In spite of all these criticisms, this volume has many advantages. Someone wanting a book dealing with the whole subject would not go far wrong in using it. Even the workers who prefer to have separate monographs dealing with each of the aspects of this subject would find this book an excellent reference volume, particularly as a source of German literature on the subject.

Dr. J. L. Raven is to be congratulated for having carried out a superb translation from the German.

R. B. McCONNELL

Biochemical Methods in Red Cell Genetics. Edited by Jorge J. Yunis. (Pp. xiii + 530; illustrated + tables. \$25.00.) New York and London: Academic Press. 1969.

The time when the genetics of red cells were equated with the knowledge of blood groups is long past, and this book deals with many inherited red cell characters, none of them being a blood group. Yet even this book is not fully comprehensive because there are many characters which are at present only inadequately described and not yet ready to be included in a work where they would have to be discussed with some finality.

Many inherited red cell abnormalities, some of which cause haemolysis, are errors of the metabolism of glucose. Thus it is appropriate that the first chapter by J. J. Yunis and W. Yasminch deals with the glucose metabolism of erythrocytes and describes which metabolic steps are more sensitive to ageing on storage than others.

G. Motulsky and A. Yoshida deal with the methods for the study of red cell glucose-6-phosphate dehydrogenase deficiency, and there is also a special study by E. Beutler of G6PD activity of individual erythrocytes and the mosaicism for this particular abnormality. The locus for the gene of G6PD deficiency is on the X chromosome, of which women have two. As one of the cell's two X chromosomes becomes functionless, some of the red cells of women who are heterozygous for an abnormal G6PD gene are enzyme deficient and others are not. Glucose-6-phosphate dehydrogenase deficiency is not, of course, the outcome of the absence of this enzyme but of a replacement of the normal enzyme by an abnormal variant of shorter life span. In older cells, therefore, glutathione is not protected from oxidation and, together with ATP, reduced glutathione is essential for the integrity of the red cell.

A chapter by H. K. Prins and J. A. Loos deals with this role of glutathione, and one by G. J. Brewer with glutathione reductase and also with 6-phosphogluconate dehydrogenase. ATP is formed by transfer of phosphate from a product of the Embden-Meyerhof cycle, phosphopyruvic acid. Difficulties can arise where there is a deficiency of pyruvate kinase, and these are discussed by K. R. Tanaka. Phosphopyruvic acid is derived from triosephosphate, and triosephosphate isomerase deficiency is described by A. S. Schneider. The role of ATP, its synthesis and regeneration, and genetic studies of the variation in concentrations of ATP are presented by G. J. Brewer. A chapter by E. R. Jaffé

deals with the deficiency of DPNH-methaemoglobin reductase (diaphorase) which causes hereditary methaemoglobinemia because methaemoglobin is not reduced to haemoglobin. Normal red cells are rich in catalase, and this enzyme is thought by many workers to protect glutathione against oxidation by H_2O_2 . H. Aebi and H. Suter describe the enzyme and the inheritance of actalasaemia. A frequent cause of jaundice and brain damage in infants is absence of galactose-1-phosphate uridyl transferase, which is described by E. Beutler. Other genetic markers reviewed in this book are the esterases and carbonic anhydrases (R. E. Tashian) and the red cell phosphatases, phosphoglucomutases, and adenylate kinases described by D. A. Hopkinson and H. Harris. G. J. Brewer and C. F. Sing provide a brief general review of isoenzymes of human erythrocytes and their genetic variation. A comprehensive and informative chapter on human haemoglobins by T. H. J. Huisman concludes this wide survey.

As can be expected from the title of the book, all contributors not only give an up-to-date account of the subject but they support them with much technical information and numerous references.

This is a most valuable volume written by the foremost authorities in the field, and both as a work of reference and as a textbook it should find its way onto the shelf of every human geneticist.

H. LEHMANN

The Handling of Chromosomes. By C. D. Darlington and L. F. LaCour. 5th Edition (revised and enlarged). (Pp. 272; illustrated + tables. 60s.) London: Allen and Unwin. 1969.

The Handling of Chromosomes was first published in 1942 and has now been revised and brought up to date five times. The preface to the fifth edition states, 'The chromosomes are responsible for heredity and variation and they control development. Their study has therefore become necessary for teaching and research in all parts of the life sciences. This book attempts to show how they have to be handled for such purposes.' The attempt has succeeded and the book is a must for all teachers and students of cytogenetics. The advent of human cytogenetics in the past 15 years has tended to overshadow much of the solid work carried out in the chromosomes of plants and insects. To the student, this manual briefly and concisely gives the equipment, techniques, and sources of material required for the study of the chromosomes of numerous different species of both plants and animals, and includes in this some of the more basic techniques and schedules for the study of human chromosomes. To those of us who have tended to become immersed in human cytogenetics, it refreshingly reminds us that man is yet another species and must take his place with *Tradescantia*, *Trillium*, *Drosophila*, *Alium*, and *Locusta*—classic species of the cytogeneticist. The book is illustrated with simple line drawings and has, as well, a superb section of chromosome photographs.

This reviewer has known this compact little manual since, as a student, he purchased the second edition in 1948 when, under one of Professor Darlington's students, he was first introduced to the fascinating subject of chromosomology. At this time it was the only manual on the subject. Now there are several, but none which covers the field so widely, clearly, or well. The authors are to be congratulated on the skill with which they have up-dated the volume which has earned its right to a place among the classics of Genetics.

JOHN L. HAMERTON

Genetic Load. Its Biological and Conceptual Aspects. By Bruce Wallace. (Pp. xi + 109; figures + tables. 60s.) New Jersey: Prentice-Hall International. 1970.

This book is an instructive account of one of the theoretical problems of population genetics. The concept of genetic load was, Dr. Wallace reminds us, introduced by H. J. Muller in 1956 to assess the extent of man's load of heritable defects. This has been split into the 'mutational' load due to mutant genes with obvious deleterious effects on the carriers, which are maintained by mutation, and the 'segregational' load due to mutant genes which are retained in the population because they enhance the fitness of their heterozygous carriers. The load was later (1956) defined by Crow and his colleagues as the proportionate decrease in the average fitness of a population relative to that of the optimal genotype.

If the fitness of the optimal genotype was regarded as unity, then the average fitness of the population where there was much polymorphism would be extremely low if the fitness of the homozygotes was even only, say, nine-tenths of the heterozygotes. Certainly the situation approached for the gene locus for the β peptide of haemoglobin in West Africa, with the S locus responsible for the early death of 1% of livebirths, is not one that could hold at many gene loci. Experimental work, however, in *Drosophila* and in man indicates that polymorphism is widespread and may be present at 30–40% of all gene loci. The conceptual difficulty arises, as Dr. Wallace and others have suggested, by regarding the fitness of the optimal genotype (the very rare individual with maximum heterozygosity) as unity and the rest as having a load in relation to them. The problem largely disappears if the individual with average fitness is regarded as having unit fitness, with fitness having something like a Normal distribution with variation above and below the population mean according to the degree of heterozygosity; very fit and very unfit individuals are exceedingly rare.

Dr. Wallace makes two further points. Firstly, in a population occupying a finite ecological niche genetic deaths and environmental deaths substitute for each other. More genetic deaths could lessen the pressure of numbers on resources (for example water or food supply) and result in fewer environmental deaths, leaving population size little altered. Second, the introduction of an allele into a hitherto homozygous population may, if

the heterozygote has greater fitness, enable the population size to increase though the genetic load (in terms of the Crow definition) has also increased. In terms of the Crow definition a population consisting entirely of albinos would have no genetic load from the gene locus involved.

The medical practitioner who, like Garrod, is conscious of the infinite variation in human beings, which must depend on multiple allelism at many loci, will find himself in sympathy with Dr. Wallace's thesis.

CEDRIC CARTER

Ophthalmic Genetics. By Arnold Sorsby. 2nd Edition. (Pp. x + 269; 235 figures + 11 tables. 110s.) London: Butterworths. 1970.

Professor Sorsby's book 'Genetics in Ophthalmology' was published in 1951, since when there have been such considerable advances in this subject that this second edition has been almost completely rewritten and given a new title. The book is in three sections: the globe as a whole, individual tissues, and generalized disorders with ocular aspects.

The first section deals with affections mainly due to arrested development (the colobomata complex, the microphthalmos complex, the complex of anterior segment anomalies), buphthalmos and glaucoma, refraction, and motor anomalies. Not surprisingly, the chapter on refraction is a concise and masterly exposition of subject to which Sorsby has contributed so extensively.

The second section contains chapters on the cornea, the lens, the anterior uvea, the retina and choroid, the optic nerve, and other tissues. Sorsby's contributions to ophthalmic genetics are again evident, particularly in the chapter on the retina and choroid where pigmentosa, both typical and atypical, is discussed informatively.

The third section deals briefly with the ocular aspects of metabolic disorders, systemic disorders, and syndromes.

This is a concise textbook of ophthalmic genetics and as such fills a gap in medical libraries. Partly as a result of its concise nature it contains a few statements that are confusing or controversial. A clear distinction is drawn between congenital and abiotrophic defects, yet one wonders whether this distinction is necessary. Retinal aplasia is used as the name for a specific entity, and also for certain of the atypical forms of retinitis pigmentosa; this is confusing. Glaucoma is stated as being dominant (p. 11), while its possible monofactorial or polygenic transmission is discussed on p. 36. Haldane's suggestion of dominant partial sex-linkage for one family with retinitis pigmentosa does not appear to merit mention; this family demonstrates autosomal dominant transmission. Certain of the terminology is old-fashioned, or even incorrect (choroidal sarcoma, p. 5), and there are several typographical errors in the text. Despite these minor criticisms this book can be recommended as being the most concise and readable text available on ophthalmic genetics.

BARRIE JAY