The former seek to avoid the plasma pressure limitations inherent in the Tokamak concept either by grossly distorting the cross-section of the toroidal plasma column, producing a belt-shaped plasma, or by programming the magnetic field in time so that a configuration is produced in which the toroidal magnetic field has opposite directions within and outside the plasma, or by adding windings which remove the axial symmetry of the magnetic field but provide stabilizing "shear". The first approach was described by Zwicker, whose device ISAR IV at Garching is now in full operation, and producing quite impressive plasmas, with pressures comparable with the confining magnetic pressure. The latter approaches are exemplified by the HβTX experiment at Culham and the various precursors to the Scyllac experiment at Los Alamos, which were reported by Jahoda. Conventional stellarators are at present rather out of favour, for their confinement properties seem to be, if anything, inferior to those of Tokamaks, and the cost and complexity of their helical windings are much greater. Some interesting work on the experimental confirmation of a theory due to Volosov of the anomalous plasma loss mechanism was reported by a group from Novosibirsk.

The only non-toroidal confinement configuration which is still a serious contender is the mirror machine. The principal problem which this approach has to face is the high rate of loss of plasma through the mirrors due to Coulomb collisions, and the possible further enhancement of this loss due to collective plasma processes. A theory of this enhancement, due to "loss-cone instabilities", was recently given by Callen and Baldwin of Livermore, and Coensgen reported some rather impressive experiments on the 2×2 device at Livermore which strongly suggest that this mechanism does account for the discrepancy which previously existed between the observed loss rate and the Coulomb collision loss rate, and it seems possible that a similar mechanism might explain the discrepancies reported by two mirror groups at Fontenay-aux-Roses.

Finally, there were a few papers on the radically different approach to controlled fusion, in which one heats the plasma extremely quickly and relies upon inertia to prevent it from expanding before the thermonuclear reactions have taken place. Three fast plasma heating procedures have been suggested —a rapid θ-pinch implosion giving a short lived axial field of several Mgauss, a relativistic beam with a high current interacting with a solid target, or a nanosecond giant laser pulse heating and compressing a spherical solid hydrogen target.

PROTEINS

Concanavalin Observed

from our Molecular Biology Correspondent

CONCANAVALIN is a name that nowadays elicits Pavlovian salivation in cell biologists and others. It is also a protein, one of the so-called lectins, which derive from plants and possess the property of agglutinating erythrocytes and other cells. They do this by binding to saccharide residues on the cell surface and, in the case of concanavalin A, the interaction with saccharides, which depends itself on the presence of calcium and manganese ions, has been studied in great detail. Not long ago, Hardman and Ainsworth (*Nature New Biology*, **237**, 54; 1972) obtained a difference electron density map between crystals of concanavalin with and without inositol and found that the sugar residue is lodged below the surface of the protein, in a cavity. The polypeptide chain contours around the binding site could be made out, and the side chains making up the cavity were identified. The manganese ion, which is indispensable for binding of the sugar, is remote from the binding site. The difference map also reveals that association with inositol causes small structural changes in the protein. An odd feature is that the binding site for an iodophenyl derivative of another saccharide, D-glucopyranoside, studied by other workers, does not coincide with that of inositol.

Now Edelman *et al.* (*Proc. US Nat. Acad. Sci.*, **69**, 2580; 1972) describe their structure of concanavalin A at 2 Å resolution, and concur in this curious finding. The protein, which in solution is predominantly a dimer, packs as pairs of dimers in the crystal, with the monomer as an asymmetric unit. The conformation contains a high proportion of antiparallel β-structure, and hardly any α-helix; β-chains extend across the interface between the subunits of the dimer. The binding sites for the manganese and calcium ions are inferred from peaks of electron density that cannot be accounted for by side chains. The manganous ion, it appears, has a more or less octahedral coordination shell, four carboxylate and one imidazole ligand contributed by the protein, the remaining two presumably by water. The calcium coordination is similar, with one backbone carboxyl and four carboxylate ligands. Two of the carboxylates, identified as aspartate side chains, are common to the coordination shells of both metal ions, which leads to a rationalization of the manganese-contingent binding of calcium, in terms of a stabilization of the local geometry by the manganese.

As to the binding site for the iodophenyl-D-glucopyranose, the iodine lies in a deep cavity, and is surrounded

Clustering of Concanavalin A Sites after Proteolysis

THE nature of the changes in the surfaces of cells, induced by transformation by tumour viruses or by exposure to proteolytic enzymes, which results in an increased susceptibility to agglutination by plant lectins such as concanavalin A is currently a matter of some controversy. One school argues that enhanced agglutinability is caused by an increase in the number of lectin binding sites exposed at the cell surface while the other maintains that changes in the topographic distribution of the sites rather than their number are responsible. In next week's *Nature New Biology* Nicolson, who belongs to the latter school of thought, reports further comparative investigations of the distribution of ferritin conjugated concanavalin A molecules bound to the surfaces of mouse 3T3 cells which have been exposed to trypsin or have been left untreated.

Nicolson reports that ferritin conjugated concanavalin A molecules are found in clusters on the surfaces of 3T3 cells exposed to mild digestion by trypsin whereas they are randomly distributed on the surfaces of untreated 3T3 cells; exposure to trypsin also, of course, renders the cells more readily agglutinable. Both the size and the distribution of the clusters of lectin bound to cells exposed to trypsin resemble those on the surfaces of 3T3 cells transformed by SV40. Moreover, when cells are exposed to extremely dilute trypsin they do not become more readily agglutinable and the ferritin conjugated concanavalin A remains randomly distributed. To show that the clustering of receptor sites is directly involved in agglutination, Nicolson examined thin sections of agglutinated cells and found that the concentration of the tagged lectin was much higher in those regions where the surfaces of two cells were in close apposition than in regions where the cell surfaces were not in contact.

These experiments strongly suggest that cell agglutination caused by polyvalent agglutinins depends at least in part on the clustering of binding sites. Such clustering implies, of course, that membrane components are free to diffuse in the plane of the membrane and presumably clustering is facilitated by mild proteolysis of cell surface. Such digestion might alter the electrostatic properties of the surface as well as the conformation of surface proteins.