Population Dynamics, Life History, and Demography: Lessons From *Drosophila*

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I. INTRODUCTION

The many scientific advances over the last 200 years have clearly been one of the greatest achievements of human civilization. Much of this scientific progress has been accomplished by employing the paradigm we call 'the scientific method.' Hypotheses are proposed, experiments are designed to test these hypotheses, then ideas are revised based on the outcome of these experiments. For many reasons this paradigm has not always been embraced by ecologists. Often scientific hypotheses must be simple and address rudimentary aspects of a problem that have not been studied previously. Many ecologists do not accept the simplicity of explicitly stated hypotheses. For example, there is the early view of Thompson (1948): "The tremendous multiplicity of factors acting on the real world has not merely the complexity of an elaborate mathematical equation, which is theoretically but not practically manageable, but implies a genuine unpredictability because the actual combination of factors has never been observed to operate and until it has, we cannot really be sure what its effect will be. Much less can we see this effect in its causes."

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Thompson's lament concerns the great complexity of nature as well as its unpredictability. Implicit in Thompson's comments is the assumption that scientific theory needs to explain nature in all its details. Think of where genetics would be, had the early geneticists required that Mendel's laws also explain the distribution of progeny height and weight from known crosses. While it would be magnificent to be able to develop ecological theories that explain all aspects of the number and distribution of organisms in their natural environment, perhaps there is some benefit to starting with more modest goals.

This compromise has been the path chosen by many theoreticians. They have focused on just a few environmental variables, e.g., population density or predators. A theoretician might be satisfied with merely understanding the logical consequences of his simple theory. However, the empiricist will want some validation that, when the model assumptions are met, biological systems will really do what the model predicts. Here is where we feel there is the greatest disagreement about what constitutes a strong empirical test. Experimental ecologists, like many of those contributing to this volume, would suggest developing an experimental system where the scientists can ensure all the assumptions of the model are met. A critical test of the theory is thus guaranteed. Other ecologists feel that the theory must be tested in a natural ecosystem, albeit one they think is congruent with as many of the model assumptions as possible. The justification for this latter approach often appeals to a visceral notion that there are some ephemeral qualities of nature that could never be reproduced in the laboratory, which makes results from the laboratory suspect.

We not only reject this point of view, we feel that many field tests hinder scientific progress. Our view follows from the ability of the field ecologist to take the results of a field study that have falsified a theory and argue that the theory is still valid, because the contrary results were simply a consequence of uncontrolled factors playing havoc with the critical observations. We believe that testing ecological theory in the laboratory will instead allow us to build an understanding of ecological phenomena that will ultimately let us understand the complexity of nature. With this perspective in mind, we review some of the important lessons in ecology and life history evolution that have been learned from studies of laboratory populations of *Drosophila*.

Having made this argument for experimental ecological research we note that there are oftentimes experiments in the field that can be particularly useful. One notably good example of this is the work of Reznick and his colleagues on the evolution of life-histories in the guppy *Peocilia reticulate*. These studies have used both observations in natural populations (Reznick and Endler, 1982; Reznick, 1989; Reznick and Bryga, 1996; Reznick *et al*, 1996) and replicated introduction experiments in natural populations

(Reznick and Bryga, 1987; Reznick *et al.*, 1990, 1997). Together these studies have developed a solid understanding of the role of predator-mediated mortality on the evolution of guppy life history. These results are not only of general theoretical interest but they can be used to understand some of the variation in life history patterns in natural populations of guppies.

II. POPULATIONS WITHOUT AGE STRUCTURE

In this section, we review topics that are motivated by theories of density-dependent population growth and selection in populations without age structure. These topics include the evolution of density-dependent rates of population growth and the evolution of population stability. In the next section, we cover material that explicitly accounts for age structure. The topics to be reviewed in that section will include the evolution of senescence, and the evolution of age-specific mortality and fecundity patterns.

In each of these sections, we first review the important theories that motivate the experimental research. An important take-home message from this review will be the manner in which experiments are designed to test the critical concepts of these theories. Potential artifacts and confounding factors are often avoided by the experimental design or can be independently investigated so their contribution to certain experimental results can be evaluated. Consequently, the results from these experimental studies will often have clear interpretations.

A. Evolution of Density-Dependent Rates of Population Growth

1. Theory

The most important event in the development of the theory of density-dependent natural selection was the book, *The Theory of Island Biogeography* by MacArthur and Wilson (1967). They called their theory *r*- and *K*-selection and many of their ideas were presented as verbal models. However, these ideas were subsequently made more rigorous, as exemplified by the work of Roughgarden (1971).

The history of *r*- and *K*-selection has been reviewed numerous times (Stearns, 1976, 1977; Boyce, 1984; Mueller, 1997; Reznick *et al.*, 2002). In many respects this particular field can be viewed as a case study for the advantages of mathematical theories versus verbal theory and the strength of controlled laboratory experiments versus field studies. A great weakness

of verbal theories concerning density-dependent selection was the attempt to extend the reasoning of MacArthur and Wilson, which was framed within the context of logistic population growth, to populations with age structure. Meanwhile, empirical studies used wild populations where only very crude inferences about past densities could be made, and there was no ability to control for factors that might affect the evolution of life history other than density (Gadgil and Solbrig, 1972; McNaughton, 1975).

The theory developed by Roughgarden assumed that populations harbor genetic variation for density-dependent rates of population growth. Under this theory the effects of density are felt adversely by different genotypes. Using the simple structure of single-locus genetics and the logistic equation, then the per-capita growth rate or fitness (W_{ij}) for genotype A_iA_j at a population density N is:

$$W_{ij} = 1 + r_{ij} - r_{ij}NK_{ij}^{-1}. \tag{1}$$

The most interesting prediction from this theory requires two assumptions: (1) W_{ij} adequately summarizes fitness, and (2) genotypes show trade-offs. Trade-offs mean that genotypes with high values of r, have relatively low values of K and vice versa. With these assumptions granted, we expect populations evolving at very high and very low population densities to become phenotypically distinct, at least with respect to their per-capita growth rates. Particular aspects of life history, like competitive ability or adult size, may have to change to accomplish these changes in per-capita growth rates but this theory says nothing about what those changes might be.

2. *Laboratory Experiments*

Given the theory above, it is apparent that a suitable test would be to create different populations that differ only with respect to the level of crowding they experience. To test whether such populations achieve the predicted phenotypic differentiation would then require measuring the density-dependent per-capita growth rates in each population. There are no other surrogate phenotypes that can be used to test this theory. If one were to measure some other phenotype, then failure of the theory could always be argued to be a consequence of measuring an inappropriate phenotype. The power of strong inference would have been effectively thwarted.

Mueller and Ayala (1981a) were the first to test this theory by directly measuring density-dependent rates of population growth in laboratory populations that had evolved at very high and low densities. These experiments revealed the trade-off assumed by the Roughgarden theory. The laboratory populations used by Mueller and Ayala differed in adult density and in effective population size during their evolution. This left open the

possibility that the observed differences might be due to inbreeding or some other aspect of the effective population size differences. A separate set of independent experiments (Mueller *et al.*, 1991) controlled for the effects of inbreeding and were able to replicate the earlier results.

While these laboratory populations appear highly simplified, there have been results that connect these studies to more complicated field environments. For instance, Borash *et al.* (1998) studied simple cultures with high larval densities. Over the course of the two-week developmental period of these larvae the levels of ammonia increased exponentially, while the levels of food and ethanol declined substantially. This environmental deterioration has important consequences for evolution in these crowded environments. Borash *et al.* (1998) document a genetic polymorphism that appears to be stably maintained in these environments. One early-developing genotype is characterized by rapid development and high feeding rates, but by low viability in ammonia-laced food. A second slow-developing genotype has longer development time and slower feeding rates, but has high survival in ammonia-laced food. Additionally, the type of within-generation temporal variation is similar to variability in many naturally occurring ephemeral habitats. Some examples include excrement from large mammals or fresh fruit that falls off trees.

There have been a number of other laboratory studies of density-dependent natural selection using *Drosophila* (Taylor and Condra, 1980; Barclay and Gregory, 1981, 1982; Sokolowski *et al.*, 1997). None of these studies measured rates of population growth directly and some of the early studies had methodological flaws that have been previously reviewed (Mueller, 1985). Sokolowski *et al.* (1997) created a set of populations similar to the *r*- and *K*-lines created by Mueller and Ayala (1981a). Sokolowski *et al.* (1997) showed that their high-density lines evolved increased foraging path lengths as did the high-density *K*-populations created by Mueller and Ayala. Consequently, we have independent corroboration of the type of phenotypic evolution that results from evolution in crowded environments.

The tension between the controlled laboratory environment and the desire to create natural conditions is reflected in the experiment of Barclay and Gregory (1982). In this study, the goal was to create experimental treatments that differed in levels of adult mortality. In most theories that permit adult mortality rates to vary, it is relatively unimportant what causes death since the event itself is fairly unambiguous. Barclay and Gregory decided to put frogs in their fly cages as a means of increasing adult mortality rather than simply removing the adults manually. In this example, we think that whatever gain in reality is achieved by having frogs kill flies does not compensate for the precision that manual control of mortality affords the experimentalist. Thus, like the decision to perform laboratory experiments versus field work, other decisions to make conditions more

natural must be evaluated with respect to their ability to permit cogent interpretation of the experimental results.

B. Evolution of Population Stability

1. Theory

Models of density-dependent population growth typically assume that per-capita growth rates decline with increasing population density. As a result there will typically be an equilibrium density at which the population will show no change in size from one generation to the next. An important property of population dynamic models is whether the population will return to an equilibrium after a small perturbation away from it. The behavior of the dynamic system around an equilibrium is called the 'stability of the equilibrium.'

It might seem as if predictions concerning population stability should follow easily from the evolution of density-dependent population growth rates (see Mueller and Joshi, 2000, Chapter 2 for a review of this theory). In the simple discrete time logistic, stability is determined by the Malthusian parameter r. Thus, once we know how r evolves we ought to understand how stability evolves. However, the evolution of stability is not that straightforward. A disconcerting complication with these simple models is that the outcome of evolution often depends on small model details. Using the discrete-time logistic, exponential and hyperbolic models, Turelli and Petry (1980) found that in variable environments the evolution of r could result in increased, decreased, or no change in stability. Turelli and Petry obtained more consistent results when they used the model,

$$N_{t+1} = N_t G[(N_t/K)^{\theta}]. \tag{2}$$

and let the parameter θ evolve. Even as $G[.]$ changed to one of the three functional forms (logistic, exponential, and hyperbolic) evolution typically favored increased stability. However, this result depends on genetic variation affecting only θ, and not r and K. This is an empirical problem, which requires an experimental resolution.

The theory developed by Turelli and Petry started with populations at a deterministically stable equilibrium. In an early study, Mueller and Ayala (1981b) examined the evolution of stability in a population undergoing a two-point cycle in population density. The results suggested that increased stability could evolve if there was a trade-off between density-dependent survival and fertility. Subsequent theoretical work has also confirmed the need for trade-offs to have stability evolve (Stokes *et al.*, 1988; Gatto, 1993). As with the theory of density-dependent natural selection, there is no

theoretical way to determine if the requisite trade-offs exist. This is entirely an empirical question.

2. *Laboratory Experiments*

To test the theory that natural selection may affect the stability of populations required, at a minimum, populations that differed in their stability characteristics but were otherwise similar. For *Drosophila* we determined that a crucial determinant of stability was the level of resources provided to adults and larvae (Mueller and Huynh, 1994). High levels of food for the larval stages but low levels for adults generally enhanced population stability. The opposite conditions, high adult food and low larval food levels, typically produced stable cycles or chaos (Sheeba and Joshi, 1998). This information was then used to design experimental populations to monitor the evolution of stability.

Twenty populations were created. Ten were created from populations called CU that had evolved at high larval and low adult densities prior to this experiment. Ten additional populations were created from populations called UU that had been kept at low larval and low adult density prior to the start of the experiment. The 10 CU populations were further divided into five populations maintained under high larval food and low adult food conditions (called HL). These are the conditions most conducive to stability. The other five CU populations were maintained under low larval food and high adult food conditions (called LH). These are the conditions that are the least conducive to stability. The 10 UU populations were divided into two groups of five in a similar fashion.

Visual inspection of the cultures suggested that the larval densities were typically much higher in the LH treatments. These impressions are confirmed by the fact that on average the adults that emerge from the LH treatments were much smaller than the adults in the HL treatments (Mueller *et al.*, 2000; Fig. 1A). The actual population size fluctuations are quite dramatic in both types of populations (Fig. 1B). However, the HL treatments tend to be more stable (Mueller *et al.*, 2000). Formally, the stability of an equilibrium is assessed by determining the rate of exponential growth either away from or towards the equilibrium in a small neighborhood around the equilibrium. The parameter describing this rate of exponential growth is often called the 'eigenvalue'. Since the eigenvalue may be an imaginary number it is common to evaluate the modulus of the eigenvalue. For real numbers the modulus is simply its absolute value and for imaginary numbers it is the square root of the sum of the squares of the real and imaginary parts of the eigenvalue. For discrete time models the modulus must be <1 to ensure stability.

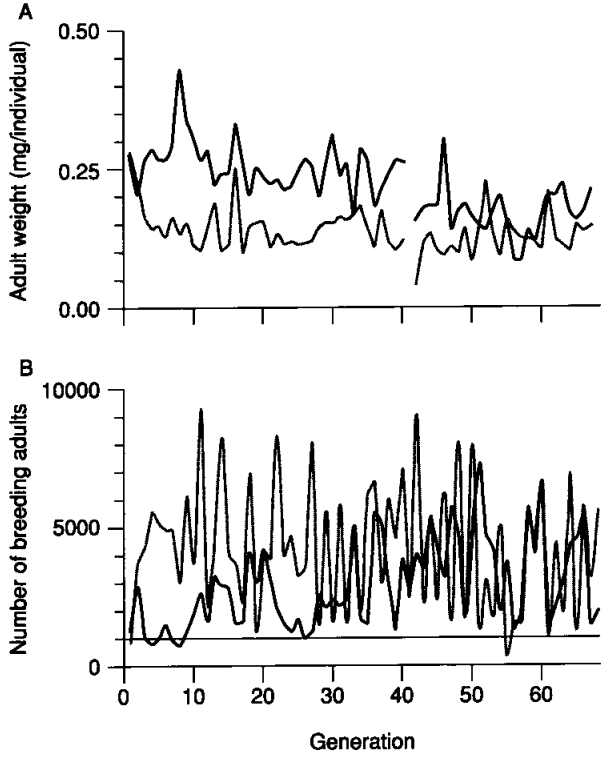

Figure 1 Adult size (A) and numbers of adults (B) for 68 generations in the experimental populations derived from the CU_5 ancestral population. The solid black lines are the HL populations and the dashed (gray) lines are the LH populations.

To assess whether the dynamics of the populations in the less stable LH treatments changed over 68 generations of lab evolution, we determined the modulus of the leading eigenvalue during the first 15 generations of the experiment and during the last 15 generations of the experiment. Although the average values of this modulus decreased from 1.00 to 0.75, that change was not significant. Likewise among the 10 LH populations, four showed increases in the modulus of their leading eigenvalue, indicating a decrease in stability. Increased stability did not generally evolve in the laboratory populations.

One interpretation of these results is that the intensity of density-dependent selection was simply too small to expect to see improvements in stability. Over the first 20 generations, we measured the larval feeding rates in each of the 20 populations. This character has been documented to increase under crowded larval conditions primarily due to its effect on larval competitive ability (Joshi and Mueller, 1988; Mueller *et al.*, 1993). We found rapid

differentiation between the HL and LH populations independent of the ancestral source of the flies (e.g., CU versus UU). In all cases the feeding rates were greater in the LH populations. Thus, the environments we imposed led to strong responses in density-dependent natural selection. But such a selection did not have measurable effects on the stability of the population dynamics. It may be that stability changes very slowly as a result of density-dependent selection. But any such change must be so slow that even after 68 generations demonstrable effects of density-dependent selection cannot be detected.

In these experiments we purposely kept the total population size at about 1,000 adults or larger to prevent inbreeding depression. In *Drosophila,* inbreeding is likely to cause reductions in female fecundity and that may have the effect of stabilizing the population dynamics. In fact, we have suggested elsewhere that the apparent evolution of more stable dynamics in Nicholson's blowfly experiments (observed by Stokes *et al.* [1988]) may have been a consequence of inbreeding during the severe population blowfly bottlenecks (Mueller *et al.*, 2000).

Prasad *et al.* (2003) studied *Drosophila* populations selected for rapid development time and their controls. The rapid developing lines show reductions in female fecundity of about 35% compared to their controls. When both selected and control populations are maintained in the HL and LH environments, the rapid developing lines show reduced population fluctuations consistent with more stable dynamics.

Population cycles may be due to a variety of causes other than density-dependent population regulation. Indeed many cycles in nature are thought to be a consequence of multispecies interactions like those between predators and prey (see Turchin 2003, for a recent review). Future experimental work on population stability will benefit from an examination of evolution in multi-species communities. An illustration of this type of work is Yoshida *et al.* (2003). They studied the consequences of evolution on the predator-prey cycles of an experimental rotifer-algal community. They demonstrated that the characteristics of the predator-prey cycle changed as the algae evolved defenses against predation.

III. POPULATIONS WITH AGE STRUCTURE

A. Age-Specific Mortality Rates

1. Theory

The theory of natural selection in age-structured populations is well developed (Norton, 1928; Charlesworth, 1994). The basic demographic parameters needed for this theory are the probabilities of an individual

surviving to age–x, $l(x)$, and the number of newborns produced by individuals age-x that survive to the first age class, $m(x)$. Populations with these demographic parameters will grow exponentially at a rate r that can be determined from the equation,

$$\sum e^{-rx} l(x) m(x) = 1, \tag{3}$$

where the summation is over all age classes. The theory of natural selection supposes that these demographic parameters vary between genotypes. Thus, in a simple single-locus setting the demographic parameters for genotype A_iA_j are $l_{ij}(x)$ and $m_{ij}(x)$. The fitness of this genotype at genetic equilibrium is,

$$w_{ij} = \sum e^{-\hat{r}x} l_{ij}(x) m_{ij}(x), \tag{4}$$

where $\hat{r}$ is the rate of exponential growth for the equilibrium population. For positive values of $\hat{r}$, fitness will be most strongly affected by the survival and fertility values at early ages. As pointed out by Hamilton (1966), mutations increasing survival at earlier ages would be most strongly selected for.

These ideas have been used to develop evolutionary explanations for the general observation that age-specific mortality rates generally increase at an exponential rate with increasing age, which can also be equated to senescence. One specific theory of senescence, called antagonistic pleiotropy (Medawar, 1952; Williams, 1957), suggests that natural selection will often view as favorable an allele that increases survival or fertility early in life even if it also has a deleterious (pleiotropic) effect late in life. A second theory, called mutation accumulation, suggests that populations will harbor a collection of deleterious alleles that affect age-specific survival and fertility (Medawar, 1952; Edney and Gill, 1968). The frequency of these deleterious alleles will be at an equilibrium dictated by the force of selection and the rate of mutation. Since the force of selection against deleterious alleles will be weak when the alleles are expressed late in life, the frequency of late-acting deleterious alleles is expected to be higher than the frequency of mutations affecting early fitness. Both the genetic theories of aging are supported by the formal theories of selection in age-structured populations. They are not even logically incompatible. Assessing the importance of these two theories is an empirical question.

2. *Laboratory Experiments*

There are a variety of possible tests of the evolutionary theories of aging. One interesting test, which can only be carried out in a laboratory setting, would be to reverse the normal way selection acts; that is, make reproduction late in life more valuable than reproduction early in life. With

natural selection acting in this fashion, there should be improvements in late life fertility and declines in later mortality rates. Rose (1984) performed this experiment with *D. melanogaster*. Control populations, called Bs, reproduced at the end of a two-week egg-to-adult life cycle. The experimental populations, called Os, were cultured from eggs laid by older females. The age of these older females was progressively increased until its present level of 10 weeks from egg. This process has lead to genetic changes in the O populations that have doubled their longevity relative to the B populations and decreased mortality rates (Fig. 2). The decreased mortality rates are not due to a simple age-independent reduction in overall mortality. The actual rate of increase in mortality with age in the O populations is one-third the rate seen in the B populations. Thus, the actual rate of aging has been genetically reduced in the long-lived O populations. Rose (1984) also documented a decline in the early fecundity of O females relative to B females but an increase in late life fecundity. This type of trade-off is consistent with the

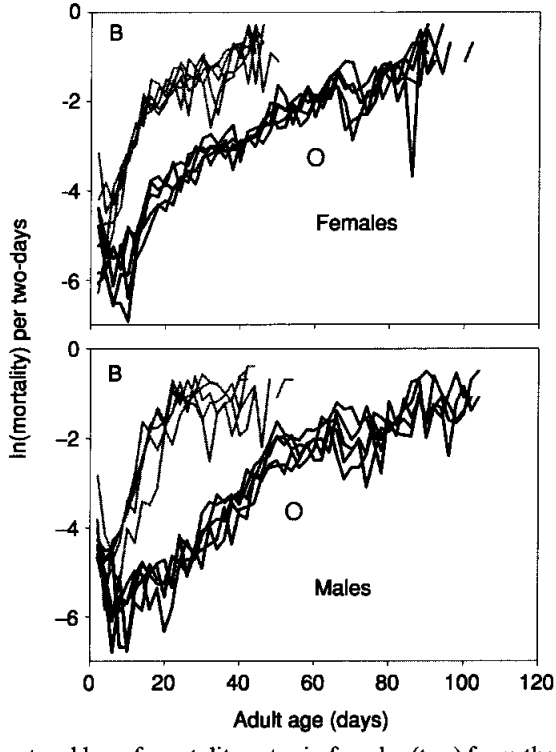

Figure 2 The natural log of mortality rates in females (top) from the B (gray) and O (black) populations and males (bottom) from the B and O populations.

action of alleles that have antagonistic pleiotropic effects on longevity and fertility.

If there are naturally occurring deleterious alleles that contribute to senescence, then their frequencies could be increased by further reducing the strength of selection against these alleles. We can accomplish this in the laboratory by permitting reproduction only at very young ages. This effectively makes late-acting deleterious alleles neutral (assuming they have no pleiotropic effects on early survival or fertility). The probability that these neutral alleles will be fixed is independent of the population size but the speed with which they move to fixation will depend on the population size. Thus, if we also make the populations small we should see a relatively rapid increase in these deleterious alleles. Mueller (1987) maintained populations under these conditions and in fact was able to document accelerated senescence due to naturally occurring deleterious alleles.

The general paradigm employed by Rose and his colleagues has been replicated many times in independent laboratories. Some of these studies have corroborated the basic results that selection for late-life fitness increases longevity but at a cost of decreased early female fecundity (Luckinbill *et al.*, 1984; Partridge *et al.*, 1999). Occasionally, selection for late-life fitness has not resulted in a measurable decline in early fecundity (Partridge and Fowler, 1992; Roper *et al.*, 1993). Some of these differences can be understood by recognizing that the laboratory is not a single environment and even subtle differences in selection protocols can significantly affect the course of evolution (Leroi *et al.*, 1994a,b).

B. Mortality-Rate Plateaus

1. Theory

Benjamin Gompertz (1825) made a seminal contribution to demography by suggesting that age-specific mortality, $u(x)$, might be modeled by the simple exponential relationship:

$$u(x) = Ae^{\alpha x}. \tag{5}$$

The two parameters of the Gompertz equation, A and α, measure age-independent and dependent sources of mortality respectively. The Gompertz equation can provide very good statistical fits to mortality or survival curves for a wide variety of species. For that reason this model has become a standard equation for summarizing the kinetics of biological mortality.

In 1992, two papers appeared that questioned the Gompertzian view of demography (Carey *et al.*, 1992; Curtsinger *et al.*, 1992). In these studies it appeared that at the most advanced ages, observed mortality rates failed to

increase exponentially or, for that matter, increase at all. After the existence of these mortality plateaus was confirmed in a number of very different organisms, like fruit flies, yeast, wasps, humans, and nematodes, an explanation for their existence was sought (Brooks *et al.*, 1994; Vaupel *et al.*, 1998).

One hypothesis posits that at the time aging commences each individual's chance of dying is described by the Gompertz equation, but that there is heterogeneity between individuals for the values of A, α, or both. This variability may be due to genetic or environmental differences. When there is variation in A of magnitude σ^2, the average mortality of individuals age x is given by (Vaupel *et al.*, 1979):

$$\bar{u}(x) = \frac{A\exp(\alpha x)}{1 + \sigma^2 A\alpha^{-1}[\exp(\alpha x) - 1]}. \tag{6}$$

This model predicts mortality plateaus if σ^2 is sufficiently large. Variation can also be incorporated in α, although analytic results are more complicated for that model.

A second explanation for mortality plateaus is that they arise as a natural consequence of selection in age-structured populations and genetic drift (Mueller and Rose, 1996; Charlesworth, 2001). Under this theory, survival shows an exponential increase at early ages due to the exponential weighting of fitness as in equation 3. At very advanced ages the strength of selection is so weak that random genetic drift dominates changes in allele frequencies (Fig. 3). Thus, mortality remains high but there is no tendency for these rates to change with age. In Fig. 3 we demonstrate how in a large population, since the effects of drift are weaker, the onset of the mortality plateau is displaced to later ages. A corollary of this theory is that the age at which the plateau starts should be a function of the age-specific strength of natural selection. If selection remains strong, even at advanced ages, the plateau in mortality rates should be accordingly later in life than the case when selection is only strong in early life. Charlesworth (2001) has developed analytical models of mutation accumulation that also predict the existence of mortality plateaus, thus supporting the general conclusions of Mueller and Rose (1996).

Tests of the heterogeneity theory are more difficult to design since the sources of heterogeneity are left vague by most theories. We have attempted to test some aspects of the heterogeneity theory but will not present the details of those results here (Mueller *et al.*, 2003).

2. *Laboratory Experiments*

The evolutionary theory of late-life mortality plateaus has been experimentally tested by Rose *et al.* (2002). The general approach was to compare populations that had been subjected to different, long-term, age-specific

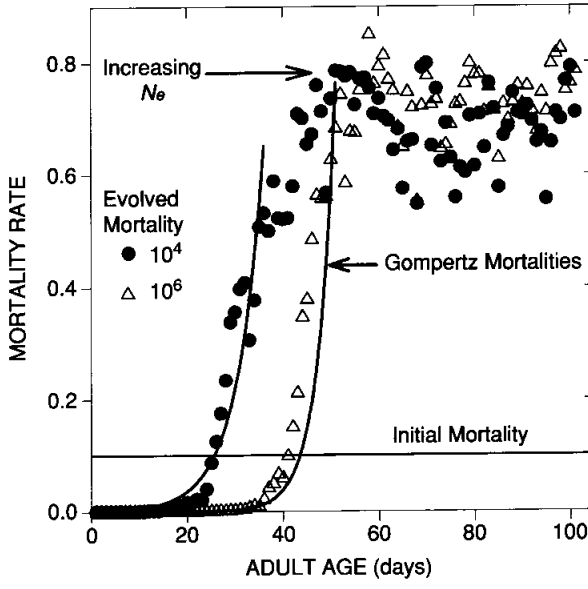

Figure 3 The evolution of mortality plateaus. In a population with no pattern of age-specific mortality (initial mortality) evolution results in the increase of mortality with age at approximately Gompertian rates until later in life. At that point drift takes over and mortality simply remains high but without an age-specific pattern (see Mueller and Rose, 1996, for additional details of these simulations).

selection regimes: reproduction early in life versus reproduction later in life. These populations comprised five sets of replicated stocks: B_{1-5}, O_{1-5}, CO_{1-5}, ACO_{1-5}, and NRO_{1-5}. The ACO and B populations have an early age of last reproduction (9 and 14 days, respectively), the CO populations have an intermediate last age of reproduction (28 days), and the O populations have a late last age of reproduction (70 days). The five NRO populations were derived from their respective O populations and have a last age of reproduction of 14 days. The NRO culture procedure was like that of the O populations, except that the flies were placed in cages at about 10 days from the egg stage with egg collection at 14 days after feeding on yeast. Except for the NRO group, these populations were each maintained for more than 100 generations at effective population sizes of ≥ 1000.

Using maximum likelihood techniques (Mueller *et al.*, 1995), we fit a two-stage Gompertz model to the cohort survival data collected from these populations. This model assumes that, up until an age called the breakday, mortality is described by the Gompertz equation. After the breakday, mortality is constant. The value of the breakday is determined from maximum likelihood techniques. The specific prediction from the evolutionary theories

Table 1 The mean longevity and breakday for populations subjected to different regimes of age-specific survival (Rose *et al.*, 2002). The pairs of selection treatments to be compared are arranged side-by-side, e.g., compare B to O, ACO to CO, and so on.

	Early reproduction	Later reproduction
	B	O
Male longevity**	20.6	52.3
Male breakday**	23.6	58.0
Female longevity**	20.8	48.2
Female breakday**	24.0	68.4
	ACO	CO
Male longevity**	26.2	44.2
Male breakday**	42.6	58.6
Female longevity**	23.5	37.2
Female breakday**	40.6	57.0
	NRO	O
Male longevity**	41.8	53.3
Male breakday**	48.2	68.6
Female longevity**	39.2	50.4
Female breakday*	54.6	67.8

*$p < 0.1$; **$p < 0.01$.

of late-life mortality plateaus is that the onset of the plateau, or the breakday, should be later in those populations experiencing strong selection for reproduction later in life. The mean longevities and breakdays for the three different pairs of selection treatments are shown in Table 1. In each case, selection for late-life reproduction increased both the average longevity and the age at which the mortality plateau begins (breakday). Except for one case, these differences are all statistically significant at $p < 0.01$.

Several studies have examined the impact of different types of heterogeneity on mortality plateaus (Carey *et al.*, 1995; Khazaeli *et al.*, 1995). Using highly inbred lines of *Drosophila*, Fukui *et al.* (1993) demonstrated that the existence of late-life mortality plateaus does not require genetic variability. Thus, for the heterogeneity theory of late-life mortality plateaus to remain viable there must be sufficient environmentally generated variability to cause such plateaus. Khazaeli *et al.* (1995) exposed *Drosophila* to variable levels of desiccation and initially interpreted their results as supporting the existence of heterogeneity. However, Curtsinger and Khazaeli (1997) later retracted that interpretation noting that the normal heat shock response of *Drosophila* made it impossible to unambiguously interpret their experimental results. The experimental results of Carey *et al.* (1995) with medflies led him to conclude that environmentally induced variation is not the likely cause of mortality-leveling in later life (Carey, 2003). While the

sources of environmental variation are effectively infinite, the inability to readily demonstrate causation of plateaus from identifiable sources of variation places the heterogeneity theory of mortality plateaus in a precarious position.

C. Fecundity Plateaus

1. Theory

The theory of fitness in age-structured populations (equation 3) controls selection on fecundity as well as survival. If the arguments presented to explain the existence of late-life mortality are essentially correct, then these same forces ought to be shaping the pattern of female fecundity in late life. That is, at sufficiently advanced ages selection for fecundity will be so weak that it cannot compete with drift. At that point, all ages become interchangeable and there should be no pattern in late-life fecundity other than it being very low (Rauser *et al.*, 2003). A well-developed formal theory about the evolution of late-life fecundity does not yet exist. This is clearly an area for additional future research. However, as with mortality plateaus, we expect that the onset of a fecundity plateau will respond to age-specific selection in much the same way mortality plateaus respond.

2. Laboratory Experiments

Female fecundity in large cohorts of flies from three ACO populations (ACO_{1-3}) was followed at daily intervals (Rauser *et al.*, 2003). The results showed a rapid decline in fecundity as the flies aged, followed by an extended period during which small but relatively unchanging numbers of eggs per female were produced (Fig. 4).

We tested the evolutionary theory that predicts level fecundity at late ages by fitting the late-life fecundity data from the three ACO populations to a two-stage linear model, which most simply summarizes our *a priori* predictions. Since our focus was on fecundity at late ages we did not try to model the age-specific fecundity patterns at early or mid-life. Our model predicts linearly declining egg numbers at advanced ages until an age at which egg numbers stop declining and plateau at low values. Specifically, this model shows that female fecundity at age x is

$$f(x) = \begin{cases} a_0 + a_1 x \text{ when} & x \leq a_2 \\ a_0 + a_1 a_2 \text{ when} & x > a_2, \end{cases} \tag{7}$$

where a_2 is the breakday or the age at which the fecundity plateau begins. We also estimated the plateau height, which was significantly different than zero

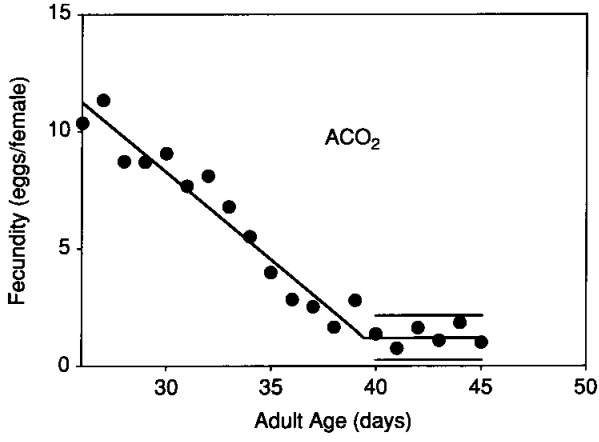

Figure 4 Late-life fecundity in the ACO_2 population. The line shows a two-stage linear model fit by non-linear least squares to the observed fecundity values (circles). In addition to the parameters for the two lines, the age of plateau onset is fit by the regression technique. A 95% confidence interval on the plateau regression is also shown to emphasize that female fecundity in the plateau is demonstrably >0.

for all three populations (Fig. 4). This result suggests that fecundity is level and non-zero at late ages as the evolutionary theory predicts.

It is important to note that the two-stage linear model does not necessarily need to support the existence of late-life fecundity plateaus. For instance, the breakday could be estimated to be at the last day females were alive, which would not be consistent with the existence of a plateau. In addition, the height of the plateau could simply be zero. This latter result is not inconsistent with an evolutionary model, but does not constitute strong support for the evolutionary model of late-life. Nevertheless, when we apply these objective criteria to our large datasets they support the overall visual impression that female fecundity reaches a non-zero plateau later in life.

IV. DISCUSSION

The use of experimental systems of *Drosophila* has contributed to our understanding of density-dependent and age-specific natural selection and population dynamics. Laboratory experiments have repeatedly allowed us to test the general theories of ecology. The use of experimental systems is not always straightforward and requires attention to details like the source of the experimental populations, effective population sizes, effects of mutant alleles, and adaptation to the laboratory environment (Rose *et al.*, 1996). However, the great strength of laboratory experimental systems is

their power when testing a theory. Oftentimes, these experimental populations can be maintained in a way that eliminates confounding factors that are present in field systems. Ultimately this makes the interpretation of laboratory experiments simpler.

Recently a number of papers have discussed the limitations of using laboratory populations for the study of evolutionary problems (Gibbs, 1999; Hoffmann and Harshman, 1999; Sgro and Partridge, 2000; Harshman and Hoffmann, 2000; Hoffmann *et al.*, 2001; Linnen *et al.*, 2001). For instance, Sgro and Partridge (2000) point out that "Conditions in the laboratory are different from those in the field and could alter both the way that genes affecting life-history traits are expressed ... and the resulting genetic correlations between them." Implicit in this statement is the idea that there are two environments: the laboratory and the field. In reality there is no single definable entity called the field. For fruit flies, the field may be almost anywhere in the world. Even if one were to focus on a specific locality, as Sgro and Partridge (2000) did, we do not expect the conditions of temperature, humidity, competitors, and disease in these localities to be precisely the same from one year to the next.

Suppose we could study life-history evolution in the field environment of Montpellier, France, in 1996. We would still not be sure that the genetic correlations and gene expression observed in France would be the same as in Irvine, California, in 2004. At least in the laboratory we can declare in a fairly precise way the quantitative state of the environment; that is, we know what the temperature, humidity, light regime, food resources, and biological competitors were during the entire experiment. In this sense, we have vastly more information about our laboratory environments than we do about any particular field environment and thus the potential to control whichever variables that matter for the evolutionary and ecological phenomena we are interested in.

The fact that the laboratory is different from any field environment should be expected. Hoffmann *et al.* (2001) document a decline in starvation and desiccation resistance of laboratory-adapted flies compared to recently collected flies from Montpellier, France. In retrospect, the laboratory must either be more or less stressful with respect to water balance compared to a particular field environment. It is unlikely to be precisely the same. Hoffmann *et al.* (2001) determined that wild caught flies are 46% more desiccation-resistant than laboratory-adapted flies. Hoffmann *et al.* (2001) suggest these declines in stress resistance reduce the usefulness of laboratory-selected populations since "... then much of the response to selection for increased stress resistance may be achieved by moving the laboratory populations back to resistance levels that approach those of recently founded populations" (Hoffmann *et al.*, 2001). However, when long established laboratory populations of *Drosophila* are selected for desiccation resistance

they improve by a factor of more than 300% not a mere 46% (Phelan *et al.*, 2003). This result also shows that the concern that laboratory populations must necessarily and quickly lose genetic variation for these stress resistant traits in benign laboratory environments is unfounded. It is true that if laboratory populations are kept at chronically small sizes, loss of variation is virtually inevitable. But the use of laboratory culture procedures that maintain large effective population sizes can minimize this effect.

One must always be careful about extrapolating conclusions obtained under one set of environmental conditions to a broader set of environments. However, it seems to us that we can determine which aspects of the environment matter only by performing experiments in which these environmental variables are carefully manipulated.

The complications of natural populations, like heterogeneous environments, migration, meteorological disaster, and so on, should not be viewed as barriers to laboratory studies but as challenges. Many interesting problems that can be addressed by laboratory populations still exist. For instance, there is still very little experimental work on the population dynamics of age-structured populations. It is unclear how age-structure affects population stability. Ultimately, we are optimistic that with the recognition of the many important contributions that can be made by experimental ecology, those employing its techniques will increase in number.

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