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The adaptive advantage of phenotypic memory in changing environments

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SUMMARY

The adaptive value of carry-over effects, the persistence of induced phenotypes for several generations despite the change in the conditions that first induced these phenotypes, is studied in the framework of a simple model. Three different organismal strategies – non-inducible (genetic), completely inducible (plastic), and intermediate (carry-over) – are compared in fitness terms within three different environments. Analytical results and numerical simulations show that carry-over effects can have an advantage in stochastic environments even over organisms with high adaptive plasticity. We argue that carry-over effects represent an adaptive mechanism on the ecological timescale that fills the gap between short-term individual adaptations and long-term evolutionary adaptations. An extension of the concept of plasticity to incorporate the time dimension and include the stability of induced phenotypes through both clonal and sexual generations, is suggested.

1. INTRODUCTION

Carry-over effects (the lingering of a particular phenotype for one or more generations despite a change in the environmental conditions which first induced this phenotype) is very common. Some of the best known cases of environmentally induced carry-over effects are when the maternal environment affects the phenotypes of the progeny (Roach & Wulff 1987; Rossiter 1991). The effects can last for only one generation or they can be more persistent (see, for example, Miao *et al.* 1991*a*, *b*). Environmental maternal effects are, however, only a special case of carry-over effects; other types of carry-over effect involve paternal effects and effects that do not depend on parental sex and are found in organisms that reproduce asexually. Plant ecologists have often noted that when plants from different environments are transplanted to common garden conditions, carry-over effects are frequent and there is uncertainty about 'the amount and persistence of variation that is carried over from the field environment to the garden' (Evans & Turkington 1988 p. 374). Carry-over effects that lingered for several sexual generations have also been reported in various cultivated plants (Highkin 1958*a*, *b*; Moss & Mulett 1982). Went (1959) demonstrated carry-over for two generations in potatoes propagated by tuber cuttings: Jollos (1921) described lingering modifications in *Paramecium*; Shaposhnikov found carry-over effects lasting for many generations in aphids (Shaposhnikov 1987); and similar observations have been made in other taxa (for reviews see Jablonka *et al.* 1992; Jablonka & Lamb 1995). In some cases the phenotypic change has been correlated with the extent of DNA methylation. For example, Sano *et al.* (1990) induced dwarfism in rice by treating germinating seedlings with the demethylating agent 5-azacytidine. The effect of the drug on both the size of the plants and the reduced level of methylation in the genome persisted for the three generations studied. A similar phenomenon in which a persistent visible phenotypic change and a reduction of DNA methylation were induced by 5-azacytidine, was found in a stable variety of Triticale (Heslop-Harrison 1990). Several different mechanisms may underlie the persistence of induced phenotypes over several generations.

1. Carry-over effects may depend on the physical transmission of material which is diluted with time (e.g. mineral composition).

2. Carry-over effects may depend on the same mechanisms as those that underlie cellular memory i.e. epigenetic inheritance systems (EISS) (Jablonka *et al.* 1992). EISS may underlie the phase variations in plants described by Brink (Brink 1962), inducible phase variations in uropathogenic *E. coli* (Van de Woude *et al.* 1992) and probably also the inducible phase transitions in the infectious yeast *Candida albicans* (Soll *et al.* 1993).

3. Carry-over effects may depend on changes in DNA sequence. Such changes can be the result of

environmentally induced, high-rate changes in DNA sequence as in the famous case of Flax genotrophs (Cullis 1984) and some cases of induced phase variations in microorganism (Robertson & Meyer 1992; Moxon *et al.* 1994). A contributing factor in organisms with somatic or late segregation of germ-line and soma may be somatic selection. Even classical, (low-rate) somatic mutations coupled with intense somatic selection in tissues which can contribute to the germ lineage can bring about rapid changes at the phenotypic organismal level.

4. Behavioural carry-over effects depending on the transmission of behavioural phenotypes through social learning, as in birds and mammals (Galef 1988; Avital & Jablonka 1994).

With the exception of maternal effects, carry-over effects seem to have been ignored by evolutionists, probably because it is assumed that they are accidental by-products of the organism's physiology and have not themselves been moulded by natural selection. An additional reason for their neglect may be that they make the distinction between the genotypic and the environmental components of variance difficult to disentangle, and are therefore treated as something of a nuisance in experiments involving transfer or transplantation between environments.

In this paper we show, by means of mathematical models and computer simulations, that carry-over effects can be advantageous in some environmental conditions and that the extent of persistence of a phenotype for a number of generations can be positively selected. Our models explore the effects of different types of environments on the performance of organisms with different types of response to the environment: non-inducible (genetic), completely inducible (plastic), and intermediate (carry-over). The probability that a phenotype will persist for a certain number of generations will be referred to as the 'phenotypic memory'. A phenotype can change spontaneously, or as examined here, by induction. We study the case when environmental factors both induce particular phenotypes as well as selecting these phenotypes. On the basis of our models we argue that carry-over effects are an important and advantageous strategy in stochastic environmental conditions, and that the consideration of these effects may have important ecological implications.

2. MODEL

We use a Monte Carlo simulation model for evaluating population growth rate for types with different extents of phenotypic memory. We assume that asexually reproducing, semelparous organisms live in an environment that may alternate between two discrete states; E1 and E2. The two types of environments both induce and select the corresponding discrete phenotypes (P1 and P2). We assume that P1 performs better in E1 (where its fitness is W_{good}) than in E2 (where its fitness is W_{bad}), and P2 performs better in E2 (where its fitness is W_{good}) than in E1 (where its fitness is W_{bad}). The change from E1 to E2 and vice versa can be (A) 'random' (i.e. the change from E1 to E2 and vice versa at every time step occurs with a probability $p = 0.5$); (B) 'temporally patchy' (i.e. a change from one environmental state to another at every time step occurs with a probability (p) different from 0.5 and smaller than 1.0); or (C) 'strictly periodic' with period length $2n$, so that the environment is E1 for n time-steps, and E2 for n time steps. The process of environmental changes in the random and temporally patchy environments is assumed to be a Markovian process.

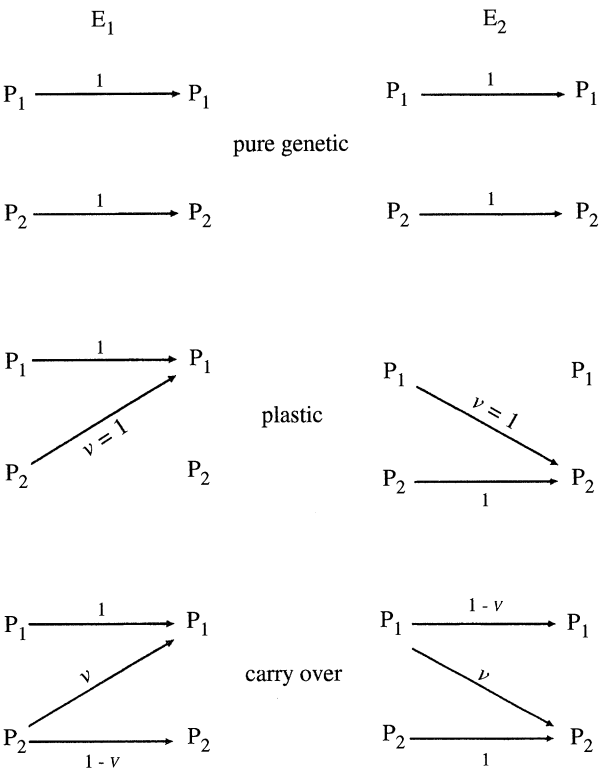


Figure 1. Three types of response to changing environments. Pure Genetic, Plastic, and Carry-over denote the purely genetic, plastic and carry-over strategies respectively. P1 and P2 are the two alternative phenotypes. For the 'pure' G strategy $\mu = 10^{-5}$, which is assumed for all other strategies. This low background mutation rate is not shown in the figure; v is the induction coefficient.

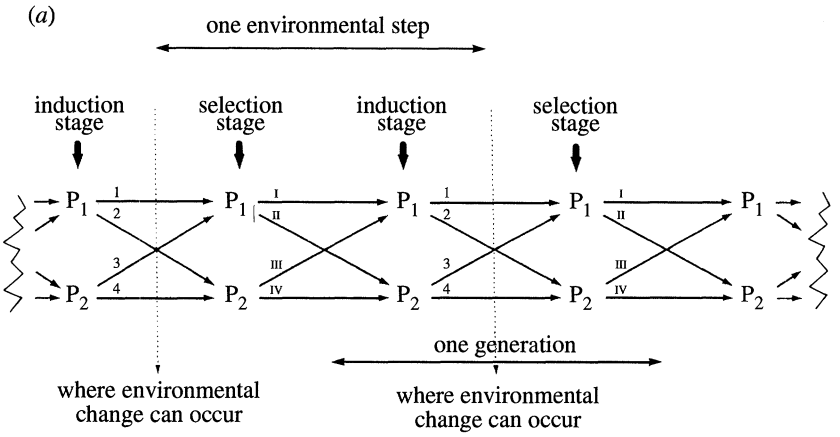
The environment can both induce and select the phenotypes; in the model we assume that there is always a time lag between induction and selection, and in the numerical simulation we also assume (for the sake of realism) that there are always spontaneous (non-induced) background mutations at a rate of 10^{-5} in both directions. Such a small mutation rate has, however, a negligible effect on our simulation results and analytical results.

We define three types of individuals with different strategies (see figure 1).

1. G – the 'genetic' strategy – the change from P1 to P2 and vice versa occurs only by mutations (we assume that $\mu_1 = \mu_2$ i.e. forward and back mutation rates are equal). When referring to a 'pure' G strategy we assume that the mutation rate is negligible i.e. mutations occur at a rate of 10^{-5} or lower.

2. P – the 'plastic' strategy – the change from P1 to P2 is induced by the environmental conditions. The whole population changes with probability 1.0 to the

(*a*)



(*b*)

arrow	in E_1	in E_2
1	1	$(1 - v)$
2	0	v
3	v	0
4	$(1 - v)$	1
I	$W_g (1 - \mu)$	$W_b (1 - \mu)$
II	$W_g \mu$	$W_b \mu$
III	$W_b \mu$	$W_g \mu$
IV	$W_b (1 - \mu)$	$W_g (1 - \mu)$

Figure 2. The model: P1, P2 are alternative phenotypes; W_{good}, W_{bad} denote fitnesses in matching and non-matching environments; μ denotes mutation rate; v denotes induction coefficient. The table shows the transition probabilities in E1 and E2, taking into account the fitnesses and the induction and mutation rates.

environmentally matching phenotype (the induction coefficients, $v_1 = v_2 = 1.0$). As León (1993) has noted the best possible strategy for any organism is to be infinitely plastic, so that induction is complete and there is no time lag. However, there is always a lag between an inducing environmental stimulus and its selective consequences. We incorporate the time lag into the models by making induction precede selection so that induction and selection occur at different phases of the life cycle of the organism. This is equivalent to ensuring the selective effects of induction occur after the inducing trigger. It is essential that the environment may change during this time lag.

3. *C* – the carry-over strategy – the organism transmits a phenotype to its progeny for several generations after the environmental change. The phenotypic memory may be long or short: a long memory means that it is difficult to induce a change in phenotype when the environment switches (the induction coefficient v, is small); a short memory means that the phenotype is expected to persist for only a few generations (the induction coefficient v, is high) and the phenotype is readily switched when the environment changes. In general, with carry-over effects, the induction coefficient v is smaller than 1.0 but larger than 0 ($0 < v_1, v_2 < 1.0$). The 'plastic' and the 'genetic' strategies are the limiting cases of the carry-over strategy, when the induction coefficients are 1.0 and 0, respectively. Phenotypic memory is defined in this system as $1 - v$. We also consider hedge-betting, a strategy in which random phenotypic switching can occur.

The population growth rate r for the different types, is computed, and depends on v (the induction coefficient), $s = W_{good}/W_{bad}$ (the selection coefficient), and the nature of the environmental sequence. We normalize fitness making the selection coefficient positive (so that $W_{bad} = 1.0$ and $W_{good} > 1.0$) to show a positive growth-rate. Population census at each generation is made after the induction phase but before the adaptive effects of induction are manifest. The general model is presented in figure 2.

The simulation software is written in Turbo Pascal for PC. We start with equal numbers of the two phenotypes: P1 = P2 = 500. The induction/selection process goes on for 1000 time-steps, and 100 independent runs with different environmental sequences are made for each parameter combination.

The population growth rate is calculated as

$$r = 1/T\{E[\log (X_t/X_0)]\},$$

i.e. the average, based on 100 runs, of the log of the growth rate over 1000 time-steps. $T = 1000$, is the number of time steps, X_T is the population size at time T, and X_0 is the population size at time 0. $E[\ ]$ denotes the average over our sample of 100 runs.

3. RESULTS

(*a*) *Random environment*

In a random environment any carry-over strategy (any value of $0 < v < 1$) is clearly superior to both the pure G and P strategies. The pure G and P strategies have the same growth rate: $(\log W_{good} + \log W_{bad})/2$ (see Appendix 1*a*). The maximal growth rate log $(W_{good} + W_{bad})/2$ is achieved for a hedge-betting (or mixed) strategy when the probability of producing each phenotype is 0.5 (Appendix 1*a*). Figure 3*a* shows the growth rate as a function of the induction coefficient v when the value of the selection coefficient is $s = 1.3$. The numerical simulation indicate that the growth rate increases steeply for very small values of the induction coefficient, and reaches a maximum at $v = 0.08$. This means that relatively long memory is advantageous under these conditions compared to alternative strategies. The same unimodal shape of the growth rate curve is achieved for all selection pressures. Providing the selection coefficient is not too small (i.e. $s > 1.1$), the majority of random environmental sequences leads to a unimodal curve. However, when s is very small, the large variance masks the curve unless a very large sample of different environmental sequences is averaged (see Appendix 1). The numerical results suggest that variance is minimal for a particular value of v which is often close to the optimal v, and it is largest for the G and P strategies. The possible significance of this result will be considered in the discussion.

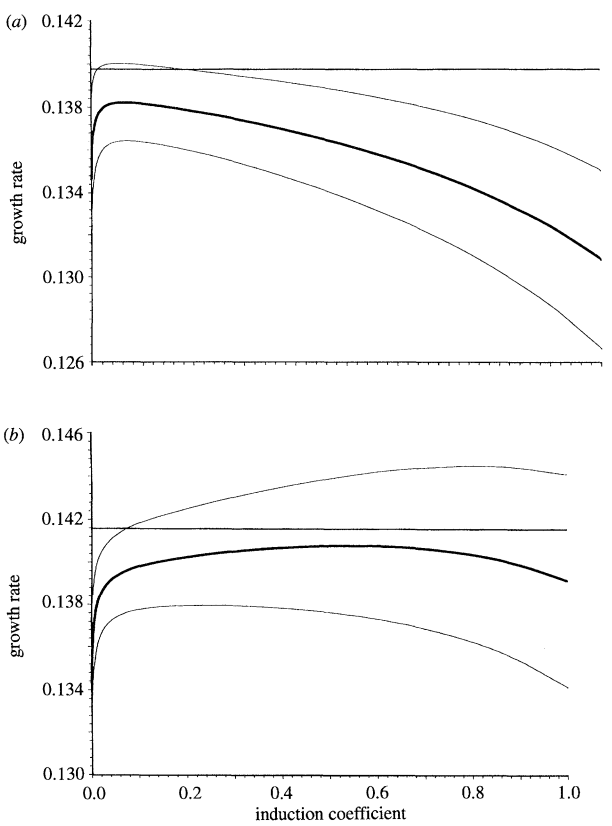


Figure 3. Growth rate as a function of the induction coefficient v for selection coefficient 1.3, and mutation rate of 10^{-5} in completely random and temporally fine-grained environments. The thicker curve is the average growth rate, the two thin curves represent one standard deviation above and below the average. The upper horizontal line gives the upper estimate of the growth rate given by equations A 3 in Appendix 1. (*a*) In a completely random environment ($p = 0.5$); (*b*) in a temporally patchy environment ($p = 0.47$).

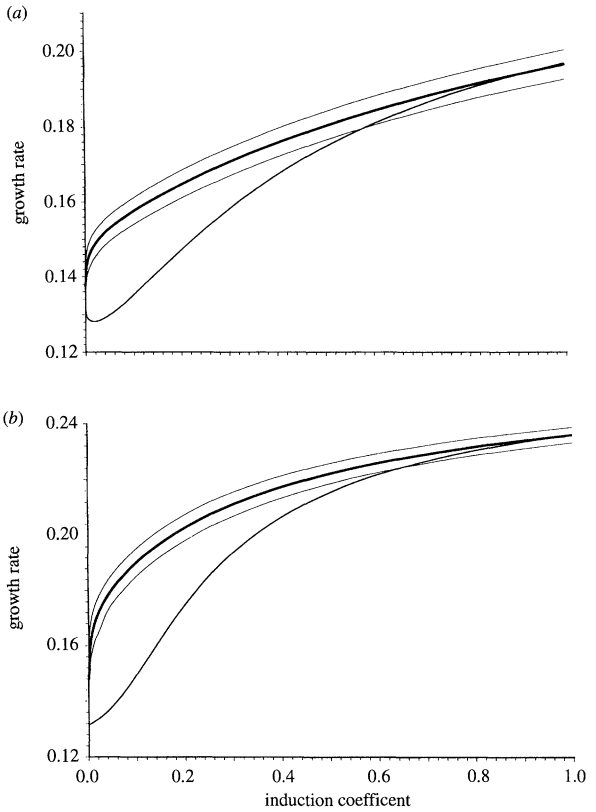


Figure 4. Growth rate as a function of the induction coefficient v, for a selection coefficeint 1.3, and mutation rate 10^{-5} in temporally coarse-grained and periodical environments. The upper thicker curve is the average growth rate in the temporally patchy environment; the two thin curves surrounding it represent one standard deviation above and below the average in a temporally patchy environment: (*a*) $p = 0.25$, (*b*) $p = 0.10$. The lower curves represent the growth rate in the corresponding strictly periodic environment ($n = 1/p = 4$ and 10 respectively).

(*b*) *Temporally patchy environment*

When the environment is temporally patchy, that is the probability of environmental changes is different from 0.5 but smaller than 1.0, the fitness of the P strategy is given by the weighted average $(1-p)\log W_{good} + p \log W_{bad}$. The value of the G strategy remains the same as in the random environment. The advantage of having phenotypic memory depends on the value of p, that is on the nature of the temporally patchy environment. When p is close to 0.5, that is if the transitions between environments is close to random and the environment is temporally 'fine grained', the shape of the curve remains unimodal and having memory is still advantageous (see figure 3*b*). When p is under or above a certain threshold the curve ascends monotonically, and the P or G strategy strategies are optimal (Appendix 1*b*). Figure 4*a*, *b* shows the growth rate in a temporally patchy environment for $p = 0.25$ and 0.1 respectively. The optimal growth rate in a fine-grained, temporally

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patchy environment (for $p > W_{bad}/(W_{good} + W_{bad})$) is conditional hedge-betting strategy, producing P1 and P2 offspring in a ratio depending on the current environmental state and correlated with the value of p and s (see Appendix 1, equation A9 for the precise formula).

(*c*) *Strictly periodic environment*

In a periodic environment the P strategy always has an advantage over any other strategy, with the exception of $n = 1$ and $n = 2$. When the period length is 1, that is the environment changes every time step, the P individuals are at a constant disadvantage as they are always adapting to an environment that has already changed. The growth rate decreases monotonously with v. When $n = 2$, the P and G strategies result in the same growth rate while C individuals have a clear disadvantage. The disadvantage of large memory is still apparent for small values of n, e.g. $n = 4$ in figure 4*a* (see Appendix 1 for the precise conditions leading to the disadvantage of memory). For larger values of n there is a monotonic increase in growth rate with increasing values of v, reaching a maximum at $v = 1$ (see Appendix 1*c*). It is interesting to compare the periodic environment with the corresponding temporally patchy environment, with $p = 1/n$ (the expected number of time-steps between environmental changes equals $1/p$). This comparison shows a definite selective advantage for the carry-over strategy in temporally patchy environments compared to the corresponding strictly periodic ones (see figures 4*a*, *b*).

4. DISCUSSION

If induced functional or structural states are transmitted to the next generation, there is usually a conflict between phenotypic memory and phenotypic plasticity. The consequences of this conflict have not been explored by evolutionary biologists because the ability of some organisms to transmit phenotypes over several generations has been almost completely ignored.

One situation in which it is intuitively clear that carry-over effects are of evolutionary importance is when current environmental cues enable an organism or a lineage to anticipate a future environmental change. Moran (1992) has shown that when there are periodical, predictable environmental fluctuations and the anticipatory cues are fairly reliable, natural selection may lead to the evolution of alternative ontogenetic programmes within the life cycle of the organism (polyphenism). However, if rather than looking at alternative developmental pathways within a single generation we consider a lineage of organisms, memory can be advantageous even when the anticipatory cue is not very reliable (see Appendix 2). In these conditions it is always advantageous to have memory because then there is no conflict between memory and plasticity.

What is surprising is that in the very common situation in which the environment both induces and selects phenotypes continuously (so that there is a conflict between phenotypic memory and phenotypic plasticity) memory can be advantageous. As our models show, it is beneficial to have memory or phenotypic persistence in a randomly varying environment (see figure 3*a*), and when the temporally patchy environment is fine-grained (see figure 3*b*). Indeed, in a random or nearly random environment, maximal growth rate is achieved when memory is relatively long (see figure 3*a*). The optimal strategy in a completely random environment changing between two states is to have a mixed, hedge-betting strategy with the probability of producing each phenotype being 0.5 (Levins 1962). This is the same as having a random mutation rate of 0.5, or exploiting developmental noise to produce alternative phenotypes with a 50% probability. Cooper & Kaplan (1982) refer to the production of two alternative phenotypes as 'coin flipping' or a 'mixed strategy'. Any switching of phenotype, any mixed strategy, is better than pure G or P in random environments. Indeed, as Cooper & Kaplan (1982) have shown, the precise value of the mixing frequency is much less important than the existence of some mixing. The carry-over strategy is a way of creating a mixed strategy. The advantages that the C strategy confers over the pure G strategy is hardly surprising because even minimal switching confers a greater advantage than genetically fixed responses. What is surprising, however, is that the C strategy confers a superior advantage to the P strategy; this is due to the lag between induction and selection. Although the completely random hedge-betting is the optimal mixed strategy in completely random environment (see Appendix 1*a*), the difference between this optimum and the best carry-over strategy is rather small for small s (see figure 3*a*). The same is true for the conditional hedge-betting strategy which is the optimal strategy for fine-grained, temporally patchy environment (see figure 3*b*, Appendix 1*b*). Hedge-betting and carry-over are both good ways of coping with random or nearly random environments. Under these conditions, it is likely that one of these strategies will evolve: which strategy that is, is dependent on the initial genetic and environmental conditions and the constraints imposed on the organism.

The advantage of having long memory in a random environment may be due to the reduction of the variance of the growth rate in long-term fitness. The advantage of having a memory in a random environment can be compared to the advantage of perennial life-form compared to the annual life-form in plants in some conditions. In a randomly changing environment, long-lived perennial plants have a smaller variance in fitness relative to annual plants because seed yield is averaged over many years (Cohen 1993). In a similar way, types with memory can be thought of as having a life span of several 'generations' (when the 'life span' is determined by the length of memory); in consequence, the long-term average fitness of the lineage (delimited by its memory span and not by conventional generations) has a smaller variance. This averaging effect of memory may also explain the reduction in variance found in our simulations for a long memory in the random environment.

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Although memory is advantageous in random and temporally fine-grained environments, and is superior to both the P and G strategies, our analytical results and numerical simulations show that it can sometimes be disadvantageous, even when compared to a pure G strategy. This is the case when the environment is strictly periodic with small period length. In such a deterministic environment any memory is bad, with (for each environment type) one particular value representing the worst possible strategy. This is because the induced phenotypic state lingers long after an environmental change so that the organism is adapted to the changing environment for only a small proportion of the time. The P strategy is superior in a periodically changing environment with $n > 2$, and in an environment that is temporally 'coarse-grained'. In these conditions the plastic organism is adapted to the prevalent environmental conditions for most of the time.

The models and simulations show that in some circumstances phenotypic memory may be selected. What are the evolutionary implications of this? One obvious conclusion is that the phenotype of the organism in a new environment, as for example after a disturbance, should reflect not only its present environment and its genotypic potential, but also the recent ecological history of its lineage. The finding (Turkington & Harper 1979; Evans & Turkington 1988) that when transplanted, the fitness of the clonal white clover *Trifolium repens* is higher when the neighbouring species are those with which it was associated in its recent ecological history, is consistent with this conclusion. The greater fitness in this case is probably the result of carry-over effects rather than genetic differences between conspecifics (Evans & Turkington 1988). Even if phenotypic memory is relatively short, lasting only few generations, it could affect the patterns of association between species in a newly established community. Past ecological associations may be reproduced.

A further implication of inducible carry-over effects is that the phenotypic memories of organisms will tend to evolve to be co-adapted. The nature and the length of phenotypic memory will be influenced by that of associated species. It is not only the genetic effects of one species on another that have to be considered, but also the inherited epigenetic effects (Molnár 1990).

Carry-over effects of various types extend our understanding of the concept of adaptation. They fill the gap between the ontogenetic or physiological adaptations that underlie individual plasticity, and the classical genetic changes associated with long-term evolutionary adaptations. They may underlie the frequently observed rapid adaptation that takes place within a few generations of a change in environment.

Even when the phenotypic variations are random rather than induced, if there is phenotypic memory, medium-term adaptation is possible. Leigh (1970) has shown that when organisms live in a selecting non-inducing environment that periodically alternates between two states every n generations the best rate at which to switch phenotypes is $1/n$. However, when n is small ($n = 10$–100), it is unlikely that the switch will be a classical genetic mutation, because rates of classical mutations are usually much lower. But as Lachmann-Tarkhanov & Jablonka (1995) have argued, if non-DNA inheritance systems such as the epigenetic systems underlying cellular memory are considered, these high transition rates have a clear biological meaning. Random epigenetic-variations, like carry-over effects, enable medium-term adaptations.

Evolutionary success involves a whole spectrum of adaptations, from short-term individual adaptations through medium-term adaptations that last only a few generations, to long-term highly stable adaptations. Induced effects are responsible for part of this continuum: for both individual adaptation and for carry-over effects. It may therefore be useful to extend the concept of phenotypic plasticity to incorporate the time dimension. Perhaps the concept should include not only the range of induced phenotypes that a genotype can produce, but also the stability of these phenotypes through both clonal and sexual generations.

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APPENDIX 1

(*a*) *Random Environment*

Let x_1, x_2 denote the population sizes of the two phenotypes P_1 and P_2 and w_g and w_b the good and the bad fitness. In environment 1, where P_1 is well adapted, we have

$$x_1' = (1-\mu)\, w_g\, x_1 + v w_b\, x_2$$
$$x_2' = \mu w_g\, x_1 + (1-v)\, w_b\, x_2 \qquad \text{(A 1)}$$

and correspondingly reversed in environment 2. In (A 1) μ denotes the mutation rate and v the combined rate of transitions (both random and induced) from the 'bad' to the 'good' phenotype. When mutation rate is small (e.g. 10^{-5}), v essentially equals the induction rate.

(A 1) can be expressed more concisely with the two matrices

$$A = \begin{pmatrix} a & b \\ c & d \end{pmatrix} = \begin{pmatrix} (1-\mu)\, w_g & v w_b \\ \mu w_g & (1-v)\, w_b \end{pmatrix} \quad B = \begin{pmatrix} d & c \\ b & a \end{pmatrix}.$$

The 'growth rate' (or 'dominant Ljapunov exponent') in a random environment is then defined as

$$r = \lim_{T\to\infty} r_T, \quad \text{with} \quad r_T = \frac{1}{T}\mathbb{E}\left[\log \|A_T \cdots A_2 A_1\|\right].$$

Here $\mathbb{E}$ means the average over all 2^T possible sequences of length T. Each A_i is either A or B. $\|.\|$ stands for any matrix norm e.g. the sum of (the absolute values of) all entries will work. It is known (see Furstenberg & Kesten 1960) that for almost all random sequences

$$r = \lim_{T\to\infty} (1/T) \log \|A_T \cdots A_2 A_1\|.$$

If the entries of A, B are positive, then a central limit theorem holds for r_T. Actually, the log of any entry of the random matrix product can be approximated by a normal distribution with mean rT and variance $\sigma^2 T$, for a certain constant σ.

An excellent review is Tuljapurkar (1990).

Let $r = r(w_g, w_b, v, \mu)$. We can show the following.

1. $r(w_g, w_b, 0, 0) = (\log w_g + \log w_b)/2$ for the genetic strategy. This follows from the law of large numbers, as each phenotype will spend approximately half the time in the good and half the time in the bad environment, as $T \to \infty$. More precisely:

$$r_T = \frac{1}{T2^T} \sum_{k=0}^{T} \binom{T}{k} \log\left(w_g^k\, w_b^{T-k} + w_b^k\, w_g^{T-k}\right)$$
$$= (\log w_g + \log w_b)/2 + [(1/2\pi T)^{1/2} + O(1/T)] \log (w_g/w_b). \qquad \text{(A 2)}$$

Therefore r_T converges rather slowly from above to its limit r. (A 2) explains the difference and the overestimate of the numerical simulations compared to the correct growth rate r. In the case $v = \mu = 0$, the

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distribution of r_T approaches a halfsided normal distribution, rather than a normal distribution as it does in the other cases when $v, \mu > 0$. For $w_g = 1.3$, $w_b = 1$ we have $r_{1000} \approx 0.1345$, which agrees well with the simulations shown in figure 3*a*, whereas the precise value is $r = 0.13118$.

2. $r(w_g, w_b, 1, 0) = (\log w_g + \log w_b)/2$ for the plastic strategy.

This is a special case of the corresponding result for temporally patchy environments discussed in the next section. In this case, $r_T - r$ is of order $1/T$ hence there is much quicker convergence and the numerical results give a more reliable estimate for r. r_T is asymptotically normally distributed, the standard deviation σ_T satisfies in this case $\sigma_T / r_T = T^{-1/2}$, independently of the selection parameters w_g, w_b. This again agrees well with the numerical results (see figure 3*a*).

3. An upper estimate:

$$r(w_g, w_b, v, \mu) \leqslant \log (w_g + w_b)/2. \tag{A 3}$$

To prove (A 3), we note from (A 1), that the total population size $x_1 + x_2$ changes after one time step into either $w_g x_1 + w_b x_2$ or $w_b x_2$ or $w_b x_1 + w_g x_2$, each with probability 1/2 (independently of the value of v, μ). If we assume w.l.o.g. $x_1 + x_2 = 1$, then the expected growth rate for one generation is given by:

$$\begin{aligned} g(x_1) &= 1/2\,[\log (w_g x_1 + w_b x_2) + \log (w_b x_1 + w_g x_2)] \\ &\leqslant \log [(x_1 + x_2)\,(w_g + w_b)/2] = \log [(w_g + w_b)/2] \end{aligned} \tag{A 4}$$

because log is a concave function. As this holds for any value of x_1, x_2, this estimate holds also for the long term growth rate r in (A 3), independently of μ and v.

This estimate is also related to the well-known fact, that the growth rate of the average population size is larger (or at most equal) to the average growth rate r of the population (see Lewontin & Cohen 1969; Tuljapurkar 1990, p. 43).

The upper bound (A 3) is important because it holds independently of the induction and mutation rates. It could be achieved only by using a bet-hedging strategy i.e. resetting $x_1 = x_2$ at every time step.

For $w_g = 1.3$, $w_b = 1$ we obtain the estimate $r < 0.13976$ (given by the upper dashed line in figure 3*a*), whereas the optimal induction rate yields a value of $r = 0.1382$.

(A 3) also gives an estimate as to how much r can be increased at all by mutation, induction, or any other mixed strategy, as compared to the pure genetic or pure plastic strategy: If $w_g/w_b = 1 + \epsilon$ (ϵ small), then the maximal 'relative gain in average growth rate', $(r_{\max} - r)/r$, is just $\epsilon/4$ (as can be easily seen from a Taylor expansion).

4. The lower estimate:

$$r(w_g, w_b, v, \mu) \geqslant (\log w_g + \log w_b)/2. \tag{A 5}$$

This means that for any of $\mu, v (\neq 0, 1)$, the carry-over strategy has a higher growth rate than the pure genetic or plastic strategy. This is again világos from (A 4): the one step average growth rate g is a concave function of x_1 (if we set $x_2 = 1 - x_1$) and hence attains its minimum value at the boundary, i.e. for $x_1 = 0$, or $x_1 = 1$, where it takes the value of the genetic or plastic strategy. The inequality is strict, whenever $0 < \mu, v < 1$.

5. One may represent r also in the following way:

$$r = \int_0^1 \phi(x)\, g(x)\, \mathrm{d}x$$

with g as in (A 4) and $\phi(x)$ denoting the invariant distribution for the relative frequency $x = x/(x_2 + x_2)$ of the first phenotype, resulting from the stochastic process. This invariant distribution ϕ is characterized by a functional equation of the form

$$\phi(x) = 1/2\{\phi[a_1(x)]\, a_1'(x) + \phi[a_2(x)]\, a_2'(x)\}.$$

The functions $a_i(x)$ are rational-linear functions, which can be determined from the matrices A, B. However, no explicit expression for this invariant distribution ϕ is known so far.

(*b*) *Temporally Patchy Environments*

Here we assume that the environmental changes follow a Markov process (with two states). The probability for a change to the other state is p, while with probability $1 - p$ it will stay the same. The concepts of the previous section apply here after appropriate modifications. The growth rate $r(p; w_g, w_b, v, \mu)$ satisfies then: $r(p; w_g, w_b, 0, 0) = \log (w_g + \log w_b)/2$, as before; and $r(p; w_g, w_b, 1, 0) = (1 - p) \log w_g + p \log w_b$ for the plastic strategy.

Although this follows easily from (A 8) below, we sketch a different argument here that also allows to compute the variance.

The probability for exactly k changes ($0 \leqslant k \leqslant T - 1$) to ocur in an environmental 0–1-sequence of length T is given by $\frac{1}{2^{T-1}} \binom{T-1}{k} p^k (1-p)^{T-k-1}$. So this follows a binomial distribution $B(T-1, p)$. The norm of the corresponding matrix product is approximately $w_g^{T-k} w_b^k$. Hence:

$$\begin{aligned} r_T &= \log w_g + \frac{1}{T} \frac{1}{2^{T-1}} \sum_{k=0}^{T-1} \binom{T-1}{k} p^k (1-p)^{T-k-1} k \log \frac{w_b}{w_g} \\ &= \log w_g + [1 - (1/T)]\, p \log (w_b/w_g) \\ &= (1-p) \log w_g + p \log w_b + O(1/T). \end{aligned} \tag{A 6}$$

For the standard deviation we obtain

$$\begin{aligned} \sigma_T &= \sqrt{[(T-1)\, p(1-p)]} \log (w_g/w_b)/T \\ &\approx (p(1-p)/T)^{1/2} \log (w_g/w_b), \end{aligned}$$

or, if $w_b = 1$, $\sigma_T / r_T \approx \sqrt{[p/(1-p)\, T]}$.

3. An upper estimate:

$$\begin{aligned} r(p; w_g, w_b, v, \mu) &< \min \{\log [(w_g + w_b)\, p^p (1-p)^{1-p}], \\ &\log w_g\} \\ &= \min \{\log (w_g + w_b) + p \log p + (1-p) \log (1-p), \\ &\log w_g\}. \end{aligned} \tag{A 7}$$

The proof of (A 7) runs similar to that of (A 3). Suppose the present state is (E_1, x_1, x_2). Then, from (A 1), the total population size $x_1 + x_2$ changes after one time step into either $w_g x_1 + w_b x_2$ or $w_b x_1 + w_g x_2$, now with probability $1 - p$ and p, respectively

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(independently of the value of v, μ). If we assume w.l.o.g. $x_1 + x_2 = 1$, then the expected growth rate for one generation is given by:

$$g(x_1) = (1-p)\log(w_g x_1 + w_b x_2) + p\log(w_b x_1 + w_g x_2), \quad \text{(A 8)}$$

which is a concave function of x_1. A simple calculation shows that for weakly correlated environments i.e. p close to 1/2. more precisely:

$$w_b/(w_g + w_b) < p < w_g/(w_g + w_b),$$

this function attains its maximum at the values:

$$x_1 = [(1-p)\,w_g - pw_b]/(w_g - w_b),$$
$$x_2 = [pw_g - (1-p)\,w_b]/(w_g - w_b). \quad \text{(A 9)}$$

The growth rate is there given by the left expression in (A 7). Therefore the optimal strategy in that case is to use a conditional mixed strategy: produce offspring in the frequencies given by (A 9) in environment E_1 and reversed proportions in environment E_2.

For $p = 1/2$ we obtain again $x_1 = x_2 = 1/2$ i.e. this conditional mixed strategy reduces to the simple hedge-betting strategy.

For highly (positively) correlated environments, that is for $p \leqslant w_b/(w_g + w_b)$, we have $x_1 \geqslant 1$ in (A 9), so that the function in (A 8) is monotonically increasing in x_1 and the optimal strategy is in fact the plastic strategy (corresponding to $x_1 = 1$). This includes also the extreme case $p = 0$, where the environment never changes.

On the other hand, for highly negatively correlated environments, that is for $p \geqslant w_g/(w_g + w_b)$, the function in (A 8) is monotonically decreasing in x_1 and the optimal strategy is the genetic strategy.

For $w_g = 1.3$, $w_b = 1$ the critical value for p is 0.435. If p is smaller than that, then the plastic strategy is the best possible.

(*c*) *Periodic Environments*

Suppose environment switches after exactly n generations. Then the growth rate is given by:

$$r = (1/2n)\log\rho(B^n A^n),$$

and can be explicitly computed as:

$$r(n; w_g, w_b, v) = \log w_g + 1/2n \log[q^n + (a_n^2/2) + (a_n^2/2)\,(a_n^2 + 4q^n)^{1/2}].$$

Here ρ is the spectral radius (= leading eigenvalue), $q = (1-v)\,w_b/w_g$, $a_n = v(w_b/w_g)\,(1-q^n/1-q)$.

Again we have the special cases $r(n; w_g, w_b, 0) = \log(w_g + \log w_b)/2$ and $r(n; w_g, w_b, 1) = [1-1/n)]\log w_g + (1/n)\log w_b$, as in the corresponding Markovian case with $p = 1/n$. Numerical simulations confirm the intuitive feeling, that for large values of n, the growth rates r for a periodic environment and the corresponding Markovian environment approach each other. The growth rate in a periodic environment seems always smaller than in the corresponding temporally patchy stochastic environment (see figures 4*a*, *b*).

For fixed w_g, w_b there are two possible shapes of the function $r(n; w_g, w_b)$. For small n, r is decreasing for small v and increasing for larger v. For larger values of n, r seems to be strictly increasing for larger v. For larger values of n, r seems to be strictly increasing for all $v \in [0, 1]$ (see figures 4*a*, *b*). More precisely, we have: r is a decreasing functionof v for small v,

$$n(w_b/w_g)^{(n/2-1)} > [1-(w_b/w_g)^n]/[1-(w_b/w_g)].$$

For fixed $w_b < w_g$, this holds for small values of n, but not for large values of n. For example, for $w_b/w_g = 0.9$, the switch is from $n = 10, 11$; for $w_b/w_g = 0.8$, the switch is from $n = 7, 8$.

APPENDIX 2

Consider a situation in which an organism acquires a particular phenotype determined by a transient anticipatory cue, and this phenotype is remembered for some number of subsequent generations which select but no longer induce the adaptive phenotype. For an organism with a plastic strategy, the production of the adaptive phenotype depends on the continuous presence of the inducing cue. Let a be the reliability of the anticipatory cue (a between 1.0 and 0), and T_i the fraction of the environmental period when the environment is both inducing and selecting. The fitness during the induction period is W_I, which is either equal to or lower than the maximal fitness. The fitness during the selecting, non-inducing environment is W_{bad} when the organism fails to match the selecting environment and W_{good} when there is adaptive matching. In this case the fitnesses of the plastic strategy and the carry-over strategy are:

$$W_{plastic} = T_i W_I + (1 - T_i)\,W_{bad}$$
$$W_{carry} = T_i W_I + (1 - T_i)\,(1-a)\,W_{bad} + a(1 - T_i)\,W_{good}.$$

It is clear that $W_{carry} > W_{plastic}$ even when a is very small.