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Theories of Life-History Evolution[1]

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SYNOPSIS. In order to assess current scientific understanding of life-history evolution, the alternative fundamental theories are formulated in a refutable form and compared with the available empirical evidence. The hypothesis that life-history does not evolve is rejected on the grounds that life-history can be readily modified by artifical selection. The hypothesis that life-history evolves according to mechanisms other than natural selection acting on genetic variation is shown to have no sound experimental basis. The hypothesis that life-history evolution depends primarily on group selection is undermined by the absence of the predicted group adaptations. The hypothesis that life-history is a unitary character which evolves in the same fashion as fitness is rejected because of the disparity between life-history genetics and basic theory concerning the evolution of fitness. The hypothesis that life-history is composed of a set of autonomous characters which are subject to mutation accumulation at later ages is refuted by the lack of any detectable increase in genetic variability with age and the evidence for the interdependence of life-history characters. It is concluded that the hypothesis of antagonistic interactions between life-history characters, generalized to take genetic variability into account, is the most satisfactory theory of life-history evolution available.

INTRODUCTION

This article attempts to give an overview and an assessment of theories of life-history evolution. Interpreted broadly, this would be quite a substantial task. In order to proceed, I will cut it down to size by adopting restricted terms of reference. A great deal will thus be neglected.

Firstly, here "theories" refers only to fundamental presuppositions, not to particular sets of equations, although such equations can, of course, embody and elucidate such presuppositions.

Secondly, I will take "life-history" to refer to two types of character only: survival and reproduction. These categories include longevity, fecundity, viability, mating success, time to sexual maturation, and the like. They exclude patterns of life cycle alternation (*e.g.*, spermatophyte and gametophyte stages), sex-determination, breeding systems (*e.g.*, monogamy, polygyny), and so on.

ANTI-DARWINIAN THEORIES

Biological theories may be readily sorted into two groups: those which are compatible with neo-Darwinism and those which are not. Here I take neo-Darwinism to be the theory that directional evolutionary change is caused by natural selection acting on genetic variation (*cf.*, Maynard Smith, 1969). Though it is easy to think of imperfections in this cursory definition, it does at least outline the central idea, and that is all I need here. Since the term "non-Darwinian" has already been adopted as a name for the neutralist view of evolution (Crow, 1972), a view which *is* compatible with neo-Darwinism, I will use the adjective "anti-Darwinian" to designate theories which are incompatible with neo-Darwinism. Anti-Darwinian theories may in turn be split into two types: those which reject the neo-Darwinian view of the evolutionary process, and those which accept this view, but reject the explanation of it provided by neo-Darwinism.

Rejection of the neo-Darwinian view of the evolutionary process

The first type of anti-Darwinism assumes that life-history is immutable, being a fixed species attribute. Unfortunately for this view, even by Darwin's time there was enough evidence available from plant and animal breeding to suggest that life-history characters evolve without speciation. A further hundred years of scientific and agricultural breeding experiments have only

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bolstered the case for the malleability of life-history under artificial selection (Falconer, 1960 *passim*). Moreover, observations of evolutionary change in nature, especially among Lepidoptera, leave little grounds for contending that natural selection, unlike artifical selection, is inflexibly conservative (Sheppard, 1964; Ford, 1971). Those who reject the hypothesis of intraspecific evolution (*e.g.*, Williamson, 1981) do so in the face of a considerable body of scientific evidence.

Rejection of natural selection

Among scientists, the most common form of anti-Darwinism is the rejection of the hypothesis that evolution is directed by natural selection acting on genetic variants ultimately supplied by arbitrary, or "random," mutation. Leaving aside those who espouse this view in general terms, it has been explicitly put forward as a theory of life-history by F. A. Lints (Lints and Hoste, 1974, 1977; Lints, 1978) and at least acceded to by Wattiaux (1968).

The argument of Lints is that his experiments on life-span and fecundity in *Drosophila melanogaster* Meigen gave results which could not be explained in terms of natural selection, and therefore some other source of cumulative parental effects must be postulated. (What this alternative source might be is left unspecified.) The experimental observations in question were of a seemingly inexplicable decline followed by a recovery in both fecundity and longevity over the course of two independent selection experiments involving bristle number (Lints and Hoste, 1974, 1977).

There were two artifactual problems with these experiments. Firstly, it is well-known that *Drosophila* life-history characters are subject to purely fortuitous fluctuations arising from seasonality, medium preparation, culture contamination, and other environmental factors, making appropriate controls an absolute necessity (Bell *et al.*, 1955; Rasmusson, 1956). Such controls were not used. Secondly, these experiments employed populations generated by mass hybridization of four *D. melanogaster* laboratory stocks only a few generations before data collection began. Better methods for disrupting genetic equilibrium are hard to imagine. When Comfort (1953) performed a similar experiment on *Drosophila* life history, using inbred lines, which were *not* crossed, to remove segregating allelic variability as much as possible, there were no effects like those observed by Lints and Hoste.

Since the case that Lints offers is based on little more than the puzzling results of technically deficient experiments, while similar *Drosophila* experiments of better design have proved readily interpretable (Comfort, 1953; Wattiaux, 1968; Rose and Charlesworth, 1981*a*, *b*), it seems clear that he has no cogent argument against neo-Darwinian theories of life-history evolution. I conclude that a successful refutation of the overall neo-Darwinian view of life-history evolution has yet to be provided. Until that event, the field of battle belongs to rival neo-Darwinian theories.

GROUP SELECTION THEORIES

"Group selection" occurs when there is sufficient differential propagation and extinction *between* groups for the attributes of individuals to evolve so that the propagation of the group *as a whole* is fostered, irrespective of the propagation of individuals. These groups may range in size from randomly-mating, communal, breeding populations (Wynne-Edwards, 1962) to species (Stanley, 1979). Though the view that group selection is the predominant force in evolution is a minority one among evolutionary biologists, it is by no means incompatible with neo-Darwinism as a whole.

The theory that group selection controls senescence and longevity dates back to Alfred Russel Wallace, but it was August Weismann who was its foremost nineteenth century exponent (Wilson, 1974). Most importantly, both men argued that senescence occurs in order to kill older individuals that would otherwise impair the fitness of younger individuals, and thus that of the group, by competing with them for food, space, etc. Though this theory has been criticized for implicitly assuming that the old must be "worn-out," and therefore senescent already, for the group selection

mechanism to work (*e.g.*, Comfort, 1979, p. 11), such "wearing out" could be due to accidental damage accumulating through time. Thus this theory is not inherently deficient.

But there is at least one respect in which this theory of longevity evolution is vulnerable to attack: empirically. If senescence is a group adaptation to kill the old, then old organisms in the wild should frequently die as a result of some programmed, internal process, after the fashion of cellular lysis. This almost never happens (Comfort, 1979, pp. 45, 140–145). Death due to senescence is a phenomenon which is almost entirely confined to organisms provided with optimal laboratory conditions or medical care. Exceptions, like that of Pacific salmon, may be explained in terms of particular individual selection mechanisms (Williams, 1966*a*, pp. 174–175). Thus senescence cannot be an adaptation for killing the old produced by group selection acting in the wild.

Wynne-Edwards (1962) has argued that characters like early fecundity are also primarily selected according to the benefits accruing to large breeding groups, rather than individuals. But, if this was generally true, then gregarious animals should exhibit large scale communal allocation of reproductive effort among unrelated individuals. There is abundant evidence against this for most gregarious breeders (Williams, 1966*a*, pp. 187–191). Although individual animals do sometimes "adopt" totally unrelated progeny, especially among vertebrates, this practice is never so widespread as to require any explanation beyond that of a maladaptive side-effect of neural programming for the care of true progeny. The eusocial insects provide the great exception to this rule, and in such cases kin selection, rather than proper group selection, is almost certainly the factor responsible for communal allocation of reproductive function (Maynard Smith, 1964; Hamilton, 1964).

Therefore, group selection does not seem to be the prevailing factor in life-history evolution. But whether or not it *ever* has a significant effect on life-history evolution is another, as yet undecided, question. There does not seem to be any fundamental reason for excluding this possibility, beyond the dictum of Occam's Razor not to multiply hypotheses beyond necessity. In any case, we are forced to turn to individual selection theories of life-history evolution. No other type of theory seems to be adequate.

INDIVIDUAL SELECTION THEORIES

The extant anti-Darwinian and group selection theories of life-history largely lack specific scientific content, having empirical significance primarily in their opposition to more conventional neo-Darwinian views. Conversely, the various individual selection theories to a large extent depend on basic neo-Darwinian theory for their substantive content. (Here, "individual selection" refers to all forms of natural selection other than group selection.) Distinguishing between them requires attention to their specific assumptions and corollaries, rather than their general line of argument.

As I hope to demonstrate, perhaps the most helpful way of distinguishing between basic individual selection theories is to examine their assumptions about the developmental foundations of life history. There are three logical possibilities for the developmental physiology of life-history: it could be a unitary character, a label for a set of particulate characters, or something between these two extremes. Indeed, all three of these views have been adopted by specific authors, implicitly or explicitly. Each merits discussion.

The unitary hypothesis

The unitary hypothesis is generally not overtly presented as a theory of life-history evolution, although it appears to be quite widely held. The central idea is that "reproduction and the survival characteristic are generally inseparable" (Giesel, 1979, p. 323). Characters like fecundity, viability, longevity, etc. are all taken to be bound up with the overall soundness of an organism's physiology in its given environment. Thus genes which enhance one of these attributes will tend to enhance all of them, according to the hypothesis. In essence, *all life-history characters are seen as positively cor-*

related components of fitness, evolving as fitness evolves (Giesel and Zettler, 1980, p. 302).

Fortunately, the evolution of fitness is something about which we know a great deal. Perhaps our most important general result concerning the evolution of fitness is Fisher's (1930) Fundamental Theorem of Natural Selection:

$$\dot{W} = V_A,$$

where $\dot{W}$ gives the rate of change in the mean relative fitness and V_A is the additive genetic variance of fitness. (The additive genetic variance of a character is that part of the total phenotypic variance which is absorbed by a generalized least-squares linear regression of phenotypes onto the allelic composition of genotypes [*cf.*, Kempthorne, 1957, pp. 320–321; Falconer, 1960, p. 123]. A beter name might be "linear genetic variance.") It is now known that this result of Fisher's is exact only under restricted conditions, but for genes of small effect it applies to at least a leading order of magnitude under a wide variety of conditions (Nagylaki, 1977). Thus there is an extremal principle for evolution analogous to that of thermodynamics: since variances are non-negative, mean fitness will tend to climb toward local peaks on what Sewall Wright (*e.g.*, 1977) has called "fitness-surfaces."

One of the central corollaries of this body of theory is that at evolutionary equilibrium the additive genetic variance of fitness will be close to zero, exactly equalling it in many cases (Ewens, 1976). Thus, if the unitary hypothesis is indeed the correct theory of life-history evolution, then additive genetic variances for life-history characters like viability and early fecundity should always be quite small among endemic populations in stable environments. Moreover, in order to cement each life-history character firmly to fitness, genetic covariances between life-history characters should be predominantly positive. That is, genes affecting life-history should affect all such characters in essentially the same fashion, whether deleterious or beneficial.

Up until the 1970s, most of the available evidence supported the unitary hypothesis. Broadly speaking, the ratio between additive genetic variance and total phenotypic variance, or "heritability," for characters like fecundity in *Drosophila* laboratory stocks, conception rate in cattle, or egg-laying in poultry is much less than that for characters like white spotting in mammals or abdominal bristle number in *Drosophila* (Falconer, 1960, pp. 167–168). Thus additive genetic variances for life-history characters seemed to be relatively small, in general, as required by the hypothesis. Moreover, several studies have shown that new mutations and the genes which are responsible for inbreeding depression exhibit positive correlation in their effects on all fitness components, be they viability, fecundity, or longevity (Temin, 1966; Mukai and Yamazaki, 1971; Giesel, 1979).

But recently these results have been found wanting in generality. Firstly, in popultions which have not been domesticated or inbred, evidence of fairly high additive genetic variances has been found for such life-history characters as developmental rate (Istock *et al.*, 1975; Dingle *et al.*, 1977), viability (Mukai *et al.*, 1974), and fecundity (Derr, 1980; Rose and Charlesworth, 1981*a*, *b*). Whereas it was once thought that such characters would always have heritabilities in the range 0.0 to 0.2, as indeed they do in domesticated and/or inbred populations, the range among consistently outbred and undomesticated populations appears to be about 0.0 to 0.6. This is contrary to those predictions of the unitary hypothesis which have been outlined above and elsewhere (*e.g.*, Istock, 1978, 1982). Secondly, it now appears that alleles which are not deleterious in their net effect on fitness, and thus achieve high frequencies in outbred populations, are often *negatively* correlated in their effects on life-history characters (Hiraizumi, 1961; Simmons *et al.*, 1980; Rose and Charlesworth, 1981*a*, *b*). Thirdly, it has been found that life-history is readily modifiable by means of artificial or natural selection (Wattiaux, 1968; Sokal, 1970; Rose and Charlesworth, 1981*b*; Doyle and Hunte, 1981), confirming the soundness of the inference that the additive genetic variances of life-history characters are not all small.

There are three immunizing strategems

which a convinced exponent of the unitary hypothesis could try. Firstly, it could be argued that environmental variation could act to maintain the variability for life-history characters which exhibit genotype-environment interaction, effectively resulting in temporal variation in genotypic fitnesses. But some of the experiments showing large additive genetic variances were performed on populations which had been maintained under stable laboratory conditions for many generations (*e.g.*, Rose and Charlesworth, 1981*a*, *b*). Secondly, it could be argued that these were all laboratory studies and "the field is different." But Noordwijk *et al.* (1980) have obtained similar results from field populations of the Great Tit. Thirdly, it could be argued that natural selection indirectly acting to eliminate genetic variability is being held in check by mutation. The problem with this view is that models with deleterious mutation show that the intensity of natural selection is inversely related to the equilibrium genetic variance (Charlesworth, 1980; Rose and Charlesworth, 1980). The present estimates of life-history genetic variances are of the same order of magnitude as those for characters less directly related to fitness (*cf.*, Falconer, 1960). One thus must argue either that *all* characters are equally fitness characters, roughly speaking, or one must invent *ad hoc* hypotheses about differences in mutation rates, with life-history being more subject to mutation. Even if that dubious step is taken, the evidence for negative genetic covariance between life-history characters must be surmounted. Such maneuvering is more evidence of the bankruptcy of a theory than a defense of it.

Thus, among outbred populations the unitary hypothesis appears to be quite wrong. That it should have been given considerable credence as a completely general theory of life-history evolution, often to the point of being assumed unthinkingly, is probably attributable to the concentration of experimentalists on both inbred populations and genes with predominantly deleterious effects on fitness, especially those produced by artificial mutagenic procedures. Unfortunately, it seems to have been the case that the most convenient biological material for experimental population genetics was not sufficiently representative of natural populations in general.

The particulate hypothesis

The particulate hypothesis of life-history evolution originates with Haldane (1941, pp. 192–194) and Medawar (*e.g.*, 1952), with subsequent formalization and clarification provided by Hamilton (1966), Edney and Gill (1968), Emlen (1970), Charlesworth and Williamson (1975), and Charlesworth (1980). (Though it has often been suggested to me that this hypothesis was never seriously entertained, personal communications with both E. B. Edney and B. Charlesworth indicate that it certainly was. Moreover, M. R. Rose can be added to the list of those who were once convinced of its general validity.) First I shall outline the basic ideas of the theory.

Assume that life-history is divided up into age-specific components, each of which evolves independently. Possible examples of such components are age-specific survival probabilities and age-specific fecundities. Hamilton (1966) and Charlesworth (*e.g.*, 1970, 1976, 1980) have shown that, under simple conditions, the evolution of genes affecting such characters depends on their effects on the Malthusian parameter, defined by

$$\int_0^\infty e^{-rx} l(x) m(x)\, dx = 1,$$

where r is the Malthusian parameter, l(x) gives the age-specific survivorship probability, m(x) gives the age-specific fecundity, and x is chronological age. The central mathematical result, on which the particulate hypothesis of life-history evolution is based, is that for genes with effects confined to a single age-class and f(x) defined by the equation

$$f(x) = \frac{\partial r}{\partial \ln P(x)},$$

it follows that

$$\frac{\partial f(x)}{\partial x} \leq 0,$$

where ln P(x) is the natural logarithm of

the survival probability at age x. Likewise, for g(x) defined by

$$g(x) = \frac{\partial r}{\partial m(x)},$$

it can be shown that

$$\frac{\partial g(x)}{\partial x} \leqslant 0,$$

providing $r \geq 0$. Thus the strength of selection acting on genes affecting single age-classes declines with the age of action. In effect, *the influence of natural selection fades out with increasing age.*

The most important empirical corollary of this is that the balance between natural selection and recurrent deleterious mutations of age-specific effects should change with age, such that the additive genetic variance for age-specific life-history characters tends to increase with age, all other things being equal (Charlesworth, 1980, p. 217; Rose and Charlesworth, 1980). Thus, on the particulate hypothesis of life-history evolution, there should be substantially greater additive genetic variance for life-history characters at later ages, all other things being equal. (Again, *ad hoc* hypotheses about mutation rates could explain any contrary variance pattern.) The only direct test of this which is known to me was one I made using an outbred *Drosophila melanogaster* population (Rose, 1979; Rose and Charlesworth, 1980, 1981*a*, *b*). I tested whether or not the additive genetic variance for *Drosophila* age-specific fecundity, corrected for scaling effects, increases with age. It did not in my experiments. Indeed, the estimated slope of the linear regression of this parameter on age was negative, though not significantly so. In addition, artificial selection experiments qualitatively corroborated this result (Rose and Charlesworth, 1981*b*). (Of course, it would still be desirable to have such tests repeated independently and with other species.)

A second corollary of the particulate hypothesis is that early and late life-history characters should exhibit little genetic correlation. Again, exactly the reverse was found for the *Drosophila* population I studied. For the time being, the particulate hypothesis of life-history evolution must be regarded as falsified.

The variable pleiotropy hypothesis

From the failure of the unitary and particulate hypotheses, it is clear that theories of life-history evolution must allow for interactions between life-history characters which are neither fully autonomous nor inextricably associated with one another. The forerunners in the development of such theory were Lack (*e.g.*, 1954) and Williams (1957, 1966*a*, *b*).

Williams (1957) was especially clear in arguing for the importance of "antagonistic pleiotropy": negative correlations between gene effects on different life-history characters. Essentially, he argued for the view that there is often a trade-off between life-history characters. Thus life-history characters are seen as multifold, but not wholly autonomous. However, because of the declining force of natural selection with age, those genes which enhance early life-history characters, such as juvenile viability, would be favored in spite of later deleterious effects on life-history. Thus he concluded that natural selection acting to increase fitness will inevitably give rise to senescence of the soma in multicellular species.

Under the rubric of "life-history theory," the pleiotropy hypothesis has been used to generate innumerable models centering around the theme of maximizing either the Malthusian parameter, r, or the carrying capacity, K, subject to fixed trade-offs between growth, survival, and reproduction (*e.g.*, Gadgil and Bossert, 1970; Schaffer, 1974; Charlesworth and Leon, 1976; Schaffer and Rosenzweig, 1977). The empirical status of these models has been thoroughly reviewed by Stearns (1976, 1977), who has emphasized the inadequacy of most attempts to test life-history theory. Certainly, no one has yet estimated the precise functions giving the interactions between life-history characters, derived quantitative predictions from a "life-history theory" model, and then exhaustively tested these predictions statistically over a range of populations.

Until we can accomplish such Herculean feats, I suggest that we might instead consider the fundamental validity of all life-history models which assume that evolution maximizes r or K, subject to pleiotropy between life-history characters. Firstly, it may be said in favor of such theory that there is clear evidence for the antagonistic pleiotropy required by the hypothesis (*e.g.*, Caspari, 1950; Simmons *et al.*, 1980; Rose and Charlesworth, 1981*a*, *b*). Secondly, there is good evidence that antagonistic pleiotropy may indeed be the cause of senescence in some species (Rose and Charlesworth, 1980). Thirdly, it is at least arguable that the pleiotropy hypothesis does least violence to intuitive perceptions of biological constraints (*cf.*, Stearns, 1980).

However, from the above discussion, it is clear that a successful theory of life-history evolution must also allow for abundant additive genetic variance in life-history characters. Fortunately, it turns out that the variable pleiotropy hypothesis implies that such genetic variability could be maintained without special mutation rates or extreme environmental variability.

Consider, for the sake of simplicity, a randomly-mating population with (a) genotypes of one locus having just two alleles, A_1 and A_2, (b) discrete generations, (c) infinite size, (d) hermaphroditism, (e) two additive fitness components, W_1 and W_2, such that total fitness is given by their sum. Now we assume a genotype-phenotype pattern specified as follows:

	W_1	W_2	$W_1 + W_2$
A_1A_1	$V + h_1\epsilon$	$f - \delta$	$V + f + h_1\epsilon - \delta$
A_1A_2	V	f	$V + f$
A_2A_2	$V - \epsilon$	$f + h_2\delta$	$V + f - \epsilon + h_2\delta$.

Elementary population genetics theory tells us that natural selection will maintain stable life-history polymorphism if

$$\epsilon > h_2\delta \text{ and } \delta > h_1\epsilon,$$

with all these parameters non-negative. In particular, for recessive deleterious gene action, h_1 and h_2 will both be close to zero, and polymorphism will be assured.

Elsewhere, I have analyzed a variety of models of this type, allowing multiplicative as well as additive fitness components, more than two alleles per locus, and more than one locus (Rose, 1982). In general, allowing pleiotropy often results in the maintenance of genetic polymorphism, when gene effects on life-history characters are sufficiently antagonistic. Moreover, the pattern of genetic polymorphism which results from such antagonistic pleiotropy often engenders large additive genetic variances for life-history characters, together with negligible additive genetic variance in fitness itself, at evolutionary equilibrium.

This type of model also readily generalizes to the treatment of a variety of patterns of life-history evolution, since by appropriate parameter choices one can generate uniform positive-correlation pleiotropy (a unitary hypothesis), absence of pleiotropy (the particulate hypothesis), along with combinations of these two patterns. Yet another possibility would be the neutrality hypothesis, which for this model would apply when

$$\epsilon = h_2\delta \text{ and } \delta = h_1\epsilon.$$

All of these possibilities are subsumed within the variable pleiotropy hypothesis, because it does not preclude any particular set of developmental constraints. All it does is characterize the patterns of allele-action which could predominate among evolving populations using elementary selection theory.

From this, it is clear that the major deficiency of the hypothesis is that it is no more than the population genetics theory of interdependent 'fitness-components. In attempting to find an acceptable theory of life-history evolution, the evidence has compelled us to abandon all simpler hypotheses. We cannot abandon this last theory without abandoning population genetics theory itself. Thus we are left with a theory of life-history evolution which is tantamount to neo-Darwinism, nothing less.

CONCLUSION

Finally, I will try to summarize what I consider our present scientific understanding of life-history evolution.

(a) Life-history characters have complex developmental interactions with one another, interactions which in turn depend on genetic segregation. Though life-history is not a generally unitary character, its component characters are also not generally autonomous of one another.

(b) In at least some species, senescence seems to be due to the declining force of natural selection with age allowing the spread of alleles which enhance early life-history characters, in spite of deleterious pleiotropic effects on later life-history characters.

(c) Antagonistic pleiotropy can in principle, and often in fact does, lead to abundant genetic polymorphism for life-history characters. Such genetic variability in turn is of a kind which allows the rapid modification of life-history by artificial or natural selection. The study of such genetic variation under selection promises to provide us with our closest view yet of the evolution of fitness itself.

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