The idea of feedback relationship suggested by Moment is probably not different from the familiar model based on the view that the selective value of a genotype (or phenotype) is determined by its frequency in the population. It is known that stable equilibrium may be achieved if a genotype enjoys selective advantage when it is rare and selective disadvantage when it is abundant. Let me illustrate. Consider a series of multiple alleles $A_1$, $A_2$, $A_3$, . . . with frequencies $p_1$, $p_2$, $p_3$, . . . where the sum of the $p$'s is unity. In the simplest case—where the selective value of $A_1A_1$ is $1 - cp_1^2$, that of $A_1A_2$ is $1 - cp_1p_2$, and so on—the stable equilibrium condition under panmixia is reached when $p_1 = p_2 = p_3 = \ldots$. If there are ten alleles, each will have a frequency of 1/10 in the population. The genotypic array of the population under random mating will then be $(.1\ A_1 + .1\ A_2 + \ldots + .1\ A_{10})^2 = .01\ A_1A_1 + \ldots + .02\ A_1A_2 + \ldots$. There are ten homozygous genotypes with a total frequency of .10 and 45 heterozygous genotypes with a total frequency of .90.

The "feedback" model described is, however, not the only one that can maintain such a diversity. If all homozygotes have the same (constant) selection value and all heterozygotes also have the same, but a superior, selection value, the same stable equilibrium condition will be reached. More generally, if we take the selection value of all heterozygotes to be unity and that of homozygotes $A_1A_1$ to be $1 - s_1$, and so on, then the equilibrium condition is $s_1p_1 = s_2p_2 = s_3p_3 = \ldots$. In other words, the equilibrium frequency of allele $A_i$ ($i = 1, 2, \ldots, 10$, say) is:

$$p_i = \left(\frac{1}{s_i}\right) \Big/ \Sigma\left(\frac{1}{s_i}\right)$$

This equilibrium will maintain the same kind of massive diversity as the feedback model. The mere fact of diversity does not discriminate one model from the other. Moment says that it does not seem to be balancing selection where heterozygotes are superior (in selection value) to homozygotes. We really have no way of telling at this stage of our knowledge.

Whatever the actual selection scheme, the diversity in color and pattern may be accounted for by postulating multiple alleles controlling them. If $A_1, \ldots, A_{10}$ is for color and $B_1, \ldots, B_{10}$ is for pattern, each with frequency 1/10, the distribution of the 3025 possible genotypes would be as shown in Table 1. Under these circumstances, we would hardly expect to find two individuals exactly alike in a sample of a few hundred. The nature of the selection scheme is admittedly very difficult to ascertain under experimental conditions, but I hope some breeding work may be done to see if essentially multiple alleles or multiple loci are responsible for the diversity in color and pattern reported by Moment (*2*).

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**Reference and Note**

1. G. Moment, *Science* **136**, 262 (1962).
2. This paper is dedicated to Professor L. C. Dunn in recognition of his long and distinguished contribution to the science of genetics.

1 May 1962

Li is entirely right in emphasizing the need for further data concerning "protective variation" and "reflexive selection," if indeed such things exist. Until more facts become available, I would like to add several brief comments. Although it may be true, as Li suggests, that semicryptic genetic variation is a very common phenomenon among animals, protective variation most certainly is not. My own observations and surveys of the literature as well as correspondence received since the publication of my report in *Science* indicate that protective variation has a frequency among animal species of about the same order of magnitude as the frequency of mimicry.

One correspondent pointed out that the essential concept of protective variation is found in a little book on evolution by P. M. Sheppard (London, 1958). It is, I have found, mentioned there briefly in connection with variation in a European land snail, only to be quickly dismissed in favor of orthodox protective coloration. Happily, this author does suggest that the idea itself is worthy of investigation.

Several correspondents have asked why protective variation should be limited to massive variation. I see no reason why it should be. It is even conceivable that the melanistic phase of the eastern squirrel derives some advantage from this difference from the common form, but I have no evidence that this is so.

In connection with the evidence since 1953 that genetic polymorphism has a basis in selection, it should be remembered that in the best understood cases the "balancing selection" is due either to a fixed advantage of the heterozygote, as in sickle-cell anemia, or to a changing environment, as in *Adalia*. This is rather different from the kind of feedback implied by the term *reflexive selection*, where the selective value of a gene is either positive or negative depending on the relation between its frequency and the frequency of other genes.

The mathematical model offered for reflexive selection should be useful. It ought to be possible to test the applicability of the nonfeedback model because it assumes a stable diversity already in existence which does not change, while the proposed theory of protective variation assumes that any mutation producing a new pattern would enter the system with a selective advantage which would decline as the frequency of the responsible gene increased. Furthermore, the nonfeedback model assumes fixed selection values for homozygotes, lower than values for heterozygotes, while the proposed theory implies that the selection values of genes will fluctuate inversely with respect to frequency and regardless of whether the pattern in question is homozygous or heterozygous.

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4 May 1962

## Radiation-Induced Chromosomal Aberrations and Lethals in Aspergillus nidulans

*Abstract:* The frequency of translocations induced in diploid conidia of *Aspergillus nidulans* by gamma rays was much higher than that of recessive mutants involving a single chromosome. More than half of the surviving nuclei contained viable translocations at radiation doses within the range normally employed for induction of mutants in microorganisms.

Uninucleate conidia from a diploid strain of the normally haploid ascomycete *Aspergillus nidulans*, in which each pair of the eight linkage groups was genetically marked, were irradiated with cobalt-60 gamma rays. The doses ranged from 30 to 50 kr and resulted in survival frequencies of approximately 10 percent to 1 percent. Normal appearing strains grown from the survivors were tested for translocations, recessive lethals, and recessive nutritional mutants. A method based on mitotic recombination was used for the detection of these mutations. It makes

Table 1. Mutations induced in *A. nidulans* by γ-rays.

| Dose (kr) | Analyzed strains | | | Mutations detected in the mutant strains | | | |
|---|---|---|---|---|---|---|---|
| | Total | Normal | Mutant | Translocations | Lethals | Translocation lethals | Auxotrophs |
| 30 | 9 | 4 | 5 | 6 | 0 | 1 | 1 |
| 35 | 3 | 2 | 1 | 0 | 0 | 1 | 0 |
| 40 | 13 | 3 | 10 | 9 | 1 | 4 | 1 |
| 50 | 2 | 0 | 2 | 1 | 0 | 1 | 0 |

use of the fact that in structurally homozygous diploids all chromosomes can be recovered in the hemizygous condition in mitotic haploids (*1*), or near haploids, produced by the process of mitotic nondisjunction (*2*). The diploid strain used contains several markers which facilitate the selection of such haploids (*3*). It is assumed that single chromosomes which cannot be so recovered carry recessive lethals. Complete linkage between two linkage groups leading to the recovery of two types (reciprocal with respect to the markers on these two linkage groups) instead of four is taken to indicate the presence of a viable translocation [as confirmed in several well-analyzed cases (Käfer, *4*)]. When more than two linkage groups show mitotic linkage, further meiotic and mitotic analysis is needed to decide whether simple translocations affecting a common linkage group or aberration complexes are present.

This type of analysis, therefore, permits the simultaneous determination, in a normally haploid species, of the frequencies of two main types of mutations, recessive lethals and translocations, which has not so far been possible in any microorganism. This will permit a direct comparison of the effects of mutagenic agents on fungus chromosomes with the effects on chromosomes in higher organisms where determination of these frequencies is usually used to analyze genetically the action of different mutagens. The viable nutritional mutants and translocations are automatically mapped as to linkage groups and can be recovered in haploids for further analysis (*5*). Recessive lethals are mapped as to chromosome, but no further isolation and analysis is possible, in contrast to Atwood's method for recovery of recessive lethals from *Neurospora* heterokaryons (*6*). Since the presented method is also somewhat more laborious, it is not very suitable for the detection of rare spontaneous mutations (*7*) but will be of value for the measurement of aberration frequencies after mutagenic treatment of fungus chromosomes.

Twenty-seven strains were analyzed, and the results are summarized in Table 1. The most striking feature of these data (*8*) is the small number of lethals not associated with rearrangements (only one) as compared with viable translocations (16 were detected). Aberrations listed as "translocation lethals" (of which seven were identified) were those cases in which only one homologue of each of two chromosome pairs was recovered. This type of abnormal segregation would also be found if two independent lethals had been induced in a single nucleus, but in view of the low frequency of lethals not associated with a translocation this is not likely for most of these cases. It cannot be decided, however, whether the lethal effect is independent of, or caused by, the translocation.

In either case the frequency of induced viable translocations is very high. These translocations are found in more than half of the diploid nuclei that survived radiation doses no higher than those generally used for induction of markers in microorganisms. However, it has to be taken into account that the treated strains are normally haploid (with a smaller number of chromosomes and chromosome breaks) and are therefore likely to show a lower translocation frequency. All the same, it becomes obvious that strains treated repeatedly with x-ray—for example, with the standard dose of 50 kr in *A. nidulans* (*9*)—have a very high probability of containing chromosomal aberrations.

A further category of induced mutations was discovered in the course of this analysis when survival rates for haploid and diploid conidia were compared to determine suitable doses of irradiation. Counts of visible colonies after 24 hours incubation were very similar, showing about 50 percent survival after treatment with 15 kr and about 1 percent after treatment with 50 kr. Prolonged incubation (up to 8 days), however, yielded at the highest doses, from diploid conidia only, large numbers of abnormal colonies, which exceeded by far the number of normal appearing ones at 50 kr. These mutant types, which often show interesting patterns of segregation and produce normally viable types, appear to contain mutations, probably mostly chromosomal aberrations, with a semidominant lethal effect (*10, 11*). Under conditions of crowding they do not form colonies and are lost along with the dominant lethals.

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6. K. C. Atwood and F. Mukai, *Proc. Natl. Acad. Sci. U.S.* **39**, 1027 (1953).
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8. A. Tector, thesis, McGill University (1961).
9. G. Pontecorvo, *Advances in Genet.* **5**, 141 (1953).
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11. The work reported here was supported by a grant from the National Research Council of Canada.
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## Evocation by Fear of a Habit Learned for Electrical Stimulation of the Brain

*Abstract.* Rats were trained to press a lever for electrical stimulation of the brain. After extinction of the habit, fear-producing stimuli (a buzzer or electrical shock to the feet) recalled the rat to the lever, although its lever pressing on these occasions never produced further intracranial stimulation. Operant levels of the lever-pressing habit were also greatly increased for long periods following the fear-producing stimuli. The phenomenon is most striking in rats trained with tegmental electrodes and is almost completely absent in rats trained with electrodes in the hypothalamus. This suggests that electrical self-stimulation in some sites may be caused by an activation of mechanisms underlying normal fear and escape.

Animals will learn to perform habits for electrical stimulation of certain parts of the central nervous system (*1*). The relation of such phenomena to those of normal drive and reinforcement is suggested by the hunger and sex differential effects reported by Brady (*2*), and by Olds (*3*). A similar but clearer relationship with normal drive mechanisms