extrinsic factor,[4] was then substituted for the crystalline B_1 in equivalent amounts. This concentrate (75 to 90 mg) plus the flavin produced a resumption of growth at an average rate of 2.3 to 2.6 g per day, respectively, for four weeks, while a lower dose level (56 mg) yielded 1.3 g per day for the same period. The different growth rates were not due to a variation of the vitamin B_1 intake, which was maintained at a constant level (6 I.U.).

Negative control rats supplied with only B_1 and flavin developed a progressive dermatitis in about five weeks, and several animals died. The swollen and inflamed paws have been cured by administration of the rice polishings concentrate which contained the dermatitis factor(s). The biological factor(s) was stable when the concentrate was subjected to ultraviolet irradiation but was partially destroyed by treatment in an autoclave.

With both types of assay the daily supplements were designed to provide optimal quantities of vitamins so that inadvertent additions with the test materials would not affect the growth rate beyond the biological error. Coprophagy did not become a problem. These methods are being used to assay experimental fractions in a study of the biological factor(s) which have a reputed relation to pellagra and pernicious anemia.

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THE DISTRIBUTION OF GENE FREQUENCIES IN POPULATIONS

THE effects of the various evolutionary factors can be reduced to common terms by considering the rates of change which they tend to bring about in the relative frequencies of alleles within a population. In the absence of such factors, there is a constancy of gene frequencies from the symmetry of the Mendelian mechanism. The frequency (q) of a given gene changes at the rate

$$\Delta q = -uq + v(1-q) - m(q-q_t) + \frac{q(1-q)}{2}\frac{\partial}{\partial q}\log \overline{W}$$

where u is the rate of mutation of the gene in question, v is the rate of mutation to it from its alleles, m is the effective amount of exchange between the local population under consideration and the species as a whole (gene frequency q_t), and $\overline{W}$ is the mean selective value of the array of genotypes characteristic of this population. Gene frequency is in equilibrium (stable or otherwise) at any point at which $\Delta q = 0$ except for variation due to the accidents of sampling among the gametes. The sampling variance for one generation is $\frac{q(1-q)}{2N}$ where N is the effective size of the breeding population. The pressure toward a stable equilibrium in value of q, due to mutation, crossbreeding and selection (assuming persistence of the same conditions for a long period), and the divergent tendency due to inbreeding should between them determine a certain probability distribution of values of q for the local population considered. The following formula is reached, assuming that the selective effects of the gene in question are independent of those of other genes.

$$\varphi(q) = \frac{Ce^{4N\int\frac{\Delta q\, dq}{q(1-q)}}}{q(1-q)}$$
$$= C\overline{W}^{2N} q^{4N[mq_t+v]-1}(1-q)^{4N[m(1-q_t)+u]-1}$$

More generally, selective values depend on the interactions of the entire system of genes. It is the harmonious development of all characteristics that determines the success of an organism, not the absolute grades of the separate characters and still less the composition with respect to a single series of alleles. The mean selective values, $\overline{W}$, of populations characterized by different sets of gene frequencies form a multidimensional surface which in general has many peaks. The joint distribution of the gene frequencies is given by the formula

$$\varphi(q_1, q_2 \cdots q_n) = C\overline{W}^{2N}\prod_{i=1}^{n} q_i^{4N[mq_t+v_i]-1}(1-q_i)^{4N[m(1-q_t)+u_i]-1}$$

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[4] D. K. Miller and C. P. Rhoads, *New Eng. Jour. Med.*, 211: 921, 1934.

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