

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  $\beta$  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.

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