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REVIEW ARTICLE

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Life history evolution: successes, limitations, and prospects

Abstract Life history theory tries to explain how evolution designs organisms to achieve reproductive success. The design is a solution to an ecological problem posed by the environment and subject to constraints intrinsic to the organism. Work on life histories has expanded the role of phenotypes in evolutionary theory, extending the range of predictions from genetic patterns to whole-organism traits directly connected to fitness. Among the questions answered are the following: Why are organisms small or large? Why do they mature early or late? Why do they have few or many offspring? Why do they have a short or a long life? Why must they grow old and die? The classical approach to life histories was optimization; it has had some convincing empirical success. Recently non-equilibrium approaches involving frequency-dependence, density-dependence, evolutionary game theory, adaptive dynamics, and explicit population dynamics have supplanted optimization as the preferred approach. They have not yet had as much empirical success, but there are logical reasons to prefer them, and they may soon extend the impact of life history theory into population dynamics and interspecific interactions in coevolving communities.

Introduction

Where life history theory fits in the history of ideas

When Darwin published the "Origin of Species" in 1859, he showed that descent with modification and natural selection could explain many of the facts of biology. However, the mechanism for inheritance that he presented was not plausible, and he came under vigorous attack on this point in the late nineteenth century. When Mendel's theory of inheritance was rediscovered in 1900 and was shown, by Fisher (1930), Wright (1931), and Haldane (1932) to be consistent with natural selection, evolutionary biologists were relieved that a major deficiency in evolutionary theory had been removed. They not only adopted genetics as the central pillar of the Modern or Neo-Darwinian Synthesis (Dobzhansky 1937; Mayr 1942); they also committed to genetics as the center of their explanatory paradigm. It became the reference point for rigor in the field.

The concentration of evolutionary biologists on genetics has lasted almost a century and continues to yield important results. Despite some claims to the contrary, however, evolutionary genetics established the consistency, not the sufficiency of genetics plus natural selection to explain evolution – particularly the evolution of whole-organism traits (phenotypic evolution), evolutionary developmental biology, and paleontology. The concentration on genetics therefore elicited a predictable reaction: What is the role of phenotypes in evolution? This reaction started to gain momentum in the 1960s and 1970s. It has both a selectionist part, which is concerned with how phenotypes are designed for reproductive success, and a developmental part, which is concerned with the restrictions placed on the expression of genetic variation by developmental mechanisms.

The selectionist part of the phenotypic reaction, which encompasses behavioral and evolutionary ecology, has developed as a theory-driven, predictive, experimental enterprise. This contrasts it with those parts of evolutionary biology that are historical, retrospective, and descriptive. Both approaches have strengths and weaknesses. The kinds of information that they yield are different and sometimes complementary.

To reiterate, life history evolution is part of evolutionary ecology, which is itself part of the more general

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attempt to explain phenotypic evolution. The developmental part of the explanation, not discussed here further, includes evolutionary developmental genetics and morphology, often now referred to as "Evo-Devo".

The nature and status of the classical explanations of life history variation

Classical life history theory, summarized in Roff (1992) and Stearns (1992), is based on optimization models. It aims to explain variation in size at birth, growth rates, age and size at maturity, clutch size and reproductive investment, and mortality rates and lifespan. By the 1990s the field had achieved consensus on the general features of a plausible explanation of the evolution of life history traits: (1) life histories are shaped by the interaction of extrinsic and intrinsic factors, (2) the extrinsic factors are ecological impacts on survival and reproduction; (3) the intrinsic factors are tradeoffs among life history traits and lineage-specific constraints on the expression of genetic variation.

Thus the classical approach makes a strong simplifying claim: you only need to understand two things to understand the evolution of life histories. One is external: how the environment affects the survival and reproduction of organisms of different ages, stages, or sizes. The other is internal: how traits are connected to each other and the constraints on how traits can vary. As we will see below, there are limitations to the classical approach that theorists are currently exploring, but all modern theory builds upon the focus achieved by this simplifying claim.

Like many simplifying claims, it is easy to think of reasons why this one may be false. For example, the manner in which limited resources are acquired and allocated to survival and reproduction varies with type of organism. This means that there is an intrinsic phylogenetic or developmental component to the way the extrinsic factors interact with the organism, and casts in doubt our ability cleanly to separate the extrinsic from the intrinsic. This comment can be translated into a research program, but it would not have been appropriate to do so at the time that life history analysis was being clarified. Then it was important to make the simplifying assumption and to push on, undaunted by the complications that were being ignored, precisely to see how much could be explained without worrying about them.

Before describing how well that classical approach has succeeded in explaining the major life history traits, I first discuss some important background: the focus of evolutionary biologists on explaining variation, the assumptions of optimality theory, the conceptual advantages of reaction norms, and bet-hedging as a method of dealing with risk.

The evolutionary emphasis on explaining variation

Evolutionary biologists differ from molecular biologists and physicists in an important basic aim. Molecular biologists want to discover the mechanisms common to all living things; they focus on what is similar and general in all cells. Physicists want to discover the general properties of matter and energy, laws valid at all places and all times. Evolutionary biologists ask questions inspired by comparisons of differences, differences between species, between populations, between individuals. They want to understand why things are different, not why they are the same. They want to understand what causes diversity. Much of their thinking is colored by this concentration on the causes of variation.

The assumptions of optimality theory

To treat life history evolution as an optimality problem, one assumes a definition of fitness, defines a relationship between traits and fitness, describes tradeoffs between traits, then finds the combination of traits that maximizes fitness. Optimality theory usually assumes that mortality and fecundity rates are constant and that the age structure of the population is therefore stable, which allows tractable measures of fitness to be defined. It also assumes that the fitness of a phenotype is adequately measured by estimating its reproductive success in a population composed exclusively of identical phenotypes. Fitness is measured as the rate at which a population of identical phenotypes would grow. Note that the assumption of identical phenotypes implies asexual reproduction or perfect heritability.

The success of the classical theory suggests that the things on which it chose to concentrate – age and size specific impacts on mortality and reproduction in a stable, asexual population – were more important for its purposes than the things it chose to ignore – such as genetics, explicit population dynamics, and frequency dependence.

The conceptual advantages of reaction norms

A reaction norm is a property of a genotype. It describes the set of phenotypes produced by that genotype across a range of environmental conditions. Reaction norms for life history traits have at least two important characteristics:

1. They clearly distinguish between the effects of "nature" and "nurture," between the component of the reaction to the environment that has evolved over many generations and the component that is due to a developmental reaction of this particular organism to this particular environment in just this generation.
2. They can be predicted by theory. This extends the range of things that the theory can predict and thus makes the theory easier to test and to improve.

Bet-hedging to deal with risk

The classical theory did make one excursion away from stable populations living in constant environments. It

dealt with the problem of the risk of reproductive failure. Evolutionary risk is variance in fitness: increases in variance reduce fitness. Measuring fitness as the geometric mean of per-generation reproductive success properly accounts for long-term risk, for this measure takes into account the effects of variance. Using this fitness measure makes clear that in a risky environment, it can pay to evolve traits that reduce the variance in fitness, even if this results in some reduction in arithmetic mean fitness.

There are three methods of dealing with risk:

1. Spread the risk by increasing the number of independent reproductive events (don't put your eggs in one basket) (Bernoulli 1738, translated 1954). Spreading risk is always preferable to not spreading risk unless there is an associated cost, in which case there will be an intermediate optimal degree of risk spreading.
2. If several traits each contribute to fitness through a mean and a variance, then a given fitness – a long-term geometric mean growth rate – can result from many combinations of means and variances of traits. Some components might contribute a low mean and a low variance, others a high mean and a high variance. The combinations that yield the same fitness value form a mean-variance fitness isocline. Investment should be distributed across components to yield maximum fitness given intrinsic constraints (Real and Ellner 1992).
3. The connection of traits to fitness is usually non-linear, as is the connection of wealth to utility in economics. If the relationship of fitness (Y-axis) to trait (X-axis) is concave down, then reducing variance in the trait will increase fitness. If the fitness-trait relation is concave up, then increasing variance in the trait will increase fitness. Thus a concave-down relationship between a trait and fitness implies risk-averse, variance-reducing behavior; a concave-up relationship implies risk-prone, variance-increasing behavior (Bernoulli 1738; Stephens and Krebs 1986).

With those concepts as background – optimality theory, reaction norms, and bet-hedging to spread risk – we can proceed to the achievements of the classical theory.

The classical explanation of the major life history traits

Age and size at maturity

The theory

Age and size at maturity are optimized when the positive difference between the benefits and costs of maturation at different ages and sizes is at its greatest. The benefits of early maturation are the costs of later maturation, and vice versa. The benefits of early maturation include a shorter generation time and a shorter period of exposure to juvenile mortality before the first reproductive event. One benefit of later maturation is a longer period in which to grow, leading to a larger size at maturity and greater fecundity, for fecundity often increases with body size. Another benefit of later maturation can be the production of higher-quality offspring, in the sense that the offspring have better survival per time unit as juveniles. If this per-time-unit effect is large enough, it can more than compensate for the risks of a longer juvenile period.

An example of a simple optimum

For many organisms the rate at which fecundity increases with size can be estimated in the field, as can the growth rate of body size, and if one assumes the population is stable, the per-time-unit juvenile mortality rate can be inferred. That is enough information, at least as a first approximation, to model the optimal age and size at maturity. When this was done for the eastern fence lizard [Stearns and Crandall (1981) using data from Tinkle and Ballinger (1972)], the result was a curve – a fitness profile (Robertson 1955) – relating fitness to variation in age at maturity. The consequences of making one or both of two assumptions were explored – effects of variation in age at maturity on (1) fecundity and (2) juvenile mortality. In this case, the model that best fit the data was one in which there were only effects of age at maturity on fecundity (Fig. 1).

This simple example illustrates the main features of the optimality approach: the costs and benefits of variation in a target trait are modeled, and the result of the model is a curve relating fitness to variation in the target trait. Because of the assumption of costs and benefits of trait variation, the fitness profile has an intermediate optimum, and the theory predicts that the trait should have the value corresponding to the peak in the fitness profile. That peak can be sharp or flat. The prediction is more robust if the peak is sharp, for then it is less likely that effects not taken into account in the

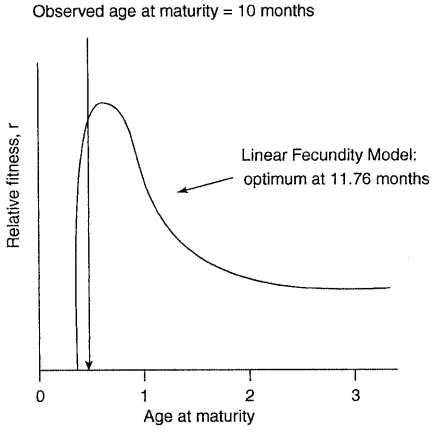

Fig. 1 An optimization model for age at maturity in the Eastern Fence Lizard. From Stearns and Crandall (1981)

model could make a big difference to the prediction. When the peak is flat, it would be relatively easy for some other factor to make a difference to the prediction. Whether the peak is sharp or flat, optimality models predict that traits involved in tradeoffs contribute most to fitness at intermediate values.

Thus the connections among traits provide a cause of stabilizing selection intrinsic to the organism. This is a general feature of trait evolution: an important component of the "environment" of a trait consists of the other traits out of which the organism is built. Its interactions with other traits generate selection pressures on the trait just as the interaction of the whole organism with the external environment generate selection pressures on the organism.

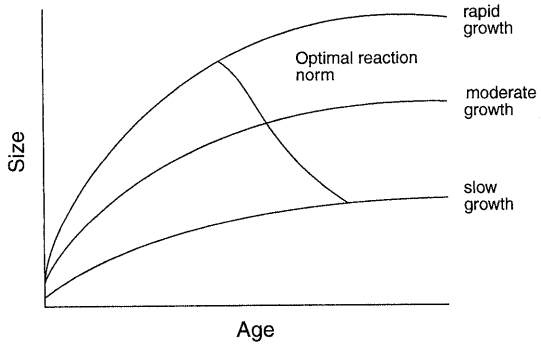

Fig. 3 The first prediction of an optimal reaction norm, in this case for age and size at maturity. From Stearns and Koella (1986)

An example of an optimal reaction norm

Optimality theory can predict not only a single optimal point, but how a trait should respond to variation in the environment. Consider a population of genetically identical organisms that is so thoroughly mixed that offspring of all individuals encounter all the kinds of microenvironments present, and all the microenvironments contribute equally to reproduction (this artificial assumption will be relaxed below). In some environments the organisms grow rapidly; in others they grow slowly. We now consider two simple maturation rules (Fig. 2): (1) always mature at the same size, and (2) always mature at the same age. Each entails a cost when the environment varies. An organism following the first rule – always mature at the same size – must wait a long time to mature in poor environments where growth is slow, and while it waits, it runs the risk of dying. The cost of this rule is the risk of mortality. An organism following the second rule – always mature at the same age – can avoid the mortality risk, but encounters a different problem: in poor environments it matures at a small size and has decreased fecundity. The cost of this rule is fewer offspring.

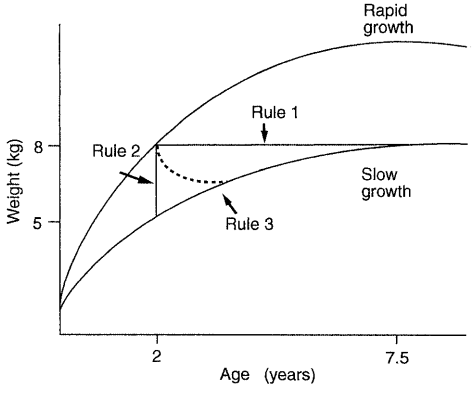

Fig. 2 The dilemmas of maturation in a heterogeneous environment where growth can be either fast or slow. From Stearns (1983)

This simple graphical analysis suggests that evolution will reach a compromise that can be predicted by a model incorporating the costs and benefits of variation in age and size at maturity in heterogeneous environments (Fig. 3). The optimal reaction norm depicted is only one of many possibilities, perhaps the simplest. It suggests that organisms should mature young and large when growth conditions are good, and old and small when growth conditions are poor (Stearns and Koella 1986). Such models, more thoroughly explored by Berrigan and Koella (1994), can explain some of the variation in reaction norms found in nature. Their predictions have attractive features that better approaches should attempt to preserve. These include:

1. The clear separation of "nature" – the shape and position of the reaction norm in phenotype space – from "nurture" – the particular point on the reaction norm at which an individual organism matures.
2. The prediction of a kind of phenotypic response that is relatively easy to measure.
3. The prediction of a kind of response that connects to population dynamics and interspecific interactions when growth, fecundity, and mortality rates depend on population densities.

Relaxing the implausible assumptions of the simple model

This simple model makes a critical and implausible assumption: that each point on the reaction norm can be treated as though it were associated with an independent, isolated, stable population. This cannot be the case. In any heterogeneous environment in which growth rates and reproductive rates vary, the contributions of the different environments to the growth rate of the whole population will also vary. In a simple, extreme case, instead of having a continuous range of environments, there are only two kinds of environment: a good environment in which most of the reproduction occurs, and a poor environment in which organisms can survive but contribute little to reproduction. There will

then be a net flow of individuals from the good environment (the source) into the poor environment (the sink). Most genes have ancestors that reproduced in the source environment. Because there has been very little evolutionary "experience" of the sink environment, the population is primarily adapted to the source, not to the sink, and in the extreme case the reaction norm will simply be a flat line with the value of the trait that is optimal in the source. In the more general case, where there are many types of environment rather than just two, the reaction norm is shaped according to the frequency with which successful reproduction occurs in each environment.

The environments that contribute most to reproduction are thus also the environments that most strongly shape the reaction norm, and those that contribute little to reproduction have correspondingly little effect on the reaction norm. This insight was arrived at simultaneously and independently by Houston and McNamara (1992) and Kawecki and Stearns (1993), who can be consulted for technical details.

Clutch size and reproductive investment

Some simple theory: the Lack Clutch

Lack (1947) published the basic idea that has formed the foundation of all subsequent theories of optimal reproductive investment. He asked, why do altricial birds not lay more eggs? His answer was, if they laid more eggs, then the per-offspring survival rate would decrease so much that they would fledge fewer young than they do with the number of eggs they actually lay. This idea can be formalized in a simple model (Fig. 4). As the number of eggs laid increases, the number of potential offspring increases in direct proportion. But if the per-offspring survival rate decreases linearly as the number of offspring to be fed increases, then the product of the number of eggs times the survival probability forms a parabola with an intermediate optimum. Lack was wrong on an important detail: fitness should be measured not as the number of offspring that leave one nest, but as the rate of production of grandchildren. The first measure ignores the subsequent survival and reproduction of the parents, whereas the second includes not only the subsequent survival and reproduction of the parents, but also the reproductive performance of the offspring, all of which can be affected by conditions in the nest. However Lack was right on the important general point: Fitness is often maximized at intermediate reproductive investments.

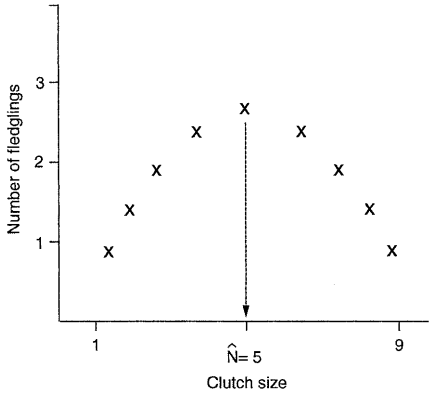

Fig. 4 The Lack clutch, the concept that introduced optimality thinking to evolutionary ecology in 1947. From Stearns (1992)

Example: kestrels in the Netherlands

If birds lay the optimal clutch, then larger or smaller clutches should yield lower fitness than the clutch actually laid. In a remarkable study, Daan et al. (1990) manipulated the clutch sizes of European kestrels (*Falco tinnunculus*) and followed the consequences for several years until they could measure the reproductive success of the offspring from clutches that had been decreased, increased, or simply subjected to a control manipulation. They reduced the size of 28 clutches, left 54 as controls, and increased the size of 20 clutches. Here is what they found:

1. The number of offspring fledged increased with clutch size, and the number of grandchildren that a clutch produced also increased with clutch size. Thus at the level of a single clutch it would have paid the kestrels to lay more eggs.
2. The survival to the next breeding season of the parents of increased clutches (0.43) was much worse than the survival of the parents of control (0.59) or reduced clutches (0.65). This large effect on parental survival more than compensated for the effects of increased clutch size. The parents of increased clutches could expect to have about one grandchild less, and the parents of reduced clutches could expect to have about half a grandchild less, than the parents of control clutches.

Thus in European kestrels the effects of changes in clutch size are felt more strongly by the parents than by the offspring. That the control group had the highest fitness suggests that kestrels optimize their reproductive investment with clutches of intermediate size. They appear to "know" how many eggs it is best to lay.

Some more advanced theory: reproductive investment

The kestrel example makes clear that reproduction has costs; this observation has been confirmed in many species. If reproduction has the benefit of producing offspring but also has costs such as increasing parental mortality or decreasing future parental reproduction, how should reproduction best be distributed over the entire lifetime? This is the general life history problem, and it has been solved for special cases with a class of models called Reproductive Effort Models. They all

share the assumption of a cost of reproduction that is paid in future mortality or reproduction or both, and the optimization produces a strategy of investing in reproduction that yields the greatest lifetime reproductive success. [The methods used include dynamic programming (Mangel and Clark 1988) and simulated annealing (Blarer and Doebeli 1996).] Here are some classical results for age-specific (not stage-specific) changes in mortality rates:

1. If mortality rates increase in one adult age class, then the optimal reproductive effort increases before that age and decreases after it (Michod 1979).
2. If mortality rates increase in all age classes, then optimal reproductive effort increases early in life and optimal age at maturity decreases (Charlesworth 1980).
3. If adult mortality rates increase, the optimal age at maturity decreases (Roff 1981).

One can interpret these statements by assuming that the life history was in a state of evolutionary equilibrium before the change in mortality rates. The response to the change in mortality rates then remodels the life history to invest more in the age classes that contribute more to reproduction under the new conditions and less in the age classes that contribute less.

An example: guppies in Trinidad

Reznick et al. (1990) carried out one of the best tests of these ideas in Trinidad. They took advantage of a system in which a small prey fish, the guppy (*Poecilia latipinna*), coexisted with one or both of two predators or lived in populations without fish predators. They could manipulate the predator–prey relationships by establishing replicate populations in various combinations above waterfalls that could not be invaded during the experiment, which started in 1976 and is continuing. Before the experiment started, they showed that guppies that coexisted with the larger, more dangerous predator, which eats guppies of all ages and sizes, matured earlier at a smaller size and produced more, smaller, offspring. They then exposed guppies that had not previously coexisted with this dangerous predator to it and followed the results for many years. The changes in life history traits were measured both in the field and in a standard laboratory environment. Evolution was rapid; significant responses occurred in less than 50 generations. As predicted, the guppies responded to increased mortality by maturing earlier at a smaller size and producing more, smaller, offspring. Within 13 years they had almost re-attained the state that long-term coexistence with the large predator had produced. The traits that changed most rapidly were those expressed early in life – age and size at maturity, size of first clutch, and size of offspring.

This study supports the predictions of the reproductive effort models. It also confirms that selection on traits expressed early in life is stronger than selection on traits expressed late in life, an idea central to the evolutionary theory of aging and lifespan.

Lifespan and aging

The life history theory of optimal lifespan

Reproductive lifespan is a byproduct of selection to maximize lifetime reproductive success. At evolutionary equilibrium there is a balance between selection to increase the number of reproductive events per lifetime and the costs of reproduction that increase the intrinsic sources of mortality with age. The first lengthen life, the second shorten it. Selection pressures that lengthen life are those that decrease the value of juveniles and increase the value of adults. These include: (1) lower adult mortality rates, (2) higher juvenile mortality rates, (3) increased variation in juvenile mortality rates from one reproductive event to the next, (4) decreased variation in adult mortality rates from one reproductive event to the next.

The effects of lowering adult mortality rates and increasing juvenile mortality rates are relatively straightforward: they increase the reproductive contributions of adults and make life as a juvenile more risky. The effects of increasing or decreasing the variation in mortality rates are perhaps less easily seen, and here it may help to refer back to the section on bet-hedging to spread risk. Variation in mortality is equivalent to risk, and strategies that minimize risk are those that reduce variation in mortality to attain the highest long-term geometric mean growth rate. If the variation in mortality is increased in an age class, investment will be shifted out of that age class and into age classes less exposed to risk, whether that risk is expressed in the mean or in the variance of the mortality rates. For multi-trait evolution under risk, see Real and Ellner (1992).

The evolutionary genetic theory of aging

Aging is a decline in internal state, manifested in the population as a reduction in survival and fecundity at later ages in adults. Thus aging not only affects survival rates: it also affects fecundity, and the effects of aging are normally reflected in age-related declines in both fecundity and survival. Questions about aging are pitched at two levels: the evolutionary – why did aging originate and why is it maintained in populations? – and the mechanistic – how does aging occur during the lifetime of an individual organism? The two levels interact: evolutionary explanations help to make sense of the mechanisms of aging in individuals at the levels of molecules and cells, while mechanisms of age-related cellular damage and its prevention provide the framework in which aging and its associated modes of genetic

effects evolve. Here I focus on the evolutionary aspects.

There are two approaches to the evolutionary theory of aging, one concentrating on phenotypic life history traits and the consequences of reproductive costs (above), the other concentrating on the genetic effects that arise when selection pressures decrease with age. The two approaches are not contradictory, but because they formulate some of the same problems in different language, they are presented separately here.

The most basic question about aging is this: What kinds of organisms should age? Are there any organisms that should not age? Williams (1957) posed the question and gave a preliminary answer. He thought that only organisms with a clear separation of germ line and soma should age. The germ line would be maintained in pristine condition, the soma could be disposed of after it served its reproductive function (Kirkwood 1987). Since then it has become clear that this criterion was too restrictive. It is now thought, following Partridge and Barton (1993), that any organisms that reproduce asymmetrically, where it can be said that there is a mother that is older than the offspring, must age. Organisms that reproduce by symmetrical division should not age; in such organisms it is not possible to decide which is the parent and which is the offspring, and whether one is older than the other. In other words, for aging to evolve, there must be an identifiable parent in which damage accumulates, and an identifiable offspring in which the damage done to the parent does not accumulate. M. Ackermann (paper in preparation) has demonstrated reproductive aging in an asymmetrically dividing bacterium, *Caulobacter*, supporting the Partridge–Barton criterion with a procaryotic example.

For organisms in which age classes can be distinguished, we can apply the next important idea. Selection pressures drop with age; sufficiently old organisms are irrelevant to evolution (Medawar 1952). This fact has consequences for two kinds of genes (Medawar 1952; Williams 1957):

1. Genes with positive effects early in life and negative effects late in life are selected for and accumulate in the genome. This effect is described as antagonistic pleiotropy – pleiotropy because the putative genes affect two or more traits, some early and some late in life, and antagonistic because the effects of the genes on fitness are positive early and negative late in life.
2. More deleterious mutations should accumulate and be maintained by selection-mutation balance when they are only expressed in older, rather than younger, age classes. This effect is described as mutation accumulation.

There is some evidence for each effect, more for antagonistic pleiotropy than for mutation accumulation, and the existence and importance of each kind of effect remains controversial and under investigation (Stearns and Partridge 2001).

Thus aging, in the evolutionary view, is a byproduct of selection for reproductive performance, no matter what the molecular mechanisms. It arises through the accumulation of many genes that have positive or neutral effects on fitness components early in life and negative effects on fitness components late in life. Experiments on *Drosophila* and *Caenorhabditis* suggest that early and late fitness components do trade off, but whether lifespan trades off with early fecundity, with size at maturity, or with development time depends on the species used, the treatment, and the laboratory doing the work.

An example: fruit flies experiencing experimental evolution in the laboratory

Aging is an increase in intrinsic mortality rates, or a decrease in intrinsic reproductive rates, or both, with age. A central prediction of the evolutionary theory of aging connects aging to extrinsic mortality rates (Williams 1957):

1. Populations encountering high extrinsic adult mortality rates should age rapidly. They should have high intrinsic mortality rates as adults.
2. Populations encountering low extrinsic adult mortality rates should age slowly. They should have low intrinsic mortality rates as adults.

The reasoning is straightforward. If, because of high extrinsic mortality, the organism is going to die soon in any case, it can gain fitness more efficiently by investing in reproduction than by maintaining the adult body.

We tested this idea in an evolutionary experiment (Stearns et al. 2000) with the fruit fly, *Drosophila melanogaster.* The lines in this ongoing experiment were derived from the same outbred, genetically variable population. There are two treatments, high and low adult mortality. Each treatment consists of three replicate lines. In the high mortality treatment, 90% of the flies are killed twice per week. The probability of surviving one week is 1%. In the low mortality treatment, 10% of the adult flies are killed twice per week. The probability of surviving one week is 81%. The densities of larvae and adults are maintained at the same level in both treatments, and all flies get the same food, temperature, humidity, and light cycle. The aim is to isolate the effects of extrinsic mortality on intrinsic mortality without confounding factors.

The experiment began in November 1993. In 1997 we performed a mortality assay involving 60,000 flies, 30,000 females and 30,000 males, with 5,000 of each sex from each of the six lines (three high mortality lines, three low mortality lines). Densities during the assay were held constant by replacing dead flies with white-eyed flies.

The result confirmed the theory (Fig. 5). Females that had evolved under higher extrinsic mortality rates had higher intrinsic mortality rates. The difference in intrinsic mortality rates was reflected in a lifespan of

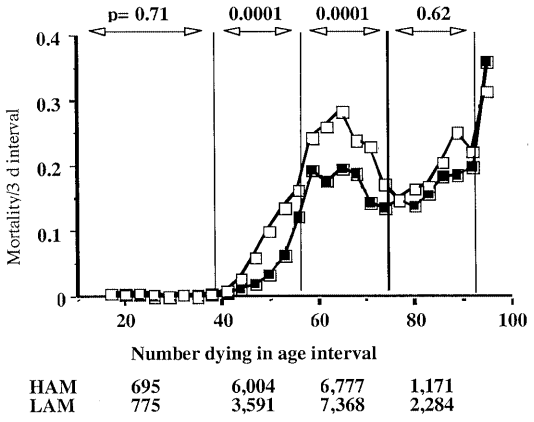

Fig. 5 The evolutionary response of intrinsic mortality rates, and hence lifespans, to changes in extrinsic mortality rates: the higher the extrinsic mortality rate, the higher the intrinsic mortality rate and the shorter the lifespan. From Stearns et al. (2000)

60 days for the females that had evolved under high extrinsic mortality and of 65 days for those that had evolved under low extrinsic mortality. That was not the only change. The flies that evolved under high extrinsic mortality also developed faster, were smaller at eclosion, and had higher fecundity early in life than the flies that evolved under low extrinsic mortality. This resembles the responses seen in the guppies in Trinidad (see above), and, like that study, this one also confirms the reproductive effort models of life history theory.

Extending the classical theory

The classical optimality theory makes restrictive assumptions, and recent theoretical work has attempted to remove those assumptions and to introduce a more realistic ecological framework into the models. That more realistic framework now includes frequency dependence, density dependence, and explicit population dynamics. Most of the motivation to proceed to these more complex models has come from logic, not from evidence. It is not as though the classical theory was confronted with empirical puzzles that it could not solve, and that the newer theory solves those puzzles. In fact, when the classical theory has been tested, it has usually done well (cf. Daan et al. 1990; Reznick et al. 1990; Stearns et al. 2000). The motivation to proceed to more complex theory has come much more from a logical consideration of how selection must work in natural populations, and from the hope that new theory will make new predictions.

Frequency dependence: ESSs for life history traits

One limitation of optimality theory is that it provides no opportunity to consider what happens when two or more life history phenotypes compete with each other within a population. That is, however, what must usually happen in nature – the phenotypes that evolved in the past are continually tested by mutants or immigrants that attempt to invade and take over the population. The theory that handles such situations is evolutionary game theory (Maynard Smith 1982).

Kawecki (1993) worked out a simple, clear case that shows how the change in approach can make a big difference to the predictions. He modeled the age and size at maturity of two individuals competing within a patch for a limited resource. In that circumstance the evolutionarily stable strategy (ESS) is to mature later and at a larger size than the age and size that maximize fitness. An analogous result for plant height under competition for light was derived earlier by Mirmirani and Oster (1978).

An example: population cycles in lizards

Sinervo et al. (2000) have discovered that two color morphs of a lizard are associated with life history phenotypes through genetic correlations. The two morphs compete with each other in a manner that generates short-term population cycles. Neither morph is optimal; each through its increase in frequency decreases its own fitness and prepares the way for the other to increase in frequency. Both density- and frequency-dependence are present and are needed to explain the behavior of the system, in which life history phenotypes cycle persistently.

Explicit population dynamics

The lizard example illustrates empirically a trend whose theoretical development began more than a decade earlier (e.g., Orzack and Tuljapurkar 1989): the emphasis on explicit population dynamics. There are good reasons for this development. First, life history traits – maturation, reproduction, and mortality – determine population dynamics. It is therefore natural to ask how population dynamics in turn shape the evolution of life history traits. Second, the study of population dynamics was invigorated by May's (1976) discovery of chaotic behavior in simple population models. That populations might behave chaotically for intrinsic reasons, rather than being driven by stochastic environmental fluctuations, together with characteristics of chaos that a mathematical mind finds beautiful, seduced a generation of theoreticians to study the new dynamics. They then naturally looked for problems that their methods could solve.

The theory of evolutionary population dynamics, called adaptive dynamics by some, makes predictions that are both intriguing and complicated (e.g., Ferrière and Clobert 1992; Ranta et al. 2000), and the mathematical nature of the general problem is now better understood (Dieckmann 1997). For a theorist, these are

exciting times, for old problems can be seen in new ways that have clear logical advantages over the old approaches. For those convinced by the approach, optimization is out; dynamics is in.

For an empiricist the excitement is also real but perhaps not as great as it is for the theorists for at least two reasons. First, the new predictions are complicated and hard to test. It is not easy to find a model system whose population dynamics can be studied in the short term, and it is even harder to find one in which one can study the evolution of the dynamics and of the life history traits at the same time. Until this technical issue is solved the theory cannot be tested. We need new model systems.

Second, perhaps we should not expect to find much chaos in natural populations. If we let the parameters that determine dynamic behavior themselves evolve, do they evolve towards or away from chaos? At least in the models studied so far, they evolve away from chaos into regions of simpler dynamics (Doebeli and Koella 1995).

Of course, the move into population dynamics is interesting and important whether it involves chaos or not. By connecting life history evolution to population dynamics, and through population dynamics to interspecific interactions and the coevolution of the life histories of several interacting species, we can hope for a large-scale integration of ecology and evolution. This ambitious vision surfaced earlier, in the 1960s, during the first big push into evolutionary ecology, but the tools available at the time were not up to the task. What is different now? In the interim, three entire fields – life history evolution, evolutionary game theory, and evolutionary population dynamics – have developed; the first two have matured with some convincing empirical success, and evolutionary population dynamics may soon move into the realm of experimental science. When that happens we will be much further along in the synthesis of ecology and evolution.

What mechanisms produce tradeoffs and constraints?

Whatever approach one takes to the theory, the simplifying claim of the classical approach still holds. To understand the evolution of life histories, we need to understand: (1) what affects the survival and reproduction of organisms of different ages or stages; (2) how traits are connected to each other, and (3) what constraints there are on how traits can vary.

To the first question we have some answers, and better ones are on the way. Our understanding of the selection pressures operating on life history traits has led to some empirical success. The second question is a question about tradeoffs. We have a lot of evidence that tradeoffs exist; we have very little understanding of the mechanisms that cause them. The explanation of variation in life histories will not be complete until those mechanisms are understood. The third question concerns phylogenetic and developmental constraints on the expression of genetic variation. Here the situation is promising. Recent progress in evolutionary developmental genetics – Evo-Devo – has been impressive. Some mechanisms that place lineage-specific constraints on the expression of genetic variation for morphological traits appear to be nearly within our reach. Taking those insights from morphological into life history traits will require a better understanding of complicated traits like fecundity and survival than is currently available, but the outlook is bright.

Thus the mechanisms that produce tradeoffs and constraints seem to be the toughest nut left to crack. That leads us towards a molecular biology informed by phylogenetics and developmental biology.

Future prospects

The rapprochement of molecular and organismal biology

The relationship between molecular and organismal biologists has often been more adversarial than cooperative. The internal logic of both fields is now, however, driving them back together. Research on the evolution and ecology of whole organisms has produced interesting questions, data, and partial answers that cry out for molecular explanations. Molecular genetics and developmental biology have explained enough at the level of the cell to make it natural to look at the whole organism as a new field to conquer. The acquisition of whole-genome sequences for model organisms and the technology of oligonucleotide microarrays to measure the expression patterns of thousands of genes are helping to push the rapprochement forward. We will see more cooperation between these fields, for it is in the interests of both to use each other's methods and to ask each other's questions, and there are solid grounds for mutual respect. People with one foot solidly in each camp will do exciting research.

The connection of micro- with macroevolution

One of the big questions that can be attacked by combining the methods of both schools is how microevolution connects to macroevolution. This has traditionally been the province of paleontology, but is increasingly becoming a problem that can be attacked with the methods and ideas of comparative biology and evolutionary developmental genetics.

When we understand the phylogenetic origins of developmental constraints on the adult phenotype, we should be in a position to explain some of the major puzzles of life history evolution – as well as many other things.

One important puzzle is that life history theory does not appear adequate to explain the diversity of life his-

tories on the planet. Part of the reason for its failure is its inability, up until now, to connect micro- with macroevolution. Consider, for example, the huge difference in adult mortality rates in the fruit fly experiment described above. That large difference in selection pressures has not yielded a comparably large difference in life history traits. The reason may be that tradeoffs hold the life histories at equilibrium values that are not far from the ancestral condition. So long as the tradeoffs do not themselves evolve, the kinds of changes that microevolution can cause in an experiment are not impressive when compared with what has actually evolved on the planet. Tradeoffs cause intrinsic stabilizing selection on characters. To bring about larger changes, the stabilizing selection must be converted into directional selection, at least for some characters. This requires that the tradeoffs break down or assume other forms. We do not yet know how that might happen.

Thus, even in the simplest selection experiments, there are components of the results that the theory cannot explain. When we consider the inability of the classical theory to explain complex life cycles, its inadequacy becomes even more apparent. Parasites with multiple hosts, algae and corals with an alternation of generations that differ in their life history, genetics and morphology, and the larvae of marine invertebrates and fish, holometabolic insects, and amphibia are not yet adequately understood – not their origin, nor their maintenance, nor their consequences for further evolution.

Other future directions

Life history theory now has a core of tested and reliable results. More work is needed on the core, but some of the most exciting work is coming from the application of the insights of life history evolution to cognate fields. The integration of the insights of life history evolution into behavioral ecology is already impressive. Other exciting work is now likely to come in population dynamics and community ecology. If we contrast populations that have plastic responses to their own density and to the density of the species with which they interact, with populations that do not have plastic responses, are the interactions more stable when the responses are plastic? Similarly, if we contrast populations with lots of genetic variation for life history traits with those that lack such variation, are the population dynamics and interspecific interactions of the genetically variable populations more stable than those of the populations that lack such variation? How do density- and frequency-dependent interactions in multi-species systems shape the coevolution of the life history traits of the participating species? Do the evolutionary responses drive the system towards regions of stability or into continued change? This list of unanswered questions suggests that our job is far from done.

Conclusions

Life history evolution, along with other parts of evolutionary and behavioral ecology, resembles physics in being theory-driven and experimental. The major life history traits are age and size at maturity, reproductive investment, reproductive lifespan, and aging. To predict the evolution of these traits it is sufficient to consider the impact on them of mortality rates, fecundity rates, and tradeoffs. There are important tradeoffs among age and size at maturity, reproductive investment, and lifespan. Variation among species and higher groups in these tradeoffs are the reason that life history traits are often found in characteristic combinations, such as slow developing, long-lived, low fecundity organisms and rapidly developing, short-lived, high fecundity organisms. Life history evolution has given phenotypes a role in the center of evolutionary theory, a center that it now shares with evolutionary genetics and development.

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