and glutamic acids, characteristic of apo HDL and apo LDL, respectively. Together they constituted 40% of the NH_2 terminals, a fact which agrees well with the immunological data, if it is assumed that most of the glutamic and aspartic acids are present as the NH_2 terminals of the apoproteins in the membrane. COOH terminals were determined by standard techniques and the results showed that these too were rich in terminal residues characteristic of the serum lipoprotein apoproteins. To discover whether the apoprotein NH_2 terminals were distributed over various molecular weight sizes, a portion of dansylated membrane proteins was fractionated by SDS electrophoresis and the amino acids from each band chromatographed on polyamide. The results showed that the characteristic NH_2 terminals of serum apoproteins were distributed over a wide range of molecular sizes, in agreement with the immunological data.

The pattern of the NH_2 terminal amino acids together with certain of the immunological data and the high molecular weights observed led Langdon to consider that the apoproteins may be linked in the membrane to form larger molecules. Previous work had shown that disulphide links are not present in membrane proteins so Langdon investigated the possibility of lysine-derived crosslinks, more usually found in connective tissue (*Biochem. biophys. Acta*, **342**, 292; 1974). He indeed found positive evidence for such links using chemical techniques similar to those used for examining proteins like collagen and elastin.

Langdon's data pose some intriguing and important questions. Are serum lipoprotein apoproteins the major intrinsic proteins of the erythrocyte by virtue of their affinity for lipids? And do they form a structural basis for the covalent attachment of enzymes and other protein types? Are the apoproteins synthesised in the haemopoietic system during erythropoiesis or are they derived from circulating lipoproteins? If Langdon's results are confirmed, they could have far-reaching implications in both membrane biochemistry and in cardiovascular research.

Functions of the cell periphery

by Peter Newmark

By shrewd invention of a title the organisers of a discussion meeting held at the Royal Society on June 19–20 managed to attract a diverse range of speakers on the pericellular environment and its regulation in vertebrate tissues. Within this framework many topics were touched on although most contributions were concerned with connective tissue cells and matrix.

The problems of understanding the metabolism of the matrix are based partly on the complexity of its component proteoglycans, glycosaminoglycans and tensile fibres and partly on separating the synthetic and degredative parts that connective tissue cells play in the turnover of the matrix. H. Muir (Kennedy Institute for Rheumatology, London) reported on the disproportionate importance of the minute amounts of hyaluronic acid recently shown to be present in cartilage matrix. It seems that the distinct proteoglycan aggregates found there are formed by virtue of the affinity of one end of the proteoglycan's core protein for multiple sites on the hyaluronic molecules. An additional important role for hyaluronic acid in cartilage is implied by the demonstration that it inhibits the production of proteoglycans by chondriocyte suspensions, possibly by means of a cell membrane effect.

The role of the membrane in matrix turnover was also raised by the studies of J. T. Dingle (Strangeways Research Laboratory, Cambridge). In addition to reporting on two new cathepsins that may play a part in proteoglycan breakdown, he also elegantly demonstrated that cathepsin D is localised on or around the cell membrane during its stimulated secretion from cells. A functional importance of this phenomenon was suggested by it also being demonstrated in cartilage taken from human rheumatoid arthritic joints. The importance and site of action of the cathepsins, all of which have an acidic *p*H optimum, however, remain to be elucidated.

P. Davies (Clinical Research Centre, Harrow) drew parallels between the development of glomerular nephritis and asbestosis. The activation of lysosomal enzymes by immune complexes in the first case and by asbestos particles in the second disease can result in the breakdown of the basement membrane. This, perhaps with contributory mechanisms, can trigger off the overproliferation of epithelial cells and thickening of the basement membrane that is associated with both diseases. Cell proliferation, in this case of the arterial smooth muscle cells, was also one of the topics discussed by R. Ross (University of Washington). When grown in culture, these cells, like so many others, only proliferate in the presence of serum. It seems, however, that the important growth factor(s) is initially derived from platelets. The importance of this factor could be that, when released from platelets at a site of arterial injury, it causes the local proliferation of subendothelial cells in atherosclerosis.

In a speculative vein J. E. Scott (MRC Rheumatism Unit, Taplow) contemplated the evolutionary forces that may have determined the universality of polyanionic carbohydrates in cell membranes and matrix. In prebiotic times they would have produced an excellent shield against radiation-produced hydrated electrons; with the advent of aerobic conditions they would still be very stable; and, finally, they possess excellent properties for withstanding the wear and tear to which extracellular molecules are subjected.

In his continuing studies of cell–cell recognition, M. M. Burger (University of Basel) has recently turned his attention to a model system, the sponge. Suitable treatment of sponge cells results in their releasing an aggregation factor and a base plate. If the latter is covalently attached to cell-sized solid beads, the addition of the aggregation factor and calcium results in bead aggregation. This neat system promises to assist in the elucidation of the molecular basis of cell interactions.

Orientation to odours by insects

from our Insect Physiology Correspondent

It has long been known that insects fly upwind to attractive odours—mosquitos finding their hosts, drones locating the queen bee, fruit flies seeking fermenting fruit—and that the maintenance of the direction of flight is a visual response to the pattern of the ground below. In other words that anemotaxis in the flying insect is an optomotor response. But a year or two ago (*Science*, **173**, 67; 1972; **180**, 1302; 1973) Farkas and Shorey claimed that in response to the sex pheromone of the female, flying males of the pink bollworm moth *Pectinophora* are guided by a chemotactic mechanism which can operate in still air. The existence of such a mechanism of oriented flight in the absence of a moving air stream (as opposed to a kinetic mechanism dependent on gradients in the concentration of odours, or a visually oriented mechanism in an air stream) could be disturbing for accepted theories of orientation by flying insects.

Kennedy and Marsh now point out (*Science*, **184**, 999; 1974) that in the experiments of Farkas and Shorey the males were already in flight and may already have been oriented anemotactically before the air stream was stopped. They have therefore repeated the experiments on males of the dried fruit moth *Plodia interpunctella* and related moths; the movements of the insects were accurately recorded in a wind tunnel and the results were analysed statistically. The flight of male insects in a stream of air in a wind tunnel could be completely controlled by the