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NATURAL GENETIC VARIATION OF LIFE SPAN, REPRODUCTION, AND JUVENILE GROWTH IN *DAPHNIA*

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Abstract.—The evolutionary theory of senescence predicts that high extrinsic mortality in natural populations should select for accelerated reproductive investment and shortened life span. Here, we test the theory with natural populations of the *Daphnia pulex-pulicaria* species complex, a group of freshwater zooplankton that spans an environmental gradient of habitat permanence. We document substantial genetic variation in demographic life-history traits among parent and hybrid populations of this complex. Populations from temporary ponds have shorter life spans, earlier and faster increases of intrinsic mortality risk, and earlier and steeper declines in fecundity than populations from permanent lakes. We also examine the age-specific contribution to fitness, measured by reproductive value, and to expected lifetime reproduction; these traits decline faster in populations from temporary ponds. Despite having more rapid senescence, pond *Daphnia* exhibit faster juvenile growth and higher early fitness, measured as population growth rate (r). Among populations within this species complex we observed negative genetic correlations between r and indices of life-history timing, suggesting trade-offs between early- and late-life performance. Our results cannot be explained by a trade-off between survival and fecundity or by nonevolutionary theories of senescence. Instead, our data are consistent with the evolutionary theory of senescence because the genetic variation in life histories we observed is roughly congruent with the temporal scale of environmental change in the field.

Key words.—Aging, habitat duration, life history, reproductive value, senescence, trade-off.

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Senescence is a postmaturation decline in the physiological state of organisms as they grow older. From an evolutionary perspective, this decline is most relevant as a decrease in age-specific rates of survivorship and fecundity. The evolutionary theory of senescence (ETS) argues that the ultimate cause of senescence is a decline in the force of selection as organisms age (Medawar 1952; Williams 1957; Hamilton 1966; Charlesworth 1980). The force of selection declines because phenotypic effects at later ages influence smaller and later portions of reproduction relative to early phenotypic effects. Furthermore, these later effects are not exposed to selection in individuals who die before expression. Populations facing a greater risk of extrinsic mortality have a stronger decline in the force of selection, thus greater senescence should evolve in them. ETS has substantial support from laboratory experiments that have tested predictions about the response to selection on late-life performance (e.g., Rose and Charlesworth 1980; Mueller 1987; Partridge and Fowler 1992; Zwaan et al. 1995a). Tests of critical assumptions, including the existence of mutations with age-specific effects (Pletcher et al. 1998) and age-specific patterns of genetic variance in mortality (Promislow et al. 1996) and fecundity (Tatar et al. 1996) also support ETS. Research has focused on understanding and discriminating among the genetic mechanisms that permit the evolution of senescence (e.g., Rose and Charlesworth 1981; Rose 1984; Partridge and Fowler 1992). However, ascertaining the relative contributions of the nonmutually exclusive genetic mechanisms will not illuminate whether the general theory adequately explains the wide diversity of senescence patterns found in nature. Relatively little work has examined the ecological context of senescence evolution (Reznick 1993; Tatar et al. 1997; Dudycha 2000).

Tests of ETS by artificial selection on stock laboratory populations have dealt with situations where selection is direct and constant. Natural systems may be more complicated due to indirect and changing selection pressures (Abrams 1991, 1993), but, in general, we expect higher extrinsic mortality to select for greater senescence (Williams 1957; Hamilton 1966; Edney and Gill 1968; Charlesworth 1980, 1993). However, if the mortality falls predominantly on juveniles, reduced senescence is expected because adults are evolutionarily more valuable (Hamilton 1966; Charlesworth 1980; Abrams 1993). Although there is no general reason to expect that the relative magnitude of juvenile versus adult mortality is correlated with the absolute magnitude of extrinsic mortality in nature, they are often confounded in selection experiments (Clark 1987). Spatial or temporal changes in factors exerting mortality in nature may shift the net direction of selection on senescence through changes in the age-specificity of mortality or changes in the total level of mortality. It is not clear that the success of ETS in explaining senescence evolution in relatively straightforward laboratory settings will translate into success in explaining senescence as it has evolved in the wild.

Comparisons among taxa have suggested that maximum or mean life span, which represent only one aspect of senescence, are consistent with ETS. The ability to fly, a trait that presumably reduces extrinsic mortality risk, is associated with longer maximum life span in mammals (Prothero and Jurgens 1987) and birds (Holmes and Austad 1995). Keller and Genoud (1997) demonstrated a cross-taxon association between social system and estimated mean life span of female ants. They argued that, in captivity, queens live longer than reproductive females from solitary taxa because queens' life

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spans have evolved in a relatively protected colonial environment. A recent analysis of avian and mammalian mortality schedules (Ricklefs 1998) moves beyond life span and shows, under reasonable extrinsic mortality assumptions, that acceleration of adult mortality rate is positively related to the extrinsic mortality risk faced by young adults. These studies used organismal traits to approximate vulnerability to extrinsic mortality and estimate risk based on those approximations. One study (Tatar et al. 1997) has looked at an environmental axis to make predictions based on exposure to mortality risk. Tatar et al. (1997) showed that, when raised in a common environment, male grasshoppers from low elevations (i.e., a long season) live longer and have slower increases in age-specific intrinsic mortality than those from high elevations.

Complete life-table data are difficult to acquire, particularly when quantifying senescence, because one normally needs to shield individuals from extrinsic mortality (but see Promislow 1991; Ricklefs 1998). This difficulty places limits on the interpretation of comparative studies, which have usually relied on summary statistics such as the maximum or mean life span. First, senescence is a nonlinear change over time whose trajectory can vary in shape and magnitude. Simple summary statistics may not capture this variation. Second, mortality is only part of the fitness equation. Testing predictions of ETS has often yielded supportive data, but many empirical reports (e.g., Carey et al. 1992; Curtsinger et al. 1992; Tatar et al. 1993) have revealed patterns of mortality not completely predicted by ETS. The unexpected patterns may be due in whole or in part to shortcomings of the theory (Blarer et al. 1995; McNamara and Houston 1996; Pletcher and Curtsinger 1998) or it may simply be that the expected senescence pattern was expressed in unmeasured traits. Reproduction must be included for an accounting of fitness reduction due to senescence because age-dependent changes in a trade-off between survival ability and reproduction may cause declines in either ability (Partridge and Barton 1993, 1996). We are unaware of comparative work specifically incorporating reproductive declines that evolved in nature.

Objectives of This Study

We compare senescence patterns in closely related taxa living across a known gradient of mortality risk. First, we quantify the magnitude and distribution of natural genetic variation in senescence using full mortality and fecundity data collected under standard conditions that shield the animals from extrinsic mortality. Second, we look for tradeoffs (negative genetic correlations) between composite indices of senescence and early-life performance. Such tradeoffs are a mechanism by which rapidly senescing genotypes could persist in the face of invasion by long-lived genotypes and provide a framework for understanding how senescence is integrated into the total life history. Third, we consider the potential for further evolution of senescence in nature by examining differentiation of senescence among populations within species. We focus on multiple populations of the species complex *Daphnia pulex-pulicaria*, a group of freshwater microcrustaceans that are well suited to studies of senescence and ecology (Bell 1984; Reznick 1993; Dudycha 2000).

Daphnia and the Pond-Lake Gradient

The taxonomic status of *Daphnia pulex* and *D. pulicaria* is controversial. Two recent mtDNA phylogenies conclude that *D. pulex* and *D. pulicaria* are not distinct clades (Lehman et al. 1995; Crease et al. 1997). However, the most recent taxonomic revision (Hebert 1995) considered them separate species, based on differences in habitat occupied (pond or lake) and fixed allelic variation at the *Ldh* allozyme locus. Because we are mainly interested in the habitat differences among taxa, here we discuss them as distinct species. Our view is that the nominal species are ecologically distinct, but extremely close genetic relatives. Naturally occurring hybrid (*Ldh* heterozygotic) populations are also fairly common and so are included in our study.

Daphnia pulex and *D. pulicaria* are small (< 3.5 mm) crustaceans, which appear to segregate along an environmental gradient of habitat permanence (Deng 1997). *Daphnia pulex* is typically found in temporary ponds, whereas *D. pulicaria* is found in deep lakes. Populations of *D. pulex* must recruit from diapausing eggs in spring and undergo a variable period of reproduction via immediately developing eggs before producing diapausing eggs. Anoxia or desiccation kills individuals that survive to early summer. Variation in pond size, local hydrology, and annual weather patterns create variation in the length of time any one pond is habitable by *D. pulex*, but all of our study ponds dry by summer. Thus, maximum life span of *D. pulex* is constrained to a few months by the temporary nature of their habitat. In contrast, *D. pulicaria* lives in a permanent and environmentally more stable habitat and can persist year-round in deep lakes (Geedey et al. 1996; Geedey 1997). In the absence of any strong abiotic constraints, resources and predators regulate *D. pulicaria* population dynamics, generating predictable peaks of habitat quality in the spring and fall (Hutchinson 1967). Planktivorous fish constitute the most important predator, but *D. pulicaria* can effectively avoid them in deep water (Tessier and Welser 1991). Populations of *D. pulicaria* display low birth and death rates even at the time of peak planktivory (Leibold and Tessier 1998). Perennial populations of *D. pulicaria* also produce both immediately developing and diapausing eggs, but we have observed that diapause investment is much less than in *D. pulex* (Geedey 1997; J. L. Dudycha, unpubl. data).

Hybrids of *D. pulex* and *D. pulicaria* occur in a broader range of waterbody type than either parent, including both shallow ponds and lakes. In some cases they co-occur with one of the parents, but they typically occupy habitats poorly exploited by either parent, such as permanent fishless ponds. Little is known about hybrid population dynamics, however, a few local populations become undetectable in late summer. (C. Steiner, pers. comm. 1998)

Gradients of habitat permanence cause strong differences in extrinsic mortality faced by different populations if individuals have no mechanism to escape habitat loss (e.g., migration). As habitat duration lengthens, the time until certain death is extended and, therefore, the duration of potentially useful adult life span is also extended. Based on an expected life span driven by abiotic and biotic factors regulating population dynamics, we predicted the evolution of

faster senescence in pond *D. pulex* than in lake *D. pulicaria*. We made no a priori predictions regarding the senescence of hybrids, due to uncertainty regarding how the parents' genomes would combine and to the lack of any reasonable expectation for adaptation to their place on the habitat duration gradient.

Methods

Clonal Isolation and Lineage Establishment

Daphnia will reproduce in the laboratory by strictly ameiotic parthenogenesis, allowing us to capture the genetic variation in nature by randomly sampling a population and establishing isogenetic lines. We isolated five randomly chosen clones from each of three populations of *D. pulex*, three populations of *D. pulicaria*, and two populations of naturally occurring hybrids. We did not choose populations randomly; rather, we chose populations of the parent species to represent the extremes of the habitat permanence gradient. This consideration did not have much effect on the choice of *D. pulex* populations, but we chose *D. pulicaria* populations based on data indicating they were perennial populations (Geedey 1997). We chose hybrid populations from permanent waters free of fish planktivory. All populations are in southwestern Michigan, except one of *D. pulex*, which is in Michigan's Upper Peninsula. Clones were isolated in April and May 1996, except for a few clones isolated in spring 1995. Collecting clones at this time of year maximizes the genetic variation we sample due to recent hatching of sexually produced diapausing eggs (Lynch, 1984; Tessier et al. 1992; Geedey et al. 1996). We established lineages from all clones and acclimated them to laboratory conditions (20–22°C, satiating food) for at least three generations prior to life-history trait measurement (Tessier and Consolatti 1989, 1991). Routine monitoring and microscopic examination (at 460×) verified that lineages were free of parasites.

Life-History Trait Measurement

We started life tables with neonates whose mothers were all raised simultaneously from birth for several weeks at low density (1/50 ml) to minimize variable maternal effects. Life tables began concurrently with neonates (~ 12 h old) from the third or later clutches of offspring. For each population, three cohorts consisting of two neonates from each of the five clones (30 neonates/population) were set up. Some minor deviations from this ideal occurred because a few clones failed to produce an adequate number of female neonates on the day the experiment started. Animals were changed to fresh water every two days, fed a satiating food level (20,000 cells/ml *Ankistrodesmus falcatus* daily), and incubated at 20°C on a 16:8 L:D cycle. Water volume was adjusted as animals grew and died such that a cohort could not filter more than 50% of their water between feedings (increased from 10 ml/neonate to 300 ml/animal at the largest size; Knoechel and Holtby 1986). We recorded mortality daily and fecundity every two days until all animals died.

Daphnia have two modes of reproduction, parthenogenetic reproduction that produces immediately developing offspring and (usually) sexual reproduction that produces diapausing eggs. Only immediately developing offspring were included in our estimates of age-specific daily fecundity (m_x). When a female produced an ephippium, the easily identified structure into which diapausing eggs are deposited, we adjusted fecundity values by considering that female to be unavailable for reproduction until the next instar (Lynch 1989). Thus, m_x was calculated by dividing the number of directly developing offspring produced by the number of live females that were not producing an ephippium. Normally, no eggs will be deposited in the ephippium when males are not present. We assumed that individual females did not engage in continuous ephippial production.

Juvenile survival was nearly perfect for most populations. As an additional measure of juvenile performance, we estimated juvenile growth rates of 50 clones of *D. pulicaria*, 11 clones of *D. pulex*, and 16 hybrid clones. These clones were isolated as part of another project from a broader array of Michigan populations than those for the life tables were. Mothers were raised as in the life tables. For each clone, 18–24 randomly chosen neonates were immediately harvested; an additional 10 neonates were raised to maturity (here, defined as the day eggs were released into the brood chamber) under the same conditions as in the life tables, except they were fed a higher food level (40,000 cells/day). This food level is functionally equivalent (satiating) to the food level used in the life tables (Lampert 1987). Neonates and mature animals were dried overnight at 55°C, then weighed (to within 0.1 μg) on a Cahn microbalance. We used time to maturity in conjunction with neonatal and mature mass to calculate juvenile specific growth rate ($\mu g\ \mu g^{-1}\ d^{-1}$; Tessier and Goulden 1987).

Quantifying Aspects of Senescence

Mortality data reflects the capability of the average individual to maintain its body under the daily rigors of life. Any degradation in that capability appears as an increase in the age-specific *conditional* probability of mortality (given survival to the beginning of the age class), commonly called the hazard function. Even if maintenance capability is constant, survivorship will still decline if it is imperfect. Thus, it is an increasing hazard that signifies decreasing maintenance capability and is considered evidence of senescence. Fecundity data reflects the capability of the average individual to produce progeny. Any degradation of this capability is manifest simply as a decline in the age-specific fecundity rate and is considered evidence of senescence.

Observing a decline in either survival ability or fecundity alone may be evidence of senescence, but it may instead reflect a reallocation of effort between these two fitness components. As an organism ages, it may trade off mortality risk (i.e., somatic maintenance) with fecundity. Therefore, when comparing rates of senescence among taxa it is important to examine age-specific patterns of survival and fecundity jointly. We use two general approaches in our comparison of populations of the *D. pulex-pulicaria* complex.

We first analyze age-specific patterns of survivorship and fecundity as separate fitness traits. Separately comparing the degradation of each trait across taxa is straightforward because each reflects a component of age-specific fitness (mor-

tality hazard or per capita fecundity) for the average individual. However, only when both traits lead to the same conclusion will this approach address senescence satisfactorily. Therefore, we also compare the taxa by combining survival and fecundity into single measures of age-specific fitness.

One of the most widely used summary measures of age-specific fitness is reproductive value, which specifies the contribution of each age class to r, the rate of population increase. Reproductive value (v_x) has been suggested as a useful measure for senescence studies (Partridge and Barton 1996). However, interpretation of v_x with respect to senescence as a process of physiological degradation is not at all straightforward. Because v_x weights each age class's expected fecundity by its contribution to r, early reproduction has greater "value" than equal reproduction at a later age. Therefore, as an alternative measure of age-specific fitness, we also examine the unweighted contribution of each age class to R_0, the total lifetime reproduction. We refer to this age-specific measure as the intrinsic value (i_x).

Because our newborns enter age class 1, we calculate reproductive value, v_x, with this equation (Goodman 1982):

$$v_x = \frac{e^{r(x-1)}}{l_x} \sum_{j=x}^{\infty} e^{-rj} l_j m_j, \tag{1}$$

where l_x (cumulative survivorship) and m_x (per capita fecundity) are taken directly from the life-table data and r is iteratively calculated by Newton-Raphson approximation. Intrinsic value, i_x, is calculated as the ratio of current and expected future reproduction to the expected lifetime reproduction of an individual:

$$i_x = \sum_{j=x}^{\infty} l_j m_j \Big/ \sum_{j=0}^{\infty} l_j m_j. \tag{2}$$

We scale by expected lifetime reproduction (R_0) to simplify comparison across taxa that differ in overall capability. The scaling by R_0 causes the intrinsic value to reflect relative changes in age-specific fitness rather than absolute changes. In some cases, comparison of absolute changes may be appropriate; however, some trade-offs (e.g., between offspring size and number) may bias an absolute comparison.

Statistical Analyses

To evaluate whether there was potential evidence for senescence in the mortality data alone, we pooled the data for each taxon and fit it to a Weibull model. Weibull models normally express age-specific mortality (μ) at age x as $\mu_x = \lambda \gamma(\lambda x)^{\gamma-1}$ (Lee 1980). When γ, the dimensionless "shape parameter," equals one, the hazard function is constant and there is no evidence of senescence. If γ is greater than one, the hazard function increases with age; because this indicates that the conditional per capita mortality rate increases with age, it is potentially (pending analysis of fecundity) evidence of senescence. The "scale parameter" λ scales the model to a baseline rate of mortality, but is not indicative of senescence. We estimated γ with the SAS version 6.09 Lifereg procedure.

We applied two types of regression model to the mortality data to test for differences among taxa in the pattern of mortality hazard. First, we used an accelerated failure time (AFT) model to test for differences in the timing of high mortality hazard; AFT models assume that a factor (in this case, taxon) affects *failure time* (life span) multiplicatively, shifting hazardous periods along the timeline (Kalbfleisch and Prentice 1980; Fox 1993). Analyses of failure-time data in other fields normally address shifts in the time period when failures occur (Kalbfleisch and Prentice 1980; Lawless 1982; Fox 1993), but this has rarely been done in investigations of senescence. Instead, life span is commonly examined due to its inherent interest, but it serves to identify only the endpoint of senescence. To apply the AFT model, we chose an underlying gamma distribution because its flexibility allows, but does not require, the theoretical expectation of monotonically increasing age-specific mortality (for a general discussion of survival distributions, see Kalbfleisch and Prentice 1980; Fox 1993). We also ran comparisons using alternative biologically plausible underlying distributions, including the Weibull distribution, and found our results were robust regardless of distribution. Second, we used a proportional-hazards (PH) model to test for differences among taxa in the magnitude of mortality hazard increase. PH models assume that a factor affects *hazard functions* multiplicatively, changing the magnitude of hazard in a given period (Kalbfleisch and Prentice 1980; Fox 1993). Juvenile mortality was excluded from analyses. All survivorship analyses were run in SAS version 6.09 with the Lifereg and PHreg procedures.

We used repeated-measures ANOVA to test for differences among taxa in the pattern of age-specific fecundity. A significant time × taxon interaction indicates differences in age-specific changes of fecundity profiles and may be evidence of differences in senescence if horizontal (age) compression of one taxon's profile relative to the other's is evident. If there is a significant interaction and mortality analyses show the same relative contrast, then we can conclude there is evidence of differences in overall senescence. We also used repeated-measures ANOVA to test for differences among taxa in the shapes of their reproductive value and intrinsic value curves. For the combined measures, a significant time × taxon interaction indicates differences in the rate of change of age-specific fitness with respect to r (reproductive value) or R_0 (intrinsic value).

We tested for correlations between r and two indices that summarize the major differences among populations in temporal distribution of age-specific fitness. The first index was the age when v_x peaks, which we determined by fitting cubic polynomials, the simplest equations allowing asymmetry, to each reproductive value curve. The second index was the age when only 25% of intrinsic value remained. We chose 25% to allow most of the divergence between taxa to occur, while minimizing any influence of "tails" in the data.

Results

Mortality and Fecundity

Daphnia pulex had a substantially shorter intrinsic life span (median = 38 days, maximum = 54 days) than *D. pulicaria* (median = 62 days, maximum = 99 days; Fig. 1A). Both species showed decreasing ability to maintain their bodies as

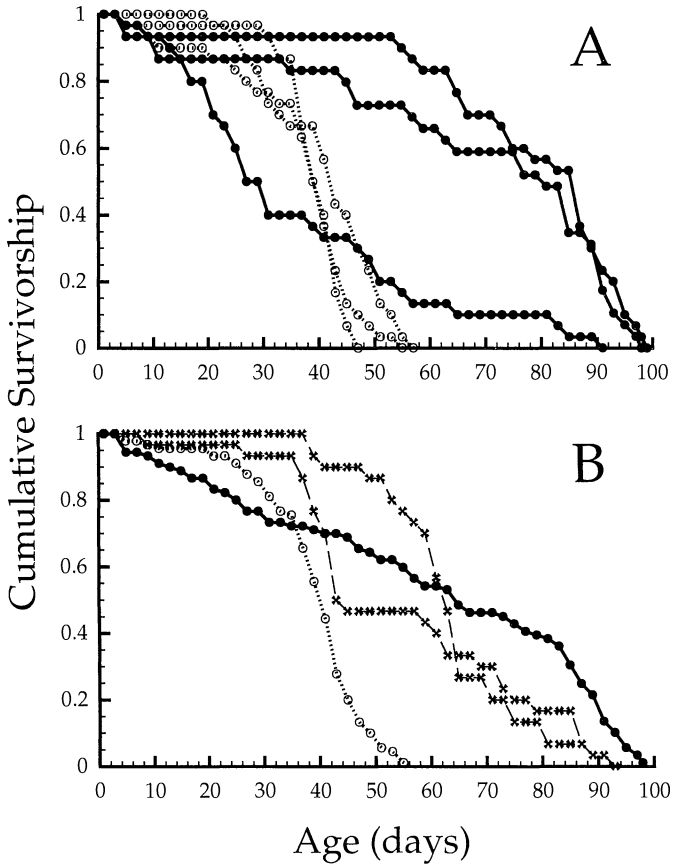

FIG. 1. Survivorship of *Daphnia pulex-pulicaria*. Median life span is the age where survivorship equals 0.5. (A) Three replicate populations of *D. pulex* (open circles and dotted lines) and three of *D. pulicaria* (closed circles and solid line). (B) Data in top panel pooled by species (dotted and solid lines) and two replicate populations of naturally occurring hybrids (crosses and dashed lines).

they aged. This was evident from both the increasing mortality hazard functions (Fig. 2) and Weibull shape parameters that were greater than one (*D. pulex* $\gamma = 7.7$, 95% CI = 5.1, 15.5; *D. pulicaria* $\gamma = 2.9$, 95% CI = 2.4, 3.6). The timing of high mortality hazard was significantly earlier in *D. pulex* than in *D. pulicaria* (AFT model; $\chi^2 = 566.2$, df = 1, $P < 0.0001$). The magnitude of mortality hazard increase was also significantly greater in *D. pulex* than in *D. pulicaria* (PH model; $\chi^2 = 57.6$, df = 1, $P < 0.0001$). Thus, intrinsic adult mortality increased both more and earlier in *D. pulex* than in *D. pulicaria*. The survivorship curve of hybrids (median life span = 58 days, maximum = 91 days; Fig. 1B) had a roughly similar shape to *D. pulex* (hybrid $\gamma = 6.1$, 95% CI = 5.1, 7.7), yet it was delayed, similar to *D. pulicaria* (Fig. 2). Within both parent species, there was also significant among-population variation in the timing of high mortality hazard (*D. pulex*: $\chi^2 = 16.3$, df = 2, $P < 0.0003$; *D. pulicaria*: $\chi^2 = 33{,}125.1$, df = 2, $P < 0.0001$). Similarly, variation in the magnitude of mortality hazard increase was significant (*D. pulicaria*: $\chi^2 = 18.7$, df = 1, $P < 0.0001$) or marginally so (*D. pulex*: $\chi^2 = 3.4$, df = 1, $P = 0.066$; Fig. 1A).

The shape of the fecundity curve for *D. pulex* was significantly compressed horizontally (changes occurred faster) relative to *D. pulicaria* (time × taxon interaction; $F = 4.12$; df = 1, 46; $P < 0.0001$; Fig. 3A). The difference between the species in their degradation of reproductive output was striking, because over the entire time that *D. pulex* was sharply declining (ages 35–55 days), *D. pulicaria* continued to improve its daily fecundity, declining moderately only after reaching 65 days old. Hybrids had a greater overall fecundity rate than either parent (Fig. 3B). The shape of the hybrid curve was strongly peaked, like that of *D. pulex*, but extended to much later ages like that of *D. pulicaria*. There was also significant variation in the shapes of the fecundity curves among populations within *D. pulicaria* and marginally significant variation within *D. pulex* (*D. pulicaria*: $F = 1.94$; df

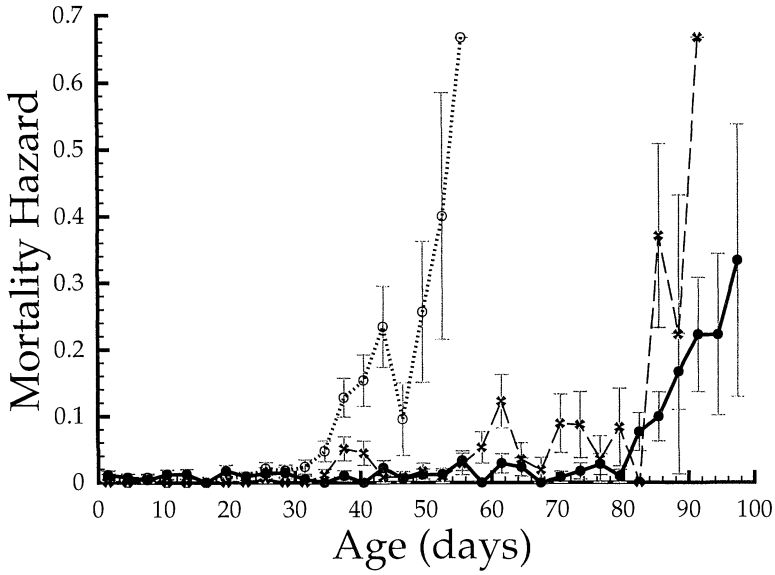

FIG. 2. Mortality hazard function of *Daphnia pulex, D. pulicaria*, and hybrids. Symbols as in Figure 1. Standard error bars are based on replicate populations.

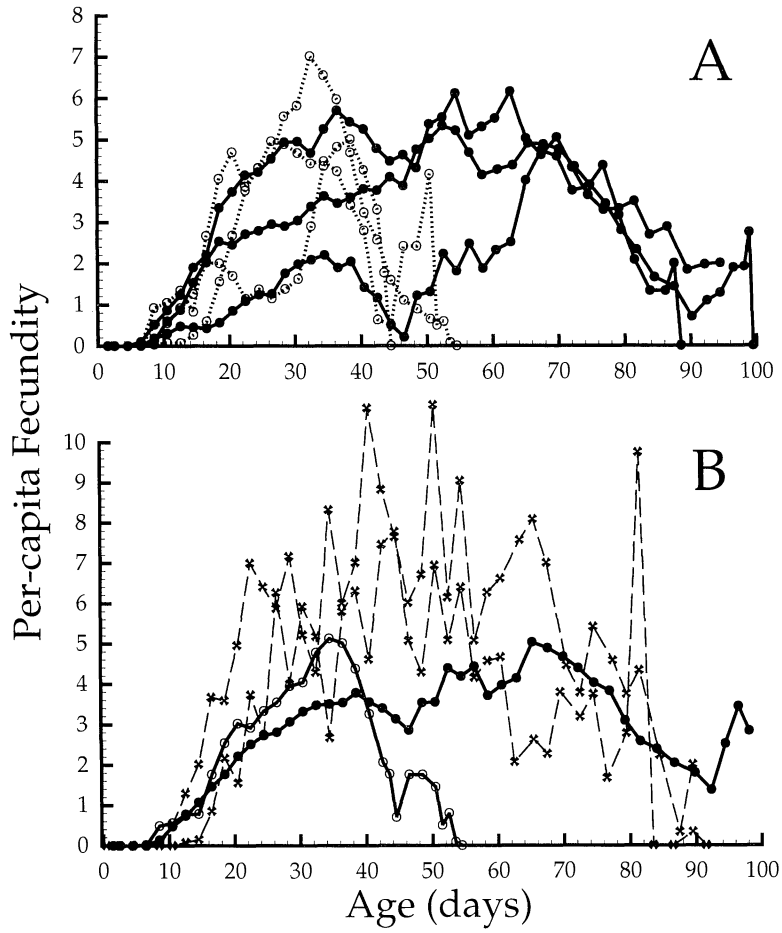

FIG. 3. Average daily fecundity of *Daphnia pulex, D. pulicaria*, and hybrids. (A) Three replicate populations each of *D. pulex* and *D. pulicaria*. (B) Data in top panel averaged by species and two populations of hybrids. Symbols as in Figure 1, except in B *D. pulex* and *D. pulicaria* have solid lines.

= 2, 94; $P < 0.0001$; *D. pulex*: $F = 1.38$; df = 2, 54; $P < 0.069$).

Combined Fitness Values

Despite exhibiting an earlier and steeper increase of mortality hazard and a more rapid decline of fecundity, *D. pulex* had a higher r than *D. pulicaria* ($t = 2.654$, df = 2, $P = 0.057$). This was a consequence of *D. pulex*'s more rapid increase in fecundity early in life (Fig. 3). There was no obvious pattern of difference in expected lifetime reproduction (Table 1).

The shape of the reproductive value profile for *D. pulex* was significantly compressed horizontally relative to *D. pulicaria* (time × taxon interaction; $F = 7.43$; df = 1, 46; $P < 0.0001$; Fig. 4A). Note that by the age when *D. pulicaria* peaks in its ability to contribute to population growth, *D. pulex* has lost essentially all of its ability, even though both species achieved similar maximal age-specific contributions. Therefore, *D. pulex* lost its reproductive value faster. The reproductive value curve of hybrids (Fig. 4B) rose rapidly like *D. pulex*, peaked midway between the parents, and declined at late ages similar to *D. pulicaria*. There was also significant variation within each parent species in the shapes of the reproductive value profiles (*D. pulex*: $F = 4.87$; df = 2, 54; $P < 0.0001$; *D. pulicaria*: $F = 1.45$; df = 2, 94; $P = 0.010$).

TABLE 1. Values of fitness measures and the timing of fitness contribution in *Daphnia pulex* and *D. pulicaria*. See text for explanation of variables.

	D. pulex			*D. pulicaria*			Hybrid	
Population	WIS	OL3	MCP	WAR	PIN	LAW	WHT	WIN
r	0.54	0.45	0.50	0.41	0.41	0.29	0.43	0.47
R_0	108	58	180	232	218	34	240	245
Age of v_x peak (days)	18.5	29.5	23.5	48.5	44.5	62	34.5	42
Age of 25% i_x (days)	29.5	35	34	63	62	54.5	50	51

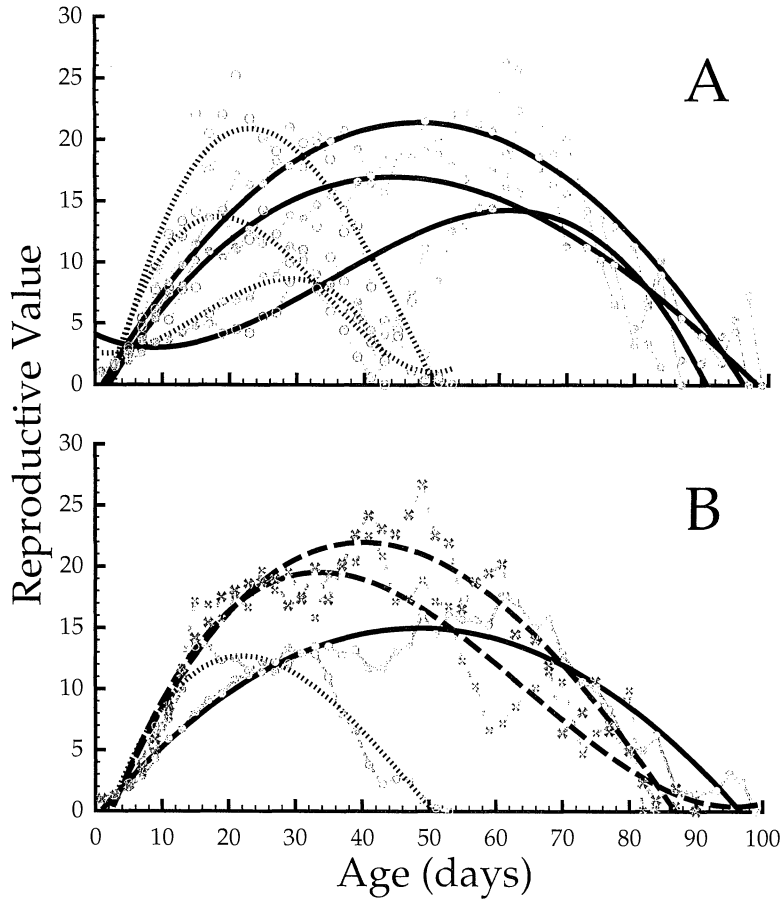

FIG. 4. Reproductive value of *Daphnia pulex-pulicaria*. Gray symbols and lines are reproductive value data calculated as described in text. Black curves are cubic polynomials fit to each population. (A) Three replicate populations each of *D. pulex* and *D. pulicaria*. (B) Mean values by species of data in top panel and two populations of hybrids. Symbols as in Figure 1.

Intrinsic value is defined to be one at age 0 and it remained one until animals matured, then declined roughly linearly until about 95% of intrinsic value had been lost. However, *D. pulex* lost its ability to contribute to R_0 substantially faster than *D. pulicaria* (time × taxon interaction; $F = 23.24$; df = 1, 46; $P < 0.0001$, Fig. 5A), creating a difference of more than 30 days between the ages when 25% of intrinsic value remained. Again, the hybrid populations were intermediate to the parental taxa (Fig. 5B).

There was a strong, significant negative correlation between r and the age when v_x peaks ($r = -0.923$, $P = 0.001$) and a weaker correlation between r and the age when 25% of intrinsic value remained ($r = -0.679$, $P = 0.064$; Table 1 and Fig. 6). These correlations both suggest a genetic trade-off between early life fitness (which maximizes r) and late life fitness (which extends the age when v_x peaks and when 25% i_x remains). Because r is used in the calculation of v_x, we looked for any mathematical dependence between r and the age when v_x peaks by running simulations with hypothetical life-history data (combinations of four l_x and 28 m_x profiles). In all life histories, reproduction began at age 11 and continued until age 50. With these hypothetical life histories, we generated points that evenly filled the range of parameter space (r = 0.0–0.5; age when v_x peaks = 10–50 days) defined by our empirical data (J. L. Dudycha, unpubl. data). This was true for both nonsenescent (constant or decreasing age-specific mortality rate and constant or increasing fecundity) and senescent (increasing age-specific mortality and/or decreasing fecundity) life-history profiles. These simulations also confirmed that the age when v_x peaks is substantially affected by late-life traits, whereas r is not. Thus,we are certain that the trade-off we inferred is biological, not mathematical, in nature.

Juvenile Size and Growth

In our comparison of juvenile performance, we observed that *D. pulicaria* matured in 6.4 ± 0.1 SE, *D. pulex* in 5.4 ± 0.2 SE, and hybrids in 6.0 ± 0.2 SE days. These differences are small relative to life span, but are a substantial portion of the juvenile period. One-way ANOVA revealed that per offspring investment, measured as neonate mass, varied among the taxa ($F = 11.8$, df = 2, 77, $P < 0.0001$). *Daphnia pulex* had the smallest neonates (2.4 ± 0.2 SE μg), followed by hybrids (2.9 ± 0.2 μg) and *D. pulicaria* (3.5 ± 0.1 μg). Post hoc pairwise comparison showed that *D. pulicaria* was significantly different from *D. pulex* ($P < 0.0001$) and from hybrids ($P = 0.0243$). Differences in per offspring investment

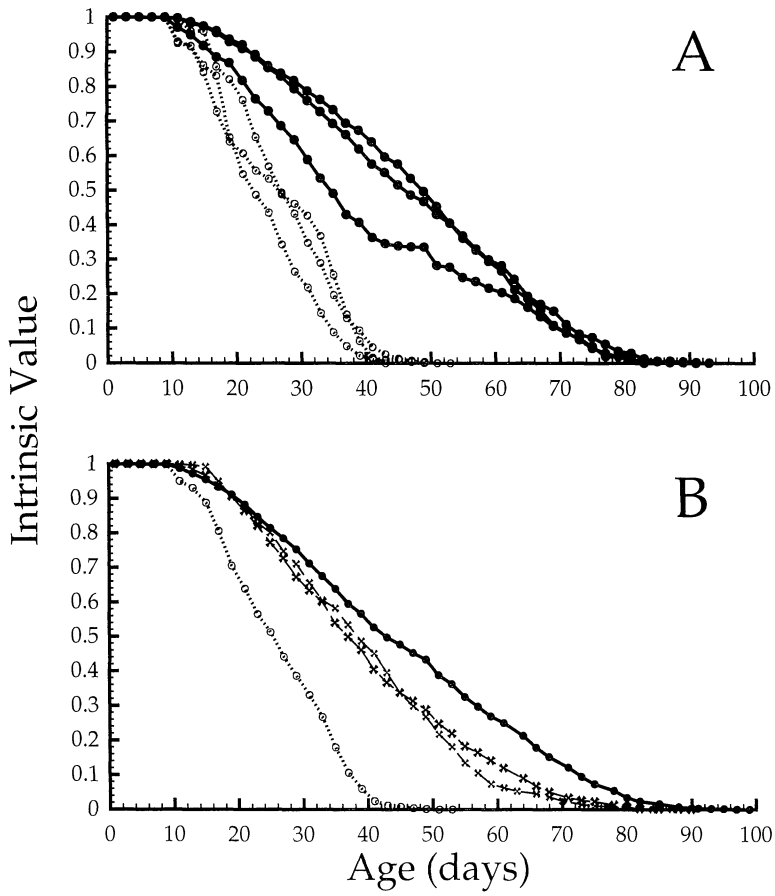

FIG. 5. Intrinsic value of *Daphnia pulex-pulicaria*. See text for explanation of intrinsic value. (A) Three replicate populations each of *D. pulex* and *D. pulicaria*. (B) Mean values by species of data in top panel and two populations of hybrids. Symbols as in Figure 1.

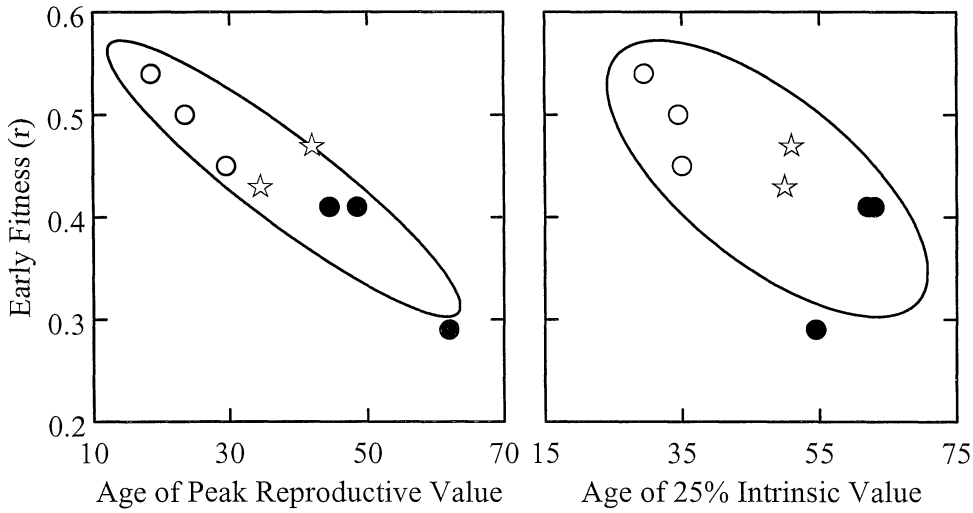

FIG. 6. Relationship between early fitness and life-history timing in *Daphnia pulex-pulicaria*. Age of peak reproductive value is the age when a cubic equation fit to reproductive value peaks. Age of 25% intrinsic value is the age when only 25% of the intrinsic value remains. Black circles, *D. pulicaria*; open circles, *D. pulex*; stars, hybrid. Each symbol represents a replicate population. One standard deviation ellipses are drawn around the data.

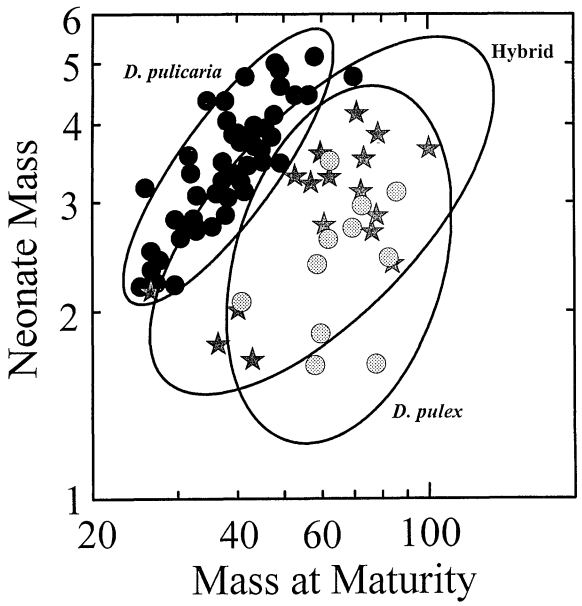

FIG. 7. Neonate and adult mass (in μg) of clones in the *Daphnia pulex-pulicaria* complex. Each symbol represents a separate clone; 90% confidence ellipses are drawn around each taxon. Black circles, *D. pulicaria*; light circles, *D. pulex*; stars, hybrid.

between the parents are even more obvious when comparing clones with similar mass at maturity (Fig. 7). *Daphnia pulicaria* had a significant correlation between neonatal and adult mass ($r = 0.83$, $P < 0.001$; Fig. 7). The relationship was isometric (reduced major axis regression slope = 1.0 ± 0.08), showing that *D. pulicaria* clones had the same juvenile specific growth rate (0.378 ± 0.003 SE μg μg^{-1} d^{-1}), regardless of their mass at maturity. Neonatal and adult mass of hybrids were also correlated ($r = 0.68$, $P = 0.004$) and isometric (RMA regression slope = 0.98 ± 0.19). However, the elevation of the hybrids' slope was distinctly below that of *D. pulicaria* (ANCOVA: $F = 88.2$; df = 1, 63; $P < 0.001$) and they matured slightly earlier, contributing to an overall faster juvenile specific growth rate in hybrids (0.514 ± 0.009 SE μg μg^{-1} d^{-1}). Our sample of *D. pulex* clones spanned a limited range of adult sizes, and there was no significant correlation between neonate and adult mass ($r = 0.3$, $P = 0.37$). These *D. pulex* clones largely fell within the 95% probability ellipse for the hybrids (Fig. 7), and because they had an even shorter maturation time, their juvenile specific growth rates were faster (0.613 ± 0.015 SE μg μg^{-1} d^{-1}) than *D. pulicaria* ($F = 611.9$; df = 1, 59; $P < 0.0001$).

DISCUSSION

We conclude that senescence occurs in all of our study populations because both the hazard functions increased and fecundity rates declined over later life. Our analyses of mortality and fecundity combined also showed a degradation of age-specific fitness in later life, excluding the possibility that the separate degradations of survival and reproductive capabilities create an illusion of senescence via a changing trade-off between them. Furthermore, our data show that *D. pulex*, a resident of ephemeral habitats, experiences greater senescence than *D. pulicaria*, a resident of permanent habitats, as expected by the evolutionary theory of senescence. Specifically, *D. pulex* has a shorter life span, an earlier and steeper rise in mortality rate, an earlier and sharper decline of fecundity, a horizontally compressed reproductive value, and a more rapidly declining intrinsic value than does *D. pulicaria*.

Our approaches to analyzing life-history data with respect to the concept of senescence are complementary. The separate mortality and fecundity analyses document that both survival and reproductive abilities degrade in concert, supporting a common assumption (Partridge and Barton 1993). Our analysis of reproductive value specifies how much the combined degradation of fitness components affects age-dependent loss of the ability to contribute to population growth. This view of senescence highlights how the contribution of alleles to phenotypic evolution changes with age and may be especially important in studies that seek to measure selection on senescence. Finally, our analysis of intrinsic value gives a summary picture of biological abilities, showing the overall degradation with age of an average individual. Under laboratory conditions, where survivorship declines primarily due to intrinsic breakdown, this measure is closer to a physiological view of senescence than reproductive value.

Studies that examine traits under a single set of laboratory conditions, but deduce predictions from field conditions, are potentially biased by genotype-by-environment interactions. We conducted our life tables under abiotic conditions that are benign and at a satiating food level. These conditions are not unusual for any of our populations in the field. However, the average temperature *D. pulex* experiences in ponds is warmer and closer to 20°C than the temperature *D. pulicaria* experiences in lakes, because *D. pulicaria* spends summer in cold deep water (Leibold and Tessier 1998). Resource levels can be high in lakes or ponds, but are usually lower in lakes than in ponds (Leibold and Tessier 1998; A. J. Tessier, unpubl. data). Therefore, the laboratory environment resembles the pond habitat of *D. pulex* more closely than the lake habitat of *D. pulicaria*. It is unlikely that an even closer match to the pond environment would improve late-life fitness in *D. pulex* enough to reverse the fundamental differences between the species' senescence patterns through a genotype-by-environment interaction.

Assuming that the laboratory environment is sufficiently similar to natural conditions to make an unbiased extrapolation to the field, we can ask to what degree the observed life-history differences are functionally significant in the field. The life spans of *D. pulex* seen in the laboratory are shorter than the duration of their habitats (typically, snowmelt occurs in late February/early March and the ponds dry in mid-June, making ponds habitable by *D. pulex* for about three months). An exact match is not expected because there is interannual variation in the duration of ponds and most individuals are not born on the earliest possible day, therefore potential life span is less than a pond's average duration.

In contrast, lakes may be a permanent habitat, but habitat quality is not uniform over the year. *Daphnia pulicaria* population density is often greatly reduced in summer and under ice in winter due to low resource availability in the former and the combination of low resources and very cold tem-

perature in the latter. Spring and fall are predictable periods of high algal (resource) availability, and consequently *Daphnia* population growth is also high (Hutchinson 1967; Sommer 1989). Individual daphnids who can survive from spring to fall (or vice versa) may have a considerable advantage over genotypes who rely on diapause to get through summer and winter. Diapause is disadvantageous because these eggs depend on imperfect environmental cues to decide when to leave diapause. The life spans we saw in the laboratory suggest that substantial numbers of *D. pulicaria* may live long enough to cross the seasonal troughs of habitat quality because the average temperature and resource level they experience in the field is lower than laboratory conditions. Daphnid life spans have been shown to lengthen in colder temperatures (MacArthur and Baillie 1929; Ingle 1933) and under reduced resources (Ingle et al. 1937). When resources increase in spring and fall, a population of *D. pulicaria* will be composed mainly of large adults that persisted from the last good season. These adults can now resume a high level of reproduction, which had necessarily declined during the poor resource period (Leibold and Tessier 1998). Because this "peristent" demography will be more rapidly responsive to resource change than a "diapause" demography, the population can exploit the resource flush better than a population constrained to hatch from diapausing eggs or even one whose age structure is relatively juvenile.

We have argued that the differences in senescence between *D. pulex* and *D. pulicaria* have evolved as a result of differences in their habitats. However, we do not present data to show that mortality imposed by habitat loss (or shielded by habitat maintenance) is the direct mechanism responsible for the genetic differences in senescence. Other differences between the habitats may influence senescence evolution. For example, temporary habitats force *Daphnia* populations to use diapause. Senescence differences could evolve as a correlated effect of selection on investment in diapause through costs of reproduction. This selective pathway may contribute to the different senescence patterns expressed by *D. pulex* and *D. pulicaria* in our study because *D. pulex* produced more diapause structures than did *D. pulicaria* (1.2 vs. 0.3 ephippia per female per lifetime). Size-selective predation may also vary between temporary ponds and permanent lakes. Because size and age are correlated, size-selective predation could exert selection on senescence. We do not know whether size-selective predation systematically varies among our study populations. If predation falls mainly on large individuals in temporary ponds and on small individuals in permanent lakes, this could contribute to the evolution of the senescence differences we found.

Trade-offs

What allows rapidly senescing organisms to persist despite the existence of long-lived genotypes? One elaboration of ETS is based on the hypothesis of genetic trade-offs, namely that poor late-life fitness is genetically linked to good early-life fitness through genes that are antagonistically pleiotropic (Hamilton 1966; Rose 1991; Stearns 1992). In this scenario, selection for a good early-life phenotype will indirectly select for a poor late-life phenotype. Optimality approaches to senescence inextricably tie senescence to development (Hamilton 1966; Partridge and Barton 1993, 1996), making ETS also a theory of *early*-life performance. The developmental theory of senescence hypothesizes that pleiotropic genes benefit development rate at the expense of extended adult survival and reproduction (Zwaan et al. 1991, 1995b). The simple prediction is that juvenile performance should be better in taxa that have faster senescence.

Life-history theory focusing on offspring quality also predicts a link between early- and late-life traits. Winkler and Wallin (1987) modeled the relationship between total reproductive effort and per offspring investment. They predicted that organisms investing heavily in reproduction would have smaller optimal per offspring investment. Their model explicitly assumed a trade-off between reproduction and adult survival. Therefore, their model also predicts a positive relation between optimal per offspring investment and life span.

We demonstrated genetically faster juvenile specific growth in *D. pulex* than in *D. pulicaria*, confirming the developmental theory's hypothesized relationship between early performance and senescence. We also showed greater per offspring investment, measured as neonate mass, in *D. pulicaria* than in *D. pulex*, supporting the Winkler and Wallin (1987) model. Hybrids are intermediate to the parental species for both of these traits and for senescence. *Daphnia pulex* produced relatively small neonates who rapidly grew large, whereas *D. pulicaria* produced large, slowly growing neonates. *Daphnia pulex* evidently gains its advantage in r by producing cheap, rapidly growing offspring. This negative relationship between per offspring investment and juvenile performance is contrary to the usual expectation that greater investment in each offspring produces juveniles who are more fit. However, through joint consideration of the developmental theory and the per offspring investment model, this negative relationship becomes intelligible, because juvenile performance and per offspring investment are both mediated by a trade-off with the senescence of adults.

In a recent review of empirical research on growth rates, Arendt (1997) argued that research rarely addresses trade-offs between juvenile growth rate and adult fitness (reproduction or maintenance), despite the possibilities that organisms may be unable to reallocate resources perfectly from growth to adult fitness or that rapid growth may build a less sturdy body. In other words, rapid juvenile growth may be advantageous to early adult fitness, yet cause poor late-age fitness. Arendt (1997) also emphasized that genetic variation in growth rate is often overlooked on the assumption that there is no fitness advantage to submaximal growth rates. Our juvenile growth experiment clearly shows that there is variation in specific growth rate among these physiologically similar taxa. In combination, our data showing associations between genetic variation in the packaging of reproductive effort, senescence, and juvenile growth suggest that exploration of the mechanisms linking them will be a strong approach to understanding life history. A large literature discusses optimal life histories from the separate perspectives of senescence and per offspring investment, but these perspectives rarely intersect. Our results suggest that empirical work in other taxa would benefit from a joint consideration of senescence and per offspring effort.

The trade-offs we discovered would prevent invasion of temporary ponds by long-lived genotypes because their reduced r places them at a disadvantage when the pond dries, and genotypes can locally persist only by generating a diapausing stage. At this critical accounting period, the number of individuals of high r will be greater than those of low r. Assuming that the genotypes have equivalent diapause, the long-lived will never catch up because the cycle is reset every year. This rationale may explain the puzzle of rapid senescence, but it highlights a more general problem in evolutionary biology: why genotypes with low r are not displaced by genotypes with high r. Although it is speculative, we suspect that the inability of short-lived, high-r genotypes to invade lakes may be related to their inability to persist across habitat quality troughs. If an individual's life span precludes survival through an extended period of poor habitat quality, its genotype can persist only through offspring production or through diapause. Neither strategy is likely to be effective in lakes. Offspring cannot survive and grow well in comparison to adults during poor resource conditions, and diapause is complicated by the need for a reliable, accurate cue of improving habitat conditions. Furthermore, a diapause strategy involves a time lag, delaying the ability of diapausing genotypes to respond to the habitat improvement compared to genotypes that persist as surviving adults. In sum, the ability of long-lived individuals to span summer or fall may outweigh the advantages of high laboratory-based r because high r is genetically linked to other traits, that is, short life span and small offspring, that are disadvantageous during low resource periods in lakes.

We focused our study on several populations of a single species complex to make detailed life-history comparisons under common conditions. At this time, we do not have a phylogenetic estimate for these populations and cannot say definitively whether our comparison includes independent evolutionary divergences or the differences we found may be partly due to historical constraints. However, the local existence of interspecific hybrids, which occupy an intermediate position along trade-offs between r and senescence, suggests that the species are genetically very similar and historical constraint on life-history evolution may be minimal. Even if our populations prove to be phylogenetically nonindependent, the principal alternative theory for explaining senescence, that degradation is caused simply by metabolic exhaustion (Comfort 1979), cannot explain the variation we found. A review of the rather extensive work on metabolic rate in *Daphnia* (Peters 1987) found no evidence for taxonomic differences; metabolism scaled negatively with size. Therefore, if senescence were ultimately caused by metabolic exhaustion, we would have seen no variation between the species or faster senescence in *D. pulicaria* because *D. pulicaria* adults are on average smaller than *D. pulex* (Fig. 4; Hebert 1995).

Our results indicate that there is potential for continued evolution of senescence in these taxa because we observed nontrivial extant genetic variation for age-specific fitness and life span within gene pools. Moreover, at the macroevolutionary level, these species are not reproductively isolated, so one could argue that the variation throughout the species complex is available for further evolution in a single gene pool. However, because we only examined variation at the level of populations (and only three populations per species), we are unable to generate reliable estimates of the heritability of senescence.

A recent study by Lynch et al. (1998) proposes an alternative interpretation to standing genetic variation of life-history traits. By comparing the genetic variation of life-history traits in a natural population of *D. pulex* to that created by a laboratory mutation accumulation experiment, they concluded that much of the natural genetic variation may be due to continually arising, transient deleterious mutations. Our study differs by examining among-population variation (rather than within) and by emphasizing late-life traits, whose mutations will be less transient than the early-life traits studied by Lynch et al. (1998). Variation in late-life traits will be less transient than in early-life traits because selection is less effective at late ages. Furthermore, among our populations senescence trades off with early life history, a pattern that cannot be explained by uniformly deleterious mutations.

Prior tests of ETS have included laboratory selection on senescence, which give a detailed picture of senescence, and broad comparisons of many taxa, usually addressing only mean life span. Our report complements these tests by extending the understanding of senescence evolution to detailed life-history changes in the face of contrasting patterns of natural selection and by jointly addressing mortality and fecundity. We conclude that the pattern of senescence exhibited by *Daphnia pulex-pulicaria* is consistent with ETS. This result is limited, however, in that the selection pressures are historical, and thus cannot be directly quantified, and that our desire for thorough senescence data limited the number of populations we could study concurrently. It will be possible to expand our findings because there are many instances of *Daphnia* sister species inhabiting different parts of the habitat duration gradient. Our results additionally suggest a relationship among senescence, offspring investment, and juvenile fitness (specific growth rate). This raises the questions of what physiological traits proximately cause the large differences in life history, whether the different life-history traits are affected by the same physiological traits, and what ecological consequences emerge from that life-history variation. A finer understanding of genotype-by-environment interactions and of age-specific mortality risk in *Daphnia* are the next steps to making the link.

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