with certain amino-acids (*Nature*, **219**, 285; 1968).

Two configurational models have been constructed for the peptidoglycan of *Staphylococcus aureus* in which the polysaccharide chains are aligned head to tail and are hydrogen bonded together (Kelemen and Rogers, *J. Gen. Microbiol.*, **57**, xiii; 1969). An α-helical model accorded little hydrogen bonding, but a β-configuration in the form of a pleated sheet possessed *trans* carboxyl groups which accommodated a regular net-like organization of the peptidoglycan in which 60 per cent of the amino groups participated in hydrogen bonds.

Although acknowledging that peptidoglycan is essential in determining cell morphology, Braun and Shwarz (*J. Gen. Microbiol.*, **57**, iii; 1969) argue that this function is a reflexion of its association with other cell wall components. They have analysed the interaction between peptidoglycan and lipoprotein in *Escherichia coli* and proposed a model in which about 10^5 lipoprotein molecules are distributed regularly over the surface of the peptidoglycan. One lipoprotein moiety appears to be associated with every tenth repeating unit of the peptidoglycan and is linked by an N-terminal lysine to the carboxyl group of diaminopimelic acid residues. Under the microscope, cell walls treated with trypsin were seen to be split into two layers. This, coupled with a rapid decrease in the optical density of the treated material, suggested to Braun and Shwarz that lipoprotein, too, plays a significant part in stabilizing the cell wall.

VIROLOGY

Fusion Within and Without

from our Cell Biology Correspondent

In the past five years the use of inactivated parainfluenza viruses to effect the fusion of somatic cells into viable hybrids has emerged as a powerful biological tool. The technique has already transformed somatic cell genetics from the emeritus professor's pipe dream to a rapidly expanding experimental discipline; it is also increasingly being applied in virology and viral oncology, for example in promoting infection of refractory cells with such viruses as polio and polyoma, and no doubt there are many more applications to come. Most of the credit for this innovation belongs, of course, to Henry Harris and his collaborators at Oxford; it was they who realized (*Nature*, **205**, 640; 1965) the potential for cell biology of Okada's discovery (*Exp. Cell Res.*, **26**, 98; 1962) that high multiplicities of Sendai virus induce cell fusion. But the way in which these viruses induce fusion remains obscure for perhaps the good reason that, for most people who use the technique, all that counts is that it works; how it works is of secondary importance. The mechanism of fusion is not, however, without its intrinsic interest and Bratt and Gallaher (*Proc. US Nat. Acad. Sci.*, **64**, 537; 1969) suggest that viral lipids or possibly lipoproteins are the fusion factor, at least in Newcastle disease virus (NDV).

While investigating the infection of secondary cultures of chick embryo cells with NDV they noticed that different strains of the virus vary in their ability to induce cell fusion; the strains seemed to fall into two classes, those which induce fusion directly and those which induce fusion only after the cells are infected and viral materials have been synthesized. Bratt and Gallaher call the two mechanisms fusion from without and fusion from within respectively. Fusion from without is a property of NDV–HP and NDV–EH strains; the fusion induced is as great 2·5 hours as 7 hours after virus has been added; it is independent of the *p*H of the cell's medium; it is greatest at the highest multiplicities of infection; and, most important of all, the virus does not have to be infectious to induce fusion.

Fusion from within by NDV–AV, on the other hand, requires at least 6 hours, is dependent on *p*H—there is most fusion at high *p*H—and the virus must be infectious. Inhibition of protein synthesis by cycloheximide either before or after infection has no effect on fusion from without by NDV–HP; fusion from within is inhibited by the drug if it is added early, but susceptibility decreases as addition is progressively delayed after infection. Both fusion processes are, however, completely suppressed by pretreatment of the virus with antiviral antibody. When antibody is added after infection, fusion from without becomes completely insensitive to inhibition between 20 and 40 minutes after infection even though fusion does not reach its maximum until 2 to 3 hours after infection. Fusion from within, on the other hand, remains completely susceptible to inhibition by antibody for 5·5 hours after infection.

Clearly Bratt and Gallaher have established that the two types of fusion are quite distinct. Fusion from without seems to result from the direct interaction of cell and virus, and the fact that it occurs at 38° C and 43° C but not at 23° C brings to mind two other temperature dependent interactions of cells with NDV virus-penetration and cell interference. Fusion from within, on the other hand, seems dependent on viral protein synthesis but not DNA-dependent RNA synthesis. The two types of fusion seem to be strain specific and, because the capacity to induce fusion is restricted to viruses with lipoprotein envelopes and because agents which alter these lipids are known to diminish fusion from without, Bratt and Gallaher suggest that either viral lipids or lipoproteins are the fusion factor. The fact that fusion from either within or without is strain specific and the lipid composition of NDV envelopes is also strain specific, of course, supports this notion.

CANCER

Oncogeny and Ontogeny

from a Correspondent

The considerable problem of defining the chemical events involved in carcinogenesis was tackled at the second meeting of the annual winter symposium of the Department of Biochemistry of the University of Miami and the Papanicolaou Cancer Research Institute in Miami on January 22 and 23 (see *Nature*, **225**, 498; 1970, for report of first meeting). One approach to this problem is to use a minimal deviation hepatic tumour, the Morris hepatoma, which in its well differentiated form differs from other experimental rodent tumours by its low glycolytic capability, high respiration and use of fatty acids as metabolic fuel. Many years of study of this hepatoma has led Professor Van R. Potter (Madison) to the concept that "oncogeny is blocked ontogeny".

Long term evolutionary changes in gene expression