inheritance of Hunter disease the authors rightly suggest that this might exist in females in association with a chromosome anomaly. However the two patients cited here were in fact due to multiple sulphatase deficiencies.

The glycoproteinoses are probably the least common of the lysosomal storage disorders. But, due to the substrate linkages acted upon, they form an interesting link between the sphingolipidoses and mucopolysaccharidoses. Covered in this section also are the steps involved in glycoprotein processing, although this could equally well have been discussed in the following chapter dealing with the mucolipidoses, where a defect in post-translational modification has been identified. But, contrary to the statement made here, carrier detection is possible, but most reliably by assay of the primary gene product rather than by indirect means.

A number of attempts have been made to correct these lysosomal defects and the chapter dealing with treatment provides an honest but rather depressing account of the state of affairs. For most of us the blood – brain barrier provides an important protective mechanism but for patients with neurological involvement it presents a major obstacle to effective enzyme replacement therapy. However, patients without brain involvement may benefit from treatment such as bone marrow transplant. In the future, as pointed out, this may provide a useful vehicle for gene correction by DNA-insertion into stem cells, but such techniques are likely to be tested out on animal models first and a useful list of these is appended. However, we must hope that the confusion shown by these authors concerning $\alpha$- and $\beta$-mannosidosis in Angus cattle or Nubian goats is rectified first.

In conclusion then, in many respects this book has achieved its aims of being an up-to-date account of lysosomal storage diseases and their biochemistry. It is good value for money and with the caveat in mind that we should not believe everthing we read, I would recommend it to students and teachers alike; for those of us more intimately involved in the subject, there is still much to recommend it.

GUY T. N. BESLEY
*Biochemical Genetics Unit*
*Department of Pathology*
*Royal Hospital for Sick Children*
*Edinburgh DH9 1LF*

*The Consequences of Chromosome Imbalance: Principles, Mechanisms and Models.* By CHARLES J. EPSTEIN. Cambridge University Press, 1986. £45·00, $59·50. Developmental and Cell Biology Series No. 18. ISBN 0 521 25464 7.

Other books concerned with aneuploidy have been published, but Professor Epstein is to be congratulated for covering an area of the subject which is so extremely complex that it has previously been largely omitted from the reviews. By gathering together a vast amount of information from the literature (nearly 100 pages of the book are devoted to references alone), and calling on his own expertise in the field, he has addressed the major question of how and why chromosome imbalance is deleterious. Earlier volumes have concerned themselves mainly with the much more straightforward issues of how aneuploidy might arise, and its frequency in the human population in which it is such a major source of genetic burden. Here, the major concern is to try to reduce the complex phenotypic effects of an aneuploid state to separable elements which can be attributed to the imbalance of a specific gene locus or sets of loci. In doing so, the author also suggests that more will be learnt about normal development and function.

Early in the book, the author considers what general principles, if any, are to be deduced from all the available clinical data on human syndromes, pointing out the problems of subjective reporting of morphological defects which so often make the data difficult to interpret. Characteristic, albeit, variable syndromes are nevertheless associated with different chromosome abnormalities, in spite of the fact that few if any physical features are exclusive to any particular chromosome anomaly. Distinctive features arise also for example, from very small deletions such as the del 15q11-q12 or 13 in Prader–Willi syndrome.

Central to the understanding of how monosomy and trisomy for various loci or segments of chromosomes produce their consequences are gene dosage effects, and a section of the book is devoted to a consideration of the secondary consequences of decreasing or increasing enzyme activities by about 50% on metabolic reactions in which they are involved. The pros and cons of the suggestion that aneuploidy produces its effects by altering rates of cell proliferation are argued out.

Animal models for the study of mammalian and human aneuploidy receive a good deal of attention, and a clear account of the use which can be made of stocks of mice carrying Robertsonian translocations with mono-brachial homology in the generation of monosomic and trisomic conceptuses, is given. The mouse is still seen by the author as the best available model system for the study of the disturbances associated with aneuploid development in man. This in spite of the fact that no whole chromosome homologies (with the possible exception of the X chromosome) are to be found between the two species: Homology has been shown however, for many small syntenic groups of genes. Finally, the book deals with three specific clinical problems in human aneuploidy, namely trisomy 21 (Down syndrome), XO (Turner syndrome, gonadal dysgenesis) and the role and importance of aneuploidy in cancer progression, in the latter case, an understanding of the mechanisms involved perhaps being relevant to the prevention or control of the effects.

After reading the book, one is impressed by the way in which the author has tackled such an enormous and complex area of research. It nevertheless leaves one with the feeling that a long road still lies ahead in the understanding of development to the aneuploid phenotype, with a great deal of research still to be done. Nevertheless, it could act as a springboard for further work, and I feel sure that the volume will serve as a most useful addition to the collections of cell biologists, developmental geneticists and clinical cytogeneticists.

ANN C. CHANDLEY
*MRC Clinical and Population Cytogenetics Unit, Western General Hospital, Grove Road, Edinburgh EH4 2XU*

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