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Feedbacks between ecology and evolution: interactions between ΔN and Δp in a life-history model

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ABSTRACT

Questions: In a growing population, is there generation-by-generation feedback between population density, the strength of natural selection, and the rate of evolutionary change? What are the overall effects of natural selection and increasing population size on the total change in mean fitness?

Mathematical Methods: Numerical iterations of equations for Δp and ΔN, coupled with Frank and Slatkin's method for dissecting Fisher's fundamental theorem of natural selection.

Assumptions: Large density-regulated populations where genetic drift is minimal. Populations begin at the carrying capacity for homozygotes for one allele, but can increase to a higher carrying capacity as a beneficial life-history allele spreads.

Results: (1) Carrying capacity (K) increases as a beneficial allele spreads to fixation. (2) The increase in density increases the strength of selection as well as the additive genetic variance for fitness, leading to a more rapid spread of the favoured allele, which further increases the rate of population growth. (3) The negative change in mean fitness due to increasing population size is a time-lagged mirror image of the positive change in mean fitness due to natural selection.

Conclusion: During life-history evolution, generation-by-generation feedback can exist between population density (ecology) and allele-frequency change (evolution).

Keywords: eco-evolutionary feedback, fundamental theorem of natural selection, population ecology, theoretical ecology, theoretical population genetics.

INTRODUCTION

Theoretical population genetics forms the conceptual and mathematical framework for modern evolutionary thought (Provine, 1971; Michod, 1981). In particular, the one-locus, two-allele model for the spread of a beneficial allele under the action of natural selection is the launching point for many textbooks and courses on evolution. Similarly, the logistic model of population growth forms the launching point for theoretical ecology (Verhulst, 1838; Pearl and Reed, 1920). A formal fusion of the two disciplines began in the 1960s, with a one-locus, two-allele model that allowed genotype fitnesses to be density-dependent (MacArthur, 1962). The

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model showed that natural selection could lead to an increase in carrying capacity, and proved ecological analogues to Fisher's fundamental theorem of natural selection (Fisher, 1958). Similar theoretical results, using different approaches, were provided by Roughgarden (1971) and Charlesworth (1971). León and Charlesworth (1978) also allowed for density-dependent differences in fitness, and similarly showed that the spread of a beneficial allele could result in an increase in total density; they also showed that the change in density each generation (ΔN) is approximately equal to the additive genetic variance divided by $\partial \bar{w}/\partial \hat{N}$, where $\hat{N}$ is the equilibrium population size for a given allele frequency. Hence, changes in allele frequency, mean fitness, and total population size are all intimately related (Charlesworth, 1971; Roughgarden, 1971; León and Charlesworth, 1978).

These relationships suggest that ecological and evolutionary processes interact to the extent that they cannot be safely assumed to operate on different time scales (Lewontin, 2000). They also suggest that the intensity of selection can change over time as a direct function of changes in population density. My aim for the present study was to examine a simple case where the strength of selection increases monotonically over time during a period of population growth. The scenario is much as envisioned by Fisher (1958), where the spread of a beneficial allele results in a correlated deterioration of the environment because of increasing competition for resources as population density increases. Hence, the possibility exists for rapid feedbacks between numerical dynamics (ΔN) and gene-frequency changes (Δp), which are mediated through increases in the strength of selection. Following Gandon and Day (2009), I used the method provided by Frank and Slatkin (1992) to separate the conflicting effects on mean fitness generated by natural selection and environmental change. The results give an example of an intra-specific eco-evolutionary feedback, where changes in the environment and mean phenotype are functions of each other (reviews in Bailey *et al.*, 2009; Post and Palkovacs, 2009; Schoener, 2011).

MODEL

Consider a single locus in a diploid population where there are only two alleles, 1 and 2. Let the absolute fitness of the three possible genotypes be density-dependent, as follows:

$$\begin{aligned} W_{11} &= 1 - d_{11} + b_{11} - a_{11}N \\ W_{12} &= (1-h)(1 - d_{11} + b_{11} - a_{11}N) + h(1 - d_{22} + b_{22} - a_{22}N) \\ W_{22} &= 1 - d_{22} + b_{22} - a_{22}N, \end{aligned} \tag{1}$$

where W_{ij} is the absolute fitness of the ij^{th} genotype as its per capita growth rate (following Pielou, 1969); h is the dominance coefficient (Hartl and Clark, 1989); $1 - d_{ij}$ is the survivorship probability of the ij^{th} genotype; b_{ij} is the intrinsic number of offspring produced by the ij^{th} genotype; and a_{ij} is the sensitivity of the birth rate to total density (N) for the ij^{th} genotype. To simplify the equations, I assumed that the death rate is density-independent; but this assumption does not affect the conclusions. Allele 1 is stable to invasion by allele 2, if $W_{11} > W_{12}$, which requires that

$$(1 - d_{11} + b_{11} - a_{11}N) > (1 - d_{22} + b_{22} - a_{22}N). \tag{2}$$

If we assume that the population is at carrying capacity, $N = K_{11}$, upon introduction of the mutant allele 2, we get:

$$\frac{b_{11}-d_{11}}{a_{11}} > \frac{b_{22}-d_{22}}{a_{22}}. \tag{3}$$

The left-hand side of equation (3) is equal to K_{11}, and the right-hand side is equal to K_{22} (Pielou, 1969); thus allele 1 is stable to invasion by allele 2 if $K_{11} > K_{22}$. Similarly, the allele will spread when rare when introduced into a population of size K_{22} under the same condition as in equation (3). Thus allele 1 will spread when rare and go to fixation. This result is consistent with previous studies showing that selection can lead to a stable increase in carrying capacity (MacArthur, 1962; Roughgarden, 1971; León and Charlesworth, 1978), assuming the environment is constant (Lande *et al.*, 2009). Hence, mutations that increase the birth rate (b), decrease the death rate (d), or decrease the sensitivity to competition (a) will be favoured by selection, and lead to a higher carrying capacity, assuming the population is limited by competition for food rather than space. Hence it is possible that life-history evolution would result in periods of population growth. Here we want to know how such growth might affect the rate of spread of alleles that reduce the sensitivity to resource competition (a_{ij}) at the cost of also reducing the intrinsic birth rate (b_{ij}).

The fitnesses can be rewritten in the more standard form of theoretical population genetics, where w_{ij} gives the fitness of the ij^{th} genotype relative to genotype 11, where

$$\begin{aligned} w_{11} &= \frac{W_{11}}{W_{11}} = 1 \\ w_{12} &= 1 - hs \\ w_{22} &= 1 - s, \end{aligned} \tag{4}$$

and, in the present study, the selection coefficient, s, is

$$s = \frac{W_{11} - W_{22}}{W_{11}}. \tag{5}$$

The selection coefficient is normally treated as a constant (Lande *et al.*, 2009; Orr, 2010; Uecker and Hermisson, 2011), but here it can change as the population size changes. In fact, the selection coefficient is density-dependent even when the density-sensitivity coefficients are equal (i.e. $a_{11} = a_{22}$):

$$s = 1 - \frac{1 - d_{11} + b_{11} - a_{11}N}{1 - d_{22} + b_{22} - a_{22}N}, \tag{6}$$

where

$$\frac{\partial s}{\partial N} = \frac{a_{22}b_{11} - a_{11}b_{22}}{(b_{11} - a_{11}N)^2}. \tag{7}$$

Note that the expression in parentheses in (7) is the per capita birth rate for individuals that are homozygous for allele 1, and will be positive. Hence, assuming that the numerator in (7) is also positive, the selection coefficient (s) increases with increasing population size (N).

Changes in population size and allele frequencies are expected to be interrelated as follows (following Roughgarden, 1971; León and Charlesworth, 1978; Otto and Day, 2007):

$$\Delta p = p\left[\frac{pW_{11} + qW_{12}}{\bar{W}} - 1\right] = p\left[\frac{pw_{11} + qw_{12}}{\bar{w}} - 1\right], \tag{8}$$

$$\Delta N = (\bar{W} - 1)N. \tag{9}$$

The total change in mean fitness is given by:

$$\Delta \bar{W} = \Delta \bar{W}_{ns} + \Delta \bar{W}_{ec}, \tag{10}$$

where $\Delta \bar{W}_{ns}$ is the change in mean fitness due to natural selection, and $\Delta \bar{W}_{ec}$ is the change in fitness resulting from environmental change (Frank and Slatkin, 1992). Under Fisher's fundamental theorem of natural selection (Fisher, 1958; Michod, 2000),

$$\Delta \bar{W}_{ns} = \frac{V_A}{\bar{W}}, \tag{11}$$

where V_A is the additive genetic variance for fitness. In the present model, which assumes a single locus with two alleles,

$$V_A = 2pq[\alpha + \delta(q - p)]^2 \tag{12}$$

(Falconer and Mackay, 1996), where α is half the difference in fitness between the homozygotes,

$$\alpha = \frac{W_{11} - W_{22}}{2}, \tag{13}$$

and δ is the dominance deviation (Falconer and Mackay, 1996). (Note that I changed the variable names from Falconer and MacKay to avoid confusion with the life-history variables; specifically, I substituted α for a, and δ for d). It is easy to show that the dominance deviation, δ, is equal to $(1 - 2h)\alpha$. The variable α is normally treated as a constant, but here it changes as the difference in fitness between the homozygotes changes. Specifically, α changes with increases in the total population size as long as $a_{11} < a_{22}$. Substituting for α and δ, the additive genetic variance for fitness becomes:

$$V_A = 2pq[q - h(q - p)]^2(W_{11} - W_{22})^2, \tag{14}$$

which shows how the difference in fitness between the homozygotes contributes to the additive genetic variance for fitness. It also shows that the additive genetic variance is density-dependent, unless $a_{11} = a_{22}$:

$$V_A = 2pq[q - h(q - p)]^2[(d_{22} - d_{11}) + (b_{11} - b_{22}) - N(a_{11} - a_{22})]^2. \tag{15}$$

The solution for V_A can also be written as:

$$V_A = 2pq[q - h(q - p)]^2 s^2 W_{11}^2, \tag{16}$$

which shows how the additive genetic variation is related to the selection coefficient.

I used numerical iterations of a discrete-time model to study the relationships among Δp, ΔN, and $\Delta \bar{W}$. Following Frank and Slatkin (1992), I calculated

$$\Delta \bar{W}_{ns} = \bar{W}' \mid E - \bar{W} \mid E \tag{17}$$

$$\Delta \bar{W}_{ec} = \bar{W}' \mid E' - \bar{W}' \mid E. \tag{18}$$

Here the primes indicate fitness, or the environment, in the next time step (see also Gandon and Day, 2009). So, for example, $\bar{W}' \mid E$ gives fitness at time $t + 1$ given the environment at time t.

As shown by Price (1972), the fundamental theorem is based only on $\Delta\bar{W}_{ns}$, and not the total change in mean fitness, which is the sum of $\Delta\bar{W}_{ns} + \Delta\bar{W}_{ec}$. For the one-locus, two-allele model considered here,

$$\bar{W}' \mid E = (p')^2 W_{11} + 2p'q' W_{12} + (q')^2 W_{22} \tag{19}$$

$$\bar{W}' \mid E' = (p')^2 W'_{11} + 2p'q' W'_{12} + (q')^2 W'_{22} = \bar{W}' \tag{20}$$

$$\bar{W} \mid E = p^2 W_{11} + 2pq W_{12} + q^2 W_{22} = \bar{W}. \tag{21}$$

Substituting into equation (17) from equations (19) and (21), the change in mean fitness due to natural selection becomes

$$\Delta\bar{W}_{ns} = \frac{V_A[\bar{W} + p(W_{11} - W_{22}) + W_{22}]}{2\bar{W}^2}, \tag{22}$$

where V_A is estimated from the variance in breeding values (equation 12). Following standard one-locus, two-allele models in population genetics (Hartl and Clark, 1989), p' is the frequency of allele 1 in the next generation:

$$p' = \frac{p(pW_{11} + qW_{12})}{\bar{W}}. \tag{23}$$

For the purpose of providing an illustrative example, I set the following values for individuals homozygous for allele 2: $b_{22} = 3$, $d_{22} = 1$, and $a_{22} = 0.0002$. Thus the carrying capacity for a population composed of 22 homozygotes is $K_{22} = 10{,}000$. I initiated the population as being fixed for allele 2, with the total population size at K_{22}. I then introduced a single heterozygote into the population having genotype 12, where $b_{11} = 2.25$ (which is less than b_{22}), $d_{22} = d_{11} = 1$, and $a_{11} = 0.0001$ (which is less than a_{22}). Thus the mutation decreased the intrinsic birth rate, but also decreased the sensitivity to competition (Fig. 1). Because the carrying capacity of a population fixed for the mutant allele ($K_{11} = 12{,}500$) is greater than that for the wild type allele ($K_{22} = 10{,}000$), the mutation would be expected to increase when rare and spread to fixation (see Fig. 1).

I used deterministic numerical iterations of the recursion equations for both allele frequency dynamics (equation 23) and population growth (in a randomly mating population) from K_{22} to K_{11} over time (equation 9). I also tracked population mean fitness (equation 21), the selection coefficient (equation 5), the additive genetic variance (equation 14), and the change in mean fitness over time due to both natural selection (equation 17) and environmental change (equation 18). I then compared these results to the situation where the carrying capacity was fixed at K_{22}, and thus could not increase in size. I set the dominance coefficient (h) to 0.5, so that the effect of the mutant allele was additive. Under this assumption, equation (22) simplifies to become equation (11). I used deterministic iterations, rather than stochastic simulations, which eliminated the effect of drift. However, given the strong selection and large population sizes studied here, drift would be expected to be a relatively weak force ($s \gg 1/2N_e$) (Hartl and Clark, 1989). Finally, I used 'if/then' statements to prevent negative birth rates, as well as low intrinsic birth rates to prevent unstable population dynamics (May, 1976; Case, 2000).

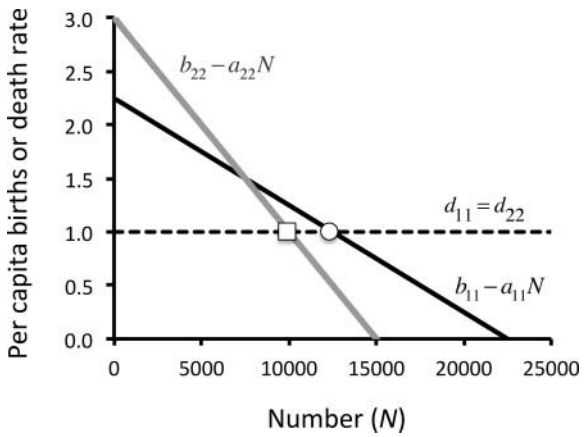

Fig. 1. Life-history trade-offs showing the per capita number of births and the probability of dying during each time step for two different life-history strategies, which are coded by a one-locus, two-allele genetic system. Homozygotes for allele 2 (shown as the grey line) produce more offspring in the absence of competition, but they also have a birth rate that is more sensitive to resource competition. Homozygotes for allele 1 (black line) produce fewer offspring in the absence of competition, but are less sensitive to competition; and they have a higher carrying capacity. For the parameter values underlying this example, allele 1 is expected to increase when rare and go to fixation in a strictly nutrient-limited population. The death rate (d_{ij}) (dashed black line) is here assumed to be density-independent and equal to unity for both genotypes, as would be the case for an annual population. The carrying capacity for 11 homozygotes is given by the open circle ($K_{11} = 12{,}500$), and the carrying capacity for 22 homozygotes is given by the open square ($K_{22} = 10{,}000$). Note that the difference in fitness between genotypes increases as the population moves from K_{22} to K_{11}. It is precisely this difference that generates the increase in both the additive genetic variance and the strength of selection as the population size increases, which underlies the feedbacks between ecological (density) and evolutionary (allele frequency) change. Parameter values: $a_{11} = 0.0001$; $a_{22} = 0.0002$; $b_{11} = 2.25$; $b_{22} = 3$; $h = 0.5$ (co-dominance).

RESULTS

As expected, the population size increased from K_{22} to K_{11} as allele 1 went to fixation (Fig. 2A). In addition, the selection coefficient (s) more than doubled from 0.20 to 0.50 as the population size increased, suggesting positive feedback between population growth and the rate of fixation of the beneficial allele (Fig. 2B). This increase in the selection coefficient was directly due to the increase in $W_{11} - W_{22}$ as the population density increased (Figs. 1 and 2B), which also increased the additive genetic variance for fitness (Fig. 3B). Hence the example shows how population growth can affect population/quantitative genetics, and vice versa.

To visualize the effect of population growth on the rate of fixation of the beneficial allele, I set the carrying capacity as fixed at K_{22} (which gives a fixed value for $s = 0.20$). In other words, the population could not increase in number, which might be expected if the population was limited by access to space rather than nutrients, such as in rocky intertidal communities (Connell, 1961). The results are shown in Fig. 3. Assuming the population is fixed at K_{22}, the beneficial allele increases in frequency over time, because it is associated with a higher birth rate at K_{22}; but the allele approaches fixation more slowly than when the

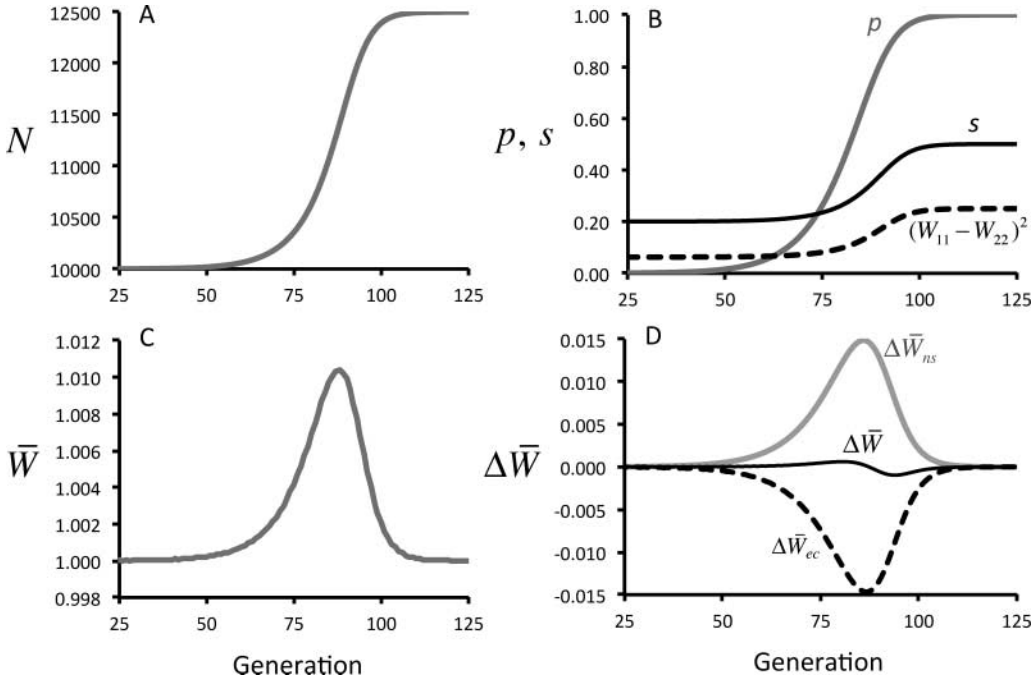

Fig. 2. Results of numerical iterations showing the relationships between N, $\bar{W}$, p, s, and $\Delta\bar{W}$. (A) Total population size (N). (B) The selection coefficient (s) (solid back line), the frequency of allele 1 (p) (grey line), and the squared difference in fitness between the homozygotes $(\bar{W}_{11} - \bar{W}_{22})^2$ (dashed line), which contributes to the additive genetic variance for fitness. (C) Mean fitness $\bar{W}$. (D) The total change in mean fitness $\Delta\bar{W}$ (solid black line), the change in mean fitness due to natural selection $\Delta\bar{W}_{ns}$ (grey line), and the change in mean fitness due to environmental change $\Delta\bar{W}_{ec}$ (dashed line). Here, the change in mean fitness due to natural selection ($\Delta\bar{W}_{ns}$) is exactly equal to $V_A/\bar{W}$, as suggested by Fisher (equation 11). Parameter values: $a_{11} = 0.0001$; $a_{22} = 0.0002$; $b_{11} = 2.25$; $b_{22} = 3$; $h = 0.5$ (co-dominance).

population increases in size (Fig. 3A). The additive genetic variance is also lower in the fixed population, and is eroded less quickly (Fig. 3B). The same result is also obtained for the change in mean fitness over time, as it is intimately related to the additive genetic variance for fitness (equation 22).

The results show how $\bar{W}$ changes over time when the carrying capacity is allowed to increase from K_{22} to K_{11}. As expected, $\bar{W}$ increases from unity, as the beneficial allele spreads in the population; but it then decreases as the population size converges on K_{11}, and the beneficial allele goes to fixation (Fig. 2C). The results also show how population density is related to the change in mean fitness due to natural selection versus the change due to environmental deterioration, where the deterioration results from greater competition for resources (Fig. 2D). During the initial spread of the beneficial allele, the change in mean fitness caused by natural selection exceeds the change in fitness due to increased competition; but as the allele goes to fixation, and the population converges on the higher carrying capacity, the change in fitness due to competition outweighs the change due to natural selection (Fig. 2D). Interestingly, the total change in mean fitness can be a small

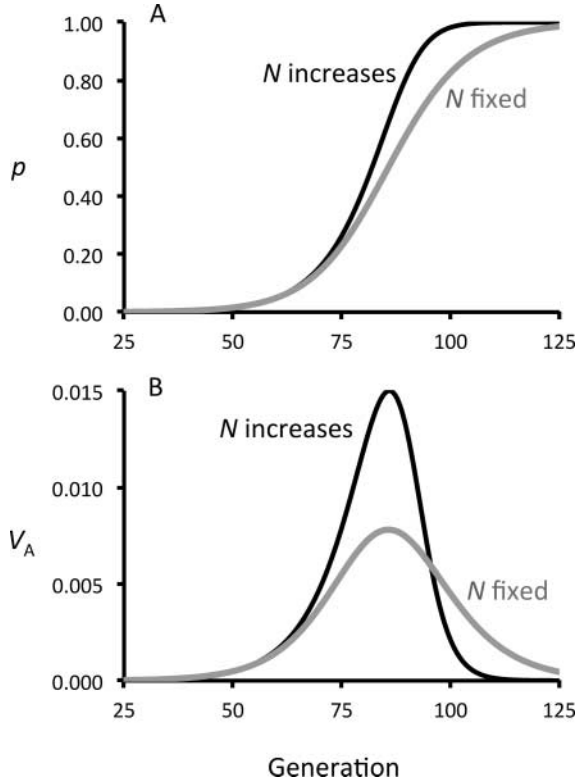

Fig. 3. Comparison of allele frequency and additive genetic variance over time for a fixed population size ($K = 10{,}000$) and a population that increases in size as allele 1 goes to fixation (from $K = 10{,}000$ to $K = 12{,}500$). (A) Allele frequency (p) over time. (B) Additive genetic variance (V_A) over time. The grey line indicates the results from iterations assuming a fixed population size.

proportion of the change in fitness due to natural selection (Fig. 2D), suggesting that ecological and evolutionary changes are occurring on the same time scale.

DISCUSSION

The goal of the present study was to examine the relationship between population growth and population genetics, including feedbacks between numerical dynamics and gene-frequency change. I used a one-locus, two-allele genetic system to examine the fate of an allele that decreased the intrinsic number of offspring produced (b), but also decreased the sensitivity of competition for resources (a), giving a clear case of K selection (Fig. 1). As expected, the allele spread when rare and went to fixation; the carrying capacity of the population also increased as shown previously (MacArthur, 1962). The gene-frequency dynamics closely mirrored the population dynamics during the spread of the allele (Fig. 2) (see also Otto and Day, 2007, figure 9.5).

Most interesting, however, is the positive feedback between population growth and gene-frequency dynamics. As the favoured allele spread, population size increased (Fig. 2A). This increase in the population size increased both the selection coefficient and the additive

genetic variance for fitness (Figs. 2B and 3B), which in turn increased the rate of spread of the favoured allele (Fig. 3A), thus completing the generation-by-generation positive feedbacks between population size (ecology) and gene-frequency change (evolution). The magnitude of the effect can be seen in Fig. 3A, which shows a faster approach to fixation of the favoured allele when the total population size is not fixed. The faster approach to fixation results from the monotonic increase in the strength of selection over time (Fig. 2B) and to greater additive genetic variance for fitness in the growing population (Fig. 3B). These results might be empirically evaluated by examining the spread of beneficial alleles in experimental evolution studies where population size is fixed versus populations in which evolution can lead to higher carrying capacities. Based on the present results, the prediction would be that the beneficial alleles (or perhaps clonal genotypes) would spread more rapidly in the unconstrained populations. Although not designed to be a direct test, a recent experimental study on clones of the Green Peach aphid clearly demonstrated that population density was positively associated with the rate of evolutionary change (Turcotte *et al.*, in press).

The results reported here rely heavily on the method derived by Frank and Slatkin (1992) for calculating the change in mean fitness due to natural selection and the change in mean fitness due to environmental change. And, as shown in a recent paper by Gandon and Day (2009), the change in fitness due to environmental change can be expanded to include multiple sources. They profitably used the method to study both mutational input and interactions between biological enemies as sources of environmental change. This is a particularly interesting situation, as antagonistic interactions can provide an ever-changing environment, and may lead to the maintenance of genetic variation in both species. In any case, as pointed out by Gandon and Day (2009), the method allows for the analysis of generation-by-generation feedback loops between natural selection and environmental change that could not be obtained by methods that employ a separation of ecological and evolutionary time scales.

The results of the present study are restricted to considering the environmental change that occurs during life-history evolution, but they nonetheless show that Fisher (1958) was correct to point out the correlated deterioration of the environment that stems from population growth. It is interesting to note that the change in mean fitness due to environmental deterioration begins almost immediately after introduction of the beneficial allele, and that it is a slightly time-lagged, mirror image of the change in mean fitness due to natural selection (Fig. 2D). The two effects tend to cancel each other, leading to a relatively small change in total mean fitness (Fig. 2D). These results then provide an illustration of Fisher's (1958, p. 51) statement that, 'Any net advantage gained by an organism will be conserved in the form of an increase in population, rather than in an increase in the average Malthusian parameter, which is kept by this adjustment always near zero.' Frank (2012) appropriately refers to this 'adjustment' as 'Fisher's conservation law for mean fitness'.

In summary, the results show generation-by-generation feedbacks between numerical change and gene-frequency change in a model of life-history evolution; they also add support to the growing field of eco-evolutionary dynamics, in which ecological and evolutionary changes operate on the same time scale and have a reciprocal influence on each other (review in Schoener, 2011).

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