ABO Blood Groups in Sarcoidosis

THE distribution of the ABO blood groups is interpreted by Vogel (1961) as the outcome of a dynamic equilibrium between the selective action of epidemic diseases of the world-populations and selection through materno-fœtal incompatibility.

The methods were described at the first and especially at the second International Congress of Human Genetics. The results were reported of investigations being conducted on the association of the ABO blood groups with the varial diseases. Our own investigations regarding the ABO blood groups in sarcoidosis are based on a sample of 518 patients suffering from these diseases.

Table 1. RELATIVE INCIDENCE OF ABO BLOOD GROUPS IN SARCOIDOSIS AND TUBERCULOSIS

	No. of sample control	No.		Comparison	Relative incidence Type A	χ^2 ($m = 1$)	P Probability	P Heterogeneity
		Patients	Controls					
Sarcoidosis	1	518	81,985	A : O	1·142	15·22	0·0001	—
Tuberculosis	10	4,505	19,883	A : O	1·1360	12·19	0·0005	0·06

The complete material for ABO blood groups is presented in Table 1. The distribution of ABO blood groups in patients affected with sarcoidosis is compared with the distribution of ABO blood groups in the Federal Republic of Germany. These statistically significant results demonstrate that the relative incidence of type A is 1·142 as against 1 in type O. It means that persons with the blood group A had a 14·2 per cent more frequent probability to acquire a sarcoidosis than persons with the blood group O.

In this connexion it is very interesting that in basal tuberculosis, too, a small preponderance of the blood group A exists. In a second investigation we were able to evaluate the data in 10 samples of former scientific publications. But we could not find a relation of sarcoidosis with the rhesus blood groups and the serum haptoglobins.

So it seems that the gene of the blood group A in the multifactorial genetic systems of sarcoidosis is of some importance.

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Mutant Expression and Canalization

EXPERIMENTS in which mutant expression was altered by selection have shown that changes in expression are more easily brought about over some parts of the range than others[1–4].

Explanation of this phenomenon, which was interpreted as a consequence of the canalization of normal development, implied that the developmental system governing the expression of the character has at least two components.

One component is sometimes represented as a morphogenetic substance[4,5]. Change of concentration of this hypothetical substance (by environmental factors or selection on expression) would cause a change in mutant expression. The amount of this substance is supposed to vary in different members of a population in a regular and continuous manner. The differences in sensitivity to change (canalization) of expression at different levels of expression, which sometimes result in irregular frequency distributions of the phenotypes[1,4], are a consequence of a second component.

This second component relates the amount of the morphogenetic substance in an individual to the phenotypic expression of the mutant in that individual. The genetic basis of this component must be—at least in part—independent of the genetic basis of the first component[4]. This second component would cause characteristic response curves relating factors which change expression with the magnitude of change in expression. It has been supposed that the mutants involved in the investigations quoted here cause only a decrease in the amount of morphogenetic substance and do not affect the response curve.

I have found[4] that in the mutant *cubitus interruptus* dominant (ci^D) of *Drosophila* genetic differences, differences in environment (temperature) and also differences operating within flies, all act according to the same characteristic response curve. If ci^D affects only the level of the morphogenetic substance, then the response curve for ci^D expression would represent the response curve of the developmental pathway to a normal longitudinal wing vein (canalization cross-section[6]). But neither these experiments nor similar experiments with other mutants could prove that the mutant had no effect on the response curve[4,7].

If the response curve of ci^D represents the canalization cross-section of fourth vein development in the normal fly, one should expect that similar curves can be obtained with certain other mutants which cause a terminal interruption of the fourth vein.

Therefore, two other mutants were tested in temperature experiments, namely, ci^{D-G} (*cubitus interruptus* dominant of Gloor, 4–0·00) and H (Hairless, 3–69·5; in a stock selected for complete penetrance of fourth vein expression).

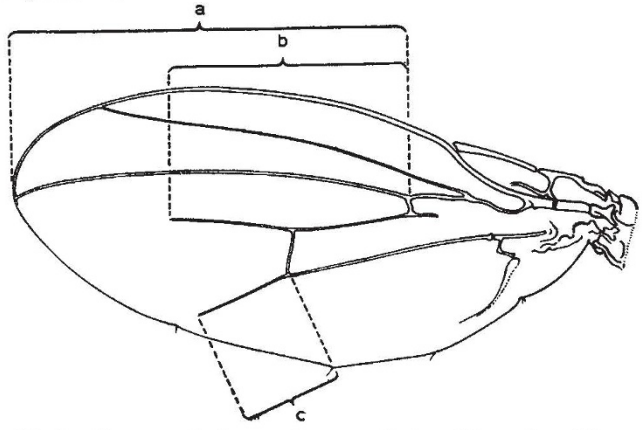

Fig. 1. Measurement of expression on a ci^D-wing. Expression of the 4th vein interruption was measured as the percentage ratio of the length of the 4th vein (*b*) to the length of the 3rd vein (*a*). Expression of the 5th vein interruption as the ratio of the 5th (*c*) and the 3rd vein (*a*)

The expression was measured as indicated in Fig. 1. At each temperature four vial cultures were reared each stocked with 70 larvæ. From each culture 20 females were measured.

The resulting response curves are given in Fig. 2 along with curves obtained earlier for two ci^D stocks with different expressions[4].

The response curves both of ci^{D-G} and H differ basically from the ci^D curves. In ci^{D-G} response is almost linear over the whole expression range. There is no indication of a region of non-linearity around the 70 value which is characteristic for ci^D (ref. 4). Furthermore, there is no indication that on approaching wild-type in expression, further response is becoming more difficult.

In the case of H, change of expression is even facilitated as wild-type is approached.