Although the accuracy of this theoretical value of $\bar{H}_{\perp}$ has been increased by direct measurements of electrical and magnetic properties of some pertinent parts, which must be as near as possible like those in Vanguard II, there is little hope for such exact magnetometry as in the case of Vanguard I. For this scientific purpose Vanguard I has the relative advantages of magnetic simplicity, as well as continuing rotation observations, both radio and optical. The practically uniform specular surface of Vanguard II would seem to preclude any rotational observations after the battery life limit shown in Fig. 1.

For future practical considerations, it is interesting to note that, if the magnetic shields used in Vanguard II (of "mumetal," a favorite material for such purposes, since $\mu > 20{,}000$) had not been well saturated by interior permanent magnets, the damping couple would have been thousands of times greater than that deduced here, and the relaxation time shorter by the same factor. In this case the entire magnetic evolution of the artificial rotational motion—damping, precession, and nutation—would have been completed in about a day, and thereafter any such motion would merely follow the local vagaries of the earth's magnetic and gravitational fields.

This remarkable sensitivity of highly permeable magnetic material to its ambient field suggests its possible exploitation for orientation control of space vehicles. The present investigation indicates, by Eqs. 2 and 5, that, if the outer shell had been of mumetal, the satellite would have become directionally "locked" in the earth's field within a second of time. Could not properly designed and oriented rings of mumetal be used as rudders to supplement more complicated gyroscopic devices for direction control of space observatories?

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Potential Genetic Variability of Wild Pairs of Drosophila melanogaster

Abstract. Eleven of 21 wild pairs of *Drosophila melanogaster* tested gave rise to at least 1 fly with crossvein defects out of 1000 F_2 progeny. Considered in the light of additional information, the results support the idea that an individual possesses to a large degree the potential variability of the population of which he is a member.

Any ordinary sexual population, if allowed to reproduce maximally, would soon give rise to a vast array of variant individuals (*1*). Even the descendants of only one pair, under similar conditions, would give rise to a great number of variant individuals. Just how the potential genetic variability of a pair compares with that of a whole population is a question that the experiment reported here was designed to approach.

Rather than attempt the impossible task of cataloging the variation produced in each case, I have taken a single example of phenotypic variation and investigated the proportion of individual wild pairs capable of giving rise to such a variant in the F_2 generation.

In wild populations of *D. melanogaster*, on the order of 1 in 1000 individuals has defective posterior crossveins. This trait has been found in *Drosophila* all over the world, and it is controlled (with rare exceptions) by a number of polygenes, probably about five, found on all three major chromosomes and studied by several workers (*2*). The rare but ubiquitous crossveinless phenodeviants (*3*) are flies in which rather common genes occur in rare combination—a situation rather analogous to that in which a spade is drawn out of a full deck of cards seven times out of ten.

If indeed the genes are so common, it follows that a large fraction of all wild pairs have the potential for producing offspring that include at least a tiny proportion of crossveinless flies. The experiment under discussion was set up to determine whether this is actually the case.

Twenty-one wild inseminated females were collected in their natural habitat, five grocery stores in Ann Arbor, Mich. Such females can be treated as wild pairs. All proved to be fertile. The 21 groups of F_1 flies were then inbred to produce 1000 F_2 flies each for analysis. The number of crossveinless flies in the F_2 generation was noted, each fly being rated as to the degree of crossvein defect, from 0 (normal) to 12 (posterior crossvein completely absent). This procedure involved two departures from natural conditions. First, as stated, the F_1 flies were inbred. Second, the flies were raised at a temperature of 18° ± 1°C. The inbreeding caused an increase in the total number of crossveinless (*cve*) flies observed but probably did not markedly alter the number of pairs giving rise to at least one crossveinless fly. Also, the *cve* genes are better expressed at 18° than at 25°C, but again this does not seriously alter the significance of the results.

Table 1 lists the strains of flies and the number of crossveinless individuals produced. It will be seen that 11 of the 21 strains contain at least one crossveinless fly in the F_2 generation. We may conclude, then, that at least half the wild pairs contain at least one of each gene necessary to produce the rare combination leading to the *cve* phenotype. These results confirm a preliminary experiment carried out in 1958 in which four out of seven wild pairs produced at least one crossveinless fly out of 1000 flies in the F_2 generation.

It is not certain that all crossveinless flies carry the same *cve* genes; indeed, this is improbable in the light of a comparison of certain results obtained by myself and others on various strains selected to breed true for the crossveinless trait (*4*). In addition, no set of *cve* genes has been so completely characterized as to permit a unique formulation of the distribution of these genes in a population.

It can easily be shown, however, that the estimate of "at least half the

Table 1. Crossveinless flies in F_2 progeny of wild pairs; 1000 flies were counted in each case. Letters represent the grocery stores from which the original females were collected. Total percentage of crossveinless flies, 0.6.

Strain	Crossveinless flies (*N*)
AP-1	1
AP-2	2
AP-3	7
AP-4	0
AP-5	1
MD-1	0
MD-2	0
MD-3	36
R-1	0
R-2	9
R-3	1
R-4	0
R-5	5
R-6	0
S-1	0
S-2	0
S-3	0
S-4	45
S-5	0
W-1	7
W-2	5

wild pairs" represents a minimum, for it appears likely that pairs with only one of each necessary gene between them would produce far fewer than 1 crossveinless fly in 1000 F_2 flies.

Support for two suggestions is lent by this experiment: (i) that of Dobzhansky and his co-workers that an individual often possesses an unexpectedly great portion of the total genetic variation of its population (*5*); (ii), that the presently observable steps in evolution are made through new combinations of common genes (*5*), which have therefore already been long since tried and tested by natural selection as members of an individual's team of genes (*6*).

Finally, it is suggested that results such as these should be borne in mind in the consideration of the many polygenic traits in human beings.

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Relationship of Stress-Induced Histidine Decarboxylase to Circulatory Homeostasis and Shock

Abstract. Histidine decarboxylase activity of mouse tissues is increased by stress and by injection of epinephrine and norepinephrine, suggesting a balance between histamine and catechol amines producing a component of circulatory homeostasis. Imbalance during intense stress might lead to failure of circulatory homeostasis and to shock. Reasons for discounting histamine as "shock toxin" may be invalid.

Workers in this laboratory have recently demonstrated that mammalian histidine decarboxylase is an adaptive enzyme (*1*); its activity in animal tissues increases in response to nonspecific stress, for example, treatment with histamine liberators, burns, delayed allergy, treatment with pertussis vaccine (*2*), exposure to cold, and injection with *Escherichia coli* endotoxin (*3*).

Among the most firmly established consequences of stress are discharge of epinephrine from the adrenal and release of norepinephrine from the sympathetic nerve endings. The catechol amines seemed likely chemical mediators of stress-induced histidine decarboxylase activity and were consequently tested.

Mice were injected intramuscularly with 20 μg of epinephrine in oil; controls received oil only. The animals were killed after 6 hours, and tissues were assayed for histidine decarboxylase activity (*4*). Results for skin of control mice were 87, 96, and 117 (av., 100); for skin of epinephrine-treated mice, 339, 348, and 466 (av., 384). Results for lungs of control mice were 42, 94, and 163 (av., 100); for lungs of epinephrine-treated mice, 367, 408, and 510 (av., 428). Thus the histidine decarboxylase activity of mouse skin and lung was increased fourfold (*5*). This has been repeatedly confirmed. Histidine decarboxylase activity of muscle was increased threefold under these conditions; other tissues are under investigation.

Results of a time study of the effects of 20 μg epinephrine in oil were as follows: for skin of control mice, 96, 97, and 107 (av., 100); for mice killed 1 hour after injection, 74, 75, and 84 (av., 78); for mice killed 6 hours after injection, 447, 454, and 575 (av., 492); and for mice killed 24 hours after injection, 97, 104, and 139 (av., 113).

Norepinephrine also increased histidine decarboxylase activity. Mice were injected subcutaneously with aqueous solutions of DL-norepinephrine equivalent to either 20 μg or 60 μg of the *l*-isomer. Each received three injections 2 hours apart. After 6½ hours the skins were assayed. For controls the values were 89, 104, and 107 (av., 100); for mice receiving three 20-μg doses of norepinephrine, 253, 312, and 375 (av., 313); for mice receiving three 60-μg doses of norepinephrine, 436, 493, and 593 (av., 507).

The earlier demonstration (*2*) that activity of histidine decarboxylase increases in response to diversified forms of stress becomes more comprehensible in view of the fact that catechol amines, known to be released in stress, also increase enzyme activity. These observations suggest that there may be a balance between the two catechol amines on the one hand, and newly synthesized histamine on the other, producing a component of circulatory homeostasis which operates under conditions of stress.

It is further suggested that in those conditions of stress where there is ultimately a failure in circulatory homeostasis, for example, traumatic shock, one of these amines may be a causative factor.

The intense adrenergic stimulation preceding stress-induced shock is well recognized; now we have demonstrated that stress increases the activity of the enzyme which synthesizes histamine. Although it has not been proved that increased in vitro histidine decarboxylase activity in the mouse is a measure of the rate of histamine synthesis in the tissues of the living animal, (*6*, *7*) this has been done in the rat (*1*). The importance of the shock problem and the attractiveness of a concept involving new formation of histamine seem to justify speculation based on the assumption that stress increases histamine synthesis in vivo. The hypothesis is therefore proposed that if during stress the supply of either histamine or the catechol amines fails, the remaining amine may be extraordinarily toxic to the cells of the small blood vessels and cause shock. The lethal effect of either amine when acting on such cells in the absence of its natural antagonist may be much greater than heretofore suspected, since in experimental tests on these drugs there is probably always some compensation by release or formation of the antagonist.

There are two obvious possibilities for explaining certain features of some types of shock in terms of an imbalance in the catechol amine-histamine relationship. First, histamine may not be formed in adequate supply in strategic locations, so that it cannot cope with catechol amines from the adrenals and sympathetic nerve endings. In this case, excessive vasoconstriction may produce hypoxia with resulting damage to the cells of the small blood vessels.

Second, intense stress may result ultimately in some degree of depletion of the catechol amines from their depots. Thus a highly active mechanism for synthesizing histamine might be left in operation. Injected histamine can produce shock in animals of many species; if formed throughout major tissues of an animal whose defenses are to some extent exhausted, it may cause severe damage.

Adrenal steroids released during stress also oppose some actions of histamine in some species; however, the degree of depletion and rate of resynthesis of these steroids during stress is not clear and their role cannot be evaluated at this time.

In certain types of shock there is good reason to believe that histamine may be the shock "toxin." Adrenalectomized animals are highly sensitive to shock and to histamine; yet they