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Population Dynamics of Sperm and Pollen Killers*

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Summary. A model of segregation distortion is assumed in which the action of the distorter when heterozygous is to render dysfunctional those gametes that carry its allele. Two gamete killers when homozygous are assumed to distort each other. Individuals that carry the gamete killer suffer a reduction in the number of functional gametes they produce, but this deleterious effect is counterbalanced by the segregation ratio advantage of the distorter. The dynamics of such a system are analyzed in terms of a generalized fecundity function, which is defined as a function which assigns to any individual his relative fecundity in terms of the fraction of functional gametes he produces. Three general classes of fecundity functions are considered: (a) proportionality, in which the relative fecundity of an individual is proportional to the fraction of functional gametes he produces, (b) compensation, in which the relative fecundity of an individual is always greater than the fraction of functional gametes he produces, and (c) mass action, in which the relative fecundity of an individual is less than or greater than the fraction of functional gametes he produces according to whether the fraction of functional gametes is less than or greater than some threshold. In case (a) all gamete killers are always at neutral equilibria and gene frequency changes at the locus are governed by random drift. In case (b) all gamete killers will be fixed if the fecundity function is such that its second derivative is negative, whenever its argument is greater than one-half. And in case (c) some gamete killers will converge to stable equilibria, others will be fixed. If a gamete killer is homozygous lethal it will almost always converge to a stable equilibrium.

Several well-known cases of meiotic drive have as their specific mechanism the dysfunction of those gametes which carry the favored gene's allele. In *Drosophila melanogaster*, sperms that carry the homologue of the *segregation distorter* chromosome fail to function properly (Hartl, Hiraizumi, and Crow 1967; Nicoletti, Trippa, and DeMarco 1967); Erickson (1965) has shown that males hemizygous for the *recovery disrupter* X-chromosome have abortive differentiation of some of the Y-bearing sperms. In *Drosophila simulans*, Faulhaber (1967) has studied a recessive autosomal gene, homozygous males of which produce mostly female progeny. She has found morphologically abnormal sperms in sufficient proportion to account for the sex ratio effect and has therefore concluded that the abnormal sperms are Y-bearing. Gametic dysfunction is also a consequence of the *sex-ratio* gene in *Drosophila pseudoobscura* (Policansky and Ellison 1970), of the *pollen killer* loci in tobacco (Cameron and Moav 1957) and wheat (Loegering and Sears 1963), of *male producer* in the mosquito (Hickey and Craig 1966), and it seems also to account for t-allele segregation distortion in the house mouse (Braden and Gluecksohn-Waelsch 1958).

This article presents a theoretical analysis of populations which contain such "gamete killers." The discussion is made in terms of a generalized fecundity function, which is defined as a function which assigns to any individual his relative fecundity on the basis of the number of functional gametes he produces. Three classes of fecundity functions are examined In one class, gamete killers tend to be retained at polymorphic equilibria solely by reason of their mechanism of action; in another, all gamete killers become fixed in the population; and in the third, a gamete killer may be fixed or it may reach a stable equilibrium. The main conclusion is that gamete dysfunction, as a mechanism of meiotic drive, does not produce an overall adverse selection on the gamete killer even in cases in which gamete dysfunction occurs in homozygotes. The segregation ratio advantage in the heterozygotes tends to balance off any fecundity disadvantage suffered by the gamete killer when heterozygous and homozygous. In the simplest case the balancing is exact, and the equilibrium established is neither stable nor unstable, but selectively neutral, like the Hardy-Weinberg equilibrium.

Throughout I consider only autosomal genes, as sex-linked gamete killers require a rather different approach. For the most part I will also assume that the dysfunction occurs to the same extent in both sexes.

Gamete Production

The mechanism of gametic dysfunction has been worked out in only a few special cases (*SD*, *RD*, *sex-ratio*, etc.). Although the details of the process are possibly different in the different systems, in broad outline they seem to be much the same. The time and place of action of the gamete killer is in the primary meiocyte. In meiocytes in which the gamete killer is operative, it does something either to its homologous chromosome or to the pole to which its

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homologue will proceed at anaphase I. Whatever the case, the gametes formed around the telophase II nuclei that contain the homologue of the gamete killer are rendered dysfunctional. Thus in each cell in which distortion occurs two gametes are killed.

This is the model of gamete dysfunction that will be assumed, but two things should be noted. The first is the obvious point that other models of gamete dysfunction can easily be devised. One might assume, for example, that meiocytes in which the distorter is active produce only one dysfunctional gamete instead of two. These possibilities will be ignored because the model above is the only one for which there is experimental precedent. The second point is that not all meiotic drive implies gamete dysfunction. If the mechanism of the meiotic drive involves the orientation of its own bivalent so as to consign its homologue to a polar body nucleus, then no reduction in the number of functional gametes is consequent to its action (Rhoades 1952; Novitski 1967). This situation can arise whenever there are regularly nonfunctional products of spermatogenesis, as was postulated in *Drosophila melanogaster* (Novitski and Sandler 1957) and as is known in coccids and some rotifers. Only when the segregation distorter causes the dysfunction of an otherwise normal gamete is the model in the present paper applicable.

Let A_1 and A_2 be two alleles at the locus of the gamete killer and suppose that A_1 renders A_2-bearing gametes dysfunctional. Let ϱ be the probability that the gamete killer is operative in a given meiocyte. Then an A_1/A_2 individual has two kinds of meiocytes: those that produce dysfunctional gametes (probability ϱ), and those that are normal (probability $1-\varrho$). Since only one-half of the gametes from the first class of meiocytes is functional, the overall fraction of functional gametes, $\bar{x}$, is given by $\bar{x} = 1 - (\varrho/2)$. The segregation ratio, denoted by k, is the proportion of functional A_1-bearing gametes among all the functional gametes produced by an A_1/A_2 genotype, hence we have

$$k = \frac{1}{2\left(1-\frac{\varrho}{2}\right)}$$

and the fraction of functional gametes is given by $\bar{x} = 1/(2\,k)$.

Assume that dysfunction occurs in A_1/A_1 homozygotes to the same extent as it occurs in heterozygotes and that the A_1 chromosomes act independently of one another. (This aspect of the model is prompted by the observation that the *SD* and *t*-allele sperm killers are male sterile when homozygous.) Then there are three kinds of meiocytes in A_1/A_1 individuals: those in which through mutual distortion of the A_1 chromosomes all of the gametes are rendered dysfunctional (probability ϱ^2); those in which only one of the gamete killers is operative, hence only half of the gametes are dysfunctional (these occur with probability $2\,\varrho\,(1-\varrho)$); and those meiocytes that are normal (probability $(1-\varrho)^2$). In homozygotes the fraction of functional gametes is therefore $1-\varrho\,(1-\varrho)-\varrho^2$, which equals $(1/k)-1$.

In A_2/A_2 homozygotes the fraction of functional gametes is 1 because none of the gametes undergoes dysfunction.

Putting all this together, for a specified value of k, the fraction of functional gametes produced by A_2/A_2 individuals is 1, by A_1/A_2 individuals is $\bar{x}$ $[= 1/(2\,k)]$, and by A_1/A_1 individuals is $2\,\bar{x}-1$ $[= (1/k)-1]$.

Fecundity Functions

Assume that the only component of fitness in which the three genotypes at the locus of the gamete killer differ is fecundity, and that the differences arise from the different proportions of functional gametes produced by the genotypes. Although the fecundity of an individual must be related in some way to the proportion of functional gametes he produces, it is not to be expected in general that these two things will be equal. Nevertheless, if the fraction of functional gametes produced by an individual is x, then there must exist a function $f(x)$ which assigns to each value of x the actual fecundity of an individual which produces that many functional gametes. $f(x)$ is assumed to be measured relative to a value of 1 for wild type. Any function of this type will be called a *fecundity function.*

Theoretically, a fecundity function could have any form whatsoever. But many candidates can immediately be excluded because their biological implications are unreasonable. To be consistent with our biological intuition a fecundity function would have to satisfy three additional conditions: (1) $f(0) = 0$; (2) $f(1) = 1$; and (3) $f(x_1) \leq f(x_2)$ if and only if $x_1 \leq x_2$. The first of these requires that an individual that produces no functional gametes be sterile; the second states that an individual that produces as many functional gametes as wild type has a fecundity equal to that of wild type; and the third requirement is that the fecundity function be a nondecreasing function of x.

In real organisms the fecundity function is certainly sex-specific and possibly age specific as well. One can also imagine conditions in which the fecundity depends on population density. Except for sex-specific fecundity functions, one case of which will be discussed later, these kinds of complications will be ignored. It goes without saying that the fecundity function is an important aspect of any reproductive system and is useful to know in connections other than segregation distortion. In particular, the fecundity function tells one how intensely selection operates against partial sterility factors in nature.

Inasmuch as no specific fecundity function is known that is applicable to any real organism, one is reduced to guesswork. Below is a list of several forms of function which seem to me to be plausible. I will discuss their possible range of application and their evolutionary consequences with respect to gamete killers. The functions to be considered fall into three categories.

A. *Proportionality*

In this case one assumes that the fecundity of an individual is proportional to the number of functional gametes he produces, i.e. $f(x) = x$. At first thought, this function appears to be so restrictive as to have no validity. There are reasons for thinking that it might have wider applicability than it first appears, however. The function might be appropriate in the female sex of most animals and most higher plants, because in these cases the dysfunction of an egg corresponds immediately to one fewer progeny. This is true in maize, where an aborted embryo is one less viable seed on an ear of corn, or in the mouse, where an inviable egg is one less offspring in a litter, or in humans, where an egg's dysfunction may preclude impregnation in a given month. This fecundity function might also be appropriate in species in which the gametes of many individuals are physically pooled, so that each functional gamete has about the same chance of survival as any other. This is the case with respect to the sperms of several marine animals and with respect to the pollen of many plants.

B. *Compensation*

In this case one assumes that the fecundity function, in addition to the three requirements listed previously, also has the property that $f(x) > x$. That is to say, an individual is always more fecund than would be expected on the basis of how many functional gametes he produces. This is the case in males of Drosophila where, under natural conditions as well as in population cages, the females produce fewer eggs than they have sperms stored in their seminal receptacles and spermathecae. The function also applies to the females of mammals whenever egg abortion occurs early enough for the dysfunctional egg to be replaced by another one. In general, one would expect most departures from the proportionality function to be in the direction of compensation.

The specific functions one might choose could be arbitrary. I have found two that have some biological meaning, however. These were obtained by arguing that a compensation function should approach 1 more and more slowly as x approaches 1, which is to say that its slope should decrease as the fraction of functional gametes increases. The first two functions below were obtained by assuming that the slope of the function decreased linearly or as the square of the value of the function itself. The functions are not only quite convenient, but their overall shape appears to be reasonable.

(1) A solution of the differential equation $f'(x) = \alpha - \beta f(x)$ that satisfies the conditions $f(0) = 0$ and $f(1) = 1$ is

$$f(x) = \frac{1 - e^{-ax}}{1 - e^{-a}} \quad (a > 0)$$

with $\alpha = \beta = a$. It is easy to show that $f(x)$ is monotone increasing, that $f(x) \geq x$, and that $\lim\limits_{a \to 0} f(x) = x$.

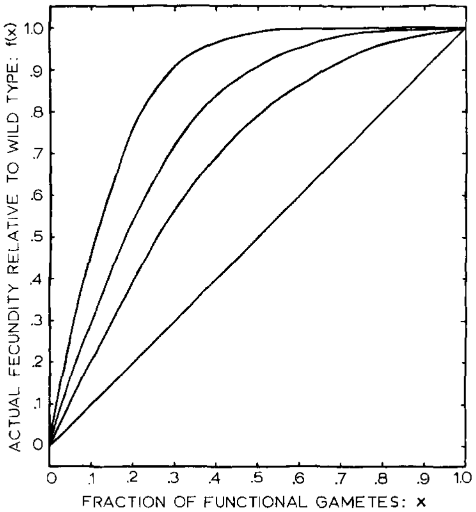

Fig. 1. The curves above are the function $f(x) = \tanh(a\,x)/\tanh(a)$. From left to right the curves correspond to $a = 5$, $a = 3$, and $a = 2$. The straight line is $\lim\limits_{a \to 0} f(x)$

(2) A solution of the differential equation $f'(x) = \alpha - \beta f^2(x)$ that satisfies $f(0) = 0$ and $f(1) = 1$ is

$$f(x) = \frac{\tanh(a\,x)}{\tanh(a)} \quad (a > 0),$$

where $\alpha = a/\tanh(a)$, $\beta = a\,[\tanh(a)]$, and $\tanh(y) = (e^{y} - e^{-y})/(e^{y} + e^{-y})$.
This function also satisfies $f(x) \geq x$, it is monotone increasing, and $\lim\limits_{a \to 0} f(x) = x$.

An idea of the shape of this function can be gotten from Figure 1. The three curves from left to right correspond to $a = 5$, $a = 3$, and $a = 2$. The straight line is $f(x) = x$.

(3) A third form of compensation function is a special case of the mass action function discussed below. Its equation is

$$f(x) = x + (1 - x) \cdot \tanh(a\,x) \quad (a > 0)\,.$$

It also has all of the properties desired.

C. *Mass action*

This is a threshold model in which individuals that have fewer than some threshold number of functional gametes have very low fecundity, and individuals that have more than the threshold number have practically normal fecundity. This model might be applicable to the sperm in mammals, in which the sperm release an enzyme, hyaluronidase, that facilitates fertilization. Because so few sperms are actually used in fertilization, the fecundity will be practically normal if the amount of enzyme is above the threshold level required for fertilization and it will be practically zero if the amount of enzyme is below the threshold.

If f is continuous and $f(x) < x$ for x close to 0 and $f(x) > x$ for x close to 1, then there must exist a point b for which $f(b) = b$. One function, obtained by trial and error, which possesses these properties is

$$f(x) = \begin{cases} x + x \cdot \tanh\,[a\,(x - b)] & \text{for } x \leq b \\ x + (1 - x) \cdot \tanh\,[a\,(x - b)] & \text{for } x > b\,. \end{cases}$$

Several examples are shown in Figure 2. From left to right the sigmoidal curves correspond to $a = 10$, $b = .3$; $a = 10$, $b = .5$; and $a = 10$, $b = .7$. The line $f(x) = x$ is the mass action function when $a = 0$. Thus by choosing b properly one can select the threshold, and by making a large enough one can make the rise in the vertical portion of the curve as abrupt as desired. The mass action function is continuous at $x = b$, but because $\underset{x \uparrow b}{limit}\, f'(x) = 1 + ba$ whereas $\underset{x \downarrow b}{limit}\, f'(x) = 1 + (1 - b)\,a$, the first derivative is not continuous at $x = b$ unless $b = .5$.

If $b = 0$ then this is a compensation function.

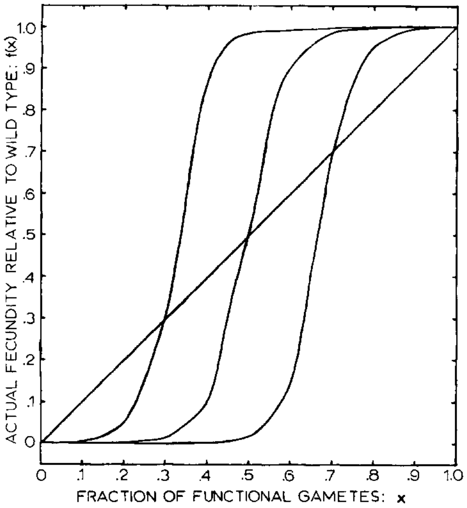

Fig. 2. Graphs of $f(x) = x + x \cdot \tanh\,[a\,(x - b)]$ for $x \leq b$ and $f(x) = x + (1 - x) \cdot \tanh\,[a\,(x - b)]$ for $x > b$. From left to right the parameters in the curves are $a = 10$, $b = .3$; $a = 10$, $b = .5$; and $a = 10$, $b = .7$. The straight line corresponds to $a = 0$

The Model

Assume a gamete killer A_1 at gene frequency p_1 and its normal allele A_2 at frequency p_2 $(p_1 + p_2 = 1)$ in a large population undergoing random mating. If k is the proportion of functional A_1-bearing gametes among the functional gametes produced by an A_1/A_2 individual, and if f is the fecundity function, then the following discrete model where $\bar{x} = 1/(2\,k)$ is appropriate:

Genotype	$A_1\,A_1$	$A_1\,A_2$	$A_2\,A_2$
Relative frequency	p_1^2	$2\,p_1\,p_2$	p^2
Relative fitness (fecundity)	$f\,(2\,\bar{x} - 1)$	$f(\bar{x})$	1
Proportion of A_1-bearing among all functional gametes produced	1	k	0 .

The change in the frequency of the gamete killer in one generation is

$$\Delta p_1 = \frac{p_1 p_2 \{[2\,k\,f(\bar{x}) - 1] - p_1\,[2\,f(\bar{x}) - f\,(2\,\bar{x} - 1) - 1]\}}{p_1^2\,f\,(2\,\bar{x} - 1) + 2\,p_1\,p_2\,f(\bar{x}) + p_2^2}, \tag{1}$$

and the nontrivial fixed point is

$$\hat{p}_1 = \frac{2\,k\,f(\bar{x}) - 1}{2\,f(\bar{x}) - f\,(2\,\bar{x} - 1) - 1}$$

$$\hat{p}_2 = \frac{2\,(1 - k)\,f(\bar{x}) - f\,(2\,\bar{x} - 1)}{2\,f(\bar{x}) - f\,(2\,\bar{x} - 1) - 1}\,.$$

Two sets of conditions lead to nontiivial fixed points:

(1) Case 1.

$2\,k\,f(\bar{x}) > 1$ and $f\,(2\,\bar{x} - 1) < 2\,(1 - k)\,f(\bar{x})$,

(2) Case 2.

$2\,k\,f(\bar{x}) < 1$ and $f\,(2\,\bar{x} - 1) > 2\,(1 - k)\,f(\bar{x})$.

Except for minor changes in terminology these are the results of Hiraizumi, Sandler, and Crow (1960) and of Scudo (1967).

Cannings (1969) has pointed out that an equation in the form of (1) is not sufficient to prove convergence to stable points. To investigate the convergencc properties of this model put $\xi = p_1/p_2$ and let ξ' be the value of ξ in the next generation and let $\hat{\xi}$ be the equilibrium. Consider the function $\Psi(\xi) = (a\,\xi + b\,\xi^2)/(1 + c\,\xi)$ where $a = 2\,k\,f(\bar{x})$, $b = f\,(2\,\bar{x} - 1)$, $c = 2\,(1 - k)\,f(\bar{x})$, so that $\xi' = \Psi(\xi)$. Since $d\Psi(\xi)/d\xi = (a + 2\,b\,\xi + bc\,\xi^2)/(1 + c\,\xi)^2 > 0$ it follows that $\Psi(\xi)$ increases monotonically. Because $\Psi(\hat{\xi}) = \hat{\xi}$ we have that $\xi < \hat{\xi} \Rightarrow \xi' < \hat{\xi}$ and $\xi > \hat{\xi} \Rightarrow \xi' > \hat{\xi}$.

Let $\theta(\xi) = \Psi(\xi)/\xi$. Then $d\theta(\xi)/d\xi = (b - a\,c)/(1 + c\xi)^2$ where $b - a\,c = f(2\,\bar{x} - 1) - 4k(1 - k)\,f^2(\bar{x})$. It can be shown that the assumptions of case 1 above imply that $b - a\,c < 0$ and that the assumptions of case 2 imply $b - a\,c > 0$. Thus in case 1 $\theta(\xi)$ decreases monotonically. Since $\theta(\xi) = \xi'/\xi$ and $\theta(\hat{\xi}) = 1$ it follows that $\xi > \hat{\xi} \Rightarrow \xi' < \xi$ and $\xi < \hat{\xi} \Rightarrow \xi' > \xi$. In case 1, therefore, we have that either $\xi < \xi' < \hat{\xi}$ or $\xi > \xi' > \hat{\xi}$. ξ converges to $\hat{\xi}$ and $\hat{\xi}$ is stable.

In case 2, $\theta(\xi)$ increases monotonically and $\xi < \hat{\xi} \Rightarrow \xi' < \xi$. Also $\xi > \hat{\xi} \Rightarrow \xi' > \xi$. We have either $\xi \to \infty$ (if $\xi_0 > \hat{\xi}$ where ξ_0 is the initial value of ξ) or $\xi \to 0$ (if $\xi_0 < \hat{\xi}$). Thus in case 2 $\hat{\xi}$ is an unstable equilibrium.

The argument above is not valid if $f\,(2\,\bar{x} - 1) - 4\,k\,(1 - k)\,f^2(\bar{x}) = 0$. In such a case $\theta(\xi) = \text{const}$ and since $\theta(\hat{\xi}) = 1$ we have $\xi' = \xi$ for all ξ.

In summary, except for the case $f\,(2\,\bar{x} - 1) - 4\,k\,(1 - k)\,f^2(\bar{x}) = 0$, we have (1) $2\,k\,f(\bar{x}) > 1$ and $f\,(2\,\bar{x} - 1) < 2\,(1 - k)\,f(\bar{x})$ leads to a stable equilibrium, (2) $2\,k\,f(\bar{x}) < 1$ and $f\,(2\,\bar{x} - 1) > 2\,(1 - k)\,f(\bar{x})$ leads to an unstable equilibrium.

In the two remaining cases it can be shown that (3) $2k\,f(\bar{x}) > 1$ and $f(2\bar{x}-1) > 2(1-k)\,f(\bar{x})$ implies $p_1 \to 1$, and (4) $2k\,f(\bar{x}) < 1$ and $f(2\bar{x}-1) <$ $> 2(1-k)\,f(\bar{x})$ implies $p_1 \to 0$.

For any given value of k we are interested in only two points on the fecundity curve, namely $f(\bar{x})$ and $f(2\bar{x}-1)$ where $\bar{x} = 1/(2k)$. These two points determine a straight line, the equation of which is $g(x) = \alpha x + \beta$ with $\alpha = [f(\bar{x}) - f(2\bar{x}-1)]/(1-\bar{x})$ and $\beta = f(\bar{x}) - \bar{x}\,[f(\bar{x}) - f(2\bar{x}-1)]/(1-\bar{x})$. Clearly $g(\bar{x}) = f(\bar{x})$ and $g(2\bar{x}-1) = f(2\bar{x}-1)$. We now prove the following:

(a) if $g(\bar{x}) > \bar{x}$ and $\beta < 0$ the distorter converges to a stable equilibrium,
(b) if $g(\bar{x}) < \bar{x}$ and $\beta > 0$ the distorter is fixed or lost depending on its initial frequency,
(c) if $g(\bar{x}) > \bar{x}$ and $\beta > 0$ the distorter becomes fixed,
(d) if $g(\bar{x}) < \bar{x}$ and $\beta < 0$ the distorter is lost.

Since $g(\bar{x}) = f(\bar{x})$ and $\bar{x} = 1/(2k)$, then $g(\bar{x}) > \bar{x}$ if and only if $2k\,f(\bar{x}) > 1$, and $g(\bar{x}) < \bar{x}$ if and only if $2k\,f(\bar{x}) < 1$.

Assume now that $f(2\bar{x}-1) < 2(1-k)\,f(\bar{x})$. This is the same as $[g(2\bar{x}-1)/g(\bar{x})] < (2\bar{x}-1)/\bar{x}$. Because $g(x) = \alpha x + \beta$ we can write $[\alpha(2\bar{x}-1) + \beta]/[\alpha\bar{x}+\beta] < (2\bar{x}-1)/\bar{x}$. Since both $\alpha\bar{x}+\beta > 0$ and $\bar{x} > 0$ we have after some cancellation and rearrangement that $\beta(1-\bar{x}) < 0$, or $\beta < 0$. Starting with the assumption $\beta < 0$ and proceeding in the reverse direction leads to the result $f(2\bar{x}-1) < 2(1-k)\,f(\bar{x})$.

In a similar way one can show that $f(2\bar{x}-1) > 2(1-k)\,f(\bar{x})$ if and only if $\beta > 0$.

Thus the cases (a), (b), (c) and (d) correspond respectively to (1), (2), (3), and (4) in the paragraph immediately above. The population consequences of a gamete killer depend only on whether $f(\bar{x})$ lies above the diagonal and on the sign of the y-intercept of the straight line passing through $f(\bar{x})$ and $f(2\bar{x}-1)$.

Special Cases

These general results will now be applied to the three classes of fecundity functions discussed previously.

A. *Proportionality*

$f(x) = x$. This case has an interesting evolutionary property, but how important it is in practice depends on how widely applicable the fecundity function might be. The result is that

$$f(2\bar{x}-1) - 4k(1-k)\,f^2(\bar{x}) = 0\,.$$

Therefore $\Delta p_1 = 0$ irrespective of the value of k, and the system is always at a neutral equilibrium. Any evolutionary changes at the locus of the gamete killer are governed by random genetic drift. When $k = 1/2$ the model reduces to the familiar Hardy-Weinberg model. Scudo (1967) was the first to point out that equation (1) implies the existence of a family of nontrivial neutral equilibria.

This result is not restricted to models in which the degree of distortion is the same in both sexes. All that is required is that the fecundity function be $f(x) = x$ in both sexes. Let p_1 be the frequency of A_1 in functional sperms, q_1 be the frequency of A_1 in functional eggs, k be the degree of distortion in males and k^* the degree of distortion in females. Then when $f(x) = x$ for both sexes, random mating leads to

$$\Delta p_1 = \frac{p_1 - q_1}{1 + (2k-1)(1-p_1-q_1)}\,[p_1(2k-1) - k],$$

$$\Delta q_1 = \frac{q_1 - p_1}{1 + (2k^*-1)(1-p_1-q_1)}\,[q_1(2k^*-1) - k^*].$$

When $p_1 = q_1$, $\Delta p_1 = \Delta q_1 = 0$ irrespective of the values of k and k^*. I have not been able to prove in general that $|p_1 - q_1| \to 0$, however. But in one important case, that in which distortion occurs in only one sex, it is possible to prove convergence. To see this let $k^* = 1/2$ and put $\delta_n = p_n - q_n$ and $\sigma_n = p_n + q_n$. For convenience put $b = 2k/(2k-1)$. Then the equations above lead to

$$\delta_n = \frac{\delta^2{}_{n-1}}{2(b - \sigma_{n-1})}$$

$$\sigma_n = \sigma_{n-1} + \delta_n\,.$$

Since $k > 1/2$, then $b > 2$, and $b - \sigma_n > 0$ for all n. Therefore for all n except possibly for $n = 1$, $\delta_n > 0$. Now $b - \sigma_n = b + \delta_n - \sigma_{n-1} > \delta_n$, or $1/(b - \sigma_{n-1}) < 1/\delta_n$. Thus $\delta_n = \delta_{n-1}^2/2\,(b - \sigma_{n-1}) <$ $< \delta_{n-1}^2/2\,\delta_n$. We then have that $\delta_n < \delta_{n-1}/\sqrt{2} <$ $< \delta_{n-2}/(\sqrt{2})^2 < \cdots < \delta_2/(\sqrt{2})^{n-2}$. Hence $\delta_n \to 0$ geometrically. Since $\Delta\sigma_n = \delta_n$, it follows that σ_n geometrically converges to its limit.

An extensive survey of initial values of p_1 and q_1 has indicated that the convergence of σ_n is extremely rapid. In most cases $\Delta\sigma_n < 10^{-3}$ in 3 or 4 generations, the only exceptions occurring when k is close to 1. Furthermore, unless k is close to 1 the equilibrium frequency of the distorter is close to the arithmetic average of the initial gametic frequencies.

B. *Compensation*

The analysis of the three compensation functions $f(x) = (1 - e^{-ax})/(1 - e^{-a})$, $f(x) = \tanh(a\,x)/\tanh(a)$, and $f(x) = x + (1-x)\cdot\tanh(a\,x)$ all follow the same logic and lead to the same conclusion. The argument will therefore be presented in terms of a generalized $f(x)$, but this $f(x)$ should be interpreted to be any one of the three above.

Consider $R(x) = f(x)/x$ and examine specifically $dR(x)/dx = [x\,f'(x) - f(x)]/x^2$. Write $dR(x)/dx =$ $= A(x)/B(x)$ where $B(x) > 0$ for all $x > 0$. We seek to know whether $R(x)$ is a monotone function of x. The sign of $R'(x)$ is determined by the sign of $A(x)$, so we are led to examine $dA(x)/dx = f''(x)$. In all three cases above, $dA(x)/dx < 0$ so that $A(x)$ is a decreasing function of x and for $x \in [0, 1]$ reaches its maximum at $x = 0$. For all three compensation functions $A(0) = 0$ so that $A(x) < 0$ for $x > 0$. Thus $dR(x)/dx < 0$ for all $x > 0$ and $R(x)$ is a decreasing function of x. (This argument is valid for *any* compensation function with the property that $f''(x) < 0$ whenever $x > 1/2$.) Therefore $R(x_0) > R(x_1)$ if and only if $x_0 < x_1$. Now let $x_0 = 2\bar{x} - 1$ and $x_1 = \bar{x}$ where $\bar{x} > 1/2$. Then $2\bar{x} - 1 < \bar{x}$ and therefore $R(2\bar{x} - 1) > R(\bar{x})$. Substituting, we obtain $f(2\bar{x} - 1)/(2\bar{x} - 1) > f(\bar{x})/\bar{x}$. This implies that $f(2\bar{x} - 1)/f(\bar{x}) > (2\bar{x} - 1)/\bar{x}$, and this in turn implies that β, the y-intercept of the straight line passing through $f(2\bar{x} - 1)$ and $f(\bar{x})$, is positive. Since $f(\bar{x}) > \bar{x}$ and $\beta > 0$ we have from the results of the previous section that all gamete killers will become fixed in the population.

As a specific example consider $f(x) = \tanh(a x)/\tanh(a)$. In this case $R'(x) = A(x)/B(x) = [a x - \tanh(a x) - a x \cdot \tanh^2(a x)]/x^2 \tanh(a)$ and $A'(x) = -2a^2 x \cdot \tanh(a x)\,[1 - \tanh^2(a x)] < 0$.

The conclusion that none of the compensation functions leads to a stable equilibrium is somewhat disturbing. The fact that gamete killers will always be *fixed* makes it even more so, because the fixation of a gamete killer that produced, say, 25% functional gametes when homozygous would lead to a substantial decrease in the average fecundity of the population. If the fixation of gamete killers occurred on any appreciable scale in evolution, then one would expect to find in existing species that a measurable fraction of the gametes would be dysfunctional. This is clearly not the case, because seed set in plants, for example, is quite high, so the number of dysfunctional gametes must be small.

A population can possess a compensation form of fecundity function and still avoid the fixation of gamete killers. This could happen in several ways. First, the population might have in it preexisting or newly arisen suppressors of the gamete killer. As the gamete killer increases in frequency the frequency of the suppressor — which may be merely an insensitive allele at the locus of the gamete killer — will also increase, until the suppressor comes to predominate and then the frequency of the gamete killer will decrease owing to its reduced fitness when homozygous. Suppressors and insentitive alleles are commonly associated with gamete killers in natural populations (Stalker 1961; Katoaka 1967; Hartl 1970a).

A second mechanism that will prevent the fixation of a gamete killer is the incorporation into the system of a recessive lethal. This is also a common occurrence in natural populations (Dunn 1956; Sandler, Hiraizumi, and Sandler 1959; Hiraizumi and Nakazima 1967), and it appears to be fostered by natural selection because the lethals are either physically close to the locus of the gamete killer on the chromosome, as is the case with the *t*-alleles, or the lethals are genetically coupled with the locus of the gamete killer, the linkage coming about by the presence of inversions. This is the case with most naturally occurring *SD* chromosomes.

Fixation of all gamete killers is an inevitable consequence of the three compensation functions I've discussed, provided that complications such as suppressors and linked lethals are ignored. This is obviously not true of *all* compensation functions. It is easy to construct examples that permit stable equilibria of some gamete killers. Consider, for example, the continuous function

$$f(x) = \begin{cases} x & \text{for } x \leq (1/2) \\ 2x - (1/2) & \text{for } (1/2) < x < (3/4) \\ 1 & \text{for } x \geq (3/4)\,. \end{cases}$$

For this function $f(x) > x$ whenever $x > 1/2$. A gamete killer will be fixed if $x > (9 + \sqrt{17})/16 = .82$ and it will be retained at a stable equilibrium if $x < (9 + \sqrt{17})/16$.

C. *Mass action*

Two different possibilities arise in this case according to whether $b \leq .5$ or $b > .5$. In the latter case, as x goes from $1/2$ to 1 the population consequences of gamete killers that produce a fraction x of functional gametes changes from those for which only unstable equilibria exist to those for which only stable equilibria exist, and finally, for x sufficiently large, all gamete killers will be fixed. On the other hand, $b > .5$ seems too restrictive for most real species. One might expect that the mass of functional gametes required for effective mass action would be something less than half the normal number.

When $b \leq .5$, then as x goes from $1/2$ to 1 the gamete killer passes from cases in which only stable equilibria exist to those in which the distorter is always fixed (this is quite obvious from the geometry of the mass action curves in Figure 2). This being the case, there exists a value of x, call it $\tilde{x}$, such that whenever $\bar{x} < \tilde{x}$ the gamete killer will reach a stable equilibrium and whenever $\bar{x} > \tilde{x}$ the distorter will be fixed. Corresponding to $\tilde{x}$ is a value of $\tilde{k}\ [= 1/(2\tilde{x})]$ such that $\bar{x} < \tilde{x}$ if only and if $k > \tilde{k}$ and $\bar{x} > \tilde{x}$ if and only if $k < \tilde{k}$. The value of $\tilde{x}$ is very difficult to obtain analytically, but I have been able to find it numerically. The results for several values of b and a are shown graphically in Figure 3. As can be seen, $\tilde{x}$ depends strongly on b, but it appears to be relatively insensitive to changes in a. That $\tilde{x} = 1/2$ whenever $b = 0$ is a numerical illustration of the analytic results reached previously for compensation functions.

The population consequences of a gamete killer with any particular degree of distortion, k, can be deduced geometrically without having to perform any calculations. This is illustrated for two cases in Figure 4. The curves A, B, and C superimposed

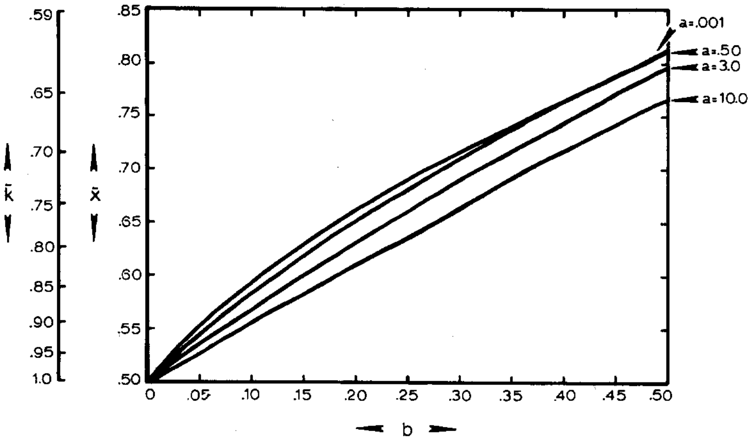

Fig. 3. Values of $\tilde{x}$ below which a gamete killer will converge to a stable equilibrium and above which it will be fixed. $\tilde{k}$ is the segregation ratio in heterozygotes corresponding to $\tilde{x}$, and a and b are the parameters in the mass action function (see Figure 2)

on the graph are as follows: A. $k(x) = 1/(2x)$; B. $f(x) = x$; and C. $f(x) = 2x - 1$. Given any value of k one can perform a set of reflections off of these curves to obtain both $f(\bar{x})$ and $f(2\bar{x} - 1)$ pertaining to the selected value of k. Consider the dashed line that proceeds horizontally from $k = .75$. This reaches curve A at a point whose x-coordinate is $\bar{x} = 1/(2k)$. The dashed line ascending to the round dot therefore identifies $f(\bar{x}) = .977$. The point corresponding to $f(2\bar{x} - 1)$ can be found by dropping a vertical from $(\bar{x}, k)$ on curve A to $(\bar{x}, 2\bar{x} - 1)$ on the line C, and then proceeding horizontally toward the y-axis. This horizontal dashed line intersects the curve B at the point $(2\bar{x} - 1, 2\bar{x} - 1)$, so a vertical line from the point of intersection to the curve of f identifies $f(2\bar{x} - 1) = .023$, the other round dot. The equation of the line through $f(\bar{x})$ and $f(2\bar{x} - 1)$ is $g(x) = 2.862\ x - .931$, but it is obviously not necessary to know the equation of the line to see that its y-intercept is negative and that therefore the distorter will reach a stable equilibrium.

The dotted lines in Figure 4 trace the reflections that generate $f(\bar{x}) = .998$ and $f(2\bar{x} - 1) = .805$ corresponding to $k = .64$. The equation of the line passing through the square dots is $g(x) = .884\,x + .308$. In this case the distorter is fixed.

Homozygotes Lethal

So far I have assumed that the only differences in fitness between the genotypes in the model arose from differences in fecundity which could in turn be traced back to the action of the gamete killer. As mentioned previously, several important examples of gamete killers, notably *SD* and the *t*-alleles, are often found in nature associated with recessive lethals. A logical extension of the simplest model would therefore be to assume that the homozygous gamete killer is lethal.

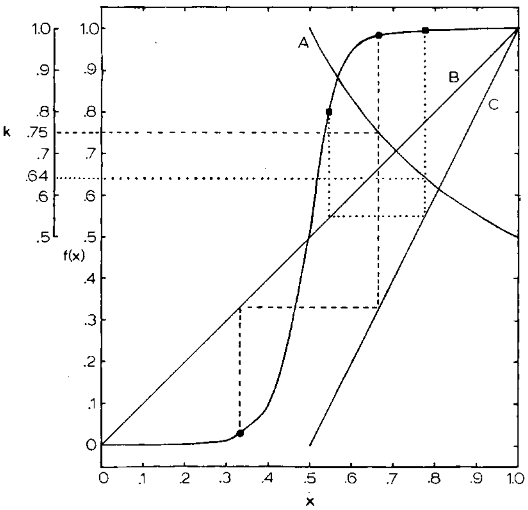

Fig. 4. The curves A, B, and C are A: $k(x) = 1/(2x)$; B: $f(x) = x$; and C: $f(x) = 2x - 1$. The sigmoidal curve is the mass action curve when $a = 10$, $b = .5$ (see Figure 2). A gamete killer with $k = .75$ converges to a stable equilibrium; one with $k = .64$ is fixed. See text for explanation

Let k and k^* be the proportion of functional A_1-bearing gametes from A_1/A_2 males and females, respectively, and let $\bar{x} = 1/(2k)$ and $\bar{x}^* = 1/(2k^*)$. Let f and f^* be the fecundity functions in males and females.

This is a special case of a model that has been examined previously (Hartl 1970b). The result pertinent to the present discussion is that, under random mating, the distorter reaches a stable equilibrium if and only if

$$\frac{f^*(\bar{x}^*)}{\bar{x}^*} + \frac{f(\bar{x})}{\bar{x}} > 2 \tag{2}$$

and the distorter is lost otherwise.

Three special cases deserve mention.

(a) If the degree of distortion and the fecundity functions are the same in both sexes, then (2) becomes

$$f(\bar{x}) > \bar{x}\,,$$

which is consistent with the previous results.

(b) If the degree of distortion is the same in both sexes but the fecundity functions differ, then (2) becomes

$$f^*(\bar{x}) + f(\bar{x}) > 2\,\bar{x}\,.$$

(c) If distortion occurs in only one sex, then we may put $f^*(x^*) = x^* = 1$ into (2), and the condition then becomes

$$f(\bar{x}) > \bar{x}\,,$$

which is the same as (a) above.

Thus a population will retain at stable equilibria all homozygous lethal gamete killers which act in only one sex if and only if the fecundity function in that sex has the property that $f(x) > x$ whenever

$x > 1/2$. Note that this inequality characterizes *all* compensation functions, and it is also true of the mass action function if $b \leq .5$.

Homozygotes Normal

One simple alternative to the assumption that the homozygous gamete killer is lethal is to assume that this genotype is completely normal. No gamete killers are known with this property, but this is not to imply that they do not occur. Their absence from contemporary populations may reflect the fact that any gamete killer which is homozygous normal will either be fixed or lost. This can be seen by going through the analysis of equation (1) with $f(2\bar{x}-1) = 1$. Because $k > 1/2$ and $f(\bar{x}) \leq 1$, we have that $2(1-k)f(\bar{x}) < 1 = f(2\bar{x}-1)$. Thus only two cases are possible: either (a) $2kf(\bar{x}) < 1$, in which case there is an unstable equilibrium at $\hat{p}_1 = [2kf(\bar{x}) - 1]/2[f(\bar{x}) - 1]$, or (b) $2kf(\bar{x}) > 1$, in which case the distorter is fixed. The fixation of the gamete killer in this case is not particularly eventful, however, because it brings about no decrease in the average fitness of the population.

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