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Effective population size when fertility is inherited*

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Kimura & Crow (1963) have shown that the variance effective size of a population is given by

$$N_e = \frac{N\bar{k}}{1-\alpha+(1+\alpha)\,V_k/\bar{k}}$$

where N and α are the actual population size and departure from Hardy–Weinberg proportions of genotype frequencies, respectively, in the parental generation, and $\bar{k}$ and V_k refer to the mean and variance of progeny number. This formula is satisfactory when progeny number or fertility is not inherited. There are, however, many evidences that fertility is inherited (Fisher, 1930, and others).

When fertility is inherited, the progeny of an individual which gives rise to a large number of progeny again tends to produce a large number of progeny because of transmission of the genes controlling fertility. This process continues until the average fertility of the progeny reaches the population mean through segregation and recombination. Thus, any genes which are associated with a high-fertility gene, i.e. the genes which are carried by individuals with a high-fertility gene, will, in the long run, be represented in a higher frequency than those associated with a low-fertility gene. Hence, the probability that such genes become homozygous due to inbreeding is larger than that expected when fertility is not inherited. In other words, the variance of frequency of genes other than the fertility genes will be increased, and the effective size is correspondingly reduced.

The rigorous mathematical treatment of the effect of fertility inheritance on the effective size is not easy, but an approximate solution is readily obtained. Robertson (1961) studied a similar problem arising in artificial selection for a quantitative character, but he was not interested in the inheritance of fertility itself, which appears very important in natural populations.

Let us first consider a population of monoecious organism, in which the number of progeny per individual is affected by genetic and environmental factors as well as sampling errors. Thus, the variance of progeny number can be written as

$$V_k = V_g + V_e + V_s$$

where V_g, V_e, and V_s refer to the additive genetic, environmental (including the non-additive genetic), and sampling variances, respectively, and no interaction between genotypic and environmental effects is assumed. In the following it is also assumed that the frequencies of fertility genes are in equilibrium owing to the balance between mutation and selection, so that V_g remains the same for all generations.

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As mentioned previously, if fertility is inherited or V_g is not negligible, the genes associated with high-fertility genes are destined to increase in frequency in the subsequent generations, while those genes associated with low-fertility genes are to be reduced. This has an effect *equivalent to increasing* the additive genetic variance. In obtaining the increased additive genetic variance, the argument analogous to that made by Robertson (1961) in connexion with artificial selection may be used. As is well known, the first generation progeny of an individual with breeding value g with respect to progeny number has the breeding value of $\frac{1}{2}g$, the second generation has $\frac{1}{4}g$, and so on, the breeding value being halved in each generation (cf. Falconer, 1960). So the accumulated breeding value will increase as $1g, \frac{3}{2}g, \frac{7}{4}g, \ldots$ to a limiting value of $2g$. Thus, the additive genetic variance to be obtained is $4V_g$. Substituting V_g by $4V_g$ in Kimura and Crow's formula and assuming that $\alpha = 0$, we have

$$N_e = \frac{N}{(1+3h^2)C^2 + 1/\bar{k}}$$

where h^2 and C are the heritability and coefficient of variation of progeny number, respectively. If $\bar{k} = 2$, this formula turns out to be

$$N_e = \frac{4N}{(1+3h^2)V_k + 2}$$

It is important to note that this formula refers to genes other than fertility genes.

In dioecious organisms with equal numbers of male and female V_k in Kimura and Crow's formula should be replaced by $\frac{1}{2}V_{k(m)} + \frac{1}{2}V_{k(f)}$ where $V_{k(m)}$ and $V_{k(f)}$ refer to the variance of progeny number for male and female, respectively. In the case of monogamy $V_{k(m)}$ and $V_{k(f)}$ are the same. Further, there is a possibility that a completely different set of genes control the male and female fertilities. To examine this effect, let the breeding value for a male be g_m and g_f, where g_m is the effect on male fertility and g_f on female fertility, and the corresponding value of his mate be g'_m and g'_f. One-half of the progeny will be male with expected fertility $(g_m + g'_m)/2$ and one-half female with expected fertility $(g_f + g'_f)/2$. The accumulated breeding value of the male is then

$$g_m + \frac{g_m + g_f}{4} + \frac{g_m + g_f}{8} + \ldots = \frac{3g_m}{2} + \frac{g_f}{2}$$

Thus, if g_m and g_f have equal variances, the increased additive genetic variance is given by $\frac{1}{2}(5+3r)Vg$, where r is the correlation between g_m and g_f. The same arguments apply to the variance of female, whose accumulated breeding value is $\frac{1}{2}(g'_m + 3g'_f)$. Therefore, the effective population size is given by

$$N_e = \frac{N}{\frac{1}{2}[1+\frac{3}{2}(1+r)h_m^2]C_m^2 + \frac{1}{2}[1+\frac{3}{2}(1+r)h_f^2]C_f^2 + 1/\bar{k}}$$

where subscripts m and f refer to the values of male and female, respectively. If the same set of genes exert the same effect on male and female fertilities, r is unity and the above formula becomes equal to that in the case of monoecious organisms. On the other hand, if a completely different set of genes control the two, r is zero and the effects of heritabilities of fertility are reduced to a half.

Fisher (1930), Huestis & Maxwell (1932), and Berent (1953) studied the mother–daughter correlation of sibship size in man and obtained 0·21, 0·12 and 0·19, respectively. These data suggest that the heritability of female fertility is 0·2 to 0·4. No data on male fertility are available. Crow & Morton (1955) estimated the value of $V_k/\bar{k}$ to be 1·5 when $\bar{k}$ is adjusted to 2. Thus, V_k is 3. Therefore, if we take $h^2 = 0{\cdot}3$ for both male and female

and assume that the same set of genes control the male and female fertilities, N_e becomes $0{\cdot}52N$. On the other hand, if h^2 were 0, N_e would become $0{\cdot}8N$, so that the heritability of 30% has reduced the effective size by 28%. Note, however, that the estimate of $h^2 = 0{\cdot}3$ was obtained from data on families with $\bar{k}$ much larger than 2. It seems that in a population with $\bar{k} = 2$ the heritability is smaller than 0·3.

Most natural populations are considered to be in approximate equilibrium with respect to gene frequencies. It is, therefore, important to know how the fertility or fitness is inherited at equilibrium. If the genes controlling the fitness are maintained in the population by the mechanism of overdominance, there remains no additive genetic variance at equilibrium, so that the heritability of fitness is 0. On the other hand, if the genes are maintained by the balance of mutation and selection, there may arise a considerable amount of additive genetic variance. Studying the genotypic parent–offspring correlation of fitness at equilibrium, Haldane (1949) showed that, if the mutant genes are completely recessive, the correlation is practically 0, while if they reduce the fitness of heterozygotes by h relative to the homozygotes for normal genes, the genotypic correlation becomes $\frac{1}{2}\sqrt{(1-h)}$ approximately. This formula is satisfactory if h is larger than 6%. However, the data on lethal and semi-lethal genes in *Drosophila* suggest that h is 2–12% on the average for female fertility (Hiraizumi & Crow, 1960; and others). Thus, we examined the genotypic parent–offspring correlation for small values of h with mutation rate of 10^{-5}. The result obtained is graphically shown in Fig. 1. In this figure s represents the selection coefficient for mutant homozygotes. It is clear that the correlation is quite high for h of 2–12%. This suggests that the heritability of fertility could be appreciably high, although in reality the environmental and sampling variations dilute its effect.

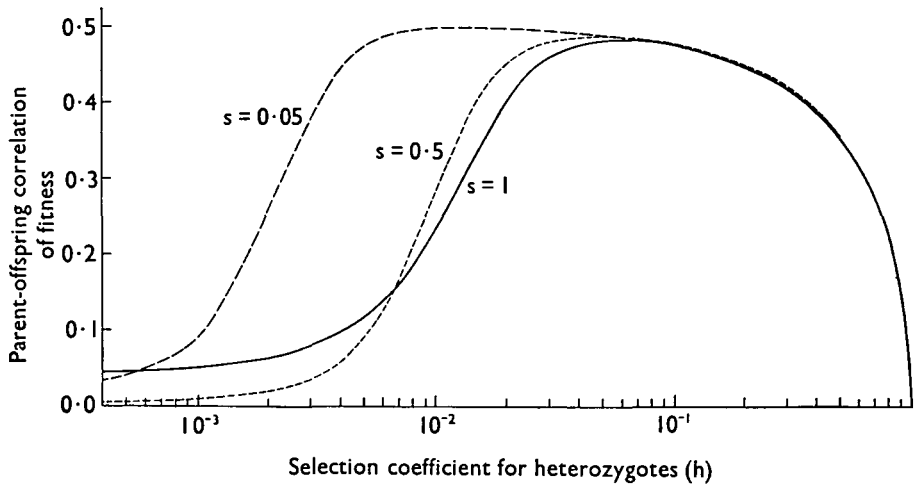

Fig. 1. Selection coefficient for heterozygotes and genotypic parent–offspring correlation. Symbol s stands for the selection coefficient for mutant homozygotes. The abscissa is on a logarithmic scale.

SUMMARY

A formula for effective population size when fertility is inherited is worked out. It is shown that the effective size decreases as the heritability of fertility or progeny number increases.

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