sis, and these grow only on bacterial strains containing the UGA suppressor and are therefore UGA mutants. One mutant is in the phage RNA polymerase cistron, the other six are mutations in the maturation protein cistron. So far, no UGA mutants of the phage coat protein have been isolated. In a cell-free protein synthesizing system lacking UGA suppressor *t*RNA, RNA from the UGA polymerase mutant stimulates the synthesis of 20 to 30 per cent of the polymerase made with wild type f2 RNA as messenger. The RNA from an amber mutant in the polymerase cistron, by comparison, does not stimulate synthesis of polymerase. The UGA codon is apparently leakier than the amber codon *in vitro*.

When the cell free system is supplemented with extracts of UGA suppressor cells, the UGA codon is read, more intact polymerase is made and there is a corresponding decrease in the amount of polymerase fragments synthesized. All this, of course, indicates that, like UAA and UAG codons, UGA acts as a chain terminator, and premature termination can be prevented by a suppressor *t*RNA which reads UGA as sense, inserting an amino-acid at the site of the mutation.

The leakiness of the UGA codon suggests that strains lacking the UGA suppressor *t*RNA nevertheless contain *t*RNAs capable of weak base-pairing with, and low frequency translation of, UGA codons. Presumably in the cell UGA is a more efficient terminator than it is in cell-free systems; during isolation of the cell extracts, for example, the termination machinery might be inactivated to a greater extent than the *t*RNAs capable of reading UGA at a low frequency, and this would therefore magnify the leakiness. As for the suppressor *t*RNA, it probably translates the UGA codon as either cysteine or tryptophan, but this cannot be known until mutants of the UGA coat protein are found.

VIROLOGY

Large RNA Viruses

from a Correspondent

THE biology of large RNA viruses was the theme of a meeting held in Cambridge from July 21 to 25. The viruses involved—influenza, paramyxoviruses, vesicular stomatitis and RNA tumour viruses of avian and murine origin—are all far more complicated structurally than the small RNA viruses, such as poliovirus, and much less is known about their replication. Several large RNA viruses have fragmented genomes, and the host cell frequently plays a crucial but obscure part in replication.

In keeping with the importance of influenza as the most widespread and potentially most serious epidemic disease, approximately half the papers were concerned with influenza viruses. It was generally agreed that the genome of these viruses is in pieces, and R. D. Barry (University of Cambridge) drew attention to the very heterogeneous nature of the RNA found in different strains and in different particles of the same strain. Various reports concerning the internal RNA-containing protein component (RNP) indicated that it usually occurs as a small flexible helical structure, probably variable in length and with the RNA located externally. Both electron microscopic and physicochemical data suggest that the virus particles contain several pieces of RNP, but these may be joined end to end in a continuous linear structure within the particle. The RNP of the paramyxoviruses, on the other hand, seems to be a single, continuous helical structure 1·1 microns long.

There is considerable doubt about the number of structural proteins in influenza viruses. Estimates varied from three to six, although the most frequently reported number was four. W. G. Laver (Canberra) and R. G. Webster (Memphis) presented chemical and biophysical data which suggest that the neuraminidase and haemagglutinin functions of the virus reside in separate structural entities on the virus surface. There were also several reports of an arginine-rich protein of unknown function.

The events concerned in the replication and assembly of influenza viruses were discussed at length. It seems clear that during the first hour after infection, cellular DNA function is essential. Furthermore, I. Macpherson (Imperial Cancer Research Laboratories, London) presented data that suggest that the early function can be induced by pre-infection of cells with either DNA or RNA tumour viruses. N. J. Dimmock (Canberra) demonstrated the appearance of a new non-virion antigen in the nucleolus early in infection, and various reports indicated that the RNP accumulates in the nucleoplasm. Despite the apparent importance of the nucleus in the initiation of infection and the accumulation of RNP, RNA polymerase paradoxically occurs predominantly in the microsomal component of the cytoplasm. Several extremely detailed studies of this polymerase and its *in vitro* product were presented, and C. Scholtissek (Giessen) has used the polymerase product of various strains in annealing experiments using native virus RNA to determine the percentage base homology between strains of virus. Assembly of whole virus is completed at the cell surface, and this process was illustrated by some electron microscopic studies. Unfortunately it has not been possible to use this technique to trace the intracellular sites of synthesis of virus components or to detect their accumulation or transport.

Relatively little was said about the structure of RNA tumour viruses; attention was concentrated on events occurring in infected cells. F. Lacour (Villejuif) indicated that DNA synthesis always occurs in cells infected with avian myeloblastosis virus. H. M. Temin (Madison) studied the progeny of cells transformed by avian and murine sarcoma viruses, and concluded that these cells receive the information for conversion and formation of virus by means of an inherited "pro-virus", a piece of DNA formed after initial infection by virus, and integrated into the host cell genome by recombination. M. A. Baluda (Los Angeles) demonstrated the presence, in infected cells, of DNA which hybridizes specifically with AMV–RNA, and found that there are at least five copies of this DNA per transformed cell.

EVOLUTION

Dichotomies in Mammal History

from our Vertebrate Palaeontology Correspondent

IN an article on early mammal history, J. A. Lillegraven has combined some new information about the mam-