AGE SPECIFICITY AND ECOLOGICAL THEORY[1]

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Abstract. This paper attempts first to deal with the question of aging as a byproduct of natural selection, drawing on the theories of Medawar, Williams and Hamilton, and second, to apply the conclusions to considerations of population and behavioral ecology. It is concluded that:

(1) Age-specific mortality should drop to a minimum prior to earliest reproductive age and then rise with age.

(2) Age-specific fecundity should rise with age to a peak, which may occur at almost any age depending on the sort of organism considered, and then fall.

(3) A sudden increase in mortality at a given age will result in natural selection favoring higher relative mortality at immediately preceding and following ages, and lowered fecundity immediately after that age.

(4) A sudden increase in fecundity at a given age will result in natural selection favoring relative higher mortality in early life and immediately after that age, as well as relaxed selection for increased fecundity, especially at middle and late ages.

(5) Selection acts to make increasingly steep the survivorship curve of a population declining or fluctuating due to changes in mortality.

(6) Selection acts to postpone reproductive effort in populations declining or fluctuating due to changes in fecundity.

(7) If mortality is very low, animals, as they age, should ideally take greater risks to secure reproductive success.

(8) The nature of altruistic behavior may be age specific, with older individuals more characteristically altruistic and younger individuals more characteristically pampered.

(9) Intergroup hostilities should be largely directed toward certain age groups.

Ecologists have for years worked with survivorship curves and age-specific fecundity curves and several papers dealing with evolution and the aging process have appeared in the literature (Williams 1957, Medawar 1957, Hamilton 1966). It does not seem to have occurred to many ecologists, however, that the mortality and fecundity changes with which they deal are, in fact, defined by the aging process, and that a knowledge of the natural-selective processes responsible for aging can profitably be applied to an understanding of birth and death rates. To my knowledge only one recent publication has even attempted to deal with this notion (Slobodkin 1968). It is the purpose of this paper to discuss the subject at some length, deriving expressions for selection on mortality and fecundity schedules and then applying these expressions to ecological theory.

AGING

In 1957, Medawar described a manner in which selection might act to influence the age of onset or demise of genetically determined traits. He used as his basis of thinking the "reproductive value," V_x, curve of Fisher (1958), which is a measure of the expected remaining contribution to the ancestry of future generations by an individual of age x (see Fig. 1). (A hypothetical set of data—see appendix—has been used to generate the curves in this and subsequent figures, except for Figure 11). The argument can be paraphrased as follows: To the extent that variation in the age of appearance of some given trait can be ascribed to additive genetic variance, the mean age of the appearance will be influenced by natural selection. Since the total genetic contribution of individuals which acquire the trait at an age of high reproductive value should be more greatly affected by the trait than the total genetic contri-

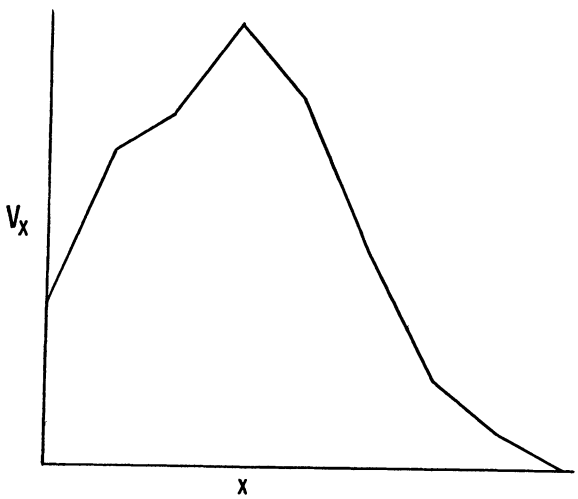

FIG. 1. The relationship between Fisher's reproductive value, V_x, and age, x.

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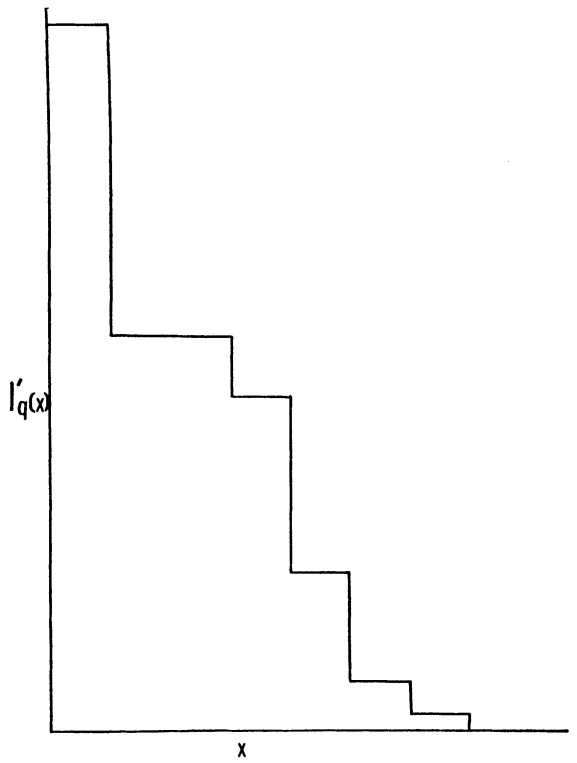

FIG. 2. Selection intensity with respect to age-specific mortality, $I'_q(x)$, as a function of age, x.

reproductive life as well as old age. As suggested by Hamilton (1966), parents which produce defective young will show higher fitness if those young die as soon after zygote formation as possible. Because of the advantage of efficient parental time and energy budgeting as well as rapid gene passage, selection will favor, on this basis, the precession in age of deleterious traits. This, of course, holds only for the parental care period which includes the time between zygote formation (or, in a sense, gamete production) and the severing of parental ties, and which in the case of some species may be almost negligably short. In this argument selection acts on individual genotypes through their parental stock—i.e. by kin selection (Fisher 1958; Haldane 1955; Hamilton 1964; Maynard Smith 1964). Mechanical considerations may lead to the same conclusion. Time is required for the development of maximum resistance to death and in viviparious species the demands of gestation on the parent do not allow for full development at birth. In other species, high larval mortality may be a necessary compromise brought on by the physical nature of a selected dispersal life-form.

There is another consideration which also leads to the same conclusion. We note that U_a, U_b, Z_a, Z_b are not always disjoint as assumed above. The life-long ontogenetic process also includes the co-development of many characteristics present throughout life, each at its own rate. Natural selection will then act to decrease or increase age-specific resistances to mortality by altering the rates of development, or age at which development starts, for each of these characteristics. It is the integration of these characeristics that affects susceptibility to stress and there may be one or several ontogenetic "adaptive peaks" associated with particular developmental configurations. If natural selection acts to push those peaks occurring late in life towards early reproductive maturity, some may be pushed beyond that age into the prereproductive period. Maximum realizable fitness will be achieved when these peaks become maximally compressed into the period up to and including earliest reproductive maturity. Maximum resistance to mortality should occur sometimes during this period.

Age-specific mortality, in other words, should generally be high at conception, fall to a minimum during prereproductive life, and then, after the age of first reproduction, rise with age. This pattern is beautifully borne out by data organized by Pearl (1940) on man (Fig. 3) (also see more recent demographic tables and Keyfitz 1968), and would appear to be true for mammals generally (Caughley 1966). Farner's (1945) data on the American Robin show a steady rise in q_x with age. High nestling mortality combined with these results yields the predicted mortality curve. In data

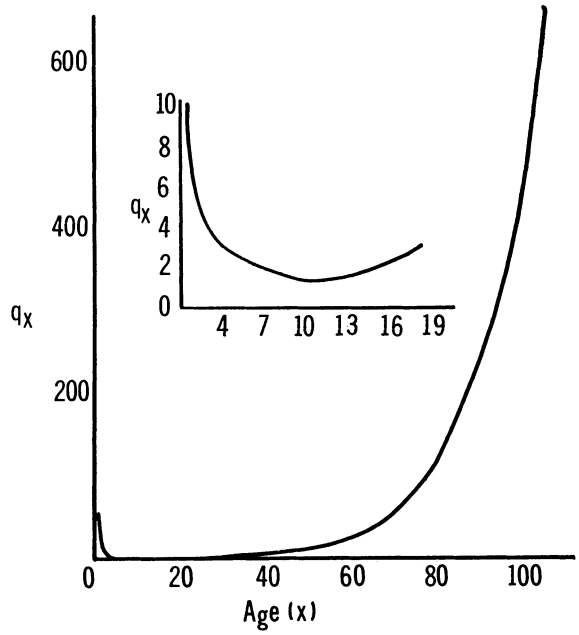

FIG. 3. The relationship between age-specific mortality, q_x, and age, x, in man.

gathered on birds by Lack (1943a, b, c) q_x appears to drop for the first year or first few years, remain fairly level (with considerable variability) and then rise in old age, but these trends are not clear cut and the final rise may be a statistical artifact. In rotifers the mortality curve rises with age yet appears to fall in very early age (Edmondson 1945). The data for the barnacle, *Balanus balanoides,* in France, show the same pattern (Hatton 1938, after Deevey 1947), as do those for *Daphnia pulex* (Frank, Boll, and Kelley 1957) and *D. obtusa* (Slobodkin 1954). After the initial drop in q_x following the fingerling stage, mortality also rises with age in the East Anglian herring (*Clupea harengus*), the Pacific herring (*C. pallasii*), the whitefish (*Coregonus ciupeaformis*), the sauger (*Stizostedian canadense*) (Beverton and Holt 1957), and the bluegill (*Lepomis machrochirus*) (Gerking 1962).

An interesting and pertinent observation less directly connected with mortality, per se, is that the ability of the prawn (*Hippolyte*) to change its protective coloration degenerates with age (Portman 1959). Still less directly connected, but perhaps significant, is the fact that, in man, learning ability, measured by two methods, rises with age until about 11, remains fairly constant until roughly 30 (see mortality curve, Fig. 3) then declines (Inglis, Ankus, and Sykes 1968). Cases such as those of many invertebrates with indeterminate growth, in which mortality falls off steadily with age—at least until very old age—can be explained on the basis of mechanical limitations [one component of $\Phi_q(x)$]: younger individuals by virtue of their smaller size are more easily destroyed than their larger, older relatives. It would be instructive to examine susceptibility to physiological, rather than physical or predative factors in such species.

Fecundity

Selection for age-specific birth rates can be handled in the same manner as selection for age specific mortality rates, where:

$$\Delta m_x = \frac{1}{R}\frac{\partial R}{\partial m_x}\Phi_m(x) \quad \text{(same as equation 2b)}, \quad (5)$$

$$I'_m(x) = \left|\frac{1}{R}\frac{\partial R}{\partial m_x}\right| = \frac{R^{-x}l_x}{\sum_{y=0} yR^{-y}l_y m_y}. \quad (6)$$

A selection intensity curve for fecundity is given in Figure 4.

Clearly, traits increasing fecundity will be pushed to earlier and earlier ages until stopped by opposing forces. These forces may be of several kinds. Selective factors may retard maturity. For example, a young animal has available to it only a finite amount of energy which must be used for growth, regulation and maturation. Survival is an obviously important factor in natural selection and must figure significantly in the evolution of systems of priority for energy appropriation. Obviously, if survival is threatened, maturation need not merit top priority. Another consideration is exemplified in the many bird species characterized by delayed maturity in the males. That is, males start mating at significantly later ages than females. Since late mating is disadvantageous, there must be some compensating factor that encourages waiting. It appears that young males often cannot compete with older males in the defense of territories, cannot so successfully defend their nests from predatory or conspecific attack, or cannot so efficiently care for their young (for example, see Coulson 1966; Coulson and White 1958). In short, females will generally have greater reproductive success if they mate with the older males (which they do). In addition, where this is likely to occur, the advantages of conserving time and energy and cutting risk may militate against even an attempt to win a mate. Under these circumstances, the early appearance of reproductive behavior and plumage is selected against. The Common Redwing (*Agelaius phoeniceus*) is such a case, the first-year males retaining much of their brown, juvenile appearance and appearing on territories only very briefly (unless unchallenged) early in the breeding season (Orians 1961).

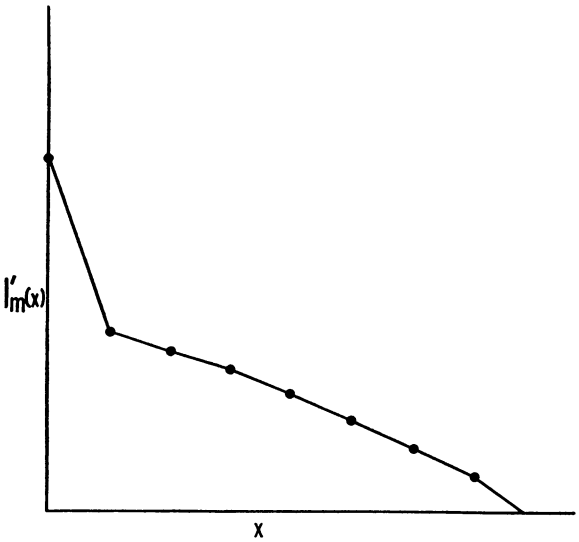

Fig. 4. Selection intensity with respect to age-specific fecundity, $I'_m(x)$, as a function of age, x.

With respect to mechanical difficulties it is

trivially apparent that individuals cannot give birth simultaneously with their own births. Reproduction cannot occur immediately, and prior to its appearance there can be no selection for successful reproduction (barring possible cases involving linkage or pleiotropy). Note also that the age at which developing characteristics integrate in such a manner that successful reproduction first becomes possible is, in fact, minimal reproductive age. Optimal reproductive success, on the basis of selective forces, and considering "ontogenetic inertia," must thus occur after minimal reproductive age.

In conclusion, fecundity should rise following earliest reproductive capability, then fall progressively with advancing age. This is true in a number of insect species, for example *Oncopeltus fasciatus* and *Aphis fabae* (Dingle 1965), *Tribolium castaneum* (Leslie and Park 1949), *Calandra oryzae* and *Rhizopertha dominica* (Birch 1953), and *Dacus tryoni* (Lewontin and Birch 1966). It is also true for *Daphnia pulex* (Frank et al. 1957), and appears to hold for rotifers (Edmondson 1945). Leslie and Ranson (1940) note the same trend for the vole (*Microtus agrestis*), and Keyfitz (1968) gives pertinent data for man. In many species, however, mechanical or experiential considerations seem to alter the picture considerably. There are many invertebrates and lower vertebrates with indeterminate growth in which egg number increases with body size. The result is that even though selection for fecundity falls off with age, individuals will produce larger numbers of young in later life. In the case of birds, too, clutch size and number of young fledged per breeding pair seems to rise with age, probably due to accumulated parental experience (Coulson and White 1958). The shape of the m_x curve may be such that fecundity reaches a peak anywhere from minimum reproductive age on. What is particularly interesting is that since selection for fecundity (which must involve selection for congenitally healthy young) falls off with age, progeny of older individuals (regardless of their numbers) would be expected to be more mortality-prone than progeny of younger individuals. To my knowledge this has been explored only in man where, as expected, the incidence of congenital defects first falls, then progressively rises with the age of the mother (Milham and Gittelsohn 1965; see Table 1).

Selection on the m_x curve may be viewed in either of two ways. In individuals which reproduce regularly, selection will tend to increase or decrease fecundity at each of those ages scheduled for reproduction. On the other hand, selection, through its effects on m_x, may be looked upon as determining those ages at which reproductive efforts should be most strongly directed. The first view is implicit in the above discussion. A particularly nice example of the second view is afforded us by species that reproduce only once during their lifetime, such as the Pacific salmon, in which potential fecundity increases with age. Here we can write, from (1), with T the age of reproduction and $m_y = 0$ for $y \neq T$:

$$1 = R^{-T} l_T m_T .$$

Hence,

$$R = \exp\left[\frac{\log(m_T l_T)}{T}\right].$$

Natural selection acts to maximize R. Thus, if we know l_T and physiological potential m_T as functions of T, in order to find optimum T, we have only to find that value of T for which R is maximum.

It may be argued that observed rises in mortality and drops in fecundity with age can be explained (except for the early decreases in death rate and increases in reproductive success) on the basis of gradually accumulated chromosomal aberrations. Clearly it would be satisfying to be able to show that under normal conditions (radiation of body tissues in the laboratory and by atom bomb explosion are not normal biological stresses) the rate at which such mutations occur falls initially with age

TABLE 1. Defects per 10,000 recorded human births[a]

Defect	Mother's age						
	15–19	20–24	25–29	30–34	35–39	40–44	45+
Anencephalus	9.8	8.7	7.2	7.7	8.8	9.7	5.5
Spina bifida	11.5	11.3	9.7	10.1	12.5	12.6	16.4
Hydrocephalus	18.0	7.2	6.9	7.6	9.6	15.2	24.5
Microcephalus	1.1	0.8	0.8	0.9	1.1	13.0	—
Heart defects	17.2	17.8	16.9	16.8	23.8	27.5	50.1
Cleft lip, palate	10.4	10.8	10.9	9.9	11.5	16.3	—
Mongolism	2.0	1.7	1.9	3.4	11.2	32.6	89.4
Club foot	14.8	13.7	11.2	11.3	12.4	14.3	17.9

[a]Data from Milham and Gittelsohn 1965.

and then rises. An examination of the information available indicates that this may be so. A number of papers (Court Brown et al. 1966, Jacobs and Court Brown 1961, Jacobs et al. 1963, Hamerton et al. 1965) show significant rises in the frequency of aneuploidy with age. Except for the data of Jacobs et al. (1963), there also appears (although in no case is it statistically significant) to be a decline in the proportion of aneuploid cells prior to ages 5 to 14. If this drop is real it means not only a tendency for the abnormal cells to be lost with age, but also a drop in the rate of appearance of these abnormalities. Furthermore, in all the above cases, the subsequent rise in aneuploidy with age appears to be linear or increasingly steep. If, as seems certain (and as suggested above), the abnormal cells are discarded, then this means that the rate of appearance of aneuploid cells increases, or the rate of their disposal decreases with age, or both.

Perturbations in the survivorship and fecundity curves

The shape of the l_x and m_x curves is the result of a balance between the selective forces described above and opposing forces which may or may not be directly related to natural selection. Thus, if $I'_q(x)$ or $I'_m(x)$ were changed, we would expect the resulting selective equilibrium values of q_x or m_x to change accordingly. Suppose, for example, that for some reason q were to increase between ages $k-1$ and k. The result of such a change is a transient $I'_q(x)$ curve which changes the rate of selection on all q_x. An examination of equation (3), for R very roughly 1, shows the resulting transient curve to drop more rapidly than the original curve for $x < k-1$, less rapidly for $x > k$. An example of the appropriate transient curve is given in Figure 5. The arrows show the directions in which selection pushes deleterious traits relative to their old equilibrium positions. This and subsequent figures are based on different sets of hypothetical data given in the appendix, but the results are general in application. As traits responsible for decreased resistance to mortality factors accumulate to either side of the interval $k-1$ to k, the selection intensity curve smooths and the survivorship curve is brought to a new equilibrium form. The interesting point is that an increase in susceptibility to one source of potential mortality at a given age tends to "attract" to the same general age traits resulting in increased susceptibility to other mortality sources.

Suppose m increases at age k. In this case the $I'_q(x)$ curve slopes less steeply than the original

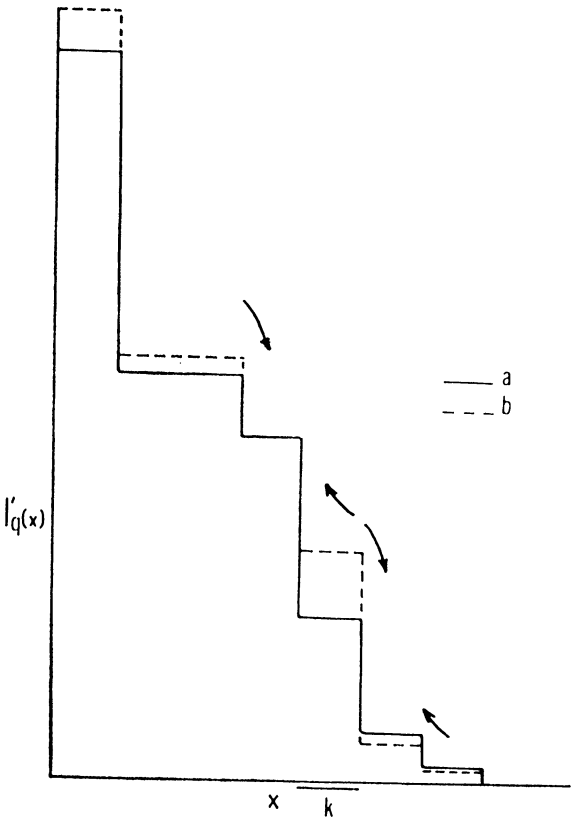

FIG. 5. Selection intensity with respect to age-specific mortality:
(a) before increase in q_k (mortality at age k),
(b) immediately after increase in q_k.

as x increases, and drops suddenly at $x = k$. The transient curve for this particular situation is shown in Figure 6.

The same sort of arguments can be applied to the $I'_m(x)$ curve. The drop in $I'_m(x)$ (transient with increase in q_k) is less steep than for the original curve and the curve jumps downward at $x = k$. Thus, there will be an increase in deleterious fecundity traits immediately following age k. The value of $\partial T'_w(x)/\partial m_k$ becomes increasingly negative with x until the trend is reversed for large x. The appropriate transient selection intensity curves are shown in Figures 7 and 8. The general results of this discussion may be summarized as follows:

(a) Increased q_k leads to selection favoring increased q_x for x close to k (Fig. 5).
(b) Increased m_k leads to selection favoring raised q_x for x immediately following k (Fig. 6).
(c) Increased q_k leads to selection favoring lowered fecundity at ages immediately following k (Fig. 7).

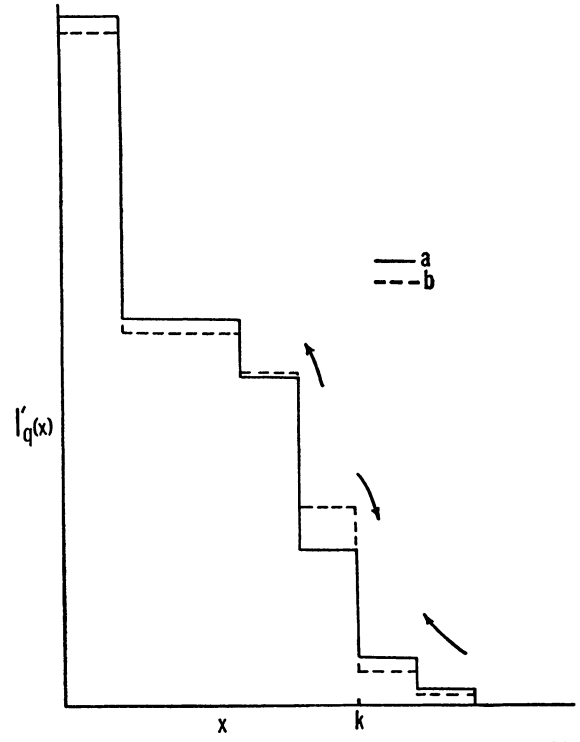

FIG. 6. Selection intensity with respect to age-specific mortality:
(a) before increase in m_k (fecundity at age k),
(b) immediately after increase in m_k.

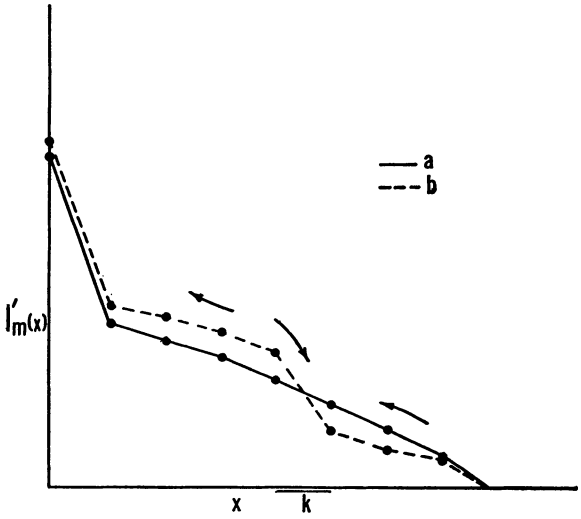

FIG. 7. Selection intensity with respect to age-specific fecundity:
(a) before increase in q_k (mortality at age k),
(b) immediately after increase in q_k.

(d) Increased m_k results in relaxed selection for reproduction, particularly in middle and late ages (Fig. 8).

The above conclusions apply to changes occurring within populations and not between species.

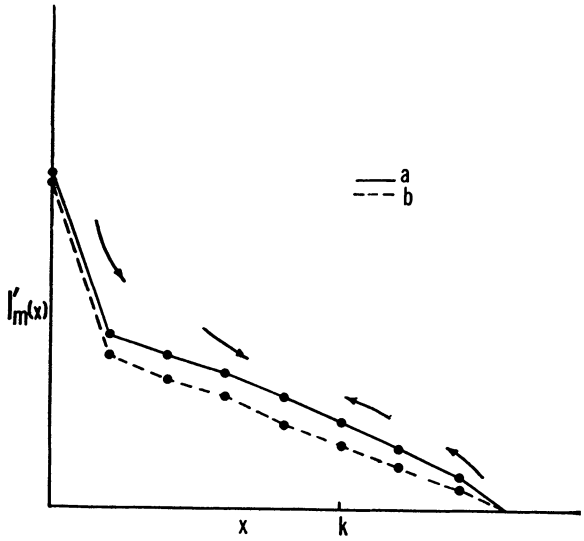

FIG. 8. Selection intensity with respect to age-specific fecundity:
(a) before increase in m_k (fecundity at age k),
(b) immediately after increase in m_k.

The use of selection intensity curves may, however, be extended to cover the latter case. For example, species which for some reason (small sized individuals, perhaps) have inevitably high mortality rates, posses $I'_m(x)$ curves which fall off rather rapidly toward old age. This, in effect, means little selection except at young ages and tends to mass all reproductive activity in early life. Early reproduction results in relaxed selection against mortality factors later in life and accentuates still further the drop in the $I'_m(x)$ curve with age. It is thus no accident that small animal species reproduce early in life (and are often semelparous) and possess short life spans even when protected from the predation or harsh climates that are the cause of their greatest mortality in the field. There is simply no selection acting to keep the physiological processes viable in old individuals. Increasingly larger species (and species inhabiting increasingly stable areas or places where they are less subject to predation) suffer increasingly less mortality. Thus, there is less selection pressure to reproduce early and selection against susceptibility to mortality factors later in life is stronger; potential longevity is greater. It is well known that larger animals tend to show greater longevity than small even under ideal conditions. It also seems generally true that marine organisms (stable environments) have potentially longer lifespans than similarly sized aquatic or terrestrial organisms (less stable environments). Similar explanations of most of these phenomena have been made previously by Williams (1957).

Fluctuations

The conditions under which equation (1) holds specify no temporal change in m_x, q_x. Suppose, however, that m_x or q_x undergo a sudden change and that the population rapidly comes to a stable age distribution and remains there during its subsequent increase or decline. Populations fluctuating in size due to repeated changes of this sort may be explored legitimately through the use of equation 1, and although such populations are not accurate reflections of real populations, they to some degree approximate them. If the l_x and m_x curves of a stable population are changed by virtue of the onset of fluctuations of the sort described above, the l_x and m_x curves of any fluctuating population should also change in, qualitatively, the same fashion. Now, populations fluctuating due to changes in selective birth and death rates will increase if and only if relaxed selection pressures allow them to do so. Selection acts most strongly during population decline when the number of selective deaths increases and/or selective fecundity falls. Thus the bulk of selected change occurs when $R < 1$, so that the net effect of selection in fluctuating populations should, unless changes in Φ are opposite to and sufficiently greater than changes in I', be as implied by the $I'_q(x)$, $I'_m(x)$ curves but with $R < 1$. In constant populations, $R = 1$. The appropriate curves are given in Figures 9 and 10. It is important to note that selection pressures may be stronger in declining populations as opposed to increasing populations only with respect to factors responsible for the population decrease (i.e. selective deaths, or selective fecundity, as the case may be.) Thus, if the fluctuations are caused strictly by changes in mortality the $I'_q(x)$ curve is affected by fluctuations (Figure 9) but not the $I'_m(x)$ curve. Similarly, if mortality remains constant and fecundity varies there will be a change in the pattern of selection due to population fluctuations (R effectively less than one) only with respect to fecundity. In the case of populations declining or fluctuating due to changes in mortality rates it is clear from Figure 9 that selection for an early age of maximum resistance to stress is relatively greater than in a constant population. Where decline or fluctuation follows from variation in fecundity (Figure 10) maximum reproductive success and, therefore, subsequently maximum reproductive effort should occur later in life than in constant populations. This is an accurate statement of the often-heard thesis that early reproduction is favored in growing, delayed reproduction in declining populations. It is doubtful that clear-cut, side-by-side cases of stable populations and populations changing due strictly to mortality changes or fecundity changes ever exist in nature, but the predictions above provide

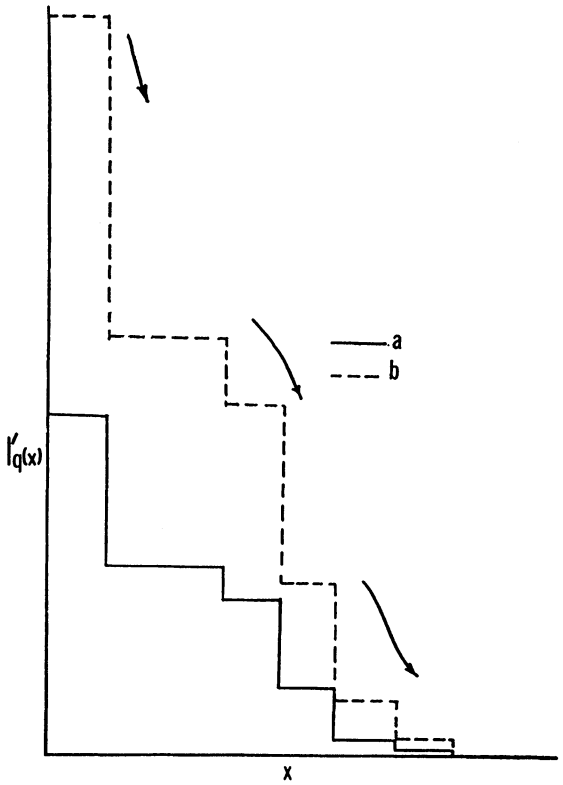

Fig. 9. Selection intensity with respect to age-specific mortality:
(a) $R = 1$ (equilibrium populations),
(b) $R < 1$ (declining or fluctuating populations).

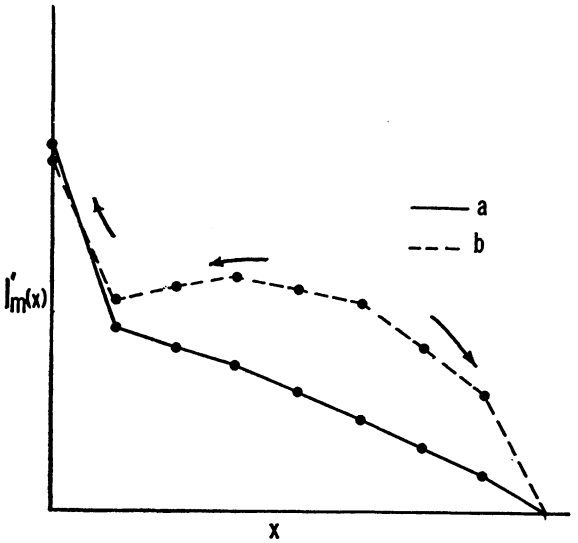

Fig. 10. Selection intensity with respect to age-specific fecundity:
(a) $R = 1$ (equilibrium populations)
(b) $R < 1$ (declining or fluctuating populations).

a rather elegant method for laboratory testing of the general approach to aging discussed in this paper. What may happen in nature is that population growth resulting from fewer genetic deaths results in stiffer competition for ability to produce offspring rapidly. Thus $I'_m(x)$ may, in actuality, be affected by mortality-induced population fluctuations. In this case selection acts on fecundity as if $R > 1$, and reproduction at earlier ages is advantageous. It has been noted before (Ford 1964) that over long periods of time (several fluctuations) selection acts more quickly to bring about changes in fluctuating than in stable environments. It appears also that selection will act in slightly different directions.

Time, Energy and Risk

In 1962 Levins introduced a technique for investigating adaptation patterns in heterogeneous environments which involves what he calls "fitness sets" and "adaptive functions." The technique has since been used elsewhere with varying degrees of success (Levins and MacArthur 1966, MacArthur 1965, MacArthur and Levins 1964). In all cases, age specificity has been ignored. In the following discussion I have applied the technique to an examination of time and energy budgeting as affected by risk factors taking fully into consideration the matter of age.

Natural selection may be thought of as acting as an efficiency expert. Thus, we should expect that an animal's budgeting of the time and energy available to it would be such as to maximize fitness. The incorporation of considerations of the risk involved with different activities, however, is much more difficult. Consider all forms of risk together as unavoidable byproducts of activity leading ultimately (and often very indirectly) toward the successful passage of genes, and assume that natural selection will tend to maximize R. Then, with respect to selected changes in R due to mortality and fecundity changes only at age x:

$$dR = \frac{\partial R}{\partial q_x} dq_x + \frac{\partial R}{\partial m_x} dm_x ,$$

so that for a given value of R, $dR = 0$, and:

$$\frac{dq_x}{dm_x} = -\frac{\partial R/\partial m_x}{\partial R/\partial q_x}.$$

Substituting from equations (3) and (6), and changing slightly the form, this becomes:

$$\frac{d(1-q_x)}{dm_x} = -\frac{I'_m(x)}{I'_q(x)} = -\frac{R^{-x}l_x(1-q_x)}{\sum_{y=x} R^{-y}l_y m_y}. \quad (7)$$

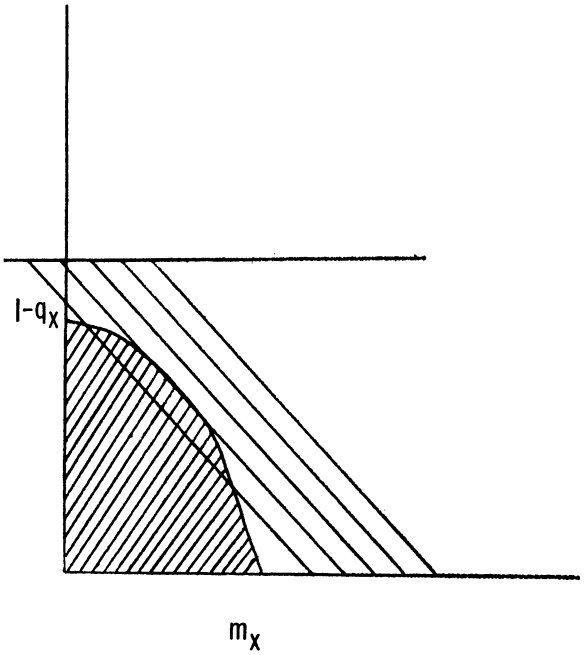

FIG. 11. The fitness set (shaded) and adaptive function (parallel lines) for fecundity-risk relations at age x.

Equation (7) provides a family of lines relating survivorship through the interval $x - 1, x$ to fecundity at age x, each of which represents a given value of R. That is, the relation between survivorship and fecundity given above defines a family of R isoclines. This is the "adaptive function" of Levins. Now consider the set of all combinations of $(1 - q_x)$ and m_x realizable in the living organism. A genotype which induces its owner to expend a maximum amount of time and energy looking after its own survival will not allow for high fecundity also. Similarly an individual which concentrates maximally on producing young has little time and energy left for defending itself. The shaded area in Figure 11, superimposed on a graph showing the adaptive function, represents the set of all possible combinations of $(1 - q_x)$ and m_x. This is Levin's "fitness set." Since selection acts to increase fitness (R), it chooses that genotype in the fitness set tangent to the highest isocline of fitness. This approach allows us to make predictions concerning optimal priorities in an animal's use of time and energy in the face of defined risks. Without quite elaborate data, quantitative application of the model is impossible. But concrete uses of the above technique do not concern us here. What does concern us is the fact that the adaptive function changes with age. The change in $d(1 - q_x)/dm_x$ between ages $x - 1$ and x can be written:

there will be no differential behavior between altruists and recipients of different ages. Where age recognition occurs, however, selection should favor discriminatory reactions in individual encounters and a tendency to change individual biases with age; each age group will come to behave altruistically toward individuals of some but not all other age groups. This situation is shown graphically in Figure 12. For any altruist of age a, there will be a corresponding value of $I'_q(a)$, so that for given values Δq_a, Δq_b, and α, there also will be a corresponding value of

$$1/\alpha\, I'_q(a)\, \frac{|\Delta q_a|}{|\Delta q_b|}.$$

$I'_q(b)$ (selection intensity on the recipient) will meet the required conditions for altruistic treatment for all ages prior to k (see equation 9a, shaded portion of Fig. 12). For a recipient of age b (Fig. 13, equation 9b) there are corresponding values of $I'_q(b)$, and

$$\alpha\, I'_q(b)\, \frac{|\Delta q_b|}{|\Delta q_a|}.$$

All individuals older than k (shaded portion of Fig. 13) are potential altruists with respect to individuals of age b.

Parental care and the subsequent, often dramatic, rebuff of young as they grow older are examples of age specificity in altruism so well

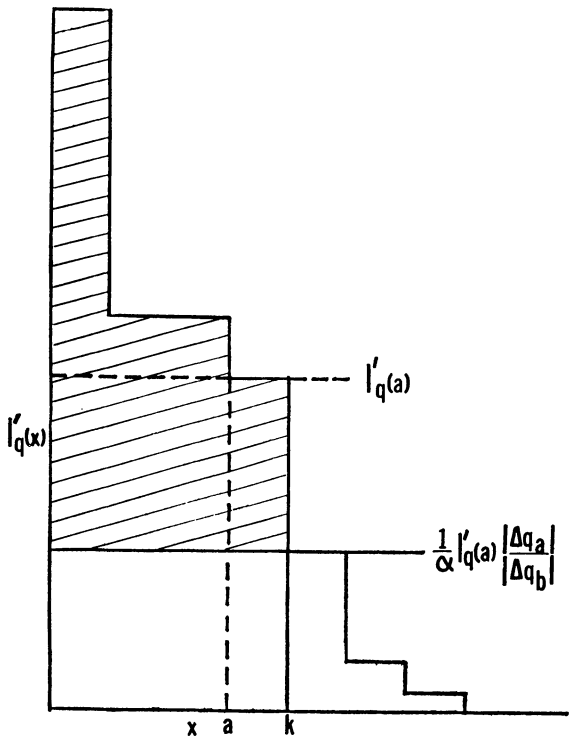

FIG. 12. Age ($< k$) of potential recipients of altruism from individuals of age a.

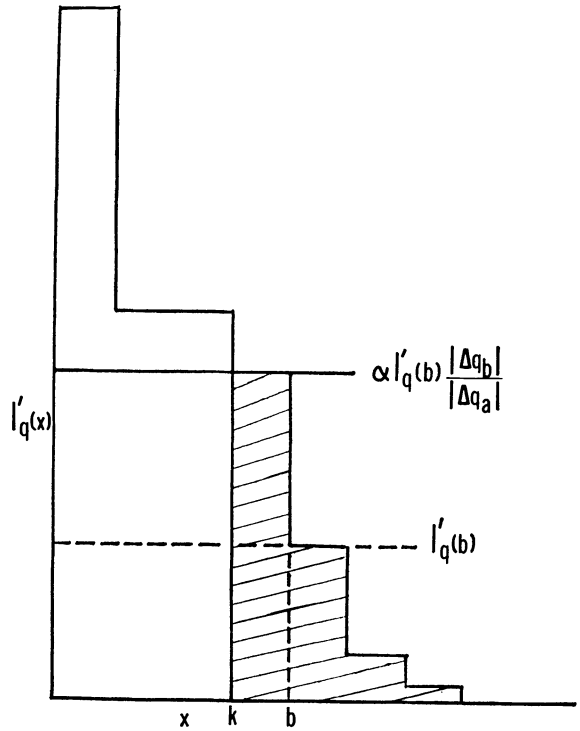

FIG. 13. Age ($> k$) of potential altruists towards individuals of age b.

known that they appear almost trivial. The protection and attention afforded young animals by adult males in some higher primate species is another example. Beyond these rather straightforward cases, though, information is difficult to find. For one thing, until the benefits accruing to actor and receiver are known, there is no way to categorize a trait as altruistic or selfish. Since these benefits are known only for a number of rather obvious traits (for example, some aspects of physical protective behavior, alarm calls) it is hard to know where to look for information on age specificity in altruism. Is grooming behavior in monkeys altruistic or not? Another difficulty is that selflessness is often hard to identify even when the benefits mentioned above are known. Is the willingness of one individual to share a food source, when another is unwilling to do so, a sign of altruism or an indication that the individual is incapable of displaying dominance? It is often unclear as to whether an altruistic act affects the recipient's survival or fecundity more importantly. In the former case protective behavior should be most directed toward young, prereproductive animals [$I'_q(x)$ is maximum]; in the latter case, towards individuals in their reproductive prime [$I'_m(x)$ is maximum]. Recognition of specific behavior patterns which are altruistic and the subsequent examination of those patterns for any