maio has worked has lost the heavy satellite DNA, but has not lost any of its vitality. What is perhaps even more intriguing is that Ritzi and Levine, last year, reported that cellular DNA synthesis is not induced when these BSC1 cells are infected by SV40 virus. But on the other hand, they found that SV40 infection does induce the synthesis of DNA in CV1 cells and Maio has shown that these cells have the ρ satellite. Furthermore, also last year, Smith, albeit working with a different system, reported that mouse satellite DNA is amongst the first cellular DNA which is induced to replicate after infection with polyoma virus. By contrast, when mouse cells are induced to replicate their DNA by exposure to serum—the usual procedure for encouraging cells to multiply in culture—satellite DNA is replicated at later times.

And there is yet one more twist to this plot. In 1966, Hatanaka and Dulbecco wrote that their BSC1 cells were induced to replicate DNA after infection with SV40 virus. The apparent discrepancy between this finding and that of Ritzi and Levine seems to have been resolved amicably ; it turns out that there are two BSC1 lines abroad which differ in provenance—one is induced to replicate its DNA after SV40 infection and the other is not.

It will be interesting indeed to see whether the BSC1 line which is inducible for DNA replication after infection with SV40 is also like CV1 cells and has the ρ satellite DNA. If this proves to be the case, the temptation will be irresistible to speculate that two properties go together because the ρ satellite plays an essential role in the induction of DNA synthesis by SV40. And what would happen in a hybrid cell produced by fusing together the two sorts of BSC1 cell and then infected with SV40? Would both or only one parental genome (that retaining ρ satellite DNA) be replicated ? The future for research about satellite DNA seems brighter now than it has for a long time.