Book reviews

Journal of Medical Genetics, 1982, **19**, 159–160

Guidelines for the Testing of Chemicals for Mutagenicity

Committee on Mutagenicity of Chemicals in Food, Consumer Products and the Environment. DHSS Report on Health and Social Subjects No 24. (Pp 95. £4.50.) London: HMSO. 1981.

Much of the anxiety about the effects of ionising radiation in causing mutation has been replaced by anxiety about the possible effects of chemical exposure in inducing mutation. It is therefore appropriate that the DHSS has produced a Grey Book on the Guidelines for Testing of Chemicals for Mutagenicity. The point is made that mutagenesis and carcinogenesis may originate in similar processes, but it is nevertheless recommended that mutagenic hazards should be considered in their own right.

Four test procedures are considered: firstly for gene mutation in bacteria; secondly for chromosome damage in mammalian cells grown in vitro; thirdly for mutation induced in either mammalian cells or for recessive lethal mutations in *Drosophila*; and fourthly in vivo tests on a mammal, for example the metaphase analysis of bone marrow or the dominant lethal test in rat or mouse.

The pamphlet contains useful tables of the frequency of chromosomal anomalies, monogenic disorders, and congenital malformations, an extensive bibliography of test procedures for mutagenesis, and a succinct summary of basic genetics for the benefit of lawyers, administrators, and others without biological training who may be concerned with the hazards of exposure to chemical mutagens.

C O CARTER

Genetic Engineering 1

Edited by Robert Williamson. (Pp xi+167; figures + tables. £9.80, \$24.00.) London: Academic Press. 1981.

This is the first book in a series of rapidly published reviews on specific aspects of genetic engineering, and if the other books follow the same pattern it is likely to prove a very useful series indeed. My initial reaction to a review series on a subject that is changing so rapidly was that no matter how fast it is published it could not compete with special review

issues of weekly journals, such as the issue of *Science* devoted to recombinant DNA. Such special issues probably are the best way to present broad reviews of recent discoveries to a wide audience, but the present book fulfils a rather different function.

There is emphasis on the evaluation of different recombinant DNA techniques, and research strategies are often described in sufficient detail for the reader to understand how particular conclusions were reached. This allows the reader, who is not actually using recombinant DNA techniques but understands the general principles, to have some insight into what sort of problems can be solved by these powerful techniques and perhaps to pose some imaginative new questions from his own field of genetics. It would be wrong to imply that this book is a basic introduction to genetic engineering; the reader needs at least a general knowledge of molecular genetics.

The book consists of three long contributions. J G Williams describes the preparation and screening of a cDNA clone bank, that is, the preparation of bacterial cells transformed by a plasmid containing the DNA copy of an RNA molecule. Like the other contributions, this one ends with a brief look at the future possibilities. The challenge, it seems, is to improve the screening of cDNA or clones so that eventually it will be possible to isolate a sequence from mRNA that represents less than 0.1% of the total mRNA population of the tissue. Many mRNAs of great interest to medical geneticists are in this category.

P F R Little describes the application of restriction enzyme mapping in the prenatal diagnosis of the haemoglobinopathies. He works through several simple mapping experiments that employ the Southern blotting technique. Reliable prenatal diagnosis of a homozygous affected fetus can either be by direct detection of the gene deletion, as in α -thalassaemia, or by demonstrating that the fetus has inherited the same two lengths of DNA containing the gene locus in question as a previously affected family member. The latter approach relies on the existence in the family of polymorphic variation at a endonuclease restriction site and classical linkage analysis.

In discussing linkage analysis, Dr Little rightly distinguishes between those situations where there is extreme linkage disequilibrium, as with the sickle gene and a *HpaI* restriction site nearby, and those