White Fife, bzw. Tuscan × Hunters oder White Fife × Hunters in ihrem Allelgehalt durch Kreuzungen mit der mutanten Linie getestet würden. Die bisher von Frankel vorgelegten Daten stehen mit dieser Hypothese durchaus im Einklang, sie genügen jedoch nicht als Beweis für ihre Richtigkeit. Sicherlich ist aber das Zusammentreffen des Chlorophylldefektes mit der Strukturmutation nicht zufällig. Dies bezieht sich auch auf den Hinweis, dass eine Linie mit einem telozentrischen Chromosom, bei dem der Arm mit der Duplikation fehlt, chlorophylldefekt ist : nicht " incidentally ", sondern " necessarily ".

(1) FRANKEL, O. H. 1949. A self-propagating structural change in Triticum I and II. *Heredity*, *3*, 163-194, 293-318.
(2) FRANKEL, O. H. 1950. A polymeric multiple gene change in hexaploid wheat. *Heredity*, *4*, 103-116.
(3) FREISLEBEN, R., UND LEIN, A. 1943. Vorarbeiten zur züchterischen Auswertung röntgeninduzierter Mutationen I und II. *Zts. f. Pflanzenzüchtg.*, *25*, 235-283.

## REVIEWS

**INHERITANCE IN DOGS, with Special Reference to Hunting Breeds. Ojvind Winge. 1950. Comstock Publishing Company. $3.50.**

A new book by Ojvind Winge cannot fail to be something of an event in genetic literature, for perhaps no living author has added so much to the diversity of genetic knowledge, or to our understanding of such a variety of genetic situations. It is particularly valuable that he has in this book put together what is known of the genetics of the dog, which has shared with many other domestic animals a neglect from which it is quite time they were rescued.

Most conclusions in this field are without explicit experimental verification, for I believe that nowhere have identifiable genes been deliberately collected, whereby authentic specimens of the various breeds might be tested. Knowledge has been accumulated piecemeal, and identifications have not always been verified by breeding tests. Nevertheless, the author has been able to list sixteen factors affecting colour, and to give some account of their interaction, and these should cover the principal segregations observable in the breeding of gun-dogs, to which the illustrative material is principally devoted. This list is a step in the direction of a standard notation for dog genetics, though no very radical reform of current usage is here proposed. For example, the extreme dilution of the Samoyed, caused by a recessive gene *f*, is here spoken of as albino, although true pink-eyed albinos admittedly occur, and are ascribed to a different recessive gene. It is not stated whether or not it is allelomorphic.

A clear introductory account of genetic principles is given, with illustrations of factor interaction and epistasy. This should enable the intelligent dog breeder to see what he is doing, and to make an analysis of his stocks so far as colour inheritance is concerned.

No case of linkage is mentioned, apart from Hutt's remarkable finding of sex-linked hæmophilia. This is not surprising as there are 38 pairs of autosomes. Analogies with colour factors in other species are not discussed, though the factor for white mottling, *t*, is closely analogous to the factor *s* in mice for recessive pied, and this analogy discourages the suggestion that the great range in the extent of colour in mottled dogs is due to multiple

alleles at one locus. All the factors listed have two known alleles except the *C*-series, including black and tan, in which six are listed. The albino-like series, smoked—Burmese—Siamese in cats seems to have no clear analogue in dogs. One could wish that the author should some day consider the canine factors with the analogy of other mammals in view.

The book is finely produced with helpful colour-plates.

R. A. FISHER.

PAPERS OF THE ROYAL COMMISSION ON POPULATION. Vol. IV : Reports of the Biological and Medical Committee. H.M.S.O. 1950. Pp. iv 52.

The Biological and Medical Committee's terms of reference were " to formulate for the assistance of the Royal Commission on Population, the biological and medical factors relevant to the Commission's enquiry and generally to advise the Commission on the biological and medical aspects of the inquiry."

The Committee divided the field between (1) reproductive wastage and (2) human fertility. The first of the three papers in the present volume deals with *Reproductive Wastage* which is due to five main causes : induced and spontaneous abortion, stillbirths, neonatal mortality and mortality between 4 weeks and 12 months, accounting for a total wastage of from 15 to 22 per cent. of all children conceived. The uncertainty in the estimate is largely due to the difficulty of estimating the extent of abortion, which contributes a wastage of from 9 to 16 per cent. So far as the other groups are concerned, 2·3 per cent. is due to stillbirths, 2·0 per cent. to neonatal deaths, and 1·4 per cent. to deaths between 4 weeks and 12 months. The possible saving due to a reduction in spontaneous abortion is considered to be small, and while induced abortion could no doubt be materially diminished by an increased knowledge of birth control methods, this would not lead to an increased number of births. Stillbirths and infant deaths all show appreciable social grading, being greatest for Class V and least for Class I. If the social and medical welfare available to the two top classes were made generally applicable then it is considered likely that wastage from those sources could be reduced from $5\frac{1}{2}$ to 4 per cent., giving an additional recruitment to the population of $1\frac{1}{2}$ per cent. The actual extent to which this gain could be realised depends, as the authors point out, on whether parents tend on the average to produce families of a predetermined size or whether they are discouraged by infant deaths. The former would tend to cancel the effect of a reduction in death rates, while the latter would enhance the additional recruitment obtained. There would probably be some average gain, but it is uncertain just how much. Another effect, which might depress the expected gain, is that the social gradings observed may be due to genetic rather than environmental differences. In the opinion of the reviewer, the report, which concludes " that there is no satisfactory evidence to show that social class differences in survival have an hereditary basis ", dismisses this possibility too readily. The facts discussed, while certainly consonant with the view that environment is the major influence, are by no means incompatible with a mainly genetic interpretation, in spite of assertions to the contrary. We do not know what would happen if we performed the crucial experiment of transferring at birth large numbers of children from Class V to a Class I environment, and conversely. Moreover, the death-rate from congenital malformations varies in the ratio of 1 : 1·4 from Class I to Class V. The