tion suggested by Bakay *et al.* and by Croce *et al.* for this phenomenon is to suppose that the mutation causing loss of rodent HGPRT in the parental cells is located in a regulator gene, perhaps comprising a deletion which can therefore never revert. Fusion with human (or with chick) cells restores a regulator product able once again to switch on the rodent HGPRT genes. This would imply that regulator genes in rat, mouse, man and chick may specify proteins sufficiently alike to substitute for each other, a conclusion with implications of obvious importance for models of eukaryotic gene control.

An alternative explanation, however, was considered by Watson *et al.* and is supported by the results reported by Shin *et al.* in *Nature New Biology* (**241**, 194; 1973) earlier this year. Reversion from $HGPRT^-$ to $HGPRT^+$ in rodent cells may depend not upon the formation of a hybrid cell as such but rather on some feature of the fusion process. Shin *et al.* found that A9 and RAG mouse cells, both $HGPRT^-$, were induced to revert at frequencies of 1.8×10^{-8} and 9.0×10^{-8} respectively by subjecting cells to the procedures used in cell fusion, that is bringing the cells to a high concentration and exposing them to a thermal shock.

These results imply that the mutation causing loss of HGPRT in the mouse cells does not represent a deletion, but takes some form which can be reverted under the environmental conditions used to form hybrid cells. Reversion *per se* cannot, therefore, be used as a test for hybrid formation, an important limitation on the use of this technique for genetic mapping. And the hybrid rodent–human cells possessing rodent $HGPRT^+$ activity may, of course, survive on HAT medium because of such reversions rather than because of the presence of human chromosomes.

The immediate implication of these results for cell genetic studies is that the technique of selection for production of enzyme by one partner to make up for a deficiency in the other can be used only when it is possible to distinguish the two enzymes unambiguously, so that reversions may be distinguished from cells possessing the (usually) human enzyme. This may well limit the exploitation of this technique in future. Another implication is that non-reversion of a mutation in normal conditions of growth can no longer be taken to support the concept that the mutation represents a deletion of the structural gene. More than one apparently well established tenet of cell genetics is therefore made dubious by the results obtained so far, and the nature of the $HGPRT^-$ mutation and the reasons for its unusual conditions of reversion may yet cast an important light on the constitution of the mammalian genome.

GENETICS

Environmental Mutagens

from a Correspondent

CONCERN about environmental mutagens is widespread and justified. The problem of devising valid tests for potential mutagens among the vast array of new chemicals in the human environment is an urgent one. It is also beset with great difficulties, requiring as it does extrapolation from data gained *in vitro* to effects suspected *in vivo*; from lower organisms to man; from somatic cells to germ cells; from cell cultures to cells in the normal environment of the body; from easily scored events with little relevance to the population as a whole to the significant genetic changes which are far more difficult to score simply and cheaply. In order to coordinate the efforts of geneticists and cytologists in this area, the Environmental Mutagen Society of the United States was founded in 1969 by Dr Alexander Hollaender; the European EMS was founded in 1970; the Japanese EMS in 1972; and this year (August 31 to September 1) the first international conference on environmental mutagens was held in Asilomar, California.

The meeting demonstrated the rapid and extensive progress in this new field. Topics ranged widely from mutagenesis at the molecular level (F. Sherman, University of Rochester) and the role of repair in the prevention and realisation of mutations (T. Kada, Mishima, Japan; S. Kondo, University of Osaka; B. S. Cox, University of Oxford; F. J. de Serres, National Institute of Environmental Health Sciences, Durham, USA; S. Wolff, University of California, San Francisco; F. H. Sobels, State University of Leiden) to tests of specific drugs or pesticides. It was realised, however, that as in cancer therapy, fundamental work is necessary.

At present an urgent task is to provide industry with easily handled testing techniques and with training courses for personnel. Also important is the construction of a workable test protocol. Papers on a possible tier system of testing were presented by G. Flamm (Food and Drugs Administration) and by C. F. Arlett on behalf of B. A. Bridges (University of Sussex). Much work is directed towards validating and improving the presently recommended techniques. B. N. Ames (University of California, Berkeley) described bacterial strains that are made hypersensitive to mutagens by genetically built-in devices for easy penetration and for lack of genetic repair. U. H. Ehling (Society for Radiation and Environmental Research, Neuherberg) dealt with the problem of correlation in mice between the easily detected but genetically not very hazardous dominant lethals and the gene mutations and small deletions that constitute one of the main genetic risks. The even more difficult problem of extrapolating from somatic cells to germ cells and from one mammalian species to another has been tackled by J. G. Brewen and J. Preston (Oak Ridge National Laboratory).

Equally important for the drawing of inferences from experimental to real-life situations are studies on the effects of dose (dealt with by D. R. Parker, University of California, Riverside, in a general way; by Brewen for rodents; by J. Patterson and D. Clive, NIEHS, for mouse lymphoma cells) and of dose

How to See the Proteins Spin

THE mobility of proteins embedded in bilayers is a preoccupation of many workers in the membrane field. It is known that in many membranes proteins are capable of rather free translational diffusion, and in these circumstances it would also be expected that their rotation would be little impeded. Cone in fact showed by flash experiments that rhodopsin rotates rapidly in the retinal rod membranes.

Naqvi *et al.*, writing in *Nature New Biology* next Wednesday (October 24), point out that any spectroscopically distinguishable species formed from a protein chromophore by electronic excitation or by photochemical reaction can be used as the basis of such measurements, as long as its lifetime is sufficiently long for rotation to occur before it decays. Experiments based on the depolarisation of an excited singlet state are in this regard misconceived, since the lifetime is no more than say 10^{-8} s, whereas in a viscous medium, rotational relaxation times of microseconds and up are to be expected.

With the retinal chromophore of the protein in purple bacterial membrane Naqvi *et al.* find that flash irradiation produces an unknown species of 10 ms lifetime. In the membrane its absorption is strongly dichroic, and the dichroism does not decay measurably during this time. It follows that the rotational relaxation time of the protein is greater than about 20 ms, and is thus much less mobile than rhodopsin in the rod outer segment.

Naqvi and his colleagues are also examining the possibility of performing similar experiments based on triplet states which have a long lifetime, and can occasionally be observed in absorption. Experiments with eosin bound to serum albumin give the right order of rotation rates in viscous solutions. The possibility of using the tryptophan chromophore of the proteins is also being explored.