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CHAPTER 5

The Evolution of Thermal Sensitivity in Changing Environments

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1. Introduction

Temperature sensitivity is defined as the physiological or behavioral response of an organism to changing temperature conditions. A high degree of temperature sensitivity implies that a small change of temperature causes a dramatic response, whereas a low degree of sensitivity suggests that the response is small. Temperature can influence the fitness of an organism by directly causing mortality, or indirectly by reducing the performance capacity of the organism to forage, mate and reproduce. The tolerance curve, a plot of survivorship as a function of temperature, describes the temperature sensitivity of mortality, whereas a performance curve (fig. 5.1) describes the sensitivity of fitness-related performance (Huey and Kingsolver, 1989).

Performance traits include locomotory ability, foraging rate, growth rate, etc., that have a primarily additive influence on fitness within a generation. An organism can stop foraging, growing, or ovipositing when conditions are temporarily unfavorable and then start again when conditions improve, so fitness accumulates from one favorable time period to the next. In contrast, tolerance traits which directly impact mortality have a primarily multiplicative effect on fitness within a generation; when conditions are temporarily unfavorable, an organism cannot stop surviving and start again when times are better. Many plants and animals have evolved a rich array of tolerance traits, including dormancy, diapause and hibernation, that allow them to survive a very broad range of temperatures. The "preferred" temperature range where performance traits contribute to growth and reproduction is generally much narrower, even in species adapted to environments with significant daily and seasonal fluctuations (Andrewartha and Birch, 1954). Clearly it is important for an organism to have a broad tolerance curve in a variable environment, but why should growth or reproduction be restricted to a relatively narrow window of thermal conditions?

Physiological studies of the enzymatic basis of thermal sensitivity suggest that performance breadth may reflect a trade-off at the molecular level between catalytic efficiency and thermal breadth. Several studies demonstrate that the thermal stability of enzymes is positively correlated with the adaptation temperature of animals (reviewed in Somero, 1995). For example, the thermal stability of the glycolytic enzyme lactate dehydrogenase (LDH-A) in different vertebrate species increases over a range of body temperatures ranging from −1.86°C (Antarctic notothenoid fishes) to 47°C (desert iguana) (Somero et al., 1996). Conversely, ligand binding ability (estimated by the Michaelis–Menten constant for pyruvate for LDH-A) at 20°C is highest for the cold-adapted species and lowest for the warm-adapted species (Somero et al., 1996). Cold-adapted species, such as Antarctic fishes, are generally far more sensitive to increasing temperatures than are desert iguanas and other warm-adapted species.

These data suggest that thermal stability is inversely correlated with the rate of formation of enzyme-ligand complexes (Hochachka and Somero, 1984; Yancey and Siebenaller, 1987; Somero et al., 1996) such that "the jack-of-all-temperatures is master of none" (Huey and Hertz, 1996). Biological enzymes undergo rapid, reversible changes in protein conformation, however the stabilization of such enzymes against denat-

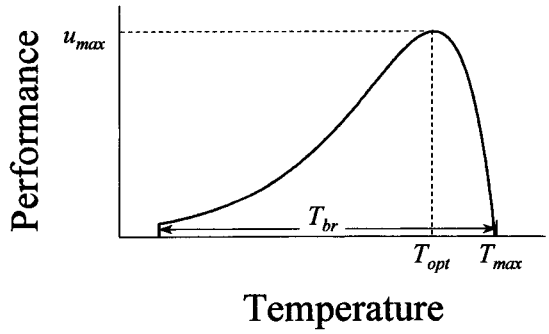

Fig. 5.1. Key parameters of the thermal performance curve described by the Logan et al. (1976) equation. The degree of thermal specialization is determined by performance breadth (T_{br}), the difference between T_{max} and T_{min}. T_{max} and T_{min} are the minimum and maximum temperatures at which performance is greater than zero, T_{opt} is the temperature at which performance is maximized (u_{max}).

uration involves stiffening the molecule through various noncovalent bonds. Somero (1995) argues that this limits the ability of organisms to achieve high levels of metabolic performance over a broad range of temperatures. The amino acid sequence in the active site of most enzymes is highly conserved across species with large differences in temperature sensitivity, but significant changes in thermal stability of proteins may require only a few substitutions in more variable parts of the polypeptide (Somero, 1995). More information on the comparative biology of enzyme structure is needed to determine whether or not such basic constraints at the molecular level can account for thermal constraints at the level of the whole organism.

Our limited mechanistic understanding of whole-organism thermal sensitivity has also constrained theoretical exploration of evolutionary patterns. Previous models of natural selection imposed by climatic variation have focused on the evolution of tolerance limits rather than performance curves. Richard Levins (1968) pioneered evolutionary models of physiological tolerance in changing environments, postulating the existence of a fundamental trade-off between "efficiency" and the range of nonlethal temperatures available to the organism. Lynch and Gabriel (1986, 1987) explored tolerance curve evolution in spatially and temporally variable environments with elegant models based on a haploid, asexual genetic system. These models show that temporal variation, and particularly variation at the within-generation (WG) timescale, favors the evolution of broad tolerance curves.

Optimality models of performance curve evolution assume that enhanced performance increases fitness through reproductive success and that the tolerance curve must be broader than the performance curve (Gilchrist, 1995). Fitness within generations is additive in performance curve models, whereas it is multiplicative in tolerance curve models. Constant environments obviously favor the evolution of performance specialists with a narrow range of preferred temperatures, but the results suggest that many patterns of within-generation (WG) and among-generation (AG) environmental variation also favor specialists (Gilchrist, 1995). But how rapidly can such specialists evolve under different temporal patterns of temperature variation? Optimality models (Gilchrist, 1995) show that selection for specialization in variable environments can be relatively weak. Evolution towards the optimum may also be hindered by low additive genetic variance as a result of constant abiotic selection pressures (Fisher, 1958; Lande, 1976; Turelli. 1984). Finally, functional constraints and the "jack-of-all-temperatures" trade-off within the performance curve itself may constrain the rate of evolution towards the optimum.

The model developed in this paper examines the evolution of both the performance curve and the genetic variation available for adaptive evolution. I model the performance curve using a physiological equation (Logan et al., 1976), treating the model parameters as quantitative genetic traits (Bulmer, 1980; Falconer, 1989) that evolve via a genetic algorithm. The results show how selection of varying intensity, imposed by the pattern of diurnal and seasonal changes in temperature, affects the rate of evolution and the maintenance of genetic variation in a finite population. Specifically, I will address how evolved genetic and intrinsic functional constraints might limit the rate of adaptation towards the physiological optimum.

2. The model

Imagine a population of insects inhabiting a seasonal environment where fitness depends on oviposition success in females and on mating success in males. Mates and oviposition sites are randomly distributed throughout the habitat. The rate at which each insect encounters oviposition sites or mates determines the frequency of oviposition and mating for that individual. Locomotion in insects and other ectotherms is strongly temperature-dependent (Casey, 1981), so fitness depends on an individual's performance over the distribution of temperatures encountered during its lifetime. The performance curve defines an individual's thermal dependence of locomotion. I assume that performance genes and a nongenetic component of variation combine to determine the phenotypic performance curve. Females and males mate at random and pass their performance genes on to their offspring. The number of eggs deposited or mates encountered during a lifetime determines an individual's fitness. Thus, individuals having a "good" performance curve for the environmental conditions they encounter will contribute more offspring to the following generation. The simulation is a model of mutation-selection balance (Lande, 1976; Turelli, 1984), with a constant mutational input and a temporally varying intensity of selection.

2.1. *The environmental model*

The seasonal change in mean environmental temperatures is modeled as a sine wave with a periodicity of $2\pi = 360$ days. The mean temperature on a given day is given by:

$$\bar{T} = \delta[\sin(date \times (\pi/180)) - 1/2] + 20, \quad (5.1)$$

where δ is the seasonal range of mean temperatures. The "active season" for the simulated insects covers a 180-day period (fig. 5.2). Each day is divided into 48 15-min periods that follow a normal distribution of temperatures, with a specified standard deviation, σ. The simulations were run under all combinations of $\delta = 0.0, 10.0$ or 20.0°C, and $\sigma = 0.5, 4.5$ or 16.5°C.

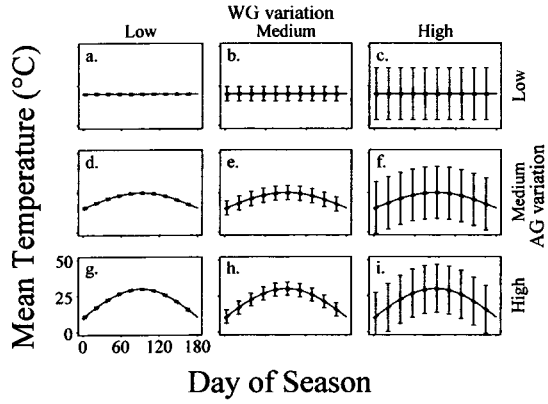

Fig. 5.2. The modeled patterns of WG and AG environmental variation over the course of a 180-day active season. The points denote the duration of each generation. WG variation, represented by the error bars, is modeled as the standard deviation of the distribution of temperatures within a day, with L = 0.5°C, M = 4.5°C and H = 16.5°C. AG variation is modeled as a seasonal range of temperatures, with L = 0.0°C, M = 10.0°C and H = 20.0°C.

2.2. *The organismal model*

The model begins with a population of 500 female and 500 male organisms. Each individual has two polygenic traits, a performance breadth (T_{br}) and a maximum temperature for performance (T_{max}). T_{max} serves to position the performance curve along the temperature axis; it is positively correlated with T_{opt}, the optimum temperature for performance (Gilchrist, 1995). Although there is no genetic correlation between these traits, the asymmetry of the performance curve imposes a functional correlation since you cannot optimize T_{max} independently of T_{br} (see Section 4). These traits determine the thermal sensitivity of mating success for males and oviposition success for females via the performance curve (fig. 5.1), given by the Logan et al. (1976) equation for insect temperature dependence:

$$f(T_i, T_{br}, T_{max}) = \Psi[e^{\rho(T_i - T_{min})} - e^{((\rho \cdot T_{br}) - 1.2\rho(T_{max} - T_i))}], \quad (5.2)$$

where

$$T_{\min} = T_{\max} - T_{\mathrm{br}}, \tag{5.3}$$

and T_i is the instantaneous body temperature. The constant Ψ (set at 4.0 in all cases) determines the minimum level of performance at $T_{\min}$. The model assumes a trade-off between performance breadth and maximum performance at the optimum, such that the area, A, beneath the performance curve is held constant (Levins, 1968; Gilchrist, 1995). This trade-off is mediated by the parameter ρ, which corresponds roughly to a Q_{10} of the organism (Logan et al., 1976). An analytical expression for ρ cannot be obtained, so a numerical solution to the expression:

$$0 = \psi\left[\frac{1}{\rho}(\mathrm{e}^{\rho\cdot T_{\mathrm{br}}} - 1.0) - \frac{0.8\bar{3}}{\rho}(\mathrm{e}^{\rho\cdot T_{\mathrm{br}}} - (\mathrm{e}^{-2.0\rho\cdot T_{\mathrm{br}}})\right] - A, \tag{5.4}$$

was obtained using the van Wijngarden–Dekker–Brent method for finding roots (Press et al., 1988).

The phenotypic variance, V_P, can be statistically partitioned into several sources of variance using standard quantitative genetics techniques (Falconer, 1989) such that

$$V_P = V_A + V_S + V_M + V_E. \tag{5.5}$$

V_A is the additive genetic variance and V_S is the segregational variance that arises through the random assortment of the chromosomes at meiosis (Bulmer, 1980). V_M is the variance introduced by random mutation (Lynch, 1988) and V_E is environmental variance. The model assumes that each of these components is independent and that there are no epistatic interactions among loci. The phenotypic values for each individual's traits in generation 0 were determined by the sum of a random draw from a normal distribution ($\sim N$ (mean, std. dev.)) of genetic variance (V_A : $T_{\mathrm{br}} \sim N(22.0, 0.5)$; $T_{\max} \sim N(31.0, 1.0)$) and a random draw from the environmental distribution (V_E : $T_{\mathrm{br}} \sim N(0, 0.5)$; $T_{\max} \sim N(0, 1.0)$).

Offspring performance traits were determined by summing genetic and environmental values, where the genetic value of an individual depends upon its maternal and paternal genotypes. The breeding value of an individual is defined as the deviation of its genetic value from the genetic mean of the population (Falconer, 1989). It describes the average effect of the individual's genes on a trait, relative to the population mean. In these models, the genetic and environmental values of each individual are known so the breeding value is obtained directly from the parents:

$$\alpha_j = z_j - \epsilon_j - \bar{z}. \tag{5.6}$$

The phenotype of offspring j is the sum of the population mean, three genetic components and an environmental component:

$$z_j = \bar{z} + \frac{\alpha_F + \alpha_M}{2} + \sigma_j + \mu_j + \epsilon_j, \tag{5.7}$$

where $\bar{z}$ is the population mean and α_x is the female or male breeding value of the parents. Two additional terms contribute to the total genetic value of the offspring: $\sigma_j \sim N(0, 0.5 \cdot V_A)$ is the added genetic value due to segregation (Bulmer, 1980), $\mu_j \sim N(0, 0.01 \cdot V_E)$, is the added genetic value due to polygenic mutation (Lynch, 1988). The final term, $\epsilon_j \sim N(0, V_E)$, is the added phenotypic value due to environmentally induced variation. The environmental variance was held constant at the initial level throughout the simulations. Genetic disequilibrium generated between the pair of traits by selection is accounted for by using individual genotypes to compute breeding values.

Male fitness was determined by assigning each male a number of mating opportunities, based on his performance phenotype. The lifetime mating success was computed by integrating the product of the performance curve and the environmental distribution:

$$L = \int_{T_{\min}}^{T_{\max}} F_n(t) \cdot f(T, T_{\mathrm{br}}, T_{\max})\,\mathrm{d}T. \tag{5.8}$$

For the parameters used here, the maximum value of this integral is 100 matings. Females selected males at random without replacement,

with a male's probability of being selected determined by his proportion of the population's unused mating opportunities (so male fitness is frequency-dependent). Females mated only once, so if a male obtained a mating, he sired all of that female's eggs. Female fitness was determined by an integration similar to that in eq. (5.8). Females could oviposit a theoretical lifetime maximum of 100 eggs.

Because of the large number of individuals involved and the limitations on computing capacity, excess offspring were terminated randomly so that no more than 500 males and 500 females formed the parental population in each generation. Population size was free to drop below 1000, but the starting conditions were chosen so that this occurred quite rarely. Each run of the model was for 20,000 generations; 5 replicates were run for each of the nine patterns of WG and AG environmental variation.

I recorded the population mean, phenotypic variance, and heritability ($h^2 = [V_A + V_s + V_M]/V_P$) for T_{br} and T_{max} in each generation. Separate records were kept for each sex. Performance breath and maximum temperature are genetically independent in this model; functional constraints, however, produce a positive phenotypic correlation. By the performance functions employed here, if two performance curves differ in breadth but share an identical optimum temperature, the broader curve will have a higher T_{max}. The strength of selection was measured with the standardized directional and stabilizing selection coefficients, or "gradients" for each trait by sex, as outlined by Lande and Arnold (1983). Selection coefficients are computed by partial linear regression of relative fitness on the values of the traits within a generation.

Both measures of selection are computed on the realized fitness; that is, the array of offspring phenotypes remaining *after* both fecundity selection and random mortality. For directional selection, negative coefficients indicate selection for smaller trait values whereas positive coefficients denote selection for larger trait values. Negative values of the stabilizing selection coefficient indicate selection for decreasing the variance, whereas positive values indicate selection to increase the variance of the trait.

The analyses in this paper are based on two subsets of the full data sets. The summary dataset consists of the mean and the variance among the replicates for each combination of WG and AG temperature variation for each variable outlined above. These data are used for computing trends and means over long stretches of the simulation. The second subset consists of the last ten generations of each replicate in each of the nine thermal environments; this is used for looking at detailed variation within a year and among the replicates.

3. Results

There were no sex differences or differences among generations in the means or heritabilities of T_{br} or T_{max} (tables 5.1–5.3), however, both directional and stabilizing selection differ between the sexes. This is because males were more variable in lifetime fitness than females. The variances for male mating success and female oviposition success were similar. A male's fitness, however, was affected by both his success in mating and his partner's oviposition success, whereas all females had one mating and varied only in oviposition success. T_{max} and T_{br} were positively and significantly correlated in all environments (table 5.4). Unless stated otherwise, only female data is plotted in the figures that follow.

In fig. 5.3, I have traced the fitness landscape for each environment as a contour map (obtained by the methods described in Gilchrist, 1995) and superimposed the trajectory of the evolving population. Each arrow (and each intervening space) spans 2000 generations of evolution. The starting point (T_{br} = 22.0°C, T_{max} = 31.0°C) of the simulations represents a combination of T_{br} and T_{max} that overlaps some of the environmental conditions encountered in each generation across all environments.

The peak on each fitness landscape indicates the phenotypic values of T_{max} and T_{br} that yield the highest geometric mean fitness over an an-

Table 5.1. Means and standard deviations of the five replicates. The score for each replicate was the arithmetic mean over the final 10 generations

		Females				Males			
WG	AG	T_{br}		T_{max}		T_{br}		T_{max}	
Mean:									
L	L	1.	36 ± 0.04	20.	39 ± 0.08	1.	36 ± 0.05	20.	39 ± 0.08
M	L	1.	96 ± 0.37	20.	70 ± 0.23	1.	96 ± 0.37	20.	70 ± 0.22
H	L	15.	39 ± 5.35	27.	98 ± 3.35	15.	39 ± 5.36	27.	98 ± 3.35
L	M	10.	44 ± 0.27	25.	10 ± 0.09	10.	43 ± 0.26	25.	11 ± 0.10
M	M	4.	82 ± 2.84	22.	48 ± 1.47	4.	82 ± 2.84	22.	47 ± 1.48
H	M	17.	15 ± 3.39	29.	25 ± 1.64	17.	15 ± 3.38	29.	25 ± 1.65
L	H	22.	34 ± 0.28	32.	34 ± 0.19	22.	34 ± 0.28	32.	33 ± 0.19
M	H	15.	75 ± 1.67	29.	23 ± 0.98	15.	74 ± 1.67	29.	23 ± 0.97
H	H	8.	36 ± 3.42	25.	36 ± 1.31	8.	36 ± 3.42	25.	35 ± 1.30
Heritability:									
L	L	0.	08 ± 0.02	0.	06 ± 0.01	0.	08 ± 0.02	0.	06 ± 0.01
M	L	0.	20 ± 0.11	0.	17 ± 0.10	0.	20 ± 0.11	0.	18 ± 0.10
H	L	0.	32 ± 0.25	0.	35 ± 0.16	0.	32 ± 0.25	0.	36 ± 0.17
L	M	0.	15 ± 0.05	0.	13 ± 0.03	0.	15 ± 0.04	0.	13 ± 0.03
M	M	0.	27 ± 0.12	0.	25 ± 0.15	0.	27 ± 0.12	0.	26 ± 0.16
H	M	0.	39 ± 0.26	0.	37 ± 0.15	0.	39 ± 0.27	0.	37 ± 0.16
L	H	0.	29 ± 0.11	0.	17 ± 0.07	0.	28 ± 0.11	0.	17 ± 0.08
M	H	0.	29 ± 0.08	0.	23 ± 0.16	0.	29 ± 0.07	0.	22 ± 0.16
H	H	0.	35 ± 0.10	0.	39 ± 0.19	0.	39 ± 0.10	0.	39 ± 0.18
Directional Selection coefficient:									
L	L	−1.	65 ± 0.51	0.	10 ± 0.01	0.	05 ± 0.16	0.	06 ± 0.01
M	L	0.	01 ± 0.01	0.	06 ± 0.00	−0.	01 ± 0.08	0.	06 ± 0.01
H	L	0.	04 ± 0.02	0.	03 ± 0.01	0.	05 ± 0.02	0.	02 ± 0.02
L	M	0.	06 ± 0.04	0.	04 ± 0.01	0.	03 ± 0.02	0.	04 ± 0.00
M	M	0.	03 ± 0.02	0.	05 ± 0.01	0.	03 ± 0.05	0.	05 ± 0.01
H	M	0.	05 ± 0.03	0.	02 ± 0.01	0.	05 ± 0.03	0.	02 ± 0.01
L	H	0.	07 ± 0.02	0.	00 ± 0.01	0.	00 ± 0.04	0.	04 ± 0.02
M	H	0.	06 ± 0.01	0.	02 ± 0.00	0.	05 ± 0.02	0.	02 ± 0.01
H	H	0.	05 ± 0.01	0.	03 ± 0.01	0.	04 ± 0.04	0.	03 ± 0.01
Stabilizing Selection coefficient:									
L	L	5.	31 ± 1.04	−0.	41 ± 0.02	0.	01 ± 0.22	0.	00 ± 0.01
M	L	0.	08 ± 0.04	−0.	02 ± 0.01	0.	02 ± 0.05	−0.	00 ± 0.01
H	L	0.	00 ± 0.01	−0.	00 ± 0.00	0.	04 ± 0.05	−0.	01 ± 0.01
L	M	0.	08 ± 0.03	−0.	02 ± 0.01	0.	01 ± 0.03	−0.	00 ± 0.01
M	M	0.	01 ± 0.02	−0.	00 ± 0.01	0.	02 ± 0.04	0.	00 ± 0.01
H	M	−0.	00 ± 0.01	−0.	00 ± 0.00	0.	02 ± 0.06	−0.	00 ± 0.01
L	H	0.	01 ± 0.02	−0.	00 ± 0.01	−0.	01 ± 0.02	0.	00 ± 0.01
M	H	−0.	00 ± 0.02	−0.	00 ± 0.01	0.	01 ± 0.03	−0.	01 ± 0.01
H	H	−0.	00 ± 0.02	0.	00 ± 0.00	−0.	02 ± 0.01	0.	00 ± 0.01

Table 5.2. Performance breadth ANOVA tables for the final 10 generations. The model is a nested ANOVA, with the effects of WG and AG variation nested within generations

	df	Sum of sq	Mean sq	F value	P
Phenotypic mean:					
Sex	1	0.000	0.000	0.001	0.979
Gen	9	0.001	0.000	0.001	1.000
WG-in-gen	20	125.685	6.284	92.537	0.000
AG-in-gen	20	320.112	16.006	235.687	0.000
WG:AG-in-gen	40	356.582	8.915	131.270	0.000
Residuals	809	54.939	0.068		
Heritability:					
Sex	1	0.000	0.0000	0.0001	0.9933
Gen	9	0.061	0.0068	0.0259	1.0000
WG-in-gen	20	71.038	3.5519	13.6138	0.0000
AG-in-gen	20	64.948	3.2474	12.4466	0.0000
WG:AG-in-gen	40	23.830	0.5958	2.2834	0.0000
Residuals	809	211.072	0.2609		
Directional selection coefficient:					
Sex	1	6.978	6.978	48.521	0.000
Gen	9	12.217	1.357	9.438	0.000
WG-in-gen	20	23.578	1.179	8.197	0.000
AG-in-gen	20	24.863	1.243	8.644	0.000
WG:AG-in-gen	40	39.967	0.999	6.948	0.000
Residuals	809	116.350	0.144		
Stabilizing selection coefficient:					
Sex	1	80.875	80.875	82.188	0.000
Gen	9	1.215	0.135	0.137	0.999
WG-in-gen	20	160.551	8.028	8.158	0.000
AG-in-gen	20	164.234	8.212	8.345	0.000
WG:AG-in-gen	40	305.972	7.649	7.774	0.000
Residuals	809	796.077	0.984		

nual environmental cycle. The steepness of the fitness surface along the T_{max} axis (indicated by the close spacing of the contour lines) is due to a rapid transition between phenotypes that never overlap the environmental conditions (and therefore have zero lifetime fitness) and those that can reproduce at least some of the time. The fitness landscape for the constant environment (fig. 5.3a) is extremely steep; increasing the amount of AG temperature variation broadens the peak somewhat (e.g. Fig 3d, g), but it is WG variation that has the most dramatic effect in flattening the landscape (e.g. fig. 5.3b, c). The difference is even more dramatic than it appears in fig. 5.3. The numbers in the upper left corner of each panel give the number of fitness units between the contour lines; there are 10 fitness units between the lines in fig. 5.3a, but only 0.5 units in fig. 5.3c, f, i. All else being equal, a steeper fitness landscape implies stronger selection and, ultimately, less variation about the optima in an equilibrium population.

Performance breadth (fig. 5.4) and T_{max} (not shown, but similar to fig. 5.4) changed substantially during the course of the simulations. The nine plots correspond to the nine environmental

Table 5.3. T_{max} ANOVA tables for the final 10 Generations. The model is a nested ANOVA, with the effects of WG and AG variation nested within generations

	df	Sum of sq	Mean sq	F value	P
Phenotypic mean:					
Sex	1	0.000	0.000	0.000	0.995
Gen	9	0.000	0.000	0.013	1.000
WG-in-gen	20	2.892	0.145	57.123	0.000
AG-in-gen	20	8.461	0.423	167.125	0.000
WG:AG-in-gen	40	9.761	0.244	96.402	0.000
Residuals	809	2.048	0.002		
Heritability:					
Sex	1	0.013	0.013	0.056	0.813
Gen	9	0.063	0.007	0.030	1.000
WG-in-gen	20	206.818	10.341	44.131	0.000
AG-in-gen	20	30.854	1.543	6.584	0.000
WG:AG-in-gen	40	26.417	0.660	2.818	0.000
Residuals	809	189.568	0.234		
Directional selection coefficient:					
Sex	1	0.001	0.001	0.156	0.692
Gen	9	1.687	0.188	64.892	0.000
WG-in-gen	20	1.525	0.076	26.392	0.000
AG-in-gen	20	1.026	0.051	17.759	0.000
WG:AG-in-gen	40	1.065	0.027	9.216	0.000
Residuals	809	2.337	0.003		
Stabilizing selection coefficient:					
Sex	1	0.553	0.557	94.377	0.000
Gen	9	0.017	0.002	0.333	0.964
WG-in-gen	20	0.992	0.050	8.476	0.000
AG-in-gen	20	1.006	0.050	8.590	0.000
WG:AG-in-gen	40	1.667	0.042	7.118	0.000
Residuals	809	4.737	0.006		

profiles depicted in fig. 5.2 and the nine fitness landscapes in fig. 5.3; the error bars indicate ± one standard deviation among the replicate populations. The dotted line indicates the location of the optimal breadth for each environment. Whereas the traits in environments a, b, d, g and h have approached their optima, the remaining populations are evolving quite slowly and are still some distance from the optima after 20,000 generations of selection. This is especially true of the populations with a large degree of WG variation. Both main and interaction effects between WG and AG are significant for mean T_{br} (table 5.2) and T_{max} (table 5.3). The broadest breadths occur with the highest level of AG variation, with little variation within generations. As WG variation increases, narrower performance breadths are favored in the high AG environments. Temperature variation affects T_{max} in a similar way, with the conditions favoring broad performance breadths also favoring high maximum temperatures. Natural selection during the course of the simulation produced a strong positive phenotypic correlation between T_{br} and T_{max} under all environmental conditions (table 5.4).

Table 5.4. Kendall's correlation coefficients between T_{br} and T_{max} over a random sample of 1000 generations of the simulation. The asterisk indicates the significance level using a sequential Bonferroni adjustment

WG	AG	τ
L	L	0.536*
M	L	0.954*
H	L	0.877*
L	M	0.670*
M	M	0.975*
H	M	0.724*
L	H	0.558*
M	H	0.868*
H	H	0.914*

* $\alpha = 0.001$.

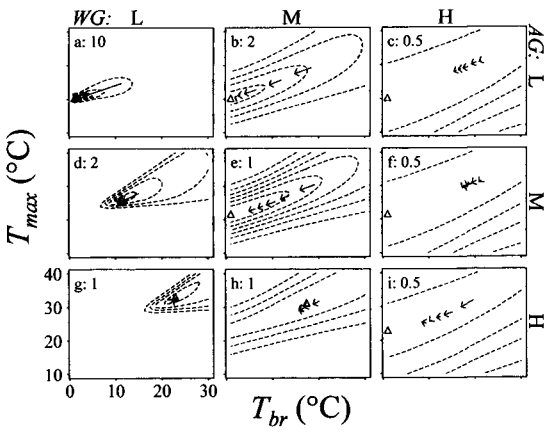

Fig. 5.3. The mean evolutionary trajectories of the populations in the nine environments pictured in fig. 5.1, superimposed on the fitness landscapes from Gilchrist (1995). The triangles mark the fitness peaks. The numbers adjacent to the letters identifying each box are the intervals, in fitness units, between the contours in that plot.

Genetic variation, as measured by the heritability, was present and highly variable over time in most populations throughout the simulation (fig. 5.5 for T_{br}; the plot for T_{max} was similar). The largest fluctuations are associated with increased WG temperature variation, with considerable heterogeneity in heritability estimates among populations even after 20,000 generations of selection. Both WG and AG temperature variation favor the maintenance of heritable variation (note the standard deviations for *Heritability* in table 5.1), as tested by the ANOVAs for T_{br} (table 5.2) and

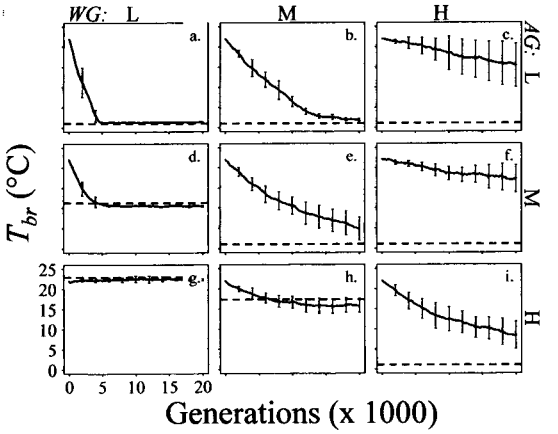

Fig. 5.4. The evolutionary trajectory of performance breadth over 20,000 generations in the nine environments pictured in fig. 5.1. The error bars give the standard deviation among the five replicate populations in each environment every 2000 generations. The dashed line indicates the location of the phenotypic optimum performance breadth.

T_{max} (table 5.3). Heritabilities were lowest in the populations occupying a constant environment (table 5.1). Increasing WG temperature variation generally decreases the intensity of both directional and variance selection. Table 5.5 shows the directional and variance selection coefficients (selection gradients; Lande and Arnold, 1983) in the first and sixth annual generations over the first 500 years of the simulations. These generations represent the extreme low and high mean temperatures in the seasonally varying environment during the years where selection was expected to be the strongest. The strength of directional selection decreases with increasing WG temperature variation, but increases with increasing AG variation, especially in the first annual generation (table 5.5(a)). Both directional selection and variance selection on performance breadth are stronger than on maximum temperature. Selection on males is about half the strength of that on females due to added variance in offspring number via female oviposition. Most interestingly, the directional selection coefficients for T_{br} and T_{max} are negatively correlated (table 5.6). Thus, when selection favors increasing T_{br}, it also favors decreasing T_{max}. Recall, however, that over the course of the simulation, a *positive* correla-

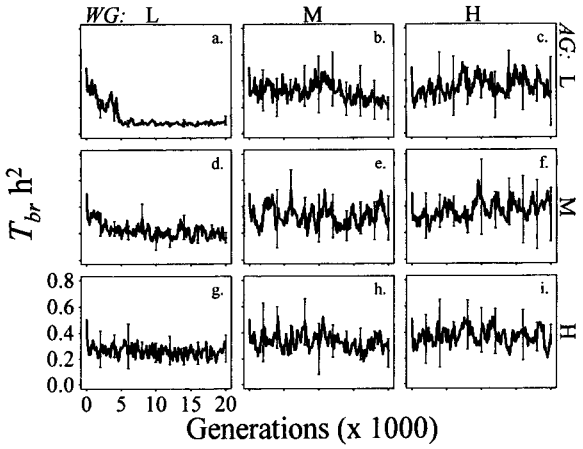

Fig. 5.5. The evolutionary trajectory of heritability for performance breadth over the 20,000 generations in the nine environments pictured in fig. 5.1. The error bars give the standard deviation among the five replicate populations every 2000 generations.

tion had evolved. This conflict will be discussed below.

4. Discussion

Understanding how natural patterns of daily and seasonal variation in temperature affect the evolution of temperature sensitivity requires consideration of several evolutionary forces (Lande, 1976; Turelli, 1984). Directional selection can alter the mean of a trait, while variance selection can alter its variability, provided there is adequate genetic variation present to allow a heritable selective response. Random genetic drift, resulting from sampling errors in a finite population, produces random changes in the mean and variance of a trait. While both natural selection and genetic drift generally deplete variation, mutation creates new genetic variation upon which selection can act. The simulation model presented here examines the role of temporal variation in selection, against the background level of mutation and random drift, in the evolution of thermal performance curves.

4.1. The evolution of tolerance curves and performance curves

Two previous models have examined the evolution of environmental sensitivity of tolerance curves (Levins, 1968; Lynch and Gabriel, 1987), mathematically equating fitness with viability or survivorship. The findings are that temporal variation within generations favors the evolution of broad tolerance curves, or "generalists"; AG variation also favors the evolution of generalists, but to a lesser degree. The result agrees with intuition and observation, however it does not explain the relatively narrow ranges of temperature that support growth and reproduction for so many species with broad thermal tolerances.

Performance curves are, like tolerance curves, a component of the niche of a species or population. The parameters for optimal performance curves in various thermal environments (computed by the methods described in Gilchrist, 1995), are shown by the contour plots in fig. 5.3. Specialists are favored in constant environments, and also in environments with significant WG temperature variation. Only AG variation favors the broadening of the performance curve. These results are dependent on two things. First, I have considered the environmental sensitivity of "fitness enhancing" traits, such as mating and oviposition, where the instantaneous contributions to fitness within each generation are additive rather than multiplicative. When WG fitness is multiplicative (as in Lynch and Gabriel, 1987), a single encounter with an unfavorable environment is catastrophic, whereas when additive, time spent in that environment simply does not contribute to lifetime fitness. The second factor is the assumed trade-off between specialist and generalist phenotypes. In a previous paper (Gilchrist, 1995), I examined the effects of relaxing the constant area assumption used in these models. Briefly, lifetime fitness is proportional to the area underneath the performance curve. If narrowing the curve increases the area, there is an added advantage to specialization that is independent of the environment. Similarly, if broadening the curve increases the area beneath

Table 5.5. (a) Mean directional selection coefficients; and (b) variance selection coefficients for T_{br} and T_{max} for the first 5000 years of simulations. The populations were sampled at the first and sixth generation each year

		Annual generation = 1				Annual generation = 6			
		Females		Males		Females		Males	
WG	AG	T_{br}	T_{max}	T_{br}	T_{max}	T_{br}	T_{max}	T_{br}	T_{max}
(a)									
L	L	−0.228	0.054	0.018	0.054	−0.251	0.056	−0.005	0.058
M	L	0.021	0.032	0.048	0.023	0.022	0.032	0.035	0.026
H	L	0.034	0.022	0.032	0.023	0.040	0.020	0.034	0.021
L	M	0.612	−0.169	0.346	−0.085	−0.307	0.153	−0.166	0.109
M	M	0.209	−0.054	0.032	0.027	−0.094	0.084	0.041	0.023
H	M	0.072	0.003	0.052	0.013	0.007	0.037	0.026	0.025
L	H	1.185	−0.566	0.384	−0.156	−0.309	0.197	−0.226	0.155
M	H	0.463	−0.179	0.132	−0.023	−0.209	0.138	−0.043	0.060
H	H	0.170	−0.038	0.131	−0.020	−0.064	0.068	−0.042	0.059
(b)									
L	L	0.148	−0.051	0.001	−0.003	0.160	−0.050	0.015	−0.005
M	L	0.016	−0.004	−0.006	0.002	0.004	−0.001	−0.002	−0.000
H	L	0.003	0.000	−0.000	−0.001	0.004	0.000	−0.003	0.001
L	M	−0.000	−0.003	−0.008	−0.001	0.081	−0.024	0.024	−0.007
M	M	−0.003	0.000	0.004	−0.002	0.017	−0.006	0.007	−0.003
H	M	0.005	−0.002	0.006	−0.002	0.003	−0.001	−0.001	−0.002
L	H	0.006	−0.005	−0.031	0.005	0.057	−0.014	0.001	0.000
M	H	−0.018	0.003	−0.006	−0.001	0.011	−0.001	−0.000	−0.001
H	H	−0.003	−0.001	0.012	−0.004	0.001	−0.000	0.003	−0.001

Table 5.6. Kendall's τ correlation coefficients between T_{br} and T_{max} for directional and variance selection coefficients. The stars give the significance level using a sequential Bonferroni adjustment. The data are a random sample of 1000 generations over the course of the simulation

		Females		Males	
WG	AG	Directional	Variance	Directional	Variance
L	L	−0.83***	−0.53***	−0.61***	−0.78***
M	L	−0.47***	−0.70***	−0.54***	−0.70***
H	L	−0.79***	−0.64***	−0.83***	−0.66***
L	M	−0.96***	−0.86***	−0.95***	−0.80***
M	M	−0.84***	−0.69***	−0.66***	−0.74***
H	M	−0.89***	−0.68***	−0.88***	−0.67***
L	H	−0.97***	−0.80***	−0.97***	−0.79***
M	H	−0.94***	−0.72***	−0.91***	−0.74***
H	H	−0.85***	−0.68***	−0.77***	−0.68***

* $\alpha = 0.05$; ** $\alpha = 0.01$; *** $\alpha = 0.001$.

it, then generalists are intrinsically favored. The critical conclusion, however, is that traits that primarily affect survivorship and mortality should differ in thermal sensitivity from traits that primarily affect fecundity and mating success. Reproductive specialists, not generalists, will be favored in most environments.

4.2. *Can temperature fluctuations maintain genetic variation?*

Intuition suggests that heterogeneity in the direction and strength of selection should counteract the depletion of genetic variation associated with stabilizing selection, but theory argues that the effect may be rare (Felsenstein, 1976; Hedrick et al., 1976; Hedrick, 1986; but see Ellner and Hairston, 1994). In the performance curve models, genetic variation in T_{br} and T_{max} is clearly maintained by both temporal components of environmental variation. The reported changes in heritability arise directly from changes in the additive components of variance (V_A, eq. (5.5)). WG and AG variation are both effective in maintaining genetic variation in T_{br} (table 5.2), however WG fluctuations contributed far more variation in T_{max} (table 5.3).

The change in performance breadth heritability over the course of the simulation is shown in fig. 5.5. All simulations began with $h^2 = 0.5$, and all experienced a sharp drop in genetic variation during the first 100–200 generations. Populations with moderate to high WG variation display a higher mean heritability over time (table 5.1), but also undergo large, aperiodic fluctuations in heritability (fig. 5.5). Within a single generation, the heritabilities among the five replicate populations within an environment might range from 0.2 to 0.8. A hundred generations later, a population with low genetic variation might rebound to a high level of variation. The fluctuations may arise from genetic drift in the face of weak directional and stabilizing selection. Two theoretical studies (Bürger et al., 1989; Houle, 1989) model the effects of stabilizing selection and mutation on polygenic variation in finite populations. When $N_e < 10^4$, both models predict large fluctuations in time and space in the levels of genetic variation, as demonstrated in the results presented here. Virtually no empirical study has measured heritability within a generation across several natural populations or within a population over many generations, so whether or not these wide fluctuations will actually be detected in nature is unknown.

4.3. *Why is performance curve evolution so slow?*

Selection clearly is weak in many environments (tables 5.1, 5.5), and often cannot be statistically distinguished from zero. Nonetheless, the trajectories of the population means in figs. 5.3 and 5.4 clearly show that all populations have moved towards their optima. The weak selection and slow evolutionary progress along the ridge of high fitness were to some degree anticipated by the flatness of the fitness landscape in some environments (fig. 5.3c, f, i). It is, however, a surprise in environments a, b, d and e (fig. 5.3), where the fitness landscape along the ridge top is somewhat steeper. The unexpectedly slow progress towards the optimum T_{br} or T_{max} is best illustrated in the most constant environment, where WG = L and AG = L and selection is the strongest. The mean directional selection coefficient over the first 5000 generations for female T_{br} is −0.228 (table 5.5(a)), with a mean heritability during this period of 0.2472. The response to selection in one generation is:

$$R = h^2 s, \tag{5.9}$$

or −0.0563 standard deviations per generation. With a mean standard deviation for T_{br} of 0.56°C over this time period, selection should carry the population from the 22°C starting T_{br} to the optimum at 1°C in less than 700 generations. In fact, it takes nearly 5000 generations for the populations to approach the optima (fig. 5.4a).

Several factors work to slow the pace of evolution. In seasonally variable environments, the direction of selection on the performance curve oscillates between negative and positive

during every year due to changes in temperature between generations. Cool temperatures (annual generation = 1) favor individuals with low T_{max}, whereas high temperatures (annual generation = 6) favor individuals with high T_{max} (tables 5.4, 5.6). Thus, the best phenotype at time t becomes the poorest a few generations later. While this may be a potent force in retarding the rate of evolution, it cannot explain the example outlined above, in which there is no seasonal variation.

Second, although a diverse range of models for the performance curve all yield similar results in terms of optima (Gilchrist, 1995), the details of the model have a dramatic effect on the rate of evolution. In particular, if the performance curve was modeled as a gaussian normal curve, the parameters (mean and variance) can evolve independently towards their individual optima. The parameters of the Logan model (or any asymmetric function) cannot be independently optimized; any change in one parameter forces the other off its optimum. Although more mathematically tractable, the normal performance curve does not represent biological reality; empirically measured thermal performance curves are always asymmetrical (Huey and Kingsolver, 1989).

Finally, a strong negative correlation exists between the selection coefficients in performance breadth and the thermal maximum for both directional and stabilizing selection in all environments (table 5.6). Yet the slope of the fitness ridge in all environments (fig. 5.3) and the correlation between T_{br} and T_{max} (table 5.3) are clearly positive. This positive slope has a simple explanation: when two performance curves are located at the same optimal temperature (T_{opt}), the broader one must, all else being equal, have a higher T_{max}. The negative correlation between the direction of selection and the direction of adaptation arises from the ridged fitness landscape. Random changes in the mean phenotype of a population can either move the population along the ridge or displace it to one side or the other. Selection to return the population to the ridge top is not only orthogonal to the direction of adaptation, but it is also stronger than selection moving the population towards the fitness peak, as indicated by the steep contours on the fitness landscape (fig. 5.3). Thus, the directional selection coefficients are dominated by evolution to maintain the population upon the ridge top rather than to move it towards the peak in the fitness landscape. The result is that selection within each generation is strongest in a direction orthogonal to the direction of adaptation. The weak selection towards the adaptive optimum coupled with the functional constraints that prevent independent optimization of the model parameters greatly decreases the rate of evolution in these performance traits (see Bossert, 1967, for a related perspective).

Ultimately, the validity of this model will depend on how genes encode the performance curve. Genetic models and selection experiments on reaction norms have produced divergent opinions about "plasticity genes" like the modeled genes controlling T_{br} (reviewed in Via et al., 1995). If, as Via (1985) has argued, plasticity is an epiphenomenon resulting from the expression of genes for performance under certain temperatures, then the models outlined here are irrelevant. If genes control the degree of plasticity, i.e. the breadth of the performance curve (Scheiner and Lyman, 1991; Scheiner, 1993), then the constraints identified here could be of importance in understanding both basic and applied physiological ecology (Lynch and Lande, 1993; Huey and Kingsolver, 1993).

4.4. Limitations of the model

The complexity of the diploid genetics, coupled with environmental variation on two timescales, precludes a simple analytical solution for this model of performance curve evolution. I presented numerical solutions to an optimality version of this simulation (Gilchrist, 1995). That model defined the fitness landscape and the optimal solutions under various patterns of temperature variation (figs. 5.3, 5.4), however it could not address the effect of genetic constraints on evolutionary dynamics. The model presented here is unique in that it uses the standard methods of evolutionary quantitative genetics within a genetic algorithm to model the evolution of two

polygenic traits. By this approach, I hope to complement existing and future empirical studies of temperature sensitivity.

The model assumes a simple polygenic inheritance and focuses on the response to selection in a finite population. A central assumption in the methodology of quantitative genetics is that a large number of loci determine the phenotype and that the phenotypic effects of the loci are normally distributed (Falconer, 1989). If relatively few loci or a few alleles with large effect determined thermal sensitivity, then the evolution of these traits might be quite different from that predicted here. These models assume no acclimational (Hochachka and Somero, 1984) or developmental (Maynard-Smith, 1985) constraints influencing the expression of the performance curve genotype. Mechanical and physical constraints on maximal performance are also ignored, except for the stipulation of a minimum performance curve breadth of 1.0°C.

The complication of overlapping generations, which may have a substantial influence on the maintenance of genetic variation (Sasaki and Ellner, 1997), is beyond the scope of this paper; the models here assume nonoverlapping generations. Some parallels exist between my model, Chesson's (1985) "storage effects" model, and Ellner's (Ellner and Hairston, 1994; Sasaki and Ellner, 1997) "seed bank" approach. In all three cases, genotypes "wait" for favorable environmental conditions. Chesson, however, focuses on the ecological consequences of different reproductive allocation strategies whereas Ellner focuses on the genetic consequences of dormancy across generations. My approach assumes a fixed dormancy strategy and focuses on the consequences of temporal variation within the "active" period of the lifecycle. Although the "organisms" modeled here have a very short lifespan, the general conclusions are broadly applicable to species with diverse life histories. Short-lived populations experience diurnal variation as WG variation and seasonal temperature changes as AG variation. Annual species, on the other hand, might experience variation on both timescales as WG and random fluctuations from year to year as the AG component. Informal explorations suggest that stochastic variation among generations produces similar results to the sinusoidal oscillations modeled here.

5. Conclusions

These genetic models show that oscillating selection imposed by daily and seasonal environmental variation is effective in maintaining heritable genetic variation for environmental sensitivity. Fluctuating temporal variation is common to all natural habitats and may contribute to the high heritabilities for fitness-related traits often found in natural populations (Mousseau and Roff, 1987). While the selection imposed by a variable environment is weak and may be statistically undetectable, its constant action over thousands of generations can result in significant evolutionary change, even in finite populations. The rates of evolution are even slower than might be expected due to conflicting selection pressures imposed by genotype-environment interactions; however, these same interactions may provide important help in maintaining genetic variation in populations.

Molecular study of the genes affecting "tolerance" and "performance" traits is very much needed. Do the molecular mechanisms that allow organisms to survive at extreme temperatures also affect the ability to develop, feed, mate, and oviposit under more moderate conditions? Can organisms generally evolve arrays of duplicated genes, each carrying alleles with different temperature sensitivities that allow them to overcome thermal trade-offs between lability and stability (Hochachka and Somero, 1984)? And if not, then why not? Can the aggregated properties of individual enzymes explain intra- and interspecific variation in temperature sensitivity? Hopefully, emerging methods in the molecular study of temperature responses will be applied to these fundamental questions that bridge the world of molecular biology, ecology and evolutionary physiology.

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