wild type responses. To obviate effects of the screening pigments in measuring spectral sensitivities the comparisons were made between the autosomal white (brown ; scarlet) and white eyed x-12 (x-12; brown; scarlet). The effects of the mutations brown and scarlet are to eliminate the screening pigments in the eye of the fly and render the eye white[4].

The spectral sensitivities of the autosomal white stock (brown; scarlet) and white eyed x-12 stock (x-12; brown; scarlet) are shown in Fig. 2. The former has two peaks, one at about 500 nm and the other at about 350 nm or somewhat below. These features are remarkably similar to the spectral sensitivity curves obtained from white eyed *Musca*[5] and *Calliphora*[6]. The spectral sensitivity of the white eyed x-12 mutants, on the other hand, has a single broad peak around 400–450 nm. For the sake of convenience the spectral sensitivity of the latter has been scaled up by about 4 log units in Fig. 2. It seems reasonably clear that the x-12 response is not generated by either of the two visual pigments present in a normal fly. Conceivably the visual pigments of x-12 are grossly abnormal. On the other hand, the extremely low sensitivity and the highly distorted waveform of x-12 response suggest that this response may not be due to a visual pigment. Thus either the visual pigments are absent in these mutants or the photoexcitation of the pigments fails to excite the active membrane of the retinula cells even though a normal complement of visual pigment is present. If the latter is true, some crucial step in phototransduction is genetically blocked in these mutants. Inasmuch as phototransduction remains the least understood of visual processes, mutants of this type would be of considerable interest in vision research.

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Selection and Covariance

THIS is a preliminary communication describing applications to genetical selection of a new mathematical treatment of selection in general.

Gene frequency change is the basic event in biological evolution. The following equation (notation to be explained), which gives frequency change under selection from one generation to the next for a single gene or for any linear function of any number of genes at any number of loci, holds for any sort of dominance or epistasis, for sexual or asexual reproduction, for random or non-random mating, for diploid, haploid or polyploid species, and even for imaginary species with more than two sexes

$$\Delta Q = \mathrm{Cov}(z,q)/\bar{z} \qquad (1)$$

The equation easily translates into regression coefficient (β_{zq}) or correlation coefficient (ρ_{zq}) form

$$\Delta Q = \beta_{zq}\sigma_q^2/\bar{z} = \rho_{zq}\sigma_z\sigma_q/\bar{z}$$

It thus has great transparency, making it useful as a tool in qualitative evolutionary reasoning. It can also be applied to non-genetical selection. For example, if students' expectations of passing a certain course vary with IQ and if student IQs do not change appreciably during the course, then equation 1 (with its variables suitably redefined) will give the difference in mean IQ between students entering the course and those completing it (and equation 4 below will apply if IQs do change during the course).

Derivation is as follows. Let P_1 and P_2 be populations of a single species, such that P_1 contains all parents of P_2 members and P_2 consists of all offspring of P_1 members. Let the number of P_1 members be N. We label these with identification numbers $i = 1, 2, \ldots, N$, assigned in any order. Let n_z be the zygotic ploidy of the species for gene A; let g_i be the dose of gene A in individual i (for example, if $n_z = 2$, $g_i = 0$, 1, or 2 according to whether i lacks gene A, is heterozygous for A or is homozygous for A); let q_i be the frequency of gene A in individual i, defined by $q_i = g_i/n_z$; and let Q_1 be the frequency of gene A in population P_1

$$Q_1 = \Sigma g_i/n_z N = \Sigma n_z q_i/n_z N = \bar{q} \qquad (2)$$

where the summations are taken over all members of P_1 ($i = 1$ to N) and $\bar{q}$ is the arithmetic mean in population P_1 (that is, $\bar{q}$ is a population variable even though I use sample variable notation).

Now we turn attention to offspring. A gamete from a P_1 member that contributes genes to a P_2 member will be termed a "successful gamete". Let n_G be the gametic ploidy for gene A; let z_i be the number of successful gametes produced by individual i (= the number of i's offspring); let g_i' be the number of A genes in the set of all of i's successful gametes; let q_i' be the frequency of gene A in this set of gametes, defined by $q_i' = g_i'/z_i n_G$ if $z_i \neq 0$, $q_i' = q_i$ if $z_i = 0$; let $\Delta q_i = q_i' - q_i$; and let Q_2 be the frequency of gene A in population P_2. The following can be seen to hold

$$\begin{aligned} Q_2 &= (\Sigma g_i')/\Sigma z_i n_G = (\Sigma z_i n_G q_i')/\Sigma z_i n_G = \Sigma z_i q_i'/N\bar{z} \\ &= \Sigma z_i q_i/N\bar{z} + \Sigma z_i \Delta q_i/N\bar{z} = [\bar{z}\cdot\bar{q} + \mathrm{Cov}(z,q)]/\bar{z} + \Sigma z_i \Delta q_i/N\bar{z} \\ &= \bar{q} + \mathrm{Cov}(z,q)/\bar{z} + \Sigma z_i \Delta q_i/N\bar{z} \end{aligned} \qquad (3)$$

where the summations are taken over all P_1 members, $\bar{z}$ is the arithmetic mean of z in P_1 and $\mathrm{Cov}(z,q)$ is the covariance (or first order central product moment) of z and q in population P_1. Subtraction of equation 2 from equation 3 gives

$$\Delta Q = Q_2 - Q_1 = \mathrm{Cov}(z,q)/\bar{z} + \Sigma z_i \Delta q_i/N\bar{z} \qquad (4)$$

If meiosis and fertilization are random with respect to gene A, the summation term at the right will be zero except for statistical sampling effects ("random drift"), and these will tend to average out to give equation 1.

Five points about equation 1 will be briefly explained. First, equation 1 in its regression coefficient form can be visualized in terms of a linear regression line fitted to a scatter diagram of z against q. (A linear regression line is the best construction in terms of the population effect ΔQ, even if it gives a poor fit in terms of individual points.) Since the regression line has slope β_{zq}, gene frequency change due to selection is exactly proportional to the slope. Therefore, at any step in constructing hypotheses about evolution through natural selection—for example, about why human canines do not protrude, why deer antlers are annually shed and renewed, why parrots mimic, why dolphins play—one can visualize such a diagram and consider whether the slope really would be appreciably non-zero under the assumptions of the theory. If there is no slope, then there is no frequency change except by Δq effects, and the hypothesis is probably wrong.

Second, equation 1 fails if gene A ploidy is not the same in each P_1 member. Suppose, for example, that the A locus is in X but not Y chromosomes in a species with XX females and XY males. Then Q_1 is redefined as $Q_1 = (\Sigma g_i)/\Sigma n_i$, where n_i is A locus ploidy in individual i (that is, $n_i = 1$ if i is male, or 2 if i is female); and Q_2, q_i, and q'_i are redefined in corresponding ways. If P_1 and P_2 have sex ratios of unity (as is commonly the case at conception), then the following can be derived

$$\Delta Q = \frac{2}{3}\,\mathrm{Cov}(z,q)_F/\bar{z}_F + \tfrac{1}{3}\mathrm{Cov}(z,q)_M/\bar{z}_M \qquad (5)$$

where $\mathrm{Cov}(z,q)_F$ is the z,q covariance and $\bar{z}_F$ is the mean in P_{1F}, the female subset of P_1, and $\mathrm{Cov}(z,q)_M$ and $\bar{z}_M$ apply to the male subset, P_{1M}.

Third, the specifications that were stated for P_1 and P_2 imply a "discrete generations model". This was done solely in order to simplify this preliminary report. Actually equation 1 can be applied to species with overlapping, interbreeding generations, and it is not necessary that P_2 should contain all offspring of P_1 members, nor that P_1 should contain all parents of P_2 members. Departure from the "all parents" condition, however, requires reinterpretation of what ΔQ means, and departure from the "all offspring" condition (meaning all zygotes conceived) must be done with insight to avoid introducing post-conceptual selection on P_2 (for post-conceptual selection would require the use of equation 4 instead of equation 1).

Fourth, as an example of how multiple gene functions can be handled, let us suppose that a regression analysis has given the relation

$$\varphi_i \approx 2{\cdot}3 + 1{\cdot}2q_{iA} - 0{\cdot}7q_{iB} + 0{\cdot}5q_{iC}$$

for the effects of genes A, B, and C on character φ. Then we may decide to define

$$\begin{aligned} q_i &= 2{\cdot}3 + 1{\cdot}2q_{iA} - 0{\cdot}7q_{iB} + 0{\cdot}5q_{iC} \\ \Delta Q = Q_2 - Q_1 &= (2{\cdot}3 + 1{\cdot}2Q_{2A} - 0{\cdot}7Q_{2B} + 0{\cdot}5Q_{2C}) \\ &\quad - (2{\cdot}3 + 1{\cdot}2Q_{1A} - 0{\cdot}7Q_{1B} + 0{\cdot}5Q_{1C}) \end{aligned}$$

and equation 1 will hold for these multiple gene functions or for any other linear function of q and Q for any numbe of genes, if it holds for each gene separately.

Fifth, it seems surprising that so simple a relation as equation 1 has not (to my knowledge) been recognized before. Probably this is because selection mathematics has largely been limited to genetical selection in diploid species, where covariance takes so simple a form that its implicit presence is hard to recognize (whereas if man were tetraploid, covariance would have been recognized long ago); and because, instead of using subscripts as "names" of individuals (as I have done), the usual practice in gene frequency equations is to use subscripts only as names of gene or genotype types, which makes the mathematics seem quite different. Recognition of covariance (or regression or correlation) is of no advantage for numerical calculation, but of much advantage for evolutionary reasoning and mathematical model building.

Some genetical selection cases (such as group selection) and many forms of non-genetical selection require more complex mathematics than that given here. I plan to discuss these and other matters in papers now in preparation.

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Toxicity of Mould-damaged Sweet Potatoes (*Ipomoea batatas*)

THE toxicity for livestock of mouldy sweet potatoes has been recognized for several decades in the United States[1] and, especially, Japan[2]. The few reports available describing toxic manifestations mention pulmonary oedema, emphysema and adenomatosis as characteristic disease signs in cattle involved in natural outbreaks[3–5]. These disease signs have also been attributed to other moulded feed products, silage and lush pasture grass[6]. Somewhat similar effects have been observed when large doses of tryptophan were administered to cattle[7].

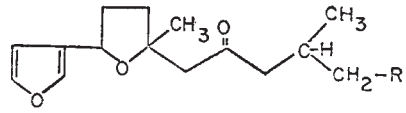

Fig. 1. Structures of ipomeamarone (R = H) and ipomeamaronol (R = OH).

Japanese workers have isolated several abnormal metabolites of sweet potatoes damaged by moulds (particularly the black-rot organism, *Ceratocystis fimbriata*[8]), insects and certain chemicals[9]. The first of these metabolites was a furanosesquiterpene named (+)-ipomeamarone[3] (Fig. 1) later shown to be an enantiomer of (−)-ngaione[10], a hepatotoxin extractable as a normal metabolite from the leaves of the Ngaio tree (*Myoporum laetum*) and other poisonous plants of Australia and New Zealand[11,12]. The toxicity of ipomeamarone for the liver and certain other abdominal organs was reported by Watanabe and Iwata[13]. This response to ipomeamarone, however, does not account for the peculiar respiratory signs that are principal pathological features in the natural disease outbreaks.

We have studied a devastating enzootic of fatal disease of beef cattle in Tifton, Georgia, associated with the consumption of mouldy sweet potato tubers. Postmortem findings were consonant with the respiratory tract pathology described in other outbreaks.

The microbial flora of damaged tubers from the Tifton outbreak consisted of several different bacteria and fungi. Some of the latter belonged to genera containing toxigenic moulds including *Aspergillus*, *Penicillium* and *Fusarium*. No *Ceratocystis* species were detected, although many of the samples had blackish discolorations. Certain toxic sweet potatoes showed only darkened areas beneath the cortex or pithy interiors, without significant colour changes.

Each fungus and bacterial isolate was grown individually on autoclaved sweet potato slurry for up to two weeks. Ethyl ether extracts of these cultures produced no toxic signs when fed to mice by stomach tube, suggesting that the spoilage microorganisms were not in themselves toxigenic.

Ether extract residues of the infected sweet potatoes were dark brown-to-yellow oils, often with pungent odours. Administration of 30–50 mg of this material to mice by stomach tube produced early generalized signs of illness, followed in a few hours by laboured breathing which became progressively more severe. Death ensued 8–24 h after extract administration, following a brief convulsion. At postmortem there was gross and microscopic evidence of lung oedema with accumulation of 1–2 ml. of clear pleural fluid surrounding the lungs. Toxic cellular changes were often seen in the liver, spleen and kidneys.

A search for toxic factors in the mouldy sweet potato extract revealed numerous pink, red or grey spots on thin layer chromatograms sprayed with Ehrlich reagent (*p*-dimethylaminobenzaldehyde). Three of these substances were studied because of their toxicity for mice. The first was purified by column chromatography,