NOTES AND COMMENTS

THE COMPLEX TRAIT AS A GEOMETRIC CONSTRUCT

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> ". . . I have studied the inheritance of a large number of characters which might be expected to show heterosis from *a priori* reasoning, but which do not show it. They are characters determined by a small number of genes and affecting particular organ systems "—E. M. East, 1936.

If a complex trait is the result of multiplicative gene action, it has a geometry. There is good reason to believe that such geometries are widespread. They are apparent in small grain where yield has been treated as the volume of a rectangular parallelepiped with edges X, Y and Z equal to number of heads per unit area, number of kernels per head, and average kernel weight, respectively (Grafius, 1956, 1959). Other geometries have been described for yield in corn (Grafius, 1959) and may be inferred from the classic work of Powers (1952) on yield in tomatoes and in the recent work of Williams (1959) also on tomatoes. This geometric attribute is not confined to plants, since the work of Gowen (1952) clearly demonstrates the possibility in the case of egg production in *Drosophila.*

Similarly Jinks (1955) has found evidence of non-allelic interactions which are quite probably of a geometric nature. He reports non-allelic interaction for height in *Nicotiana rustica*, and for yield in corn on the basis of the published data of Kinman and Sprague (1945), Nilsson-Leisson (1927) and Stringfield (Hull, 1946) and for yield in flax, Carnahan (1947). Jinks makes this penetrating observation, " Wherever we find evidence of overdominance we also find non-allelic interaction ".

In all of the cases that Jinks reported where non-allelic interaction occurred it was associated with a complex trait such as yield or height. On the other hand, the cases where he found no evidence of non-allelic interaction involved less complex traits such as flowering time, weight of individual fruits or shape of fruit.

The inference is clear. This multiplicative interaction could well be the result of the geometry of the complex trait. This is not to say that traits such as fruit weight do not have components, but only that with our present precision of measurement there is no evidence of multiplicative interaction.

A question arises regarding the apparent tacit assumption that all multiplicative interaction is between the components or edges of the geometric image and not at the locus level as in the classic sense. The multiplicative action need not be all one or the other, but for complex traits such as yield the apparent effect is not inter locus but between the edges of the geometric figure. In point of fact inter locus multiplication in the

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classic sense may lack the explosive effect necessary to explain the massive quantitative increase exhibited by complex traits such as yield of an F_1 hybrid in corn.

Starting with a simple system, let the various $A_1 \ldots A_n$ loci interact with the various $B_1 \ldots B_n$ loci in pairs and with summation of effects between pairs, *e.g.* $A_1B_1 + A_2B_2 + \ldots + A_nB_n$ where the various A_i and B_i represent loci, not alleles. As a contrasting model let the sum of all the A_i effects interact with the sum of all B_i effects. Then it is apparent that

$$\sum_{i=1}^{n} A_iB_i < \sum_{i=1}^{n} A_i \sum_{i=1}^{n} B_i. \quad [1]$$

Hence epistasis in the classic sense, as shown on the left of the inequality, is a much less potent hypothesis. It could be argued that there may be other possible types of inter locus interaction. For example

$$A_1 \times A_2 \times A_3 \times \ldots \times A_n \times B_1 \times B_2 \times B_3 \ldots \times B_n,$$

which under some conditions could greatly exceed the right hand side of [1]. One can only state that such a system would be extremely vulnerable to a locus failure and also that there is no evidence for such a system, while both types in [1] have been demonstrated for a wide variety of organisms. Other systems could include the combination of both sides of [1], or the combination of both additive and multiplication gene action, and so on. Gene action in a complex trait need not be restricted to any one type but it is proposed that major heterotic effects will be associated with the right hand side of [1].

For obvious reasons the classic type of epistasis will be designated " geometric-additive ", while the right hand side of [1] will be called " additive-geometric ".

As it now stands the additive-geometric model permits the restoration of the dominance hypothesis to a position of eminence. No satisfactory explanation has ever been advanced to refute the crucial * experiment of Richey (1931) except to show that dominance *per se* could not account for the degree of heterosis found in many instances (East, 1936 ; Hull, 1946 ; Crow, 1952) the inference being that heterosis must be due to a physiological stimulus between alleles. Admittedly, intra-allelic interactions have been demonstrated for simply inherited traits (Quinly and Karper, 1948) but in so far as the complex trait is concerned the additive-geometric concept appears, in combination with relatively small amounts of dominance for each edge, to offer a more logical explanation for the heterosis puzzle. For it should be evident that relatively small amounts of dominance at each edge could, when multiplied together, readily account for F_1 vigour in a complex trait.

An attempt has been made to illustrate the usefulness of a geometric concept in predicting components of complex traits due to multiplicative gene action (table 1). The multiplicative gene action must result in areas, volumes or higher dimensional products, otherwise the products are non-sense. Some of these values are exact, as for example yield in small grain where the product of the three components is identically yield. In this case

* The idea of the convergent improvement experiment being crucial was borrowed from W. H. Leonard.

TABLE 1

Geometrics of complex traits

Trait	Postulated components
Height	Number of internodes × average length of internode or alternately, average cell length × average number of cells on the long axis of the plant
Leaf shape as a ratio of length/width	Length × 1/width or alternately, the product of length and 1/width as functions of cell diameters and average number of cells on the long and short axis
Total leaf area per plant	Number of leaves × average leaf area
Yield *	
Small grain	Heads/unit area × kernels/head × av. kernel weight
Corn †	Ears/plant × rows/ear × kernels/row × average kernel weight
Tomatoes †	Weight/fruit × fruit/plant or alternately, weight/locule × locules/fruit × fruit/plant
Production of eggs	
Lifetime egg *number*	Length of egg laying period × average daily egg production
Lifetime egg *weight*	Length of egg laying period × average daily egg production × average egg weight
Lodging resistance in small grain	Force the culm is capable of supporting × 1/height

* Note that the geometry here is exact in most instances.
† Assuming constant stand.

the trait is a mental construct and therefore the relationship should be exact. In others, *e.g.* the components of a trait such as kernel weight, it will only approximate the true kernel weight since the kernel is only approximately ellipsoidal.

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THE FUTURE OF MAN: A REPLY

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Professor Darlington's review of my broadcast lectures on *The Future of Man* appears in the December 1960 issue of *Heredity*. In the main, he construes the lectures as an attack upon a " bogeyman ", an " enemy ", an " unidentified antagonist " who practises what I have chosen to call " geneticism ". The length and style of his review, its misrepresentations, and its agitated appeals to an unseen audience (Darlington asks no less than twenty rhetorical questions) combine to suggest that he has identified my unknown antagonist with himself.

Of my first lecture Darlington says that I advocate cohort analysis because " it is likely to succeed where other methods have failed in predicting the future numbers of our population ". The point of the first lecture has therefore escaped him. What I actually said was that " predictions founded upon cohort analysis are somewhat more exact in the sense that one can foresee a little more clearly what follows from one's assumptions; and if these predictions are wrong, as to some extent they surely will be, it will be easier in retrospect to see which assumptions were faulty and which factors changed in unforseeable ways ".

Darlington reviews my second lecture at great length. His philosophic reflections upon it have, for me, a certain self-taught quality that make them hard to follow, but he is particularly contemptuous of my saying that " it is impossible, indeed self-contradictory, that an animal should have evolved into the possession of some complex and nicely balanced genetic make-up which rendered it unfit ". This statement is true, and the term " self-contradictory " is to be taken in its strictest sense. Darlington apparently deplores the ambition to cure phenylketonuria, for if we achieve it, " shall we not in some sense be arranging for a particular type of hereditary imbecile to breed ? " As he does not answer the question, I shall do so for him : No. Darlington must distinguish between the genetic singularity and its somatic manifestations of the first or second order. He writes as if he thought the genes themselves were mentally deficient. Potential victims of phenylketonuria whose metabolic disorder has been circumvented will still suffer