

# Undermining the Baldwin Expediting Effect: Does Phenotypic Plasticity Accelerate Evolution?

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**The claim that phenotypic plasticity speeds up evolution towards a target phenotype is a recent incarnation of the Baldwin effect. To differentiate this theory from earlier interpretations of Baldwin's ideas, we name it the Baldwin expediting effect. Models that demonstrate this effect assume an extreme fitness scenario which bestows high fitness upon a single optimal phenotype and treats all other phenotypes as equal. In two modeling frameworks, we demonstrate that the effects of plasticity on the rate of evolution are highly dependent on the fitness function and population starting conditions. We argue that phenotypic plasticity does not universally facilitate evolution. Furthermore, in cases where the Baldwin expediting effect occurs, it is not necessarily correlated with increased fitness and therefore is not sufficient to explain the evolutionary success of plasticity.** © 2000 Academic Press

## INTRODUCTION

In the late-nineteenth century, James Mark Baldwin and several contemporaries sought to explain superficially Lamarckian phenomena through Darwinian theory (Baldwin, 1896; Morgan, 1896; Osborn 1896). Their mechanism of *organic selection* transforms an ontogenetically acquired trait to one that is hereditary. According to Morgan, this process, driven entirely by natural selection, requires a particular genetic and environmental background.

[B1] Suppose a population of plastic individuals encounters a new environment in which phenotype  $\Phi$  is necessary for survival.

[B2] Only those whose heritable plasticity encompasses  $\Phi$  are able to acquire  $\Phi$  and thereby survive.

[B3] There is no direct transmission of  $\Phi$ , only transmission of heritable factors determining the range of plasticity.

[B4] Any new heritable variants that more directly determine  $\Phi$ , (i.e., variants that are less plastic and

include  $\Phi$  within the range of phenotypic possibility) are favored by natural selection. Any variants that heritably determine only nonoptimal phenotypes are weeded out.

[B5] Ultimately a heritable variant that directly produces  $\Phi$  will fix in the population.

This line of thought, now called the *Baldwin effect*, was recast in the mid-twentieth century in modern genetical terminology (Simpson, 1953; Bateson, 1963; Waddington, 1942). Premised on the differentiation of germ cells from somatic cells (Weismann, 1893), Bateson (1963) based his rejection of pure Lamarckism on the consensus that somatic modifications are rarely if ever communicated back to the germline.

Waddington's (1942) *genetic assimilation* mechanistically connects the acquired and subsequent inherited versions of a trait. In general, he claims, developmental pathways are canalized via mutations and natural selection to produce regular and timely responses to environmental stimuli. Genes that replace the role of an environmental stimulus by constitutively switching development into a preferred pathway sometimes arise.

Since they determine the desired phenotype more reliably than the environment, the high fitness of these alleles will lead to their fixation. An initially environmentally-triggered trait can thereby become genetically produced. Waddington's version of the Baldwin effect has been used to explain the origin of traits ranging from ostrich callouses to multicellularity (Waddington, 1942; Wolpert, 1994).

The transition from [B4] to [B5] presumes that heritability has an advantage over plasticity. In other words, the transition away from plasticity via natural selection relies on the fitness cost of plasticity. If plasticity increased phenotypic options with no associated costs, then natural selection should produce maximally plastic organisms. Since this is not the case, scientists have sought to identify the forces that curb plasticity. Schmalhausen (1949) cited "erroneous" phenotypic changes as one cost of plasticity. Other potential costs include energetic requirements of regulation, of sensing the environment, and of producing biological structures and genetic side effects such as deleterious pleiotropy of genes involved in plasticity (DeWitt *et al.* 1998; Schlichting and Piglicci, 1998).

If plasticity is costly, why does it occur? This is critical to the Baldwin effect, since the process is predicated on initial plasticity. One explanation rests on the demands of an ever-changing environment. Several models have demonstrated the stability of phenotypic plasticity in a temporally or spatially fluctuating environment (Levins, 1968; Slatkin and Lande, 1996; Moran, 1992; Ancel, 1999). Even when developmental flexibility entails fitness costs, these models demonstrate that the ability to respond to environmental volatility has a selective advantage.

Beginning with Hinton and Nowlan, the term Baldwin effect has been applied to a different role for phenotypic plasticity. This incarnation of the Baldwin effect, which we call the *Baldwin expediting effect* is the following claim: Learning or, more generally, phenotypic plasticity expedites evolution (Hinton and Nowlan, 1987). In other words, given an optimal phenotype (which we call the target), a population of individuals with plasticity will evolve towards the target in fewer generations than a population of nonplastic individuals. A series of interesting models has further demonstrated this benefit of learning (Fontanari and Meir, 1990; Ackley and Littman, 1991; Belew and Mitchell, 1996).

In their seminal paper, Hinton and Nowlan (1987) offer the Baldwin expediting effect as an alternative explanation for the existence of plasticity. They write, "The most common argument in favor of learning is that some aspects of the environment are unpredictable, so it is positively advantageous to leave some decisions to

learning rather than specifying them genetically (e.g., Harley, 1981). This argument is clearly correct and is *one* good reason for having a learning mechanism, but it is different from the Baldwin effect which applies to complex co-adaptations to *predictable* aspects of the environment." Implicit here is the evolutionary claim that plasticity arose and is maintained because it accelerates evolution. To test this proposition, one must assess the evolutionary success of plasticity in a population consisting of both plastic and nonplastic individuals. The model frameworks that demonstrate the Baldwin expediting effect, however, consider populations evolving under plastic (or learning) genotype-phenotype maps *independent* from those evolving under nonplastic (or nonlearning) genotype-phenotype maps. We will show that the expediting effects of plasticity sometimes occur in regions of phenotype space where plasticity conveys lower fitness than non plasticity. Thus the claim that the Baldwin expediting effect underlies the presence of plasticity seems to rely on group selection.

The Baldwin expediting effect models start with a simple unimodal jagged fitness function (Fig. 1) and a simple one-to-one mapping from genotype to phenotype. They introduce plasticity (or learning) as a change in the genotype-phenotype map. In each case, the new genotype-phenotype map smoothes the fitness function (Fig. 1). The Baldwin expediting effect rests on a comparison of populations that lie in the tails of these fitness functions. Natural selection will favor variants in the plastic population that lie closer to the optimum, whereas natural selection will be ineffective in the nonplastic population. The plastic genotype-phenotype map thereby provides evolutionary foresight. In the words of John Maynard Smith (1987), "finding the optimal neural set in

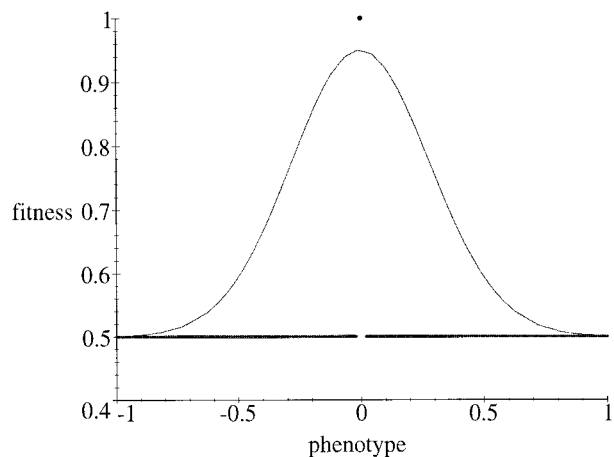


FIG. 1. Plasticity smooths the fitness function: Nonplastic (dark) vs plastic (light) fitness functions. The target phenotype here is 0.

the absence of learning is like searching for a needle in a haystack. With learning, it is like searching for the needle when someone tells you when you are getting close.”

We do not take issue with the claim that (some forms of) plasticity smooth the fitness function. This phenomenon appears also in studies of the evolution between two fitness peaks. Increases in phenotypic variance eliminate otherwise uncrossable valleys (Wright, 1931; Lande, 1980; Whitlock, 1997). Rather, we claim that “smoothness” does not necessarily translate into higher fitness or increased evolutionary velocity.

Besides the shape of the fitness function and the smoothing effects of plasticity, the Baldwin expediting effect makes three critical assumptions which we do not address further. Each considers evolution in a constant environment, a fitness cost of plasticity that is either integrated into the genotype-phenotype map or is imposed externally, and a correlation between the phenotypes accessible through plasticity and the phenotypes accessible through genetic evolution (See Anderson, 1997, for further treatment). A relatively stable environment is necessary for a population to ultimately converge on the optimal phenotype through an evolutionary reduction of plasticity. As discussed above, numerous models have demonstrated that plasticity is maintained under environmental volatility. Also, the inevitability of maximal plasticity in the absence of a fitness cost supports the second requirement, costly plasticity. Finally, several authors have analyzed the third assumption, termed *neighborhood correlation* (Mayley, 1997) and *plastogenetic congruence* (Ancel and Fontana, 2000).

We will demonstrate that although the Baldwin expediting effect occurs for a certain class of models, it is highly dependent on the shape of the fitness function, the formulation of plasticity, and the initial distribution of genotypes across the population. In two modeling frameworks we consider the rapidity of evolution towards a target in populations with and without plasticity. The first model, a quantitative genetics conception, treats plasticity as a nonevolving parameter applied uniformly across the population. We reject the Baldwin expediting effect in this context by demonstrating that plasticity uniformly slows evolution. The second model presents plasticity as an evolvable attribute that entails fitness costs. Here, the relationship between plasticity and the rate of evolution is much more complex.

## QUANTITATIVE GENETICS MODEL

The evolution of plasticity has been approached from several mathematical perspectives including optimality

theory, quantitative genetics, and multi-locus population genetic theory. First we show that a simple quantitative genetics model of plasticity precludes the Baldwin expediting effect.

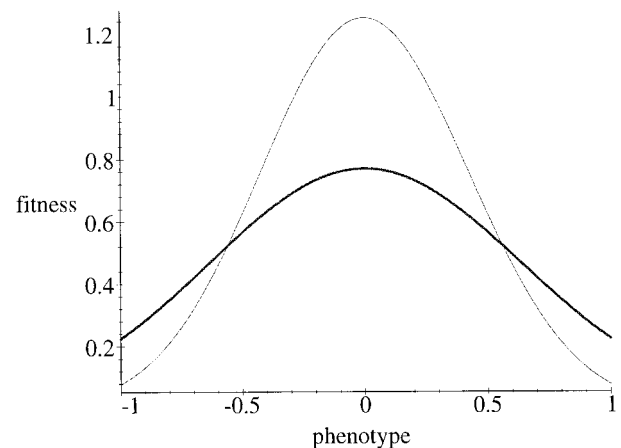
We begin with a nonplastic single-locus quantitative genetics model. An individual is described by its genotypic value  $g$  which maps one-to-one onto a real-valued phenotype  $\phi_g$ . For analytical simplicity let  $\phi_g = g \in \mathbf{R}$ . Its fitness is a Gaussian function centered on an optimal phenotype  $g_{\text{opt}}$ ,

$$w_S(g) = \frac{1}{\sqrt{2S\pi}} e^{-(g - g_{\text{opt}})^2/2S},$$

where the variance  $S$  determines the strength of the selection.

Plasticity increases the range of phenotypes and thereby may reduce the distance from an individual's phenotype to  $g_{\text{opt}}$ . To add plasticity to our model, then, we can add a variance component to individual phenotypes. Since the convolution of two normal distributions  $N(0, V_1)$  and  $N(0, V_2)$  is the normal distribution  $N(0, V_1 + V_2)$ , then this is equivalent to augmenting the variance  $S$ , which effectively weakens selection. (See, for example, Cavalli-Sforza and Feldman, 1976 and Anderson, 1995).

Figure 2 gives two fitness maps with different plasticities. The fitness graph for the nonplastic population is the curve with the higher peak. Near the target phenotype,  $g_{\text{opt}}$ , plasticity lowers fitness, while the opposite is true further from the target. Under this model then, there is a cost to plasticity. Far from the target, the benefits of experiencing higher fitness phenotypes outweigh the cost of plasticity.



**FIG. 2.** Plasticity smooths the quantitative genetics fitness function: Nonplastic (light) vs plastic (dark) fitness functions.

Near the target, however, plasticity reduces fitness and is therefore detrimental.

We assume an initially Gaussian population genotype frequency distribution centered at  $c_0$  with variance  $V_0$ . So the probability density of individuals with genotype  $g$  is

$$f_0(g) = \frac{1}{\sqrt{2V_0\pi}} e^{-(g-c_0)^2/2V_0}.$$

Assume that discrete generations are nonoverlapping. Then the distribution of genotypes at  $t = 1$  can be written as follows.

$$\begin{aligned} f_1(g) &= \frac{f_0(g) \cdot w_S(g)}{\int f_0(g) w_S(g) dg} \propto e^{-(g-c_0)^2/2V_0} e^{-(g-g_{\text{opt}})^2/2S} \\ &= e^{t(S(g-c_0)^2 + V_0(g-g_{\text{opt}})^2)/2V_0S}. \end{aligned}$$

For simplicity, assume  $g_{\text{opt}} = 0$ . Then

$$\begin{aligned} f_1(g) &\propto e^{-(Sg^2 - 2Sgc_0 + Sc_0^2 + V_0g^2)/2V_0S} \\ &= e^{-(g^2(S+V_0) - 2Sgc_0 + Sc_0^2)/2V_0S \cdot 1/(S+V_0)/1/(S+V_0)} \\ &\propto e^{-(g-(S/(S+V_0))c_0)^2/2(V_0S/(V_0+S))}. \end{aligned}$$

(Cavalli-Sforza and Feldman, 1976 and Lande, 1980). By similar reasoning, in general

$$f_t(g) \propto e^{-(g-(S/(S+V_{t-1}))c_{t-1})^2/2((V_{t-1}S)/(V_{t-1}+S))}.$$

Now we turn to the question of the speed of evolution. From this recursive formulation, we have expressions for the movement of the population mean towards the target  $g_{\text{opt}} = 0$  and of the population variance towards 0. In particular, the mean at time  $t$  is  $c_t = Sc_{t-1}/(S+V_{t-1})$  and the variance at time  $t$  is  $V_t = V_{t-1}S/(V_{t-1}+S)$ , from which we derive the closed forms:

$$c_t = c_0 \left( \frac{S}{tV_0 + S} \right)$$

and

$$V_t = \frac{V_0S}{tV_0 + S}.$$

We further calculate  $dc_t/dS = c_0(tV_0/(tV_0+S)^2)$  which implies that  $dc_t/dS$  has the same sign as  $c_0$ , and  $dV_t/dS = tV_0^2/(tV_0+S)^2 > 0$ . Recall that in this model an

increase in plasticity is tantamount to an increase in  $S$ . At any time  $t$  then, increased plasticity (higher  $S$ ) entails a wider population variance and a mean genotype further from the target. In other words, plasticity both slows down the movement of the population mean towards the target and hinders the convergence of the population variance to 0.

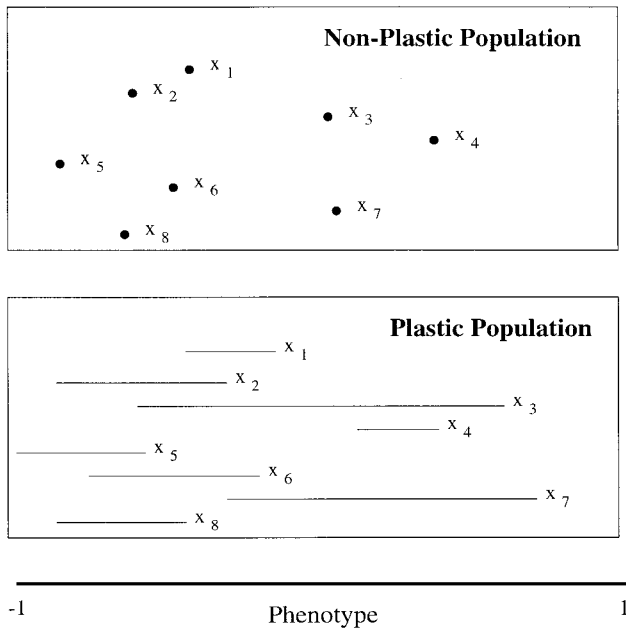
Within this infinite-population-size, Gaussian-distribution framework, the rapidity with which a population approaches a target both varies with the initial genotypic distribution and is always greater for a low plasticity population than for a high plasticity population. Note that this model differs significantly from Baldwin expediting effect models, in that here the extent of phenotypic plasticity cannot evolve. It is imposed uniformly across the population and through time. Next we describe a model that, like earlier models, permits plasticity itself to evolve.

## NORM OF REACTION MODEL

Through a second model, we address the evolution of plasticity more explicitly. See Ancel (1999) for a detailed analysis of this model system. Let  $\Phi = [-1, 1]$  be the space of all phenotypes. For a nonplastic population, an individual  $i$  is defined by a genotype  $g_i$  that maps one-to-one onto a single point phenotype  $p_i$  in  $\Phi$ . We model plasticity by replacing the point genotype–phenotype with a norm of reaction. In the plastic case, an individual  $i$  is defined by a genotype that maps to an interval of phenotypic possibilities:  $g_i \rightarrow \rho_i = [\lambda_i, Y_i] \subseteq \Phi$  where  $\lambda_i$  and  $Y_i$  represent the lower and upper bounds of the norm of reaction, respectively. In the following analysis, all populations reproduce asexually. Figure 3 illustrates nonplastic and plastic populations.

For simplicity, we consider only a finite set of phenotypes in  $\Phi$ . In particular, let  $\nu$  be the size of intervals between phenotypes. In the following numerical analysis, we set  $\nu = 0.025$ . Then phenotype space consists in 41 discrete points  $\Phi' = \{-1, -0.975, -0.95, -0.925, \dots, 1\}$ . Nonplastic individuals assume a fixed one of these phenotypic values, whereas plastic individuals can assume norms of reaction with endpoints in the subset  $\{-1, -0.95, -0.9, \dots, 1\}$ . In this way we can compare any individual norms of reaction to the nonplastic phenotype lying at its midpoint. For example we can compare  $\rho_i = [-0.4, -0.05]$  to the nonplastic phenotype  $p_j = -0.225$ .

Mutations occur at a rate  $\mu$  per individual per generation. For nonplastic populations, a mutation to  $g_i$  changes the phenotype to either  $p_i + \nu$  or  $p_i - \nu$ . For



**FIG. 3.** Populations in the norm of reaction model: Nonplastic populations consist in point-valued phenotypes (top) and plastic populations consist in interval-valued norm of reactions (bottom). Each  $x_i$  represents either a point value of an individual phenotype (top) or the range from which a phenotype is drawn (bottom).

plastic populations, mutations alter the upper and lower bounds of the norms of reaction. Each mutation is either an increment or a decrement to  $\lambda_i$  or  $Y_i$  of size  $2v$  for some  $i$ . Note that such mutations shift the midpoint of a norm of reaction by  $\pm v$ . Mutations affect the upper or lower bounds and cause shrinking and expansion with equal frequency as described by Table 1.

Fitness is a function of both phenotype and norm of reaction length. For nonplastic individuals, fitness is determined by  $w(p_i)$  where  $w: \Phi \rightarrow \mathbf{R}^+$ . The fitness of a comparable plastic individual is  $\Omega(\rho_i) = \max(w(p) | p \in \rho_i) \cdot \chi(|\rho_i|)$  where  $\chi: [0, 1] \rightarrow \mathbf{R}^+ \cup \{0\}$  is a monotonically

**TABLE 1**

**Mutation Rates**

	Old $p_i(\rho_i)$	New $p_i(\rho_i)$	Effect on $p_i(\rho_i)$	Rate
Nonplastic	$p_i$	$p_i - v$	shift left	$\frac{1}{2} \mu$
	$p_i$	$p_i + v$	shift right	$\frac{1}{2} \mu$
Plastic	$[\lambda_i, Y_i]$	$[\lambda_i + 2v, Y_i]$	shrink	$\frac{1}{4} \mu$
	$[\lambda_i, Y_i]$	$[\lambda_i - 2v, Y_i]$	extend	$\frac{1}{4} \mu$ if $\lambda_i \geq 2v$ , 0 if $\lambda_i = 0$
	$[\lambda_i, Y_i]$	$[\lambda_i, Y_i + 2v]$	extend	$\frac{1}{4} \mu$ if $Y_i \leq 1 - 2v$ , 0 if $Y_i = 1$
	$[\lambda_i, Y_i]$	$[\lambda_i, Y_i - 2v]$	shrink	$\frac{1}{4} \mu$

decreasing function that assesses the cost of phenotypic plasticity. In other words, we assume that plastic individuals will settle on the most fit phenotype within their norms of reaction and that this flexibility is costly in terms that increase with the size of the norm of reaction.

### THE EXTREME CASE

First we treat the extreme fitness scenario found in the Baldwin expediting effect models. Consider the fitness landscape depicted in Fig. 4A where

$$w(p) = \begin{cases} 1 + \kappa & \text{if } p = p_{\text{opt}}, \\ 1 & \text{otherwise.} \end{cases}$$

A single optimal phenotype, in this case  $p_{\text{opt}} = 0$ , enjoys relative fitness  $1 + \kappa$  while all others have relative fitness of 1. Figure 4B displays the comparable fitness function for a plastic population given by

$$\Omega(\rho) = \begin{cases} (1 + \kappa) \cdot \left(1 - \frac{|\rho|}{2}\right) & \text{if } p_{\text{opt}} \in \rho, \\ \frac{2 - |\rho|}{2} & \text{otherwise.} \end{cases}$$

where  $\rho$  is a norm of reaction.

The cost of plasticity, namely multiplication by  $1 - \frac{|\rho|}{2}$ , increases with norm of reaction length. In the extremes: a norm of reaction containing a single point will have a plasticity factor of  $1 - \frac{0}{2} = 1$  which imposes no cost and a maximal norm of reaction containing the entire phenotype space will have a plasticity factor of  $1 - \frac{2}{2} = 0$  which yields an overall fitness of zero. See Fig. 5 for an illustration of this fitness function.

The evolutionary trajectory of a nonplastic population towards the optimal phenotype breaks down into two episodes:

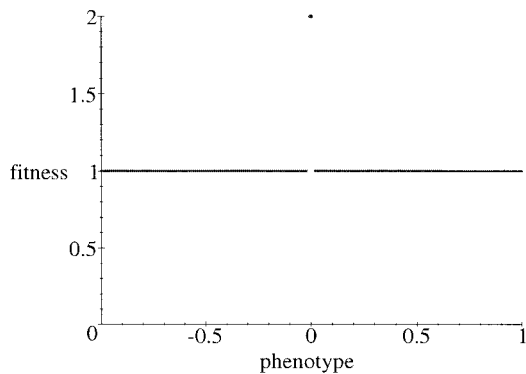
[NP1] The finite population moves at random through phenotype space until an individual mutates to the optimal type.

[NP2] At that point the lineage of the optimal individual will rapidly come to dominate the population.

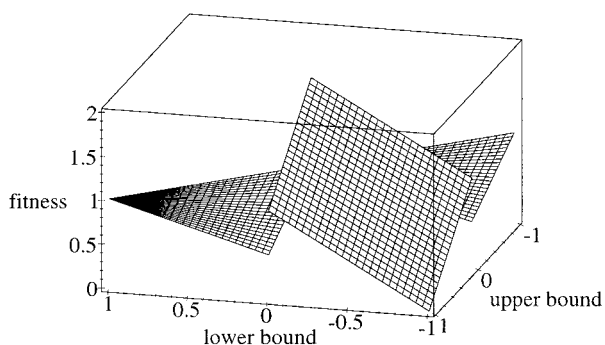
Through a two-part analysis we can approximate these times for a finite population. We label the time taken in [NP1] and [NP2],  $\tau_{NP1}$  and  $\tau_{NP2}$ , respectively.

If we assume that the population starts with a normal distribution of genotypes, then we can track movement

(A)



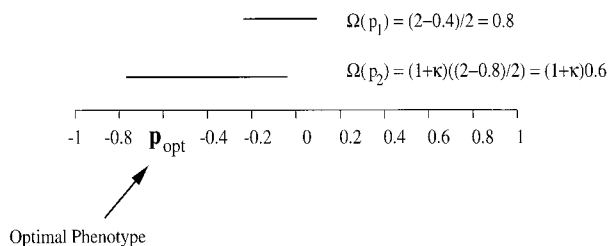
(B)



**FIG. 4.** Plasticity smooths the extreme norm of reaction fitness function: (A) Nonplastic vs (B) plastic fitness functions (right). In this case,  $\kappa=1$ . The  $x$ - and  $y$ -axes indicate phenotype and the  $z$ -axis represents fitness. The phenotype of a nonplastic individual is the average of the  $x$ -value and the  $y$ -value. The norm of reaction for the associated plastic individual has the  $x$ -value as a lower bound and the  $y$ -value as an upper bound.

through phenotype space via the evolution of the distribution variance. Let  $X = \{x_1, x_2, x_3, \dots, x_N\}$  represent the population, where each  $x_i$  denotes an individual genotype. Individuals in the population move as independent identical unbiased random walks.

If no individual has the optimal phenotype, then fitness will be constant across the population, and individuals will



**FIG. 5.** Fitness of two plastic individuals: One norm of reaction contains  $p_{\text{opt}}$  (bottom line) and the other does not (top line).

have equal probability of representation in the next generation. We model the transition of genotypes with the simplifying assumption that all lineages are maintained in subsequent generations. For a given  $i$ , the genotype  $x_i$  will either be transmitted unchanged to the next generation or mutate with probability  $\mu$ . Let  $b$  be a random variable describing the per generation changes in genotype where

$$b = \begin{cases} v & \text{with probability } \frac{\mu}{2}, \\ 0 & \text{with probability } 1 - \mu, \text{ and} \\ -v & \text{with probability } \frac{\mu}{2}. \end{cases}$$

We find the expectation and variance of  $b$ :

$$E(b) = v \cdot \frac{\mu}{2} + 0 \cdot (1 - \mu) - v \cdot \frac{\mu}{2} = 0$$

$$V(b) = [E(b)]^2 - E(b^2) = E(b^2)$$

$$= (v)^2 \frac{\mu}{2} + 0^2 \cdot (1 - \mu) + (-v)^2 \frac{\mu}{2} = v^2 \mu.$$

Consider  $x_{i_t}$ , an individual genotype at generation  $t$ . Then  $x_{i_{t+1}} = x_{i_t} + b$  represents the genotype of the lineage at time  $t + 1$ . We use this formulation to compute the changes in the variance and expectation of the  $x_i$ 's through time.

$$E(x_{t+1}) = E(x_t + b) = E(x_t) + E(b)$$

$$= E(x_t) \Rightarrow e(x_t) = E(x_0)$$

$$V(x_{t+1}) = V(x_t + b) = V(x_t) + V(b) + 2Cov(b, x_t)$$

$$= V(x_t) + V(b)$$

since the  $x_t$  and  $b$  are independent. Continuing,

$$V(x_{t+1}) = V(x_t) + v^2 \mu \Rightarrow V(x_t) = V(x_0) + t v^2 \mu.$$

Now we derive the time until the first individual mutates to the optimal genotype. The frequency of optimal individuals in the population at generation  $t$  is given by

$$\begin{aligned} f_i(p_{\text{opt}}) &= f_i(0) = \frac{1}{\sqrt{2\pi V(x_t)}} \cdot e^{-E(x_t)^2 / (2V(x_t))} \\ &= \frac{1}{\sqrt{2\pi (V(x_0) + t v^2 \mu)}} e^{-E(x_0)^2 / (2(V(x_0) + t v^2 \mu))}. \end{aligned}$$

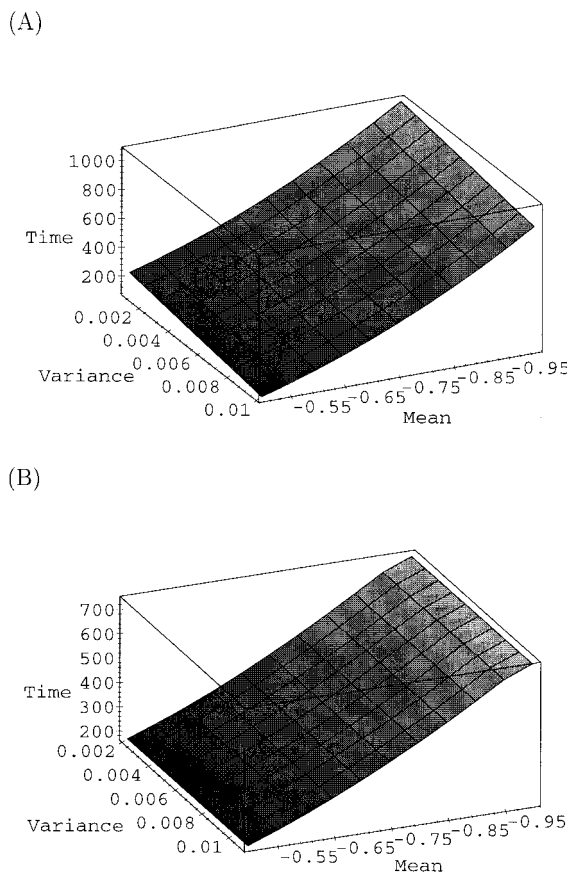


FIG. 6. Times from previous equilibrium to first encounter with the new optimal phenotype: (A) Nonplastic  $\tau_{NP1}$  and (B) plastic  $\tau_{P1}$ .

For a population of size  $N$ , the time until the first encounter with  $p_{\text{opt}}$  is the minimum time  $\tau$  such that  $f_{\tau}(p_{\text{opt}}) > \frac{1}{N}$ . For example, if a population with  $N = 1000$ ,  $\mu = 0.1$ , and  $\nu = 0.05$  begins with  $E(x_0) = -0.5$  and  $V(x_0) = 0.01$ , then the time until the first optimal phenotype arises is approximately  $\tau_{NP1} = 88$  generations. Figure 6(A) graphs  $\tau_{NP1}$  for initial variances  $V(x_0) \in [0.001, 0.01]$  and initial means  $E(x_0) \in [-1, -0.5]$ .

The second evolutionary episode consists of the newly optimal lineage overtaking the population. We estimate the timing of this transition. Let the variable  $O_t$  be the frequency of individuals in the optimal class at time  $t$  since the first encounter with  $p_{\text{opt}}$ , and let  $R_t = 1 - O_t$  be the frequency of the remaining population at time  $t$ . Then  $O_{t+1} = O_t w(p_{\text{opt}}) / (O_t w(p_{\text{opt}}) + R_t w(x \neq p_{\text{opt}})) = O_t(1 + \kappa) / (O_t(1 + \kappa) + R_t(1))$ , and similarly  $R_{t+1} = R_t / (O_t(1 + \kappa) + R_t)$ . These yield the ratio  $O_{t+1}/R_{t+1} = O_t(1 + \kappa)/R_t$ , and therefore  $O_t/R_t = (O_0/R_0)(1 + \kappa)^t = (1/(N-1))(1 + \kappa)^t$ , since the optimal lineage begins with a single individual. Neglecting mutation, the time until

all individuals are optimal is approximately  $\tau_{NP2} = \log N(N-1) / \log(1 + \kappa)$  which depends only on the population size and the relative fitness of the optimal type. For  $N = 1000$  and  $\kappa = 2$ , that is, optimal individuals are three times as fit as nonoptimal individuals,  $\tau_{NP2} = 19.9$  generations. For  $N = 10^4$  and  $N = 10^5$  the times from [NP1] until [NP2] are only  $\tau_{NP2} = 26.5$  and  $\tau_{NP2} = 33.2$ , respectively. Note that the search time,  $\tau_{NP1}$ , is approximately three times longer than the convergence time,  $\tau_{NP2}$ .

The effect of plasticity on the fitness map is to give direction to the evolving population as illustrated in Fig. 4. Norms of reaction containing the optimum have higher fitness than those that do not. For optimum-containing norms of reaction, fitness increases as the size of the norm of reaction decreases. In this model, the evolution of a plastic population towards a target occurs in three stages:

[P1] The population wanders in phenotype space until a single individual has a norm of reaction that contains the optimal phenotype.

[P2] Once the individual encounters the optimum, its lineage will quickly dominate the population.

[P3] Selection will narrow the norms of reaction around the optimum.

We use  $\tau_{P1}$ ,  $\tau_{P2}$ , and  $\tau_{P3}$  to denote the timing of each evolutionary episode. A detailed analysis of these processes appears in an earlier paper and is omitted here (Ancel, 1999). (Belew (1990) presents a comparable dissection of the Hinton and Nowlan model dynamics.) It is only during the first stage, finding the optimum, that plasticity expedites the evolutionary trajectory. Figure 6A displays the time until a plastic population first encounters a new optimum given the mean and variance of the initial normal distribution. A comparison with Figure 6B illustrates an early expediting effect of plasticity.

When we consider the latter stages of evolution to the target, however, the plastic population is slower than the nonplastic population. Large norms of reaction are more likely to contain the optimal phenotype than small norms of reaction. The first individual to have the optimal phenotype within its norm of reaction is therefore probably an individual with a large norm of reaction relative to the rest of the population. Importing the analysis of the nonplastic population, we find  $\tau_{P2} = \log N(N-1) / \log(\Omega(O)/\Omega(R))$  where  $\Omega(O)$  is the estimated fitness of the optimal lineage and  $\Omega(R)$  is the average fitness of the remaining population (Ancel, 1999). Because norm of reaction length is costly and the optimal lineage

endures a larger plasticity cost than the average of the remaining population, then  $\Omega(O)/\overline{\Omega(R)} = (1 + \kappa)(2 - |\rho_O|)/(2 - |\rho_R|) > 1 + \kappa$ , which implies that  $\tau_{NP2} = \log N(N-1)/\log(1 + \kappa) < \log N(N-1)/\log(\Omega(O)/\overline{\Omega(R)}) = \tau_{P2}$ .

The final step, [P3] is a burden for the plastic population alone. We find that  $\tau_{P3}$  decreases as the cost of plasticity increases and as the mutation rate increases (Ancel, 1999). In this model, plastic populations always spend less time in [P1] than comparable nonplastic populations spend in [NP1]. Yet they spend more time in [P2] than the nonplastic population spends in [NP2]. Overall, plasticity will only expedite evolution if  $\tau_{NP1} - \tau_{P1} > (\tau_{P2} + \tau_{P3}) - \tau_{NP2}$ , in other words, if the relative rapidity of the initial search outweighs the subsequent impediments to convergence on the new phenotype. Hence there is a paradox: a high cost to plasticity expedites  $\tau_{P3}$  (narrowing of the norms of reaction to the new optimum), yet a low cost to plasticity expedites  $\tau_{P1}$  and  $\tau_{P2}$  (finding the new optimum, then shifting over to the new optimum). The Baldwin expediting effect will therefore take place in this model only when the cost of plasticity satisfies these strict constraints and when the mutation rate is sufficiently high.

## EVOLUTIONARY RATE APPROXIMATIONS

In the next version of the evolving plasticity model, we assume Gaussian fitness functions. That is,  $w(p) = (1/\sigma\sqrt{2\pi})e^{-(p-\zeta)^2/(2\sigma^2)}$  where  $\zeta$  is the optimal phenotype and  $\sigma$  indicates the strength of selection. Again we compare populations of nonplastic single-point phenotypes to populations of plastic interval-valued norms of reactions. As above, the fitness of a nonplastic individual is just  $w(p_i)$  and the fitness of a plastic individual is  $\Omega(\rho_i) = \max(w(p) | p \in \rho_i) \cdot (1 - |\rho_i|/2)$ .

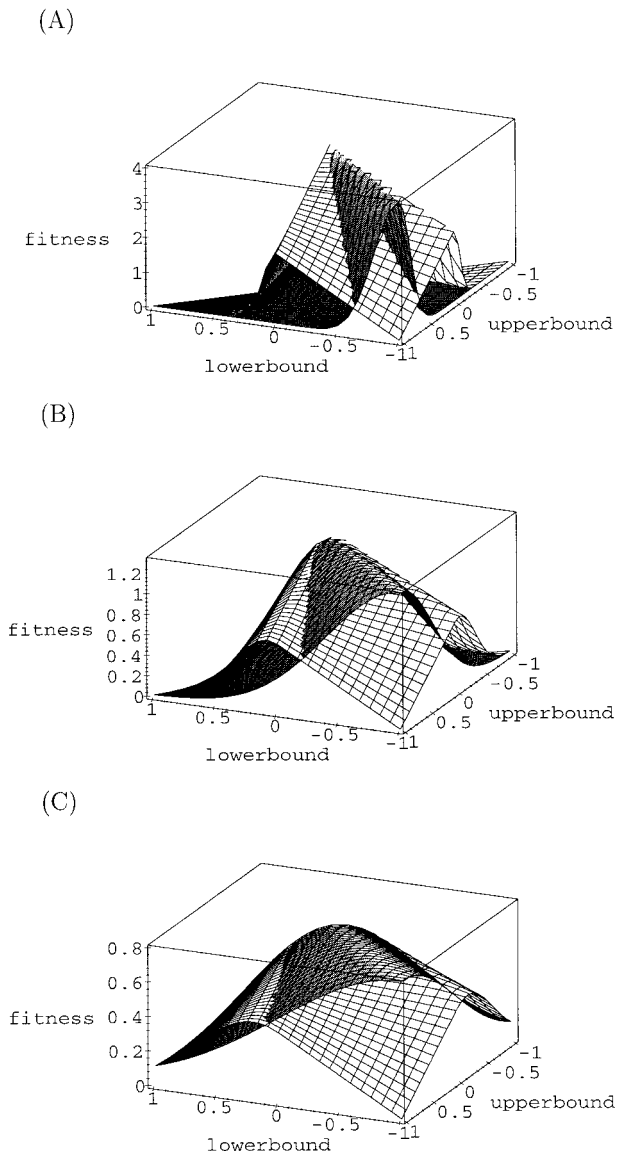
The addition of plasticity dramatically alters the shape of the fitness landscapes. Consider the pairs of fitness functions depicted in Fig. 7. These are slightly modified versions of  $w$  and  $\Omega$  described above. The light mesh surface displays the fitness function for a plastic population, whereas the dark surface graphs that for a nonplastic population. For a plastic population, the  $x$ - and  $y$ -axes are the lower and upper bounds of the norm of reaction. These bounds are sufficient to calculate an individual's fitness, which plots in the  $z$ -direction. For example the point  $(-1, 1, 0)$  means the individual with norm of

reaction  $\rho_i = [-1, 1]$  has fitness  $\Omega(\rho_i) = 0$ . In other words, the plastic population graph is of the function

$$\Omega': (x, y) \mapsto \max(w(p) | p \in [x, y]) \cdot \chi(y - x).$$

Recall that  $w$  is a Gaussian function described above. Here we use the cost function  $\chi: a \mapsto 1 - \frac{a}{2}$  since the maximum length for a norm of reaction is  $a = 2$ .

In the case of the nonplastic graphs, the  $x$ - and  $y$ -axes have a different meaning. Here the phenotype of an



**FIG. 7.** Plasticity smooths the Gaussian norm of reaction fitness functions: Nonplastic (dark surface) vs plastic (light surface) fitness functions. (A)  $\sigma = 0.1$ , (B)  $\sigma = 0.3$ , and (C)  $\sigma = 0.5$ .



individual is taken to be the midpoint between the  $x$ -value and the  $y$ -value. For example the point  $(-0.5, 0.5, 1)$  means the individual with phenotype  $p_i = \frac{-0.5+0.5}{2} = 0$  has fitness  $w(p_i) = 1$ . That is, the graphs depict the function

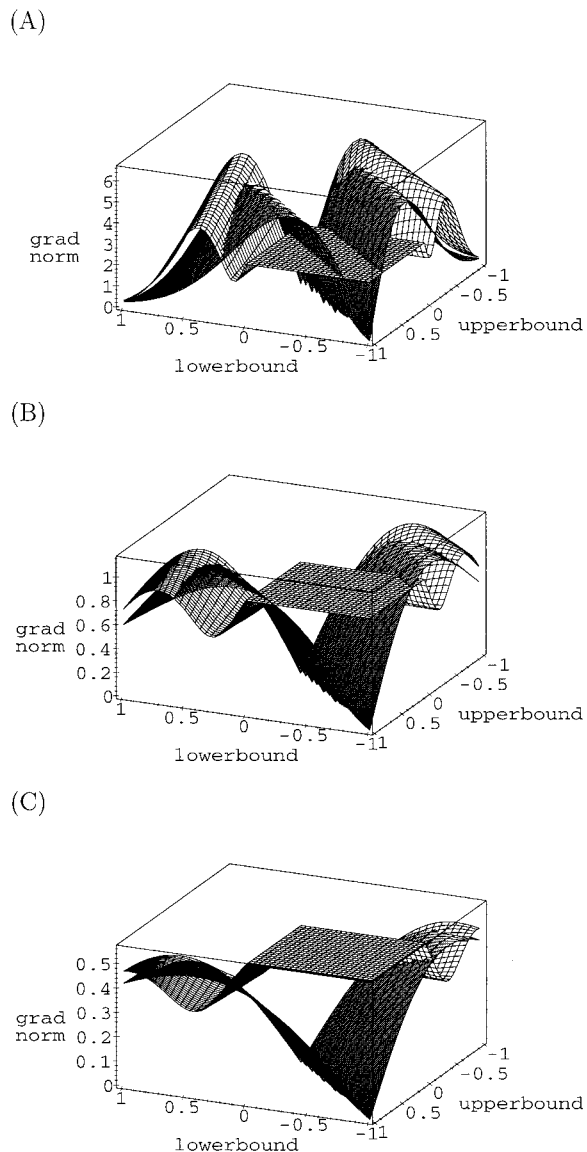
$$w': (x, y) \mapsto w\left(\frac{x+y}{2}\right).$$

Through these graphs we compare the fitness of a plastic individual to that of a nonplastic individual, where the phenotype of the latter is the norm of reaction midpoint of the former. A visual survey reveals that plasticity improves fitness in some regions of phenotype space, but not all. As the fitness function becomes more peaked, the regions in which plasticity has a selective advantage expand. This may have implications for when plasticity can invade a nonplastic population, and vice versa, but does not say anything about the relative speeds of evolution.

In this graphical format, evolutionarily adjacent types, i.e., phenotypes that are separated by single mutations, lie next to each other in the  $x$ - $y$  plane. So evolutionary trajectories are continuous on the surface of the graph. The norms of the gradients of the fitness functions,  $\|\nabla w'\|$  and  $\|\nabla \Omega'\|$ , give the slopes in the  $z$ -direction of the steepest paths towards the target. If we assume relatively homogeneous populations, that is populations consisting of genetically similar individuals, then for any location in phenotype space, the norms of the gradients roughly approximate the rapidity of evolution there.

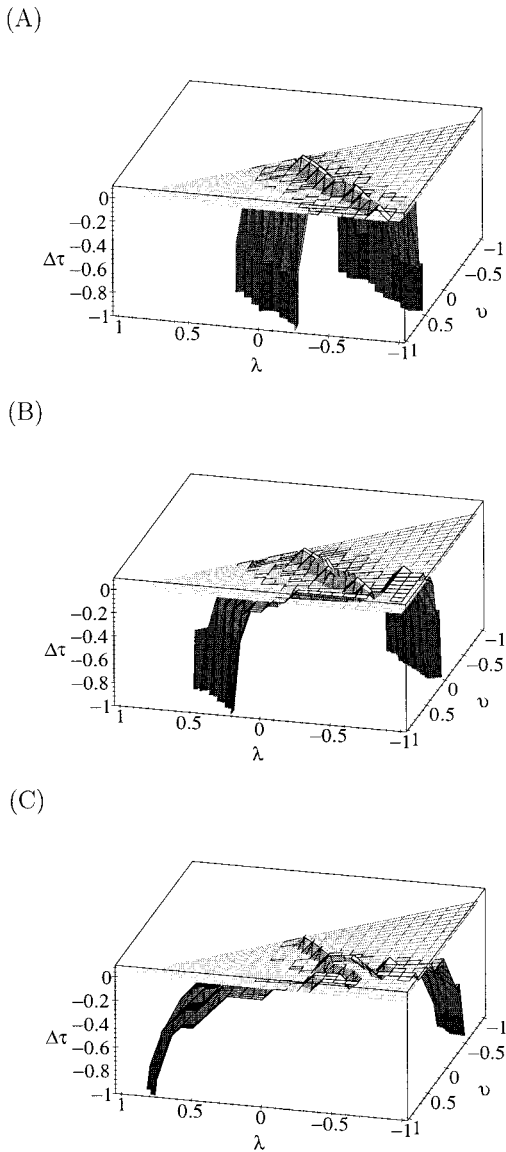
Figure 8 plots  $\|\nabla w'\|$  and  $\|\nabla \Omega'\|$  for comparable nonplastic and plastic populations, respectively. The dark surface that dips to  $(-a, a, 0)$  for all  $a \in [0, 1]$  in each illustration is  $\|\nabla w'\|$  for the nonplastic population, while the lighter surface represents the plastic population. As  $\sigma$  increases, the region of phenotype space in which plasticity provides for faster evolution increases. A comparison of Figs. 7 and 8 demonstrates that relative rates of evolution do not correspond to relative fitnesses. Furthermore, the claim that plasticity expedites evolution does not generally hold in this analytical framework.

This analysis alone is unsatisfying. Take, for example, the values of  $\|\nabla w'(-1, 1)\|$  and  $\|\nabla \Omega'(-1, 1)\|$ . In all three graphs,  $\|\nabla \Omega'(-1, 1)\| > \|\nabla w'(-1, 1)\|$  suggesting the rate of plastic evolution is faster than nonplastic evolution in this region of phenotype space. But the nonplastic population is already at the target while the plastic population is merely centered at the target. So the rate comparison alone can be misleading.



**FIG. 8.** Gradient estimates of evolutionary rates: Nonplastic  $\|\nabla w'\|$  (dark surface) vs plastic  $\|\nabla \Omega'\|$  (light surface) for (A)  $\sigma = 0.1$ , (B)  $\sigma = 0.3$ , and (C)  $\sigma = 0.5$ . The  $x$ - and  $y$ -axes indicate the phenotype of the homogeneous starting population. The  $z$ -axis depicts the norms of the gradients of the fitness functions, an approximation for the time until the population converges on the optimal phenotype.

We now extend this line of analysis to remedy the previous objection. We suggest that the length of the path from any given point in phenotype space to the target along the fitness surface is a better indication of the evolutionary rate. The gradients serve as vector fields which determine the most direct trajectory to the target. We numerically solve the system of equations given by the gradients and the starting phenotypes to determine the path and time to the target. For example, a plastic



**FIG. 9.** Solving the gradient equations to estimate the time until the population converges on the optimal phenotype: Differences in the evolutionary time to target for a plastic population and a nonplastic population. (A)  $\sigma = 0.5$ , (B)  $\sigma = 0.1$ , and (C)  $\sigma = 0.2$ . The pale surfaces depict the plane  $(x, y, 0)$ .

population beginning at phenotype  $\rho = [0.1, 0.3]$  follows the path given by the system:

$$\frac{\partial x}{\partial t} = \frac{\max(x, 0) e^{-x^2/2\sigma^2} (2 - (y - x)) \sqrt{2}}{4\sigma^2 \sqrt{\pi}} + \frac{e^{-(\min(|\min(0, y) + \max(0, x)|, |x|, |y|))^2/2\sigma^2}}{2\sigma \sqrt{\pi}},$$

$$\frac{\partial y}{\partial t} = -\frac{\min(y, 0) e^{-y^2/2\sigma^2} (2 - (y - x)) \sqrt{2}}{4\sigma^3 \sqrt{\pi}} + \frac{e^{-(\min(|\min(0, y) + \max(0, x)|, |x|, |y|))^2/2\sigma^2}}{2\sigma \sqrt{\pi}},$$

$$x(0) = 0.1 \quad \text{and} \quad y(0) = 0.3.$$

We use  $(2 - (y - x))$  as a cost of plasticity because the norm of reaction length is  $(y - x)$  which has a maximum length of 2.

Figure 9 depicts differences between path lengths to the target. We graph  $\Delta\tau$ , which is the time taken for a plastic population to reach the target minus the time taken for a comparable nonplastic population to reach the target. We assume the population moves as a single point across the fitness surface and that starting phenotypes are given by the  $x$ - and  $y$ -axes. The graph is positive where plastic populations take longer to reach the target and negative where plasticity expedites evolution. The plane at  $\Delta\tau = 0$  divides the graph into these two domains. Populations starting near the target are generally better off without plasticity, and likewise populations starting far from the target seem to greatly benefit from plasticity. As the fitness landscape becomes more peaked, the region in which plasticity is detrimental diminishes.

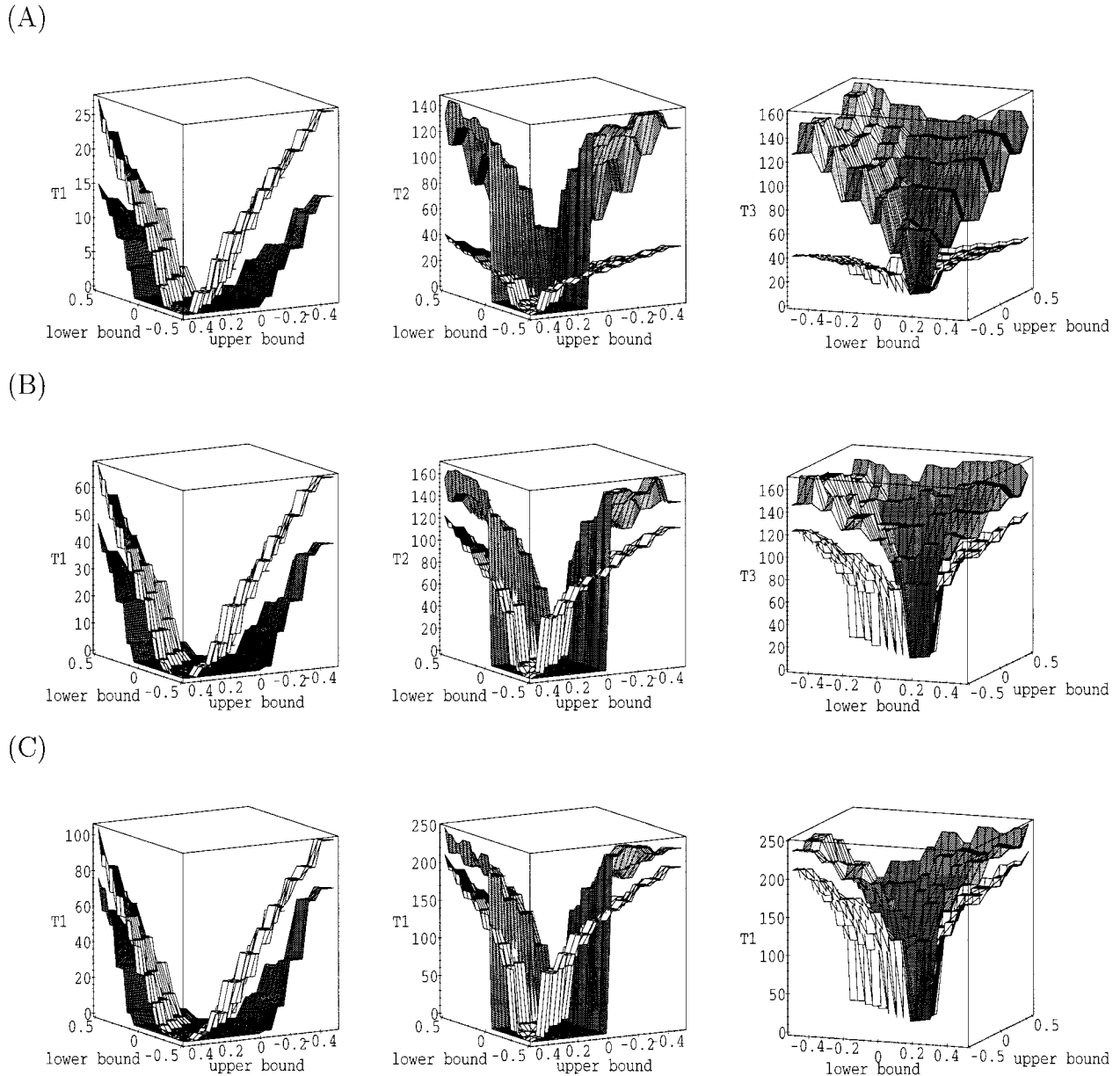
These observations support the claim that plasticity accelerates evolution in much of phenotype space. The Hinton and Nowlan model (1987) demonstrates a loss of plasticity as the populations nears the target. The regions in which plasticity appears advantageous in our graphical analysis therefore do not do much damage to the Baldwin expediting effect. In the next section we discuss the drawbacks to this analysis.

## SIMULATION RESULTS

We compare evolutionary simulations to the gradient analysis. Simulations reveal the limitations of representing a population as a point on a fitness graph. As discussed above, the evolution of a plastic population towards a target breaks down into three epochs. While the first one, the initial search for the optimal type, might be amenable to the point population approximation, the latter two can only be treated with a closer look at the within-population evolutionary dynamics. An analysis of  $\tau_{P2}$ , the time until the lineage of the first optimal individual takes over, requires a model that places the individual within a population distribution context. The subsequent time until the norms of reaction converge on the optimal phenotype depends entirely on the cost of plasticity and

the mutation rate and not on the underlying fitness landscape. We compare simulations of plastic and nonplastic populations to demonstrate the burden on plastic populations of this final component of evolution which is not reflected in the gradient framework. The disparities between the two approaches suggest that the smoothness of fitness surfaces does not sufficiently indicate the speed of evolution.

Each simulation begins with a uniform population of  $N=1000$  asexual individuals; population size then remains constant at  $N=1000$ ; the optimal phenotype is  $p_{\text{opt}}=0$  and mutation occurs according to Table 1 at a rate  $\mu=0.1$ . Figure 10 graphs the times for each of the components of the evolutionary trajectory. The axes represent the genotypes of the starting population. For plastic populations, the  $x$ - and  $y$ -axes indicate the lower



**FIG. 10.** Simulation results: The dark and light plots represent the plastic and nonplastic populations, respectively.  $T1$ , time to first encounter with the optimal phenotype;  $T2$ , time until 95% of the population either has the optimal phenotype (nonplastic population) or has a norm of reaction containing the optimal phenotype (plastic population); and  $T3$ , time to average norm of reaction within a 5% neighborhood of the optimal (plastic) or population average phenotype lies within a 5% neighborhood of the optimal (nonplastic). Note that for the nonplastic population  $T2 \sim T3$ . The fitness functions have (A)  $\sigma=0.1$ , (B)  $\sigma=0.2$ , and (C)  $\sigma=0.3$ .

and upper bound of the initial norm of reaction. For nonplastic populations, the starting genotype equals the average of the  $x$ - and  $y$ -values.

The graphs in the first column indicate the time until the first individual encounters the optimum phenotype. The plastic population (light mesh) generally takes less time to find the optimum than the nonplastic population. Recall that the first encounter means that one individual has the optimal phenotype in the nonplastic case and that one individual has the optimal phenotype within its norm of reaction in the plastic case.

The second column shows the time until most of the population attains the optimal phenotype. For the nonplastic population, this is the time until 95% of individuals have the optimal phenotype. For the plastic population, this is the time until 95% of norms of reaction contain the optimal phenotype. Plasticity shows a slight expediting effect for a subset of starting conditions. In particular, only those populations that started with norms of reaction containing  $p_{\text{opt}} = 0$  still outpace the nonplastic population. Other populations have lost their lead.

Finally we illustrate the time until the average norm of reaction lies within a small neighborhood of the optimal phenotype for the plastic population. In particular, the light graphs in the third column show the time until  $\bar{l} = \sum_{i=1}^N l_i > p_{\text{opt}} - 0.1 = -0.1$  and  $\bar{u} = \sum_{i=1}^N l_i < p_{\text{opt}} + 0.1 = 0.1$ . For the nonplastic population we graph the time until  $\bar{p} = \sum_{i=1}^N p_i \in [p_{\text{opt}} - 0.1, p_{\text{opt}} + 0.1] = [-0.1, 0.1]$ .

In accordance with our analysis for the extreme case, plasticity expedites the first part of evolution for highly peaked fitness functions. As we smooth the fitness map, i.e., increase the variance of the Gaussian function, the early benefits of plasticity remain, but time taken to first encounter with the optimal phenotype increases. Also predicted earlier, plasticity does worse in the second and third phases of the trajectory than nonplasticity. The disadvantage of plasticity in the third stage diminishes slightly as the fitness function become wider.

## CONCLUSIONS

The Baldwin expediting effect fails under a simple quantitative genetic characterization of plasticity. Other studies of evolutionary rates have further demonstrated that the effects of plasticity are highly dependent on the conception of plasticity. Whitlock, for example, models the evolution of niche breadth as a product of evolutionary rate trade-offs. The model's predictions about specialism

and generalism can easily be translated into claims about nonplasticity and plasticity. His primary result is that benefit of specialization, and analogously of nonplasticity, is a faster rate of evolution (Whitlock, 1996).

Plasticity seems to expedite evolution under restrictive conditions. We can state sufficient conditions for a limited version of the Baldwin expediting effect. These criteria must be less ambitious than the general claim that plasticity accelerates evolution.

[BEE1] Plasticity expedites the search from an initial population distribution to the first encounter with the optimum phenotype.

[BEE2] [BEE1] is observed for initial genotype distributions sufficiently distant from the target.

In our evolving plasticity model there is a large class of fitness maps under which these conditions hold. For the extreme case and highly peaked Gaussian fitness functions, both analysis and simulation exhibit this restricted Baldwin expediting effect. As the kurtosis drops, the effect dissipates.

Evolution towards an ideal phenotype, however, involves more than the initial search. In many cases plasticity retards evolution from the first encounter until fixation of the population on an optimal phenotype. When plasticity is not sufficiently costly, it protects suboptimal phenotypes from elimination by natural selection. On the other hand, when plasticity is too costly, it fails to provide an advantage in the initial search. Furthermore, the regions of phenotype space in which plasticity speeds evolution are frequently regions in which a plastic individual is less fit than its nonplastic counterpart. These observations undermine the Baldwin expediting effect as an explanation for the evolutionary maintenance of phenotypic plasticity.

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