INTEGR. COMP. BIOL., 44:498–509 (2004)

Temperature, Growth Rate, and Body Size in Ectotherms: Fitting Pieces of a Life-History Puzzle[1]

MICHAEL J. ANGILLETTA, JR.,[2] TODD D. STEURY, AND MICHAEL W. SEARS[3]
Department of Ecology and Organismal Biology, Indiana State University, Terre Haute, Indiana 47809

SYNOPSIS. The majority of ectotherms grow slower but mature at a larger body size in colder environments. This phenomenon has puzzled biologists because classic theories of life-history evolution predict smaller sizes at maturity in environments that retard growth. During the last decade, intensive theoretical and empirical research has generated some plausible explanations based on nonadaptive or adaptive plasticity. Nonadaptive plasticity of body size is hypothesized to result from thermal constraints on cellular growth that cause smaller cells at higher temperatures, but the generality of this theory is poorly supported. Adaptive plasticity is hypothesized to result from greater benefits or lesser costs of delayed maturation in colder environments. These theories seem to apply well to some species but not others. Thus, no single theory has been able to explain the generality of temperature-size relationships in ectotherms. We recommend a multivariate theory that focuses on the coevolution of thermal reaction norms for growth rate and size at maturity. Such a theory should incorporate functional constraints on thermal reaction norms, as well as the natural covariation between temperature and other environmental variables.

INTRODUCTION

The relationships among environmental temperature, organismal growth, and adult body size have intrigued biologists for over a century, but a resurgence of interest in the last decade has been fueled by the discovery of widespread patterns in diverse taxa. Species distributed over broad geographic ranges often exhibit thermal clines in body size, with the majority of species exhibiting larger adult size in colder environments (Partridge and French, 1996; Ashton, 2004). This geographic variation in body size is consistent with the intraspecific version of Bergmann's rule, which states that races of a species tend to be larger in colder environments (for reviews of this concept, see Blackburn *et al.* [1999] and Ashton [2004]). Although genetic divergence in body size among populations is not uncommon (Partridge and Coyne, 1997), phenotypic plasticity is likely to be a major contributor to geographic clines in body size because lab studies have shown that a reduction in environmental temperature causes an increase in adult size in the majority of ectotherms studied to date (Atkinson, 1994, 1995; Atkinson *et al.*, 2003). This thermal plasticity of body size—dubbed *the temperature-size rule*—has been observed in bacteria, protists, plants, and animals, making it one of the most taxonomically widespread ''rules'' in biology. As with all biological ''rules,'' clear exceptions to Bergmann's rule and the temperature-size rule exist. Still, biologists have had more difficulty finding plausible explanations for these rules than they have had finding causes for exceptions.

Relationships between environmental temperature and life history in ectotherms have puzzled biologists because of the paradoxical effects of temperature on growth rate and size at maturity: lower temperatures cause ectotherms to grow slower but mature at a larger body size. In contrast, most optimization models predict a smaller size at maturity in environments that retard growth (reviewed by Berrigan and Charnov, 1994). Despite their slower rates of growth, individuals in cold environments can reach a relatively large body size by prolonging growth and delaying reproduction relative to individuals in hot environments; nevertheless, such a strategy decreases the probability that individuals in cold environments will survive to produce any offspring. Thus, we should be puzzled even if individuals in cold environments were to reach the same size as those in warm environments.

Although temperature-size relationships are puzzling, their widespread similarity suggests that a common cause exists. In the last decade, both theorists and empiricists have responded to the challenge of identifying this cause; both nonadaptive and adaptive explanations have been offered (Atkinson and Sibly, 1997). Nonadaptive theories describe how the effects of temperature on biochemical processes can give rise to the observed temperature-size relationship. Such theories, if they are to have generality, must also describe how some species are able to circumvent these physical constraints on growth and development. Adaptive theories use the costs and benefits of particular life histories to describe why, in most species, natural selection favors genotypes that grow faster but mature at a smaller size when raised at higher temperatures. Because such a diverse array of organisms are involved, biologists have mainly considered simple, univariate explanations which are seemingly more general than complex, multivariate ones (Atkinson, 1996). Despite some claims that the puzzle has been solved, we emphasize that simple theories have not provided a general explanation for temperature-size relationships. By

[1] From the Symposium *Evolution of Thermal Reaction Norms for Growth Rate and Body Size in Ectotherms* presented at the Annual Meeting of the Society for Integrative and Comparative Biology, 5–9 January 2004, at New Orleans, Louisiana.

[2] E-mail: m-angilletta@.indstate.edu
[3] Current address: Department of Biology, University of Nevada, Reno, Nevada 89557.

refuting some of these theories, however, we have made significant progress toward a solution. In the following sections, we review nonadaptive and adaptive theories of temperature-size relationships and consider their generality in light of emerging data. We then discuss a theoretical framework that could enable biologists to achieve a general theory by focusing on more complex, multivariate explanations. Our emphasis is placed on the thermal plasticity of body size, but the theories we discuss could also be used to explain the genetic divergence of body size along geographic thermal gradients.

NONADAPTIVE PLASTICITY OF BODY SIZE

Given the magnitude and diversity of organisms that conform to the temperature-size rule, one might reasonably hypothesize that some general physical constraints operating at the cellular and molecular levels play a causal role. In this spirit, van der Have and de Jong (1996) proposed that temperature-size relationships result from unequal effects of temperature on growth and differentiation. Specifically, they modeled rates of growth and differentiation according to the Sharpe-Schoolfield equation, assuming growth and differentiation are governed by independent systems of enzymes whose performances can be summarized by single rate-limiting steps. When the effect of temperature on the rate of differentiation is greater than its effect on the rate of growth, the model predicts that increasing temperature should lead to a decrease in the size at maturity. To make a connection between the model and real organisms, van der Have and de Jong assumed that differentiation is proportional to cellular division and that growth is proportional to protein synthesis. Importantly, they argued that DNA replication is more sensitive to temperature ($Q_{10} \approx 2$) because of the high diffusibility of DNA replicase, whereas protein synthesis is less sensitive to temperature ($Q_{10} \approx 1$) because of the low diffusibility of ribosomal subunits. Thus, the theory's explanatory power hinges on the assumption that the physical capacities for differentiation and growth are set by rates of different cellular processes.

If the thermal sensitivity (*i.e.,* the Q_{10}) of cellular division does exceed that of cellular growth, another possible cause is a reduction in the availability of oxygen for cells with increasing temperature. Woods (1999) used a biophysical model to demonstrate that the maximal size of a cell—the size at which oxygen concentrations should reach zero at its center—decreases with increasing temperature. Although data on oxygen gradients in cells and their consequences for cellular function are lacking (Woods, 1999), some observations suggest that the body size of organisms is limited by the biophysical mechanisms that govern the diffusion of oxygen. For example, Chapelle and Peck (1999) noted that an increase in the threshold size of benthic amphipods (*i.e.,* the size separating the largest 5% of species from the smallest 95% of species) is associated with an increase in the oxygen content of water among 12 locations, ranging from tropical to polar regions. By extending Wood's theory from the cellular level to the organismal level, one might hypothesize that ectotherms raised at higher temperatures reach a smaller final size because of biophysical constraints on the size of their cells.

However, biophysical constraints on cellular size cannot be a general explanation for the temperature-size rule because temperature can affect both the number and the size of cells at adulthood. An implicit assumption of the theory based on constraints on cellular size is that the number of cells in adults is the same at all temperatures; otherwise, no definitive end to differentiation would exist and the rate of cellular division would need not correlate with the duration of development. For species such as *C. elegans,* in which the number of cells at adulthood is constant, larger body sizes at lower temperatures are caused obviously by an increase in cellular size (Van Voorhies, 1996). But the generality of this theory depends on whether the number of cells at adulthood is insensitive to temperature in other species of ectotherms. The cellular basis of variation in body size has been explored most extensively in *Drosophila melanogaster*. Thermal plasticity of body size is caused primarily by variation in the size of cells in some populations (Partridge and French, 1996; James *et al.,* 1997; French *et al.,* 1998; Azevedo *et al.,* 2002) and by variation in the number of cells in others (Noach *et al.,* 1997). Likewise, the cellular basis of thermal clines in body size among genetically-distinct populations of *D. melanogaster* is inconsistent; an Australian cline is caused primarily by variation in the number of cells (James *et al.,* 1995, 1997), but a South American cline is caused by variation in both the size and number of cells (Zwaan *et al.,* 2000). Even within the South American cline, the relative contribution of variations in the number and size of cells to variation in the size of an organ differs among wings, eyes and legs (Azevedo *et al.,* 2002). Among 28 isofemale lines derived from three populations, both the number and size of cells contribute to variation in body size and their relative contribution varies among lines (de Moed *et al.,* 1997). A similar diversity of proximate mechanisms underlies latitudinal clines in the body size of *D. subobscura* (Calboli *et al.,* 2003). Apparently, no general cellular mechanism causes the thermal plasticity of body size. Instead, the outcome seems to depend on the genetic variation available prior to natural selection (Zwaan *et al.,* 2000; Calboli *et al.,* 2003).

ADAPTIVE PLASTICITY OF BODY SIZE

Two lines of evidence suggest that a larger body size in colder environments is adaptive. First, genetic divergence among geographically widespread species is consistent with patterns of phenotypic plasticity; individuals from colder environments often exhibit larger sizes at maturity than those from warmer environments when all are reared in a common environment (reviewed by Partridge and French [1996]; see also

van 't Land *et al.* [1999], and Morin *et al.* [1999]). Second, studies of natural selection in the laboratory have linked environmental temperature to the evolution of body size in *Drosophila melanogaster*, with low-temperature lines being larger than high-temperature lines (Partridge *et al.*, 1994). To reach a relatively large body size, individuals in cold environments must prolong growth and delay reproduction relative to individuals in warm environments. Such delayed maturation is adaptive when a colder environment enables a larger increment in fecundity or a higher rate of survival (Stearns, 1992). Although fecundity typically increases with increasing body size (Roff, 2002), individuals in cold environments are not likely to achieve relatively large increments in fecundity because they grow slower than individuals in warm environments. Therefore, adaptive explanations for the temperature-size rule have been based on the costs and benefits of particular life histories given thermal constraints on production or thermal effects on survival (Berrigan and Charnov, 1994; Atkinson, 1994, 1996). In this section, we summarize recent efforts to evaluate these theories.

Benefits of early maturation in warm environments

Predictions of life-history theory often depend on which estimate of fitness is chosen (Kozlowski, 1993). Two common estimates of fitness are the net reproductive rate (R_0) and the Malthusian parameter (r). In analyses of life-history strategies over discrete intervals of time, the finite rate of increase (λ) is considered instead of r, but these two estimates are interrelated ($\lambda = e^r$; Charlesworth, 1980). The use of R_0 is appropriate for a closed population at an equilibrium size (Roff, 2002), or for a spatially-structured population with an average growth rate equal to zero (Kawecki and Stearns, 1993). The use of r is appropriate when the growth of a population is unchecked; however, r is also maximized when multiple generations are possible within a single active season, and mortality during the inactive season is independent of the life-history strategy (Kozlowski, 1993). To maximize r, the optimal strategy is usually to mature as early as possible because of the advantage of compound growth (*e.g.*, see Sebens, 2002), as with the compounding of interest in monetary investments. If temperature constrains the timing of maturation, by affecting the minimal size for reproduction or by limiting the rate of gonadal development, the temperature-size rule can describe the optimal reaction norm for size at maturity in populations whose growth is compounded.

The ''compound interest hypothesis'' has been offered to explain why certain ectotherms mature earlier at a smaller size in warmer environments (Partridge and French, 1996; Fischer and Fiedler, 2002; Atkinson *et al.*, 2003). When reproduction is limited to a particular season, the ability to complete multiple generations during this season favors early maturation at a relatively small size whereas the inability to do so favors delayed maturation at a relatively large size. To evaluate this hypothesis, Fischer and Fiedler (2002) compared thermal reaction norms in two univoltine and two oligovoltine populations of the butterfly *Lycaena hippothoe*. Univoltine populations were characterized by reduced thermal sensitivity of adult size compared to oligovoltine populations. Nevertheless, smaller adults at higher temperatures were observed in all four populations. The compound interest hypothesis applies only to species in which generation times are constrained, perhaps by some minimal size at maturity. Otherwise, natural selection would favor the earliest possible age at maturity in all thermal environments, and the faster growth at a higher temperature would result in a larger size at maturity. Thus, studies of developmental constraints on age and size at maturity might shed light on the generality of this explanation.

Similarly, a thermal constraint on gonadal growth can favor smaller adult sizes at higher temperatures (Kindlmann and Dixon, 1992). The optimal reaction norm for size at maturity depends on the relative rates of energetic assimilation and gonadal growth; if higher temperatures increase the maximal rate of gonadal growth more than they speed the mass-specific rate of assimilation, size at maturity should decrease with increasing temperature. This result is contingent upon the choice of r as an estimate of fitness, such that the optimal strategy is to invest maximally in gonadal growth so as to benefit from the effect of compound interest. In support of the model, Kindlmann and Dixon (1992) presented evidence that temperature impacts embryonic growth rate (an estimate of gonadal growth) more than relative growth rate (an estimate of assimilation) in several species of aphids. But why should temperature affect rates of gonadal growth more than rates of assimilation? Certainly gonadal growth relies on energetic resources that stem from the process of assimilation. In the absence of a physiological mechanism for this constraint, we wonder whether thermal effects on embryonic and relative growth rates of aphids reflect adaptive strategies of allocation rather than a constraint on the maximal rate of gonadal growth. If so, thermal sensitivities of embryonic and relative growth rates are a consequence of a strategy that diverts resources from growth to reproduction earlier at higher temperatures, rather than a cause of this strategy. A convincing evaluation of Kindlmann and Dixon's model awaits the identification of a mechanism by which low temperatures restrict the rate of gonadal growth more severely than they restrict the rate of assimilation.

Benefits of delayed maturation in cold environments

A relatively large increment in fecundity through delayed maturation in cold environments is an implausible explanation for the temperature-size rule unless one assumes thermal constraints on maximal body size. Typically, delayed maturation provides a benefit of greater fecundity because fecundity increases with increasing body size (Stearns, 1992; Roff, 2002). If this benefit outweighs the cost of reduced survival to

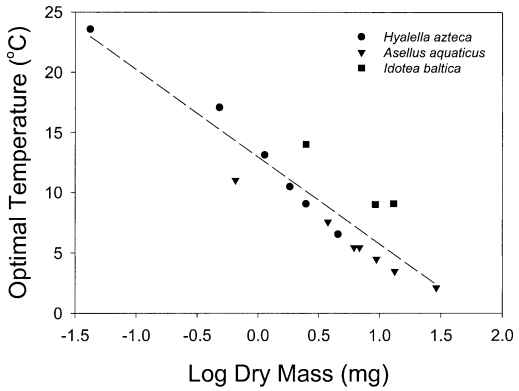

FIG. 1. In three species of isopods, the optimal temperature for growth rate decreased with increasing body size. Adapted from Panov and McQueen (1998).

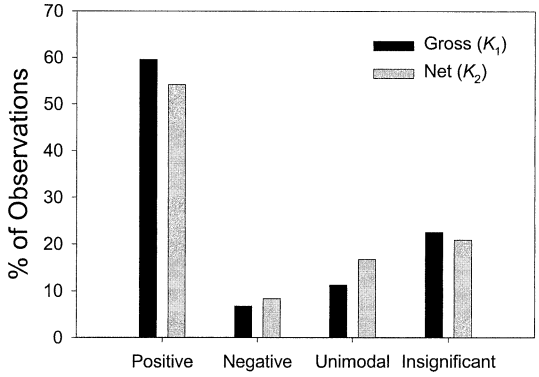

FIG. 2. In contrast to the assumption of Perrin (1995), the relationship between environmental temperature and the growth efficiency of an ectotherm is usually positive and is rarely negative. Relationships between temperature and growth efficiency (gross or net) were characterized as positive, negative, unimodal, or statistically insignificant. Data on gross and net growth efficiencies represent 89 populations of 53 species and 24 populations of 20 species, respectively. Adapted from Angilletta and Dunham (2003).

maturity, delayed maturation at a relatively large size will be favored by natural selection (Stearns, 1992; Roff, 2002). The problem is that delayed maturation should yield a relatively small increment in fecundity in cold environments because ectotherms grow slower at lower temperatures. However, thermal constraints on maximal body size can generate optimal reaction norms consistent with the temperature-size rule (Berrigan and Charnov, 1994; Atkinson, 1996; Kindlmann *et al.*, 2001). Such constraints limit growth at the end of ontogeny, and thus reduce the benefit of delayed maturation; since these constraints are assumed to be absent or less severe at low temperatures, the optimal reaction norm is a decrease in age and size at maturity with increasing temperature.

Thermal constraints on maximal body size have been explored using Bertalanffy's growth function (Bertalanffy, 1960), in which growth rate is the difference between rates of anabolism and catabolism:

$$\frac{dW}{dt} = aW^c - bW^d \qquad (1)$$

where W is body mass, a and b are coefficients of anabolism and catabolism, and c and d are exponents that define the allometry of anabolism and catabolism. Growth decelerates with age when $c < d$, and accelerates with age when $c > d$. Temperature can influence the maximal attainable body size by changing the coefficients or the exponents. Strong and Daborn (1980) suggested that smaller sizes at higher temperatures are caused by a decrease in c and an increase in d with increasing temperature. Similarly, Perrin (1995) showed that the optimal life history follows the temperature size-rule when the thermal sensitivity of catabolism (*i.e.*, the Q_{10} of b) is greater than the thermal sensitivity of anabolism (*i.e.*, the Q_{10} of a). Thermal effects on the coefficients and exponents are multiplicative, such that both can contribute synergistically to a reduction in body size at a higher temperature (Kozlowski *et al.*, 2004).

Available evidence supports Strong and Daborn's hypothesis that the allometries of anabolism and catabolism are differentially affected by temperature. In their study of the isopod *Idotea baltica* (Strong and Daborn, 1980), ingestion scaled almost isometrically ($c = 0.94$) at low temperature but allometrically ($c = 0.71$) at high temperature, whereas respiration scaled allometrically ($d = 0.68$) at low temperature and isometrically ($d = 1.00$) at high temperature. These thermal effects on allometry resulted in a decrease in the thermal optimum for growth rate throughout ontogeny. Consequently, individuals raised at high temperatures started out growing at a maximal rate and ended up growing at a sub-maximal rate, whereas those raised at low temperatures started out growing at a sub-maximal rate and ended up growing at a maximal rate. Similar trends have been observed in other species of aquatic ectotherms indicating that the phenomenon observed in *Idotea baltica* is not an isolated case (Fig. 1; see also Angilletta and Dunham, 2003). These ontogenetic shifts in the potential for growth favor early maturation in warm environments where growth decelerates with age and delayed maturation in cold environments where growth accelerates with age.

In contrast, empirical evidence does not support Perrin's hypothesis that the thermal sensitivity of catabolism is greater than the thermal sensitivity of anabolism. If this condition applies generally, growth efficiency of an ectotherm must decrease with increasing temperature. In a survey of more than 50 species, Angilletta and Dunham (2003) showed that growth efficiency usually increases with increasing temperature within the thermal range that is governed by the temperature-size rule (Fig. 2). Even when data for a particular species do support Perrin's hypothesis, the range of temperatures over which this mechanism can explain the temperature-size rule is only about 5°C. Therefore, the explanation put forth by Perrin (1995) only applies to a narrow range of temperatures in the minority of ectotherms studied to date.

A major problem with theories based on Bertalanf-

fy's growth function is that the mechanisms they assume are not sufficiently general to explain the temperature-size rule. In Perrin's model, a decelerating rate of growth must be assumed to produce an optimal strategy of a smaller size at a higher temperature. Yet, growth rates of juveniles are constant or accelerating in a diverse array of species, including cnidarians (Båmstedt *et al.*, 1999), mollusks (Laing *et al.*, 1987; Sicard *et al.*, 1999), crustaceans (Lellis and Russell, 1990; Pöckl, 1995; Lee *et al.*, 2003), insects (Tsitsipis and Mittler, 1976; Sweeney and Vannote, 1978; Ernsting *et al.*, 1992), fish (Imsland *et al.*, 1995; Martinez-Palacios *et al.*, 1996; Jonassen *et al.*, 2000; Bendiksen *et al.*, 2002), amphibians (Berven, 1982; Jørgensen, 1983), and reptiles (Dunham, 1978; Schoener and Schoener, 1978; Sears and Angilletta, 2003). Even Strong and Daborn's hypothesis, which accounts for accelerating growth at low temperatures is unsettling because this condition would favor growth to an infinite body size under a reasonable set of assumptions (Sevenster, 1995). Because many organisms do not grow asymptotically and no organism grows indefinitely, Bertalanffy's model is best treated as a phenomenological description of growth rather than a set of mechanisms that constrain growth. The parameters of Bertalanffy's model can be modified through thermal acclimation of morphology, behavior, and physiology (Sebens, 1987), such that any particular set of values are the consequence of a life-history strategy (albeit not necessarily an adaptive one). In fact, models of the optimal allocation of resources predict growth trajectories that are well described by Bertalanffy's growth function even when growth is not constrained by a maximal body size (Kozlowski *et al.*, 2004).

Although the assumption of decelerating growth is problematical for analyses of optimal age and size at maturity, the temperature-size rule can also result from the optimal allocation of resources when somatic and gametic production undergoes senescence (Kindlmann *et al.*, 2001). The senescence of production can result from a decrease in the ability to acquire resources or an increase in the required maintenance and repair with age. Kindlmann *et al.* (2001) assumed that production accelerated early in ontogeny but decelerated late in ontogeny because of senescence. Under these conditions, the growth of juveniles accelerates with age while the fecundity of adults decelerates with age. The optimal age and size at maturity depends on the rate of senescence; if senescence is faster at higher temperatures, early maturation is favored because it enables reproduction before senescence takes a major toll on fecundity. Kindlmann *et al.* (2001) claimed their model probably resolves the puzzling effects of temperature on body size. However, their conclusion depends on the validity of r as a criterion of fitness, the time-course of senescence, and the thermal sensitivities of assimilation, gonadal development, and senescence. As such, the generality of this explanation is presently unclear.

Atkinson and Sibly (1996) argued that, even when production accelerates with age, energetic costs associated with pupation, mating, or other activities of adulthood could favor the evolution of a smaller size at maturity. This idea seems like a potential cause for earlier maturation in warmer environments, but the argument is logically flawed. Fecundity should be higher for individuals that delay maturation despite the additional energetic costs associated with adulthood. For income breeders, delayed maturation will result in a higher rate of reproduction during adulthood because the rate of production accelerates with age. For capital breeders, delayed maturation will result in larger size at maturity, which provides more stored energy for reproduction. Therefore, the benefits of delayed maturation are unlikely to provide an explanation for the larger size of ectotherms in colder environments unless some convincing mechanism for a thermal constraint on maximal body size is discovered.

Costs of delayed maturation in warm environments

Since a higher survivorship of juveniles favors delayed maturation (Stearns, 1992; Roff, 2002), the temperature-size rule could be explained by a lesser risk of mortality at lower temperatures (Atkinson, 1994, 1995, 1996). Temperature can have both direct and indirect effects on the survivorship of juveniles. Direct effects are mediated by thermal sensitivities of development, physiology, and behavior. Indirect effects are mediated by changes in the quantity or quality of resources, competitors, predators, or parasites. In the latter case, temperature need only be a reliable indicator of these indirect effects for selection to favor reaction norms that are expressed in the absence of ecological factors (Perrin, 1988; Sibly and Atkinson, 1994). Finally, temperature might interact with other abiotic variables that impact survivorship, such as salinity or pH. Models that incorporate a risk of mortality predict optimal sizes at maturity that are below the sizes that maximize production (Perrin and Rubin, 1990; Perrin and Sibly, 1993; Sebens, 2002).

Very large thermal effects on survivorship are needed to produce an optimal reaction norm that conforms to the temperature-size rule (Myers and Runge, 1983). The necessary magnitude of the effect depends on the thermal sensitivity of production and the survivorship at low temperature (see Appendix). Typically, the Q_{10} of anabolism ranges from two to six, and the Q_{10} of catabolism ranges from one to three; consequently, production is slightly more sensitive to temperature than is anabolism (Table 1). Because production is very sensitive to temperature, a decrease in survivorship with increasing temperature does not guarantee that the optimal life history will accord with the temperature-size rule. Indeed, thermal sensitivities of survivorship must be quite large if they are to explain the generality of this rule (Fig. 3).

Are direct effects of temperature on survivorship large enough to explain the temperature-size rule? Because studies conducted in the laboratory exclude most ecological sources of mortality, they provide valuable

TABLE 1. *Thermal sensitivities of anabolism and catabolism in ectotherms.*

Species	Thermal Range (°C)	Q_{10} of Anabolism	Q_{10} of Catabolism	Q_{10} of Production	Reference
Acanthocyclops viridis	5–15	0.8	0.6	1.7	Laybourn-Parry *et al*. 1988
Argopecten ventricosus-circularis	16–19	9.3	2.7	12.9	Sicard *et al*. 1999
Daphnia middendorffiana	3.5–16	2.3	2.8	1.9	Yurista 1999
Macrocyclops albidus	5–20	1.1	0.9	1.4	Laybourn-Parry *et al*. 1988
Mytilus galloprovincialis	10–20	1.3	1.7	1.3	van Erkom Schurink and Griffiths 1992
Oncorhynchus mykiss	10–19	1.3	1.0	1.4	Myrick and Cech 2000
Ostrea edulis	5–15	5.7	0.8	7.8	Buxton *et al*. 1981
Ostrea edulis	5–15	10.8	3.3	11.3	Hutchison and Hawkins 1992
Ostrea edulis	14–20	3.5	2.9	3.6	Beiras *et al*. 1994
Palaemonetes pugio	15–30	2.2	1.0	6.8	Vernberg and Piyatiratitivorakul 1998
Papilio canadensis	12–30	1.8	1.8	2.3	Ayres and Scribner 1994
Perna perna	10–20	3.4	2.0	4.0	van Erkom Schurink and Griffiths 1992
Pinctada margaritifera	19–28	2.0	1.8	2.2	Yukihira *et al*. 2000
Pinctada maxima	19–28	3.2	2.0	4.2	Yukihira *et al*. 2000
Platichthys flesus	6–22	2.9	2.2	4.4	Fonds *et al*. 1992
Salmo trutta	5.6–12.8	10.5	9.0	13.4	Elliot 1976
Scottolana canadensis	20–28	2.8	2.1	3.4	Lonsdale and Levinton 1989
Simocephalus vetulus	8–28	1.8	1.3	1.9	Sharma and Pant 1984
Venerupis pullastra	10–20	6.4	3.8	7.4	Albentosa *et al*. 1994
Lower Quartile		1.8	1.1	1.9	
Upper Quartile		4.6	2.8	7.1	

Anabolism and catabolism were estimated from measures of net assimilation (or ingestion) and respiration, respectively. We excluded low temperatures at which rates of production were negative and high temperatures at which rates of production were less than maximal; therefore, estimates apply to thermal ranges described by the temperature-size rule.

information on the magnitude of direct effects. Atkinson (1994) examined the thermal effects on survivorship for 29 of the 109 cases in his data set, concluding that survivorship did not always decrease with increasing temperature. Expanding on Atkinson's analysis, we calculated the thermal sensitivities of survivorship for 130 populations of ectotherms, including 1 species of cnidarians, 4 species of annelids, 5 species of mollusks, 2 species of rotifers, 7 species of arachnids, 18 species of crustaceans, 54 species of insects, 21 species of fish, and 2 species of amphibians (data and references are available upon request). We excluded low temperatures at which rates of production were negative and high temperatures at which rates of production were less than maximal, and used daily survivorship at the two extremes to estimate a Q_{10}. Thus, our calculations provided conservative estimates of the Q_{10}'s of survivorship over the thermal range to which the temperature-size rule pertains. Although higher temperatures caused lower survivorship in 74 of 130 cases, thermal sensitivities of survivorship in most species were too small to explain smaller adult sizes at higher temperatures (Fig. 3). Thus, we can rule out direct effects on survivorship as a general explanation for the temperature-size rule. Instead, biologists should focus their attention on the mechanisms by which temperature influences ecological sources of mortality.

In natural environments, where ecological sources of mortality abound, environmental temperature is expected to have a much greater impact on the survivorship of juveniles. Presently, this impact can only be estimated by comparing rates of survival among populations or species distributed along thermal clines. Intraspecific comparisons suggest that absolute effects of temperature on survivorship are substantially greater in natural environments than they are in artificial environments (Strong and Daborn, 1980; Ebert *et al.*, 1999; Angilletta *et al.*, 2004). However, the Q_{10}'s of survivorship are still low because survivorship is relatively poor regardless of the environmental temperature. For example, the Q_{10}'s of survivorship range from 1.9 to 2.5 for fishes (Fig. 4) and copepods (Hirst and Kiørboe, 2002). An important caveat is that these examples pertain to early stages of the life cycle, which are prone to very high mortality. Either an increase in the mean survivorship or a decrease in the Q_{10} of production with increasing body size will decrease the thermal sensitivity of survivorship that is needed to explain the temperature-size rule. Thus, biologists should endeavor to characterize thermal sensitivities of size-specific rates of survival in natural or semi-natural environments. Although this goal might be attained through comparative studies, the manipulation of temperature in replicated mesocosms is a promising experimental approach.

LINKING REACTION NORMS FOR GROWTH RATE AND BODY SIZE

Existing theories demonstrate clearly that reactions norms for age and size at maturity should be strongly influenced by rates of production throughout ontogeny. When optimizing life histories, specific trajectories of growth have been assumed because either they reflect patterns observed in nature or they simplify analyses (*e.g.*, see Stearns and Koella [1986] or Berrigan and Charnov [1994]). Optimal reaction norms depend on assumptions about the thermal sensitivity of these growth trajectories. Alternatively, growth can be mod-

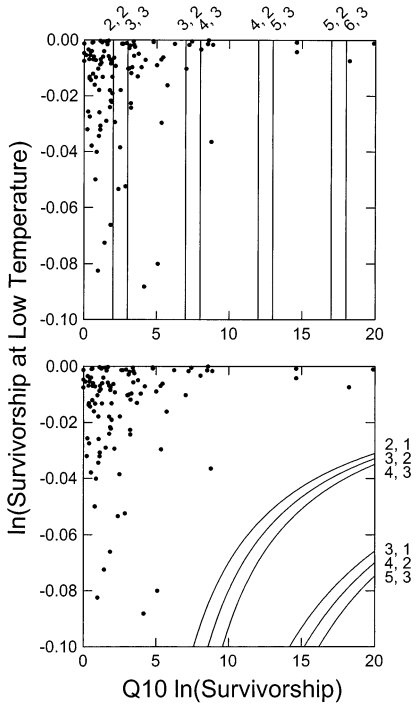

FIG. 3. The thermal sensitivity of juvenile survivorship needed to explain the temperature-size rule depends on the thermal sensitivities of anabolism and catabolism (see Appendix). The upper plot depicts the case in which the allometries of anabolism and catabolism are identical. The lower plot depicts the case in which anabolism scales allometrically with body mass (the exact relationship does not matter) and catabolism scales isometrically. The lines are isoclines for realistic combinations of thermal sensitivities of anabolism and catabolism, which are listed in the margin as [Q_{10} of anabolism, Q_{10} of catabolism]. To the right of each isocline, the optimal size at maturity decreases with increasing temperature in accord with the temperature-size rule. The points are thermal sensitivities of survivorship for 130 populations of 114 species of ectotherms (see text for details).

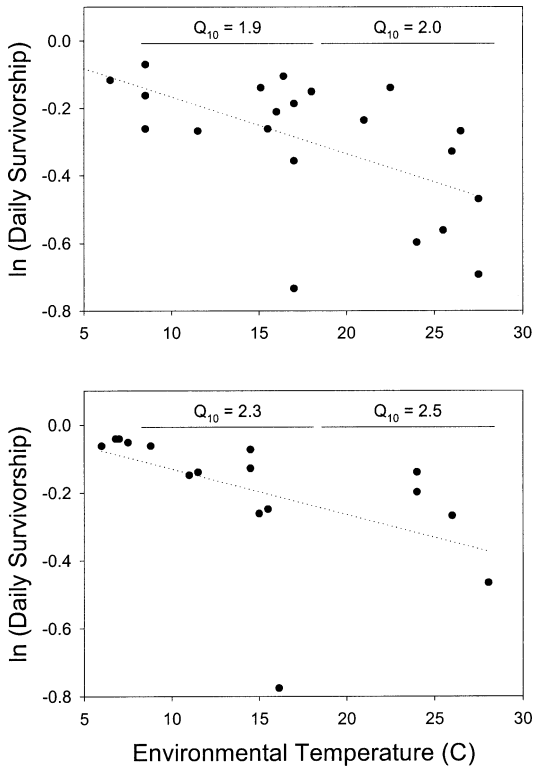

FIG. 4. Relationships between temperature and the natural logarithm of survivorship for larval (top) and post-larval (bottom) fishes in natural environments. Thermal sensitivities from 8° to 18°C and from 18° to 28°C were comparable to those observed for ectotherms raised in the laboratory (see text). Survivorships of larval and post-larval fishes were calculated from daily rates of mortality tabulated by Houde (1989) and Pepin (1991), respectively.

eled as the product of the allocation of energy among competing functions (Perrin and Sibly, 1993; Kozlowski, 1992). In models of energy allocation, the optimal reaction norm still depends on rates of energy assimilation throughout ontogeny (Kozlowski *et al.*, 2004). If assimilation is very sensitive to temperature, thermal effects on senescence or extrinsic mortality must be more extreme to favor smaller sizes at higher temperatures (Kindlmann *et al.*, 2001; Fig. 3). Therefore, thermal and allometric effects on production are key assumptions in all models of life-history evolution.

Although physical constraints on production have played a major role in previous attempts to explain the temperature-size rule, empirical evidence for these constraints remains controversial. Certain body plans place obvious restrictions on the rates of energy acquisition and assimilation at a given size (Sebens, 1987; Twombly and Tisch, 2000), but such constraints are temporary because they can be circumvented through evolutionary modifications of behavior, physiology and morphology. Some biologists have argued convincingly that allometric growth results from the allocation of energy to growth and reproduction rather than physical constraints on production (reviewed by Kozlowski *et al.*, 2004). Furthermore, comparative and experimental evidence strongly support the view that growth is a form of physiological performance whose relationship with temperature evolves by natural selection (Huey and Kingsolver, 1989; Angilletta *et al.*, 2002). Thermal sensitivities of growth rate vary considerably within and among species; among mollusks, arthropods, and fish, an increase in environmental temperature of 10°C results in a two- to sixteen-fold increase in growth rate (see Table 1 and additional references in Angilletta and Dunham, 2003). Therefore, allometric and thermal effects on growth rate cannot be viewed as constraints on the life history.

A better approach is to consider thermal reactions norms for growth rate and size at maturity in the context of a developmental reaction norm (*sensu* Schlichting and Pigliucci, 1998). The developmental reaction norm (Fig. 5) is a multivariate function linking the influence of temperature on growth rate (a labile trait) to age-specific body size (a fixed trait). As such, it provides an ontogenetic dimension to the study of temperature-size relationships. By focusing simultaneously on the evolution of thermal reaction norms for

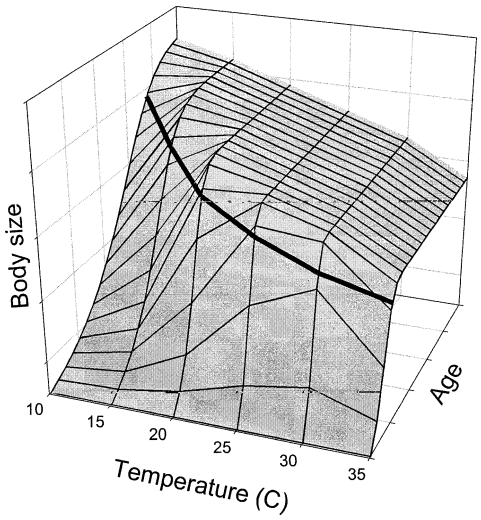

FIG. 5. A hypothetical multivariate reaction norm depicting the influence of environmental temperature on body size throughout ontogeny. Growth rate at a particular temperature is equal to the slope of the relationship between age and body size. The bold line depicts the thermal reaction norm for age and size at maturity. In this example, a lower temperature results in slower growth but a larger size at maturity, which accords with the temperature-size rule.

growth rate and size at maturity, one avoids unnecessary assumptions about constraints on growth that are common among current theories. Moreover, modeling the evolution of the developmental reaction norm forces one to confront factors that are commonly ignored in models of life-history evolution, including the roles of certain functional constraints and thermal heterogeneity in the evolution of thermal reaction norms.

Functional constraints on thermal reaction norms

Functional constraints, or tradeoffs (*sensu* Arnold, 1992), have a major influence on the evolution of the developmental reaction norm. Because growth is determined by rates of acquisition, assimilation and allocation, ectotherms can alter their rates of growth by numerous mechanisms (*e.g.,* see Bayne, 2004). Each mechanism involves a particular tradeoff that will influence the fitness of the organism (Angilletta *et al.*, 2003). For example, an ectotherm can grow faster by allocating a greater fraction of its available energy to growth. Because this increase in growth rate would occur at the expense of others functions, the individual would suffer a decrement in maintenance, activity, or reproduction (Stearns, 1992; Zera and Harshman, 2001). Alternatively, an ectotherm can grow faster by acquiring additional energy; this strategy would eliminate the need to divert resources from competing functions, but it would likely increase the risk of predation or parasitism (Werner and Anholt, 1993; Gotthard, 2000). Finally, ectotherms can grow faster through thermal specialization, which involves changes in physiology that determine the efficiency with which resources are assimilated and used for growth. However, specialization would increase growth rate at some temperatures while decreasing growth rate at other temperatures (Bennett ands Lenski, 1999; Yamahira and Conover, 2002).

Existing theories of evolution emphasize some tradeoffs while ignoring others. Models designed to explain the evolution of age and size at maturity emphasize tradeoffs that arise from the allocation of energy to competing functions (Kozlowski, 1992; Heino and Kaitala, 1999), but tradeoffs arising from the acquisition of resources for growth have also been considered (Gotthard, 2001). To our knowledge, tradeoffs related to both acquisition and allocation have not been considered simultaneously, and tradeoffs related to thermal specialization have been ignored entirely by life historians. Similarly, models designed to explain the evolution of thermal reaction norms for physiological performances, such as growth rate, emphasize tradeoffs arising from thermal specialization but ignore tradeoffs arising from acquisition and allocation (Huey and Kingsolver, 1993; Gilchrist, 1995). A wealth of comparative and experimental evidence suggests that all three kinds of tradeoffs play important roles in shaping thermal reaction norms (reviewed by Angilletta *et al.*, 2003). Therefore, theorists will need to incorporate these functional constraints in a general theory of temperature-size relationships.

Bringing natural (co)variation into focus

Optimal thermal reaction norms for growth rate depend on the temporal variation in environmental temperature and the manner in which growth contributes to fitness. If growth contributes additively to fitness, thermal specialists are favored under most patterns of temporal variation in environmental temperature; thermal generalists are favored only in environments where temperature varies greatly among generations and little within generations (Gilchrist, 1995). Consistent with this conclusion, thermal generalists are favored if environmental temperature changes systematically with time (Huey and Kingsolver, 1993). Thus, both variations within and among generations determine the optimal reaction norm if growth contributes additively to fitness. If growth is linked to thermal tolerance (and thus contributes multiplicatively to fitness), the thermal reaction norm is affected more by variation within generations than variation among generations; thermal specialists are favored in constant environments and thermal generalists are favored in variable environments (Lynch and Gabriel, 1987). Acclimation of the thermal reaction norm might also be favored if the environment varies spatially or temporally (Gabriel and Lynch, 1992). Because the pattern of environmental variation determines how natural selection acts on the thermal reaction norm for growth rate, an important task for biologists is to characterize these patterns and incorporate them into theories designed to understand temperature-size relationships.

The covariation between temperature and other environmental variables can also shape the developmental reaction norm. A higher temperature could be a reliable cue for increasing resources and hence signal

the opportunity for population growth (Atkinson *et al.*, 2003). On the other hand, if higher temperatures are usually associated with a scarcity of resources, the potential for growth observed in the laboratory might not be realized in nature. Higher temperatures could also be associated with greater risks of mortality through changes in the density and activity of predators (*e.g.*, see Lampert, 1989). If temperature covaries with the abundance of prey or predators in a particular manner, natural selection will favor reaction norms that have the greatest fitness under those conditions. This point is especially important because theory predicts that increased predation should have direct and indirect effects on size at maturity (Abrams and Rowe, 1996); the direct effect is a reduction in size because higher rates of mortality favor earlier maturation, whereas the indirect effect is an increase in size because predation decreases intraspecific competition for resources. Because resources are often limiting in nature, one might expect developmental reaction norms to be shaped by both direct and indirect effects of predation. Breaking the natural covariation among temperature, food availability, and predation risk can create a condition that never occurs in nature, which would lead to an erroneous interpretation of results (Bernardo, 1998). The way in which these variables interact to determine thermal reaction norms for growth rate and size at maturity (*e.g.*, see Weetman and Atkinson, 2002) might not make sense if one ignores the natural covariation. Presently, we know very little about this covariation in most populations of ectotherms suggesting an obvious need to pay more attention to the natural context in which temperature-size relationships have evolved.

FITTING PIECES OF THE PUZZLE

Current theories of nonadaptive or adaptive plasticity of body size in response to temperature are relatively simple, in that each focuses on only one or two of the mechanisms by which temperature can influence the life history. In reality, most variables are affected by temperature, and optimal reaction norms for age and size at maturity depend on the relative strengths of these thermal effects. Atkinson (1994) suggested that three thermal effects in particular were key to understanding temperature-size relationships: thermal constraints on maximal body size, thermal sensitivities of growth rate, and thermal sensitivities of juvenile survivorship. To this list, we add thermal effects on the frequency of reproduction and the survivorship of adults, which have not received serious consideration from life historians (but see Charnov and Gillooly, 2004). Because reproduction is typically less frequent in colder environments, natural selection could favor a larger body size to enhance fecundity at each reproductive episode. For similar reasons, a larger size at maturity might be adaptive if the survivorship of adults is lower in colder environments (Stearns and Koella, 1986). Finally, a larger body size could enable individuals to produce larger offspring or to provide better parental care, which are thought to be adaptive in colder environments (Perrin, 1988; Yampolsky and Scheiner, 1996). Like many hypotheses in evolutionary ecology (Quinn and Dunham, 1983), these mechanisms are not mutually exclusive; therefore, some or all of them could contribute to an explanation for the temperature-size rule. Moreover, the relative importance of each mechanism probably varies among species. By combining these mechanisms in a single theory, one can achieve a deeper understanding of the relationships among temperature, growth rate and body size.

When developing a multivariate theory of temperature-size relationships, biologists should focus on the developmental reaction norm because this approach forces one to consider the coevolution of thermal reaction norms for growth rate and size at maturity. Allometric and thermal effects on growth rate can be modeled by including well-established functional constraints, such as tradeoffs associated with acquisition, allocation, and specialization. The natural variation in temperature and the covariations between temperature and other environmental variables must be considered because they play important roles in the coevolution of growth rate and body size. Because current theory describes how the thermal environment shapes the optimal reaction norm for growth rate, modeling the developmental reaction norm could reveal why specific temperature-size relationships have evolved in specific environments. Such a breakthrough is needed if we are to understand not only why most species follow the temperature-size rule, but also why certain species do not.

ACKNOWLEDGMENTS

We thank the individuals that participated in this symposium for their unique perspectives and the engaging discussions. We are also grateful to Brian Helmuth, Sue Burk, and Brett Burk for their help and advice during the planning stages. The symposium was funded by the Division of Ecology & Evolution of the Society for Integrative and Comparative Biology and the National Science Foundation (IBN 0335735). Our manuscript was improved by thoughtful comments from Jan Kozlowski and an anonymous reviewer.

REFERENCES

Abrams, P. A. and L. Rowe. 1996. The effects of predation on the age and size of maturity of prey. Evolution 50:1052–1061.

Albentosa, M., R. Beiras, and A. Pérez Camacho. 1994. Determination of optimal thermal conditions fro growth of clam (*Venerupis pullastra*) seed. Aquaculture 126:315–328.

Angilletta, M. J. and A. E. Dunham. 2003. The temperature-size rule in ectotherms: Simple evolutionary explanations may not be general. Am. Natur. 162:332–342.

Angilletta, M. J., P. H. Niewiarowski, A. E. Dunham, A. Leaché, and W. P. Porter. 2004. Bergmann's clines in ectotherms: Illustrating a life-historical perspective with sceloporine lizards. Am. Natur. 164. (In press)

Angilletta, M. J., P. H. Niewiarowski, and C. A. Navas. 2002. The evolution of thermal physiology in ectotherms. J. Therm. Biol. 27:249–268.

Angilletta, M. J., R. S. Wilson, C. A. Navas, and R. S. James. 2003.

Tradeoffs and the evolution of thermal reaction norms. Trends Ecol. Evol. 18:234–240.
Arnold, S. J. 1992. Constraints on phenotypic evolution. Am. Natur. 140:S185–S107.
Ashton, K. G. 2004. Sensitivity of intraspecific latitudinal clines of body size for tetrapods to sampling, latitude and body size. Integr. Comp. Biol. 44:403–412.
Atkinson, D. 1994. Temperature and organism size—a biological law for ectotherms? Adv. Ecol. Res. 25:1–58.
Atkinson, D. 1995. Effects of temperature on the size of aquatic ectotherms: Exceptions to the general rule. J. Therm. Biol. 20: 61–74.
Atkinson, D. 1996. Ectotherm life history responses to developmental temperature. *In* I. A. Johnston and A. F. Bennett (eds.), *Animals and temperature: Phenotypic and evolutionary adaptation*, pp. 183–204. Cambridge University Press, Cambridge.
Atkinson, D. and R. M. Sibly. 1996. On the solution to a major life-history puzzle. Oikos 77:359–364.
Atkinson, D. and R. M. Sibly. 1997. Why are organisms usually bigger in colder environments? Making sense of a life history puzzle. Trends Ecol. Evol. 12:235–239.
Atkinson, D., B. J. Ciotti, and D. J. S. Montagnes. 2003. Protists decrease in size linearly with temperature: *ca.* 2.5% $°C^{-1}$. Proc. Roy. Soc. London B 270:2605–2611.
Ayres, M. P. and J. M. Scribner. 1994. Local adaptation to regional climates in *Papilio canadensis* (Lepidoptera: Papilionidae). Ecol. Monogr. 64:465–482.
Azevedo, R. B. R., V. French, and L. Partridge. 2002. Temperature modulates epidermal cell size in *Drosophila melanogaster*. J. Insect Physiol. 48:231–237.
Båmstedt, U., J. Lane, M. B. Martinussen. 1999. Bioenergetics of ephrya larvae of the scyphozoan jellyfish Aurelia aurita in relation to temperature and salinity. Mar. Biol. 135:89–98.
Bayne, B. L. 2004. Phenotypic flexibility and physiological tradeoffs in the feeding and growth of marine bivalve molluscs. Integr. Comp. Biol. 44:000–000.
Beiras, R., A. P. Camacho, and M. Albenstosa. 1994. Influence of temperature on the physiology of growth in *Ruditapes decussatus* (L.) larve. J. Shellfish Res. 13:77–83.
Bendiksen, E. Å., M. Jobling, and A. M. Arnesen. 2002. Feed intake of Atlantic salmon parr *Salmo salar* L. in relation to temperature and feed composition. Aquacult. Res. 33:525–532.
Bennett, A. F. and R. E. Lenski. 1999. Experimental evolution and its role in evolutionary physiology. Amer. Zool. 39:346–362.
Bernardo, J. 1998. The logic, value, and necessity of grounding experiments in reality. *In* W. J. Resetarits and J. Bernardo (eds.), *Experimental ecology: Issues and perspectives*, pp. 370–393. Oxford University Press, Oxford.
Berrigan, D. and E. L. Charnov. 1994. Reaction norms for age and size at maturity in response to temperature: A puzzle for life historians. Oikos 70:474–478.
Bertalanffy, L. von. 1960. Principles and theory of growth. *In* W. W. Nowinski (ed.), *Fundamental aspects of normal and malignant growth*, pp. 137–259. Elsevier, New York.
Berven, K. A. 1982. The genetic basis of altitudinal variation in the wood frog *Rana sylvetica*. II. An experimental analysis of larval development. Oecologia 52:360–369.
Blackburn, T. M., K. J. Gaston, and N. Loder. 1999. Geographic gradients in body size: A clarification of Bergmann's rule. Div. Distrib. 5:165–174.
Buxton, C. D., R. C. Newell, and J. G. Field. 1981. Response-surface analysis of the combined effects of exposure and acclimation temperatures on filtration, oxygen consumption and scope for growth in the oyster *Ostrea edulis*. Mar. Ecol. Prog. Ser. 6:73–82.
Calboli, F. C. F., G. W. Gilchrist, and L. Partridge. 2003. Different cell size and cell number contribution in two newly established and one ancient body size cline of *Drosophila subobscura*. Evolution 57:566–573.
Chapelle, G. and L. S. Peck. 1999. Polar gigantism dictated by oxygen availability. Nature 399:114–115.
Charlesworth, B. 1980. *Evolution in age-structured populations.* Cambridge University Press, Cambridge.
Charnov, E. L. and J. F. Gillooly. 2004. Size and temperature in the evolution of fish life histories. Integr. Comp. Biol. 44:494–497.
de Moed, G. H., G. de Jong, and W. Scharloo. 1997. The phenotypic plasticity of wing size in *Drosophila melanogaster*: The cellular basis of its genetic variation. Heredity 79:260–267.
Dunham, A. E. 1978. Food availability as a proximate factor influencing individual growth rates in the iguanid lizard *Sceloporus merriami*. Ecology 59:770–778.
Ebert, T. A., J. D. Dixon, S. C. Schroeter, P. E. Kalvass, N. T. Richmond, W. A. Bradbury, and D. A. Woodby. 1999. Growth and mortality of red sea urchins *Strongylocentrotus franciscanus* across a latitudinal gradient. Mar. Ecol. Prog. Ser. 190:189–209.
Elliot, J. M. 1976. The energetics of feeding, metabolism and growth of brown trout (*Salmotrutta* L.) in relation to body weight, water temperature and ration size. J. Anim. Ecol. 45:923–948.
Ernsting, G., J. A. Isaaks, and M. P. Berg. 1992. Life cycle and food availability indices in *Notiophilus biguttatus* (Coleoptera, Carabidae). Ecol. Entomol. 17:33–42.
van Erkom Schurink, C. and C. L. Griffiths. 1992. Physiological energetics of four South African mussel species in relation to body size, ration and temperature. Comp. Biochem. Physiol. 101A:779–789.
Fischer, K. and K. Fiedler. 2002. Reaction norms for age and size at maturity in response to temperature: A test of the compound interest hypothesis. Evol. Ecol. 16:333–349.
Fonds, M., R. Cronie, A. D. Vethaak, and P. van der Puyl. 1992. Metabolism, food consumption and growth of plaice (*Pleuronectes platessa*) and flounder (*Platichthys flesus*) in relation to fish size and temperature. Neth. J. Sea Res. 29:127–143.
French, V., M. Feast, and L. Partridge. 1998. Body size and cell size in *Drosophila*: The developmental response to temperature. J. Insect Physiol. 44:1081–1089.
Gabriel, W. and M. Lynch. 1992. The selective advantage of reaction norms for environmental tolerance. J. Evol. Biol. 5:41–59.
Gilchrist, G. W. 1995. Specialists and generalists in changing environments. I. Fitness landscapes of thermal sensitivity. Amer. Natur. 146:252–270.
Gotthard, K. 2000. Increased risk of predation as a cost of high growth rate: An experimental test in a butterfly. J. Anim. Ecol. 69:896–902.
Gotthard, K. 2001. Growth strategies of ectothermic animals in temperate environments. *In* D. Atkinson and M. Thorndyke (eds.), *Environment and animal development: Genes, life histories, and plasticity*, pp. 287–303. BIOS Scientific Publishers, Oxford.
Heino, M. and V. Kaitala. 1999. Evolution of resource allocation between growth and reproduction in animals with indeterminate growth. J. Evol. Biol. 12:423–429.
Hirst, A. G. and T. Kiørboe. 2002. Mortality of marine planktonic copepods: Global rates and patterns. Mar. Ecol. Prog. Ser. 230: 195–209.
Houde, E. D. 1989. Comparative growth, mortality, and energetics of marine fish larvae: Temperature and implied latitudinal effects. Fish. Bull. 87:471–495.
Huey, R. B. and J. G. Kingsolver. 1989. Evolution of thermal sensitivity of ectotherm performance. Trends Ecol. Evol. 4:131–135.
Huey, R. B. and J. G. Kingsolver. 1993. Evolution of resistance to high temperature in ectotherms. Amer. Natur. 142:S21–S46.
Hutchinson, S. and L. E. Hawkins. 1992. Quantification of the physiological responses of the European flat oyster *Ostrea edulis* L. to temperature and salinity. J. Moll. Stud. 58:215–226.
Imsland, A. K., A. Folkvord, and S. O. Stefansson. 1995. Growth, oxygen consumption and activity of juvenile turbot (*Scophthalmus maximus* L.) reared under different temperatures and photoperiods. Neth. J. Sea Res. 34:149–159.
James, A. C., R. B. R. Azevedo, and L. Partridge. 1995. Cellular basis of developmental timing in a size cline of *Drosophila melanogaster*. Genetics 140:659–666.
James, A. C., R. B. R. Azevedo, and L. Partridge. 1997. Genetic and environmental responses to temperature of *Drosophila melanogaster* from a latitudinal cline. Genetics 146:881–890.
Jonassen, T. M., A. K. Imsland, S. Kadowaki, and S. O. Stefansson. 2000. Interaction of temperature and photoperiod on growth of

Atlantic halibut *Hippoglossus hippoglossus* L. Aquacult. Res. 31:219–227.
Jørgensen, C. B. 1983. Pattern of growth in a temperate zone anuran (*Bufo viridis* Laur.). J. Exp. Zool. 227:433–439.
Kawecki, T. and S. C. Stearns. 1993. The evolution of life histories in spatially heterogeneous environments: Optimal reaction norms revisited. Evol. Ecol. 7:155–174.
Kindlmann, P. and A. F. G. Dixon. 1992. Optimum body size: Effects of food quality and temperature, when reproductive growth rate is restricted, with examples from aphids. J. Evol. Biol. 5:677–690.
Kindlmann, P., A. F. G. Dixon, and I. Dostálková. 2001. Role of ageing and temperature in shaping reaction norms and fecundity functions in insects. J. Evol. Biol. 14:835–840.
Kozłowski, J. 1992. Optimal allocation of resources to growth and reproduction: Implications for age and size at maturity. Trends Ecol. Evol. 7:15–19.
Kozłowski, J. 1993. Measuring fitness in life-history studies. Trends Ecol. Evol. 8:84–85.
Kozłowski, J., M. Czarnoleski, and M. Dańko. 2004. Can optimal resource allocation models explain why ectotherms grow larger in cold? Integr. Comp. Biol. 44. (In press)
Laing, I., S. D. Utting, and R. W. S. Kilada. 1987. Interactive effect of diet and temperature on the growth of juvenile clams. J. Exp. Mar. Biol. Ecol. 113:23–38.
Lampert, W. 1989. The adaptive significance of diel vertical migration of zooplankton. Funct. Ecol. 3:21–27.
Laybourn-Parry, J., B. A. Abdullahi, and S. V. Tinson. 1988. Temperature-dependent energy partitioning in the benthic copepods *Acanthocyclops viridis* and *Macrocyclops albidus*. Can. J. Zool. 66:2709–2714.
Lee, H.-W., S. Ban, T. Ikeda, and T. Matsuishi. 2003. Effect of temperature on development, growth and reproduction in the marine copepod *Pseudocalanus newmani* at satiating food condition. J. Plankton Res. 25:261–271.
Lellis, W. A. and J. A. Russell. 1990. Effect of temperature on survival, growth and feed intake of postlarval spiny lobsters, *Panulirus argus*. Aquaculture 90:1–9.
Lonsdale, D. J. and J. S. Levinton. 1989. Energy budgets of latitudinally separated *Scottolana canadensis* (Copepoda: Harpacticoida). Limnol. Oceanogr. 34:324–331.
Lynch, M. and W. Gabriel. 1987. Environmental tolerance. Amer. Natur. 129:283–303.
Martinez-Palacios, C. A., M. C. Chavez-Sanchez, and L. G. Ross. 1996. The effects of water temperature on food intake, growth and body composition of *Cichlasoma urophthalmus* (Günther) juveniles. Aquacult. Res. 27:455–461.
Morin, J. P., B. Moreteau, G. Pétavy, and J. R. David. 1999. Divergence of reaction norms of size characters between tropical and temperate populations of *Drosophila melanogaster* and *D. simulans*. J. Evol. Biol. 12:329–339.
Myers, R. A. and J. A. Runge. 1983. Predictions of seasonal natural mortality rates in a copepod population using life-history theory. Mar. Ecol. Prog. Ser. 11:189–194.
Myrick, C. A. and J. J. Cech, Jr. 2000. Temperature influences on California rainbow trout physiological performance. Fish Physiol. Biochem. 22:245–254.
Noach, E. J., G. de Jong, and W. Scharloo. 1997. Phenotypic plasticity of wings in selection lines of *Drosophila melanogaster*. Heredity 79:1–9.
Panov, V. E. and D. J. McQueen. 1998. Effects of temperature on individual growth rate and body size of a freshwater amphipod. Can. J. Zool. 76:1107–1116.
Partridge, L., B. Barrie, K. Fowler, and V. French. 1994. Evolution and development of body size and cell size in *Drosophila melanogaster* in response to temperature. Evolution 48:1269–1276.
Partridge, L. and J. A. Coyne. 1997. Bergmann's rule in ectotherms: Is it adaptive? Evolution 51:632–635.
Partridge, L. and V. French. 1996. Thermal evolution of ectotherm body size: Why get big in the cold? *In* I. A. Johnston and A. F. Bennett (eds.), *Animals and temperature: Phenotypic and evolutionary adaptation,* pp. 265–292. Cambridge University Press, Cambridge.
Pepin, P. 1991. Effect of temperature and size on development, mortality, and survival rates of the pelagic early life history stages of marine fish. Can. J. Fish. Aquatic Sci. 48:503–518.
Perrin, N. 1988. Why are offspring born larger when it is colder? Phenotypic plasticity for offspring size in the cladoceran *Simocephalus vetulus* (Müller). Funct. Ecol. 2:283–288.
Perrin, N. 1995. About Berrigan and Charnov's life history puzzle. Oikos 73:137–139.
Perrin, N. and J. F. Rubin. 1990. On dome-shaped norms of reaction for size-to-age at maturity in fishes. Funct. Ecol. 4:53–57.
Perrin, N. and R. M. Sibly. 1993. Dynamic models of energy allocation and investment. Annu. Rev. Ecol. Syst. 24:379–410.
Pöckl, M. 1995. Laboratory studies on growth, feeding, moulting and mortality in the freshwater amphipods *Gammarus fossarum* and *G. roeseli*. Arhiv fur Hydrobiol. 134:223–253.
Quinn, J. and A. E. Dunham. 1983. On hypothesis testing in ecology and evolution. Amer. Natur. 122:602–617.
Roff, D. A. 2002. *Life history evolution.* Sinauer Associates, Inc., Sunderland, Massachusetts.
Schlichting, C. D. and M. Pigliucci. 1998. *Phenotypic evolution: A reaction norm perspective.* Sinauer Associates, Inc., Sunderland, Massachusetts.
Schoener, T. W. and A. Schoener. 1978. Estimating and interpreting body-size growth in some *Anolis* lizards. Copeia 1978:390–405.
Sears, M. W. and M. J. Angilletta. 2003. Life history variation in the sagebrush lizard: Phenotypic plasticity or local adaptation? Ecology 84:1624–1634.
Sebens, K. P. 1987. The ecology of indeterminate growth in animals. Annu. Rev. Ecol. Syst. 18:371–407.
Sebens, K. P. 2002. Energetic constraints, size gradients, and size limits in benthic marine invertebrates. Integr. Comp. Biol. 42: 853–861.
Sevenster, J. G. 1995. Equations or organisms? A comment on Berrigan and Charnov. Oikos 73:405–407.
Sharma, P. C. and M. C. Pant. 1984. An energy budget for *Simocephalus vetulus* (O. F. Muller) (Crustacea: Cladocera). Hydrobiol. 111:37–42.
Sibly, R. M. and D. Atkinson. 1994. How rearing temperature affects optimal adult size in ectotherms. Funct. Ecol. 8:486–493.
Sicard, M. T., A. N. Maeda-Martinez, P. Omart, T. Reynoso-Granados, and L. Carvalho. 1999. Optimum temperature for growth in the catarina scallop (*Argopecten ventricosus-circularis*, Sowerby II, 1842). J. Shellfish Res. 18:385–392.
Strong, K. W. and G. R. Daborn. 1980. The influence of temperature on energy budget variables, body size, and seasonal occurrence of the isopod *Idotea baltica* (Pallas). Can. J. Zool. 58:1992–1996.
Stearns, S. C. 1992. *The evolution of life histories.* Oxford University Press, Oxford.
Stearns, S. C. and J. C. Koella. 1986. The evolution of phenotypic plasticity in life-history traits: Predictions of reaction norms for age and size at maturity. Evolution 40:893–913.
Sweeney, B. W. and R. L. Vannote. 1978. Size variation and the distribution of hemimetabolous aquatic insects: Two thermal equilibrium hypotheses. Science 200:444–446.
Tsitsipis, J. A. and T. E. Mittler. 1976. Development, growth, reproduction, and survival of apterous virginoparae of *Aphis fabae* at different temperatures. Ent. Exp. & Appl. 19:1–10.
Twombly, S. and N. Tisch. 2000. Body size regulation in copepod crustaceans. Oecologia 122:318–326.
van der Have, T. M. and G. de Jong. 1996. Adult size in ectotherms: Temperature effects on growth and differentiation. J. Theor. Biol. 183:329–340.
Van Voorhies, W. A. 1996. Bergmann size clines: A simple explanation for their occurrence in ectotherms. Evolution 50:1259–1264.
van 't Land, J., P. van Putten, B. Zwaan, A. Kamping, and W. van Delden. 1999. Latitudinal variation in wild populations of *Drosophila melanogaster*: Heritabilities and reaction norms. J. Evol. Biol. 12:222–232.
Vernberg, F. J. and S. Piyatiratitivorakul. 1998. Effects of salinity and temperature on the bioenergetics of adult stages of the grass shrimp (*Palaemonetes pugio* Holthuis) from the North Inlet estuary, South Carolina. Estuaries 21:176–193.

Weetman, D. and D. Atkinson. 2002. Antipredator reaction norms for life history traits in *Daphnia pulex*: Dependence on temperature and food. Oikos 98:299–307.
Werner, E. E. and B. R. Anholt. 1993. Ecological consequences of the trade-off between growth and mortality rates mediated by foraging activity. Amer. Natur. 142:242–272.
Woods, H. A. 1999. Egg-mass size and cell size: Effects of temperature on oxygen distribution. Amer. Zool. 39:244–252.
Yamahira, K. and D. O. Conover. 2002. Intra- vs. interspecific latitudinal variation in growth: Adaptation to temperature or seasonality? Ecology 83:1252–1262.
Yampolsky, L. Y. and S. M. Scheiner. 1996. Why larger offspring at lower temperatures? A demographic approach. Amer. Nat. 147:86–100.
Yukihira, H., J. S. Lucas, and D. W. Klumpp. 2000. Comparative effects of temperature on suspension feeding and energy budgets of the pearl oysters *Pinctada margaritifera* and *P. maxima*. Mar. Ecol. Prog. Ser. 195:179–188.
Yurista, P. M. 1999. Temperature-dependent energy budget of an Arctic cladoceran, *Daphnia middendorffiana*. Freshwater Biol. 42:21–34.
Zera, A. J. and L. G. Harshman. 2001. The physiology of life history trade-offs in animals. Annu. Rev. Ecol. Syst. 32:95–126.
Zwaan, B. J., R. B. R. Azevedo, A. C. James, J. Van 't Lande, and L. Partridge. 2000. Cellular basis of wing size variation in *Drosophila melanogaster*: A comparison of latitudinal clines on two continents. Heredity 84:338–347.

APPENDIX

Assume an organism in an aseasonal environment whose relative fitness is indexed by the net reproductive rate (R_0), which is defined as follows:

$$R_0 = \int_0^\infty l_x m_x \, dx, \tag{A1}$$

where l_x is survival to age x and m_x is fecundity at age x. Fecundity is a function of the energy available for production at age x (P_x), the fraction of this energy allocated to reproduction (f), and the energetic content of offspring (ϕ), which is assumed to be constant:

$$m_x = \frac{fP_x}{\phi}. \tag{A2}$$

Production is a function of body size (S_x) and environmental temperature (T).

$$P_x(T, S_x) = a(T)\cdot S_x^c - b(T)\cdot S_x^d, \tag{A3}$$

where S_x is size at age x, a and b are coefficients of anabolism and catabolism, respectively, and c and d are exponents that describe the allometry of these metabolic processes. Size at age x is a function of size at birth (S_0) and the allocation of energy to growth from ages 0 to x:

$$S_x = S_0 + \int_0^x (1 - f)P_y \, dy. \tag{A4}$$

For simplicity, we assume rates of survival for juveniles (s_j) and adults (s_a) depend only on environmental temperature, such that survivorship to age x is denoted as follows:

$$l_x = \{[(s_j(T))^\alpha][(s_a(T))^{x-\alpha}]\}, \tag{A5}$$

where α is the age at maturity.

The optimal strategy under these conditions is to allocate entirely to growth at early ages and allocate entirely to reproduction after maturation (Perrin and Sibly, 1993). Given that no energy is optimally allocated to growth following maturation, equation (A1) becomes

$$R_0 = m_\alpha \int_\alpha^\infty l_x \, dx. \tag{A6}$$

Furthermore, given that adult survivorship is constant, R_0 can be further simplified to

$$R_0 = l_\alpha m_\alpha C, \tag{A7}$$

where

$$C = \int_0^\infty [s_a(T)]^y \, dy. \tag{A8}$$

Thus, fitness is proportional to the product of the probability of survival to maturity and the fecundity at maturity.

To determine the age and size at maturity that result in maximal fitness, we take the derivative of R_0 with respect to age at maturity (α):

$$\frac{d}{d\alpha}R_0 = C\left(\frac{dl_\alpha}{d\alpha}m_\alpha + l_\alpha\frac{dm_\alpha}{d\alpha}\right), \tag{A9}$$

where

$$\frac{dl_\alpha}{d\alpha} = l_\alpha \ln[s_j(T)], \quad \text{and} \tag{A10}$$

$$\frac{dm_\alpha}{d\alpha} = \frac{f}{\phi}\frac{dP_a}{d\alpha} = \frac{f}{\phi}P_\alpha[ca(T)S_\alpha^{c-1} - db(T)S_\alpha^{d-1}]. \tag{A11}$$

Therefore,

$$\frac{d}{d\alpha}R_0 = Cl_\alpha\frac{f}{\phi}P_\alpha\{\ln[s_j(T)] + [ca(T)S_\alpha^{c-1} - db(T)S_\alpha^{d-1}]\}. \tag{A12}$$

Fitness is maximal (or minimal) when eq. (12) equals zero.

We explored the Q_{10} for optimal size at maturity over a realistic set of parameters: (1) isometric (c or $d = 1.0$) and allometric (c or $d < 1.0$) scalings of anabolism and catabolism with body size, and (2) different thermal sensitivities of anabolism (Q_{10}'s ranging from 2 to 6) and catabolism (Q_{10}'s ranging from 2 to 3). When c is greater than d, the optimal decision is to grow indefinitely (Sevenster, 1995). When c and d are both equal to 1.0, eq. (12) cannot be solved; thus, the optimal decision is either to grow indefinitely, or not grow at all, depending on the values of a and b. When c and d are equal, but less than 1.0, the optimal size at maturity is described by

$$S_\alpha = \left\{\frac{c[a(T) - b(T)]}{-\ln[s_j(T)]}\right\}^{1/(1-c)}, \tag{A13}$$

and the Q_{10} for optimal body size is determined by

$$Q_{10\text{size}} = \left[\frac{\dfrac{a(T_{z+10}) - b(T_{z+10})}{a(T_z) - b(T_z)}}{Q_{10\,\ln(s_j)}}\right]^{1/(1-c)}, \tag{A14}$$

where T_z is any temperature and T_{z+10} is 10°C higher than T_z. Thus, the Q_{10} for optimal body size is a function of the Q_{10} of the natural log of juvenile survivorship, the coefficients of anabolism and catabolism at each temperature, and the allometries of anabolism and catabolism. However, the conditions under which the optimal body size at T_z is equal to the optimal body size at T_{z+10} (*i.e.*, the Q_{10} for body size = 1) is not a function of c or d (Fig. 3).

The optimal body size and the Q_{10} for optimal body size cannot easily be solved for the general condition in which d is greater than c. However, under the special case when d is 1.0, the optimal size at maturity is

$$S_\alpha = \left\{\frac{ca(T)}{b(T) - \ln[s_j(T)]}\right\}^{1/(1-c)}, \tag{A15}$$

and the Q_{10} for optimal body size is

$$Q_{10\text{size}} = \left\{\frac{a(T_{z+10})[b(T_z) - \ln(s_j(T_z))]}{a(T_z)[b(T_{z+10}) - \ln(s_j(T_{z+10}))]}\right\}^{1/(1-c)}. \tag{A16}$$

Thus, the Q_{10} for optimal body size is still a function of the Q_{10} of the natural log of juvenile survivorship, the coefficients of anabolism and catabolism at each temperature, and the allometry of catabolism, but is also a function of the absolute survivorship at the low temperature (Fig. 3). Once again, the conditions under which the optimal body size at T_z is equal to the optimal body size at T_{z+10} does not depend on the specific value of c, as long as $c < 1.0$.