stages of development of the cultures. Much interest was focused on the 26S RNA which corresponds in size to myosin mRNA. Such RNA is synthesized before as well as after fusion, though the species made before fusion is much less stable. Whether this material is indeed myosin mRNA, and the relationship between these findings and those of Dr Yaffe, remain open questions. Dr D. Luzzati (Institute of Molecular Virology, Paris) obtained temperature-sensitive variants of myoblast lines. At the non-permissive temperature the cells do not fuse to form myotubes, nor do they make muscle proteins. Thus the fusion process is coupled to the formation of the characteristic muscle proteins.

Pathological aspects posed more questions than could be answered. At the molecular level, most progress was evident in the analysis of thalassaemia on which there was a conference in April (see *Nature New Biology*, **243**, 97 ; 1973).

CELL SURFACE

Adhesion of Cells

from a Correspondent

A COLLOQUIUM on specific adhesion of cells organized by the Cell Surface Discussion Group was held in London on May 18. Professor L. Wolpert (Middlesex Hospital Medical School, London) in his introductory remarks, pointed out that there is little evidence for adhesive specificity between cells analogous to that between enzyme and substrate or antigen and antibody ; cells of many different kinds can stick to one another. A recurring, if sometimes peripheral, theme of the meeting concerned the precise molecular mechanisms involved in adhesion ; models of locks and keys or locks and locks with bivalent bridging keys were equally favoured.

The strength of the cellular slime moulds as a model system for morphogenesis was shown by Dr G. Gerisch (Max-Planck Gesellschaft, Tubingen), who described the interesting work of his group on the remodelling of the cell surface accompanying morphogenesis of *Dictyostelium discoideum*. During transition from single cell growth to aggregation the cells develop or redeploy surface structures responsible respectively for terminal (A-sites) or side to side (B-sites) intercellular adhesion. The contact sites can be separated by following the developmental kinetics of the organism, by monospecific antibodies directed against those sites, or by the identification of B-sites by their requirement for divalent cations. The sites occupy at most 2% of the total cell surface and are mobile, as shown by capping with antibody. It is possible to select morphogenetic mutants which lack A-sites that reappear in revertants. Antibodies against surface carbohydrate do not block A or B-sites. By contrast, Dr J. Smart (Imperial Cancer Research Fund, London) has found that surface membranes purified from vegetative or aggregation competent cells stick to cells and block aggregation. The active factors are heat stable components and may be glycoproteins because activity is unaffected by treatment with protein reagents such as glutaraldehyde. Dr A. McLennan (Microbiological Research Establishment, Porton) tabulated the many varied sugar-containing compounds isolated by acid extraction from unicellular sponges. These materials, although probably degraded, nonetheless inhibit aggregation of sponge cells, apparently species specifically, and are heat stable polysaccharides containing negligible protein. Acidic proteins of low molecular weight catalysing aggregation of unlike sponge cells were also mentioned. Dr McLennan pointed out that many of the experiments designed to show specificity of sponge cell adhesion use organisms from different taxonomic orders (*Haliclona* and *Microciona*) and it is not perhaps surprising that specificity has been found at this level.

In higher organisms Dr J. Edwards (University of Glasgow) has shown inhibition of spontaneous BHK cell aggregation by inhibitors such as vinblastine, colcemide and colchicine at anti-mitotic concentrations. The effect is probably on microtubules and does not require protein synthesis. Unidentified factors in serum seem to amplify isotypic recognition and collection of chick embryonic cells, according to Dr B. Pessac (Institut d'Immunobiologie, Hôpital Broussais, Paris), who has shown also that hyaluronic acid induces aggregation of tissue culture cells but not of trypsin treated cells. Dr J. Pitts (University of Glasgow) summarized the properties of gap junctions formed between apparently most cells—not L-cells—in culture. Formation of junctions is rapid and does not require protein synthesis. Metabolites such as nucleotides can pass between cells through gap junctions, but RNA and proteins are not transferred. L-cells do not form junctions with BHK cells but L/BHK hybrids do and presumably therefore junction formation requires some gene function related to a product(s) on the cell surface.

The possible role of surface located glycoproteins or glycolipids in cell adhesion continues to intrigue. Dr C. H. O'Neill (Imperial Cancer Research Fund, London), discussing lectin-induced agglutination of cultured cells, proposed that two types of aggregation are involved: direct binding of cells through ligand, and an indirect effect of the ligand on the intrinsic tendency of cells to stick together. He suggested that this last property is related to growth. Dr A. Allen (University of Newcastle upon Tyne) suggested that non-specific adhesion of cells may involve hydrophobic interactions of the carbohydrate chains of surface glycoproteins. He finds rapid binding of radioactive gastric mucosal glycoprotein or glycopeptides stripped from BHK cells with trypsin to different cultured cell lines. Binding is strongly temperature dependent, requires divalent cations and is reversible.

Dr K. Chandrabose (Imperial Cancer Research Fund, London) showed that at least one glycosyl transferase is concentrated on a plasma membrane fraction, lactosyl ceramide galactosyl transferase, and in a cautionary tale Dr W. H. Evans (National Institute for Medical Research, Mill Hill) described a principal glycoprotein of rat liver plasma membranes with potent sugar nucleotide pyrophosphatase activity against, for example, UDP galactose. The enzyme is iodine-labelled with lactoperoxidase and is probably located externally on cells. Consequently re-evaluation may be required of experiments, for example, by Roth and colleagues in which surface-bound glycosyl transferases are located by utilizing sugar nucleotides added externally to intact cells. Sachs has recently reported decreased levels of sugar nucleotide pyrophosphatase in transformed cells and it is relevant to these results and the findings of Dr Evans that Dr R. Hynes (Imperial Cancer Research Fund, London) who, in describing his work on lactoperoxidase labelling of the external surfaces of normal and virally transferred cells, showed an apparent loss in the transformed cells of several surface components (including glycoproteins) that are reactive in control cells. Professor L. Mester (Institut de Chimie des Substances Naturelles, Gif-sur-Yvette) emphasized the importance of surface-located sialic acid residues for controlling platelet aggregation induced by ADP or serotonin.

Drs C. D. Forbes and C. R. M. Prentice (Royal Infirmary, Glasgow) discussed some other agents affecting platelet aggregation, namely, animal AHF and heparin. These agents probably have direct effects on the platelet membrane. Therapeutic concentrations of animal AHF produce aggregation of human platelets *in vivo* and *in vitro* and if administered *in vivo* will induce thrombocytopenia which may exacerbate bleeding.

Closing the meeting, Dr M. Raff (University College, London) observed that no system of cell adhesion is really understood and stated his belief that, in the present advanced state of technology of membrane chemistry, new approaches at the level of interacting purified membrane components are long overdue.

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