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THE EVOLUTION OF ENDOTHERMY: TESTING THE AEROBIC CAPACITY MODEL

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Abstract.—One of the most important events in vertebrate evolution was the acquisition of endothermy, the ability to use metabolic heat production to elevate body temperature above environmental temperature. Several verbal models have been proposed to explain the selective factors leading to the evolution of endothermy. Of these, the aerobic capacity model has received the most attention in recent years. The aerobic capacity model postulates that selection acted mainly to increase maximal aerobic capacity (or associated behavioral abilities) and that elevated resting metabolic rate evolved as a correlated response. Here we evaluate the implicit evolutionary and genetic assumptions of the aerobic capacity model. In light of this evaluation, we assess the utility of phenotypic and genetic correlations for testing the aerobic capacity model. Collectively, the available intraspecific data for terrestrial vertebrates support the notion of a positive phenotypic correlation between resting and maximal rates of oxygen consumption within species. Interspecific analyses provide mixed support for this phenotypic correlation. We argue, however, that assessments of phenotypic or genetic correlations within species and evolutionary correlations among species (from comparative data) are of limited utility, because they may not be able to distinguish between the aerobic capacity model and plausible alternatives, such as selection acting directly on aspects of thermoregulatory abilities. We suggest six sources of information that may help shed light on the selective factors important during the evolution of high aerobic metabolic rates and, ultimately, the attainment of endothermy. Of particular interest will be attempts to determine, using a combination of mechanistic physiological and quantitative-genetic approaches, whether a positive genetic correlation between resting and maximal rates of oxygen consumption is an ineluctable feature of vertebrate physiology.

Key Words.—Aerobic capacity, comparative method, endurance, evolution of endothermy, genetic correlation, locomotion, maximal aerobic metabolism, metabolism, quantitative genetics, resting metabolism.

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Endothermy (the elevation of body temperature by metabolic heat production) represents one of the most significant developments during vertebrate evolution. Endothermy has evolved in birds and mammals, or their ancestors, as well as in some reptiles (e.g., brooding pythons, large sea turtles, and maybe dinosaurs), scombrid fishes (e.g., tunas and billfishes), lamnid sharks, insects, angiosperms (families Annonaceae, Araceae, Cyclanthaceae, and Palmae), and cycads (Hutchison et al. 1966; Carey and Teal 1969; Friar et al. 1972; Knutson 1974; Prance and Arias 1975; Carey et al. 1981; Carey 1982; Standora et al. 1982; Tang 1987; Gottsberger 1989, 1991; Skubatz et al. 1990; Spotila et al. 1991; Block et al. 1993; Heinrich 1993). The endothermy of pythons, scombrid fishes, sharks, and sea turtles results primarily, but perhaps not exclusively, from myogenic heat production, coupled with relatively large body size (Hutchison et al. 1966; Mrosovsky and Pritchard 1971; Block 1991). The endothermy of insects is also myogenically based, but given their small size, endothermy in insects also requires a highly effective insulation (Heinrich 1993).

The endothermy of birds and mammals is distinctive, because at rest it results primarily from metabolic heat production by visceral organs (and to a lesser extent the brain), rather than by muscles (Bennett 1991; Ruben 1995). Only birds and mammals have metabolic rates high enough and insulation effective enough or body size large enough that they can maintain body temperatures elevated above ambient while at rest and in the absence of contractions by skeletal muscles. (If the slow swimming needed to ventilate the gills of obligate ram ventilators [fish that must swim to force {ram} water through their mouth and over their gills] is equated with the effort needed to ventilate the lung of a bird or mammal, then it can be argued that some fishes also maintain elevated body temperatures while at rest; i.e., during slow swimming.)

The origins of endothermy and the types of natural (or sexual) selection that gave rise to it remain controversial (e.g., Bakker 1971, 1980; Heinrich 1977; Crompton et al. 1978; McNab 1978; Bennett and Ruben 1979, 1986; Bennett 1991; Block et al. 1993; Ruben 1995). Increased resting metabolic rate has been hypothesized to be associated with selection for (1) thermal niche expansion (Bakken and Gates 1975; Crompton et al. 1978; Block et al. 1993); (2) homeothermy (stable body temperature) and increased metabolic efficiency (Heinrich 1977; Avery 1979); (3) homeothermy followed by decreasing body size (McNab 1978); (4) postural changes that enhance exercise performance (Heath 1968, cf. Carrier 1987); (5) increased brain size (cf. Hulbert 1980); and (6) increased aerobic capacity during exercise (Regal 1978; Bennett and Ruben 1979). Recent attempts to elucidate the selective regime(s) responsible for the evolution of avian and mammalian endothermy have focused principally on the last of these, termed the aerobic capacity model by Taigen (1983).

Our purpose here is to briefly evaluate the foregoing verbal models of the selective factors involved in the evolution of endothermy. Whether similar selective factors operated in all lineages that evolved endothermy is unknown (cf. Bennett 1991; Block et al. 1993; Ruben 1995). However, the vastly different thermal characteristics of water and air suggest that even if selection were similar, the possible responses to selection may have been very different for terrestrial and aquat-

ic organisms. Hence, we consider only the evolution of avian and mammalian endothermy.

Most recent attempts to test the aerobic capacity model are based on examining phenotypic correlations between resting and maximal aerobic metabolic rate, either within or among species. We contend that such information may have limited utility for distinguishing among alternative models for the evolution of endothermy. Moreover, failure to rigorously assess the underlying evolutionary mechanisms by which the aerobic capacity model could have operated may lead to the erroneous conclusion that this particular model is easier to test than are some alternatives. We briefly review six alternative models for the evolution of endothermy and critically analyze the evolutionary mechanisms that would need to have operated for the aerobic capacity model to work. We then summarize and evaluate the utility of evaluations of the model to date, and finally recommend six sources of information that may offer additional insight.

Alternative Models for the Evolution of Endothermy

At least six verbal models pertaining to the selective origins of endothermy have been proposed. Several of these emphasize endothermy as "the end product of selection for a high and stable body temperature per se" (Bennett and Ruben 1979, p. 649). Bennett and Ruben's aerobic capacity model, in contrast, emphasizes that "the endothermic condition was directly linked with the development of high activity that was sustained by aerobic metabolism" (Bennett and Ruben 1979, p. 650). In briefly reviewing five alternatives to the aerobic capacity model, we emphasize that several postulate or are at least consistent with the attainment of endothermy via more-or-less direct selection on resting metabolism, rather than on activity metabolism.

Thermal Niche Expansion Model

The thermal niche expansion model proposes that endothermy evolved because it allowed animals to be active in thermal niches from which they had previously been excluded (cf. Block 1991; Block et al. 1993). Thus, gradual increases in metabolic rate, with concomitant increases in heat production and hence thermoregulatory abilities at relatively low ambient temperatures, may have progressively expanded the thermal environments that could be exploited. For example, Regal (1975) thought that endothermy would have been a major advantage for protobirds that glided for long distances. He reasoned that gliding birds would lose body heat to the air via convection. Therefore, maintaining preferred body temperatures might require stopping and basking after a flight. Endothermy would enable the birds to maintain their body temperatures without having to bask, or bask as much, between flights.

A related but more complicated model was proposed by Crompton et al. (1978). They argued that mammalian homeothermy evolved in two steps. First, homeothermy at about 28–30°C body temperature evolved in combination with relatively low metabolic rates. "The combination of a low body temperature and a low metabolic rate restricted the first mammals to their nocturnal niche. At various times, small nocturnal mammals acquired higher metabolic rates, enabling them to regulate their body temperature at higher levels and to invade a diurnal niche" (Crompton et al. 1978, p. 333).

In general, increased metabolic heat production may have been important in enabling animals to alter rates of heating and cooling, thus allowing an expansion of the inhabitable thermal niche (Bartholomew and Tucker 1963; cf. Bakken and Gates 1975). However, based on the costs versus benefits of increasing resting metabolism, two arguments can be made against thermal niche expansion models. First, except in well-insulated and large animals, small increases in resting metabolic rate would not enable animals to elevate their body temperature significantly above the environmental temperature but would still incur the cost of a greater need for food (Stevenson 1985). Second, the cost of increased resting metabolism in terms of the additional food needed would outweigh any benefits to be gained, at least in the early stages (Bennett and Ruben 1979, Pough 1980, 1983; cf. Huey and Slatkin 1976).

Homeothermy and the Metabolic Efficiency Model

The ability to maintain a relatively constant body temperature, termed homeothermy, is one potential advantage of endothermy. Avery (1979) postulated that endogenous heat production (and insulation) may have gradually increased to help maintain a body temperature at which enzyme systems were more efficient. Similarly, Heinrich (1977) suggests that homeothermy may allow enzyme specialization, which results in greater metabolic efficiency (see also Hochachka and Somero 1984).

McNab's Decreasing Body Size Model for Mammals

McNab (1978) proposed a model for the evolution of endothermy in mammals that overlaps with the thermal niche expansion model. He argued that the large carnivorous reptilian ancestors of mammals were inertial homeotherms. McNab suggested that as the body size of these animals decreased over evolutionary time (as indicated by the fossil record), selection acted to increase mass-specific resting metabolic rate, thus preserving the ability to maintain relatively high and constant body temperatures. Body size may have decreased because competition with other predators caused a shift in food habits from eating large prey to eating small prey. In any case, as body size decreased and mass-specific metabolism increased, homeothermy based on thermal inertia was supplanted by endothermic homeothermy. A previous commitment to homeothermy (cf. Hochachka and Somero 1984, pp. 375–376) and the ability to exploit a nocturnal niche are argued to be the reasons that evolutionary decreases in size resulted in endothermy in mammals but not in lizards. McNab's model has received surprisingly little attention of late; perhaps because it deals exclusively with mammals (see also Crompton et al. 1978), it is less appealing than are such alternatives as the aerobic capacity model, which potentially can account for the evolution of endothermy in both birds and mammals, as well as other animals (Bennett and Ruben 1979).

Postural Changes Model

Heath (1968) also proposed a model for the evolution of endothermy in the lineage that led to mammals. According to Heath, the skeletons of therapsid reptiles indicate that they had an erect, not a sprawling, stance. This erect stance supposedly required greater muscle tonus, which resulted in increased metabolic rate, thus leading to the development of endothermy. Heath, therefore, argues that therapsids evolved endothermy as an accidental by-product of their change in posture. Thus, selection for the evolution of endothermy equates to selection for an erect posture. An erect posture may provide some advantage in locomotion (Bakker 1971, 1980; Carrier 1987; but see Ruben 1995). Based on studies of extant vertebrates, however, neither an erect posture nor a semierect posture has been shown to require substantially greater energy expenditure than does a sprawling posture (Bennett and Dalzell 1973; cf. Brody 1945; Full 1991). Thus, it seems unlikely that selection for an erect posture per se resulted in the evolution of endothermy.

Increasing Brain Size Model

Based on the reasoning of a number of authors, a model postulating selection for increased brain size leading to the evolution of endothermy can be formulated (cf. Hulbert 1980; Martin 1980). First, increased brain size may have been advantageous to mammals (or their ancestors) for increased sensory (e.g., aural or olfactory) acuity. For birds, increased brain size may have been advantageous in providing better neural control of muscles for maneuvering amidst foliage (Jerison 1971). Second, across species of mammals, brain size was thought to scale with an allometric exponent similar to that for basal metabolic rate (about 0.75), suggesting that "brain size may be linked to maternal metabolic turnover" (Martin 1981, p. 57). Thus, selection for increased brain size may have resulted in increased metabolic rate, leading to endothermy. However, the scaling exponent for brain mass now appears to be about 0.69 rather than 0.75 (Harvey and Krebs 1990; Harvey and Pagel 1991). More significantly, two recent interspecific comparisons, although not analyzed in a fully phylogenetic manner (see below), have reported no significant correlation between relative brain size and metabolic rate in mammals (Pagel and Harvey 1988; McNab and Eisenberg 1989). Thus, because there is no obvious mechanism linking increased relative brain size with substantially increased heat production by visceral organs (see also Harvey and Krebs 1990), because residual brain size and residual resting metabolism are not correlated, and because the brain's contribution to resting metabolism of extant endotherms is fairly modest (Hulbert 1980), the plausibility of this model is low.

Aerobic Capacity Model

Bennett and Ruben (1979) argued that selection for higher capacity for sustainable activity, supported by aerobic metabolic rate, was probably important during the evolution of endothermy. They noted that the energy (food) cost of increasing resting metabolic rate was high (cf. Huey and Slatkin 1976), compared to the thermoregulatory benefits, especially for small increases in resting metabolism, for which thermoregulatory improvements would be insignificant. In contrast, any increase in maximal aerobic capacity will be reflected in higher capacity for sustainable activity.

The aerobic capacity model has two major parts. First, directional selection related to activity capacity resulted in the evolution of a higher maximal aerobic metabolic rate that could be attained during activity (i.e., a greater aerobic capacity). An increased aerobic capacity would enable animals to exercise longer at higher levels, because sustained vigorous activity is supported by aerobic metabolism. The ability to sustain vigorous activity could have been advantageous for many reasons (e.g., Bennett and Ruben 1979, 1986; Bennett 1991; Garland 1993, 1994b). For example, more highly active animals might be better at capturing prey, defending territories, or competing for resources (cf. Heinrich and Bartholomew 1979; Vermeij 1987). Animals with higher aerobic capacities also might traverse, and hence forage over, a greater area, because they were capable of higher sustained speeds of locomotion. Nevertheless, higher aerobic capacity by itself would not result in endothermy of resting animals.

The second element of the aerobic capacity model is that maximal and resting metabolism are somehow linked in a causal, mechanistic sense; thus, evolutionary changes in the two traits cannot occur independently (Bennett and Ruben 1979; Ruben 1995). This idea is based on the observation that in vertebrates maximal oxygen consumption during exercise ($\dot{V}O_2max$) is typically 5–10 times resting oxygen consumption (Bennett and Ruben 1979; see also Bartholomew 1972). "Thus there appears to be a consistent linkage between resting and maximal levels of oxygen consumption in the vertebrates. When an animal is in any given physiological state, oxygen consumption may increase an average of only five- to tenfold" (Bennett and Ruben 1979, p. 651). Bennett and Ruben saw what they interpreted as a relatively constant factorial aerobic scope as indicative of a fundamental property of vertebrate physiology, but they concluded that the reasons for the linkage between resting and activity metabolism were not clear. A necessary mechanistic link between resting and activity metabolism has yet to be demonstrated (Else and Hulbert 1981; Hulbert and Else 1981, 1989, 1990; Bennett 1991; Porter and Brand 1993; Ruben 1995). In any case, Bennett and Ruben's (1979) model assumed that selection for increased maximum aerobic capacity also resulted in increased resting metabolic rate and ultimately endothermy.

Testing Models for the Evolution of Endothermy

General Considerations

Phenotypic evolution can occur for many reasons. Here we emphasize microevolutionary processes, especially natural selection. This emphasis is consistent with most of the models listed above, which view the evolution of endothermy as an adaptive process (but see the sections on the Postural Changes Model and the Increasing Brain Size Model). Moreover, a quantitative-genetic framework for analyzing the microevolution of potentially correlated characters, such as resting and maximal aerobic metabolic rates, is well established (cf. Price and Schluter 1991). Although a quantitative-genetic

framework provides a vehicle for extrapolating from microevolutionary processes to macroevolutionary patterns (Lande 1979, 1985; Slatkin and Kirkpatrick 1986; Arnold 1987, 1992; but see Barton and Turelli 1989 and references therein), it does not address the possibility of qualitatively different macroevolutionary processes (e.g., species selection), which may affect the patterns observed among extant species (Emerson and Arnold 1989; Vrba 1989; Gould 1990; Stanley 1990; Williams 1992). The existence of such qualitatively different processes is still a matter of debate, and any potential relationship to the evolution of endothermy has not been discussed.

Although the aerobic capacity model can be viewed as a general model for the evolution of energy metabolism, including energetic costs and locomotor and behavioral abilities that may vary in association with aerobic metabolic rate, here we are concerned specifically with the model's relevance to the evolution of endothermy in birds and mammals. Direct tests of the aerobic capacity model are difficult because it pertains to historical events. If foolproof fossil indicators of resting and maximal aerobic metabolic rates could be identified, and if the relevant fossils could be found, then direct tests of the model might be possible (see Bakker 1971, 1980; Bennett and Ruben 1986; Carrier 1987; Hillenius 1992, 1994; Ruben 1995). Alternatively, indirect tests of the assumptions of the model are possible. The primary assumptions of the model pertain to the action of natural selection on, and the genetic architecture of, resting and maximal rates of oxygen consumption.

Potential Selective Mechanisms

Any aspect of the phenotype can evolve by genetic drift, but we are unaware of any models that propose genetic drift as an important cause of the evolution of endothermy. Presumably, the energetic costs of increasing metabolic rate are seen as high enough to result in substantial countervailing selection for lower resting metabolism. If so, then endothermy would be unlikely to evolve in the absence of directional selection favoring higher metabolic rates, either directly or because they are correlated with enhanced organismal function (e.g., various behaviors, locomotor abilities, growth rate, other aspects of life history). Note that throughout we refer to phenotypic selection acting within generations (e.g., Lande and Arnold 1983), not the response to selection, which involves inheritance. Inheritance of metabolic rate is discussed below.

Given that genetic drift is unlikely to have been a major factor in the evolution of vertebrate endothermy, then what types of selection could have led to higher metabolic rates and ultimately to the evolution of endothermy? Sexual selection has been proposed as a factor influencing endurance capacities of lizards (Garland 1993; Beck et al. 1995; and references therein). Similarly, Cowens and Lipps (1982) argued that sexual selection on feather structure may have yielded synergistic benefits for mating displays, enhanced fighting ability in male-to-male combat, and thermoregulatory benefits. Nonetheless, we are unaware of any models proposing that endothermy per se evolved via sexual selection.

Several modes of natural selection could, in principle, account for the evolution of endothermy. First, selection could have acted directly to increase aerobic capacity, with resting metabolism increasing solely as a correlated response (i.e., without direct selection on resting metabolism). This would be consistent with the aerobic capacity model. Second, selection could have acted directly to increase resting metabolism, with aerobic capacity increasing solely as a correlated response (i.e., without direct selection on aerobic capacity). This scenario is the opposite of the aerobic capacity model. Third, selection could have acted directly to increase both resting metabolism and aerobic capacity. This scenario would be inconsistent with the aerobic capacity model, at least in its early stages. Although Bennett and Ruben (1979) acknowledged that other factors (e.g., selection related to thermoregulatory abilities) might also have been important in the evolution of endothermy (cf. Bennett 1991), their emphasis was on the initial stages when, they argued, increasing resting metabolism would entail high costs (increased energy and food requirements) compared to any thermoregulatory benefits. Thus, their model was an attempt to explain the initial evolutionary steps towards endothermy, in spite of what would appear to be the poor cost-to-benefit ratio of increasing resting metabolism before it reached a level sufficient to confer thermoregulatory advantages.

The foregoing paragraph describes three scenarios by which natural selection could have led to the evolution of endothermy. These scenarios deal with evolutionary change that can be explained by (1) direct effects of selection on traits, and (2) correlated responses to selection caused by additive genetic correlations among traits. Basic quantitative-genetic equations describe the responses to selection on correlated characters (e.g., see Lande and Arnold 1983, Falconer 1989). In the notation of Lande and Arnold (1983) and Arnold and Wade (1984), we can write the response to selection as,

$$\Delta\bar{z} = G\beta, \tag{1}$$

where $\Delta\bar{z}$ is a column vector of the change in phenotypic means across generations, G is the additive genetic variance–covariance matrix, and β is a column vector, termed the directional selection gradient vector, the elements of which account for the direct effects of selection on traits within generations. Because the aerobic capacity model postulates direct selection on aerobic capacity, the elements of the directional selection gradient (i.e., the β_i) are the selection coefficients relevant to the model.

In practice, measuring the elements of the directional selection gradient is more complex when traits are phenotypically correlated, as resting metabolism, aerobic capacity, and other traits may be (see table 1 and below). When traits are phenotypically correlated, the equation for responses to selection is fruitfully rewritten as,

$$\Delta\bar{z} = GP^{-1}s, \tag{2}$$

where P^{-1} is the inverse of the phenotypic variance–covariance matrix, and s is a column vector of observed selection differentials (Lande and Arnold 1983, Arnold and Wade 1984). Note that β equals $P^{-1}s$. When a single selection event affects survival (e.g., see the examples in Lande and Arnold 1983), the observed selection differentials (s_i) can be envi-

TABLE 1. Summary of intraspecific correlations between resting or standard metabolic rates and exercise $\dot{V}O_2$, or maximal oxygen consumption. The effects of body mass were controlled statistically (i.e., removed) either by analyzing residuals from regressions on body mass or by testing the partial correlation with body mass effects held constant.

Taxon	Correlation coefficient	n	One-tailed P	Source
Amphibians				
Bufo woodhousii	0.374	38	0.0104	Walton 1988
Lizards				
Callisaurus draconoides	0.224	20	0.1712	T. Garland, Jr., unpubl. data
Chalcides ocellatus	0.089	28	0.3261	Pough & Andrews 1984
Cnemidophorus tigris	−0.010	24	0.5185	T. Garland, Jr., unpubl. data
Ctenophorus nuchalis	0.219	56	0.0524	Garland & Else 1987
Ctenosaura similis	0.122	18	0.3148	Garland 1984
Dipsosaurus dorsalis	0.176	70	0.0725	John-Alder 1984
Sceloporus undulatus	0.318	47	0.0145	Pierce & John-Alder pers. comm. 1994
Snakes				
Thamnophis sirtalis	0.11	250	0.0413	B. M. Walton, A. F. Bennett, & C. C. Peterson pers. comm. 1994
Mammals				
Peromyscus maniculatus	0.318*	50	0.0122	Hayes 1989a
Spermophilus beldingi	0.31	95	0.0011	Chappell and Bachman 1995
S. beldingi	0.005*	95	0.4804	Chappell and Bachman 1995

* designates maximal oxygen consumption elicited via cold exposure.

sioned as the within-generation change in phenotypes before versus after selection. More generally, the observed selection differentials (s_i) are estimated as the covariance of each trait with relative fitness (Lande and Arnold 1983). As Lande and Arnold (1983) showed, the partial regression coefficients from a multiple regression of relative fitness on trait values can be used to estimate the elements of the directional selection gradient vector (β_i), which enables one to partition the observed selection differentials (s_i) into the direct effects of selection plus the indirect effects of selection caused by phenotypically correlated traits (cf. Mitchell-Olds and Shaw 1987; Schluter and Nychka 1994).

Bennett and Ruben (1979) did not discuss the role of indirect selection (or of correlational selection; see below) in the aerobic capacity model, at least partly because none of the data summarized in table 1 were then available, nor was the quantitative-genetic perspective common in evolutionary biology, let alone in physiological ecology (but see Lynch 1994). Nonetheless, even though evolutionary responses to selection can be modeled without reference to indirect selection or phenotypic correlation (see eq. 1), it is important to recognize that phenotypic correlations among traits complicate the measurement of phenotypic selection (Lande and Arnold 1983). Such a perspective is likely to be essential if attempts are made to study selection on resting and maximal aerobic metabolic rates, which may be correlated with each other as well as with other traits. In any case, we see indirect selection (e.g., on resting metabolic rate) as neither necessary for, nor inconsistent with, the aerobic capacity model.

The last possible mode of selection that we consider is correlational selection (Lande and Arnold 1983; Arnold 1986; Endler 1986; Phillips and Arnold 1989; Schluter and Nychka 1994; cf. Mitchell-Olds and Shaw 1987). In Lande and Arnold's (1983, p. 1216) formulation, "correlational selection" refers to the off-diagonal elements of the stabilizing selection gradient matrix, which measure "the strength of selection directly on the covariances (i.e., the intensity of selection to positively or negatively correlate pairs of characters), after allowing for the changes produced by directional selection." If the off-diagonal elements are nonzero, then there is correlational selection. In other words, correlational selection is a form of multivariate selection that favors particular combinations of traits. Correlational selection alone, however, would not result in evolutionary changes in trait means (e.g., a trend for increasing resting and activity metabolism culminating in endothermy), because estimates of correlational selection (sensu Lande and Arnold 1983) are adjusted for the changes produced by directional selection (i.e., correlational selection acts on the covariance between traits, not on the trait means).

A slightly different view of the aerobic capacity model is that direct selection favored the ability to sustain vigorous activity and that this direct selection resulted in a correlated response in aerobic capacity. This view treats organismal performance (or its behavioral correlates) as the most direct target of selection (see Arnold 1983; Pough et al. 1992; Garland 1994b; Garland and Carter 1994; Garland and Losos 1994). In Bennett and Ruben's (1979, p. 652) original words: "the evolution of increased aerobic metabolism for the sake of expanded potential for sustained activity played an important role in the development of endothermy." If this viewpoint is adopted, then the aerobic capacity model is slightly more complex. Direct selection favoring ability for sustained vigorous activity results in a correlated increase in both aerobic capacity and resting metabolic rate. Because ability for sustained activity (endurance) is generally positively correlated with aerobic capacity (Taylor et al. 1987; Jackson et al. 1990; Gatten et al. 1992; Pough et al. 1992; Garland 1993, 1994a,b; Pfeiffer et al. 1993; Garland and Losos 1994), indirect selection also would occur. Moreover, correlational selection might have acted to favor individuals with appropriate combinations of behavior and physiological capacities (cf.

Brodie 1992; Garland 1994a), such as highly active individuals with high aerobic capacities and relatively inactive individuals with low aerobic capacities. Acting alone, however, such selection on covariances would not cause evolutionary increases in metabolic rates.

Genetic Requirements of the Aerobic Capacity Model

An essential feature of the aerobic capacity model is that resting metabolic rate evolved as a correlated response to phenotypic selection on activity metabolism. This model was proposed before quantitative-genetic perspectives came to the fore in evolutionary biology. Thus, Bennett and Ruben (1979, p. 653) did not discuss the genetic implications of their model, except to claim that "the catalytic capacity of the mitochondrial enzymes . . . could have been increased with a minimum of genetic alteration by increasing mitochondrial volume."

From today's perspective, however, an implicit genetic assumption of the aerobic capacity model is the existence, in the lineages evolving endothermy, of a positive additive genetic correlation between resting and maximal rates of oxygen consumption. This requirement implies heritable variation (nonzero additive genetic variance) for both resting and maximal aerobic metabolic rate, because if either or both of a pair of traits has no additive genetic variance, then the additive genetic covariance between the traits is necessarily zero. Moreover, if the interpretation of the aerobic capacity model includes selection on behavior that involves sustained vigorous activity, then we must also postulate heritable variation for these behaviors in addition to the heritable variation for resting and activity metabolism, and we must also postulate additional genetic correlations.

Direct tests of whether ancestral populations had significant additive genetic variance or covariance are impossible (cf. Leroi et al. 1994). Indirect tests, based on quantitative-genetic analyses of extant species and using parsimony to infer ancestral conditions, are possible, but problematic, for several reasons. First, because large sample sizes are needed, it is difficult to estimate heritabilities and genetic correlations accurately, particularly in natural populations. Second, both genetic correlations and single-trait heritabilities are defined for particular environments. In a different environment, the same set of genotypes can yield different heritabilities and genetic correlations (Falconer 1989; Stearns 1992). Quantitative-genetic parameters are also sensitive to changes in gene frequency, and the magnitude and even the sign of a genetic correlation can change ontogenetically, across generations, across environments, and as a function of resource availability (van Noordwijk and de Jong 1986; Falconer 1989; Stearns 1992). Correlational selection may contribute to the maintenance of additive genetic correlations (Brodie 1992), but the length of evolutionary time over which genetic correlations are likely to persist is unknown. Theoretical treatments come to varied conclusions about the likelihood that genetic correlations will persist over evolutionary time (Lande 1980; Turelli 1988; Gillespie and Turelli 1989; and references therein), and the available data do not provide a clear answer (Lofsvold 1986; Kohn and Atchley 1988; Wilkinson et al. 1990). Thus, extant populations may or may not be representative of the genetic architecture of ancestral populations.

In general, the long-term persistence of genetic correlations appears to be most likely for traits that are closely coupled developmentally, biomechanically, or physiologically (Cheverud 1982; Riska 1989; Stearns 1989a,b, 1992). Bennett and Ruben (1979) asserted that an ineluctable feature of vertebrate physiology is a link between resting and maximal rates of oxygen consumption. If so, then this design feature should also be expressed as a persistent genetic correlation (cf. Riska 1986). The large differences in metabolic rate among extant species of reptiles, mammals, and birds indicate that ample additive genetic variation for both resting and maximal rates of oxygen consumption has been present during at least some periods of evolutionary history. But this does not mean that genetic correlations must have existed.

The organisms in which mammalian and avian endothermy evolved are not the same as any extant terrestrial vertebrates, and we have no guarantee that the latter are accurate physiological or genetic models for those extinct forms. Thus, it is possible that a mechanistic link—and a positive genetic correlation—existed while endothermy was evolving in "transitional" forms but does not exist in living mammals, birds, or reptiles; the converse is also possible.

Previous Attempts to Test the Aerobic Capacity Model

General Considerations

Since Bennett and Ruben (1979) first proposed the aerobic capacity model, several studies have reported data on the relationship between resting or standard metabolic rate and maximal aerobic metabolic rate during exercise or during cold exposure, either within or among species. These measures of metabolism are often strongly correlated with body mass; thus, the effect of body mass is controlled statistically (i.e., removed), either by analyzing residuals from regressions on body mass, by testing the partial correlation with body mass effects held constant, or by dividing whole-animal metabolic rate by body mass raised to an exponent derived from an empirical allometric relationship.

Which taxa and which measures of metabolism should be used to test the aerobic capacity model (cf. Block et al. 1993)? As noted above, whether any living organisms can serve as useful models for understanding physiological evolution in extinct forms is debatable, but extant organisms are all that physiologists have. If a positive relationship between resting metabolic rate and aerobic capacity is a fundamental feature of vertebrate physiology (e.g., Bennett and Ruben 1979), then this relationship should hold at least for all amniotes. Alternatively, maybe one should study only those species that are perceived as being most similar phenotypically to the ancestral forms, perhaps the tuatara, some lizards, or some "primitive" mammals (e.g., Dawson et al. 1979; Schmidt-Nielsen et al. 1980). Or, possibly one should focus on species that exhibit exceptionally high aerobic capacities. Given such uncertainty, here we consider all data of which we are aware.

Whether maximal aerobic metabolic rate measured during cold exposure is relevant to the aerobic capacity model depends on whether it is a good indicator of maximal aerobic

metabolism measured during exercise (cf. Hayes 1989b; Bozinovic 1992; Hinds and Rice-Warner 1992; Sparti 1992). Across a substantial range of environmental conditions, maximal aerobic metabolic rate during cold exposure is significantly correlated with maximal aerobic metabolic rate during exercise in *Peromyscus maniculatus* (Hayes and Chappell 1990), but the generality of this result remains to be tested. We include information on aerobic metabolism measured during cold exposure in our analysis. The appropriateness of including tests based on measuring maximal aerobic metabolic rate via cold exposure can be reevaluated in the future, when the relationship between maximal aerobic metabolic rate during cold exposure and maximal aerobic metabolic rate during exercise is more clearly understood.

Quantitative Genetics of Aerobic Metabolic Rates

Although the effects of selection on genetically correlated variables are complex (Kirkpatrick and Lande 1989; Charlesworth 1990; Houle 1991), Bennett and Ruben's (1979) formulation of the aerobic capacity model clearly requires there to have been a positive additive genetic correlation between resting and maximal rates of oxygen consumption. Unfortunately, information on the additive genetic correlation between resting and maximal oxygen consumption is not available for any species, and even estimates of the heritability of metabolic rate are uncommon. Low but significant narrow-sense heritabilities of basal oxygen consumption have been reported for domestic house mice (references in Lynch 1994), and significant realized heritabilities for oxygen consumption have been reported for chickens (e.g., MacLaury and Johnson 1972). Resting metabolic rate may also be heritable in humans (e.g., Bouchard et al. 1989). Twin studies suggest that $\dot{V}O_2$max (the maximal rate of oxygen consumption during exercise) is heritable in humans (Bouchard et al. 1986), and full-sib analyses suggest genetic variation for $\dot{V}O_2$max in garter snakes (*Thamnophis sirtalis*; Garland and Bennett 1990).

Intraspecific Phenotypic Correlations of Aerobic Metabolic Rates

Although no estimates of genetic correlations are available, phenotypic correlations between resting and maximal rates of oxygen consumption are available for at least 11 species (table 1). Mass-corrected resting and maximal oxygen consumption were significantly (one-tailed test; $\alpha = 0.05$) positively correlated in one species of amphibian (Walton 1988), in one of seven species of lizards (Garland 1984; John-Alder 1984; Pough and Andrews 1984; Garland and Else 1987; T. Garland, Jr. unpubl. data, V. Pierce and H. B. John-Alder pers. comm. 1994), and in one species of snake, *Thamnophis sirtalis* (B. M. Walton, A. F. Bennett, and C. C. Peterson pers. comm. 1994). Note that Feder (1987) also found a significant positive Spearman's rank correlation ($r = 0.71$) between resting metabolism and aerobic capacity in *Bolitoglossa subpalmata*. However, because his data were not mass corrected, we did not include them in table 1. In Belding's ground squirrel (*Spermophilus beldingi*), basal metabolic rate (BMR) is significantly correlated with $\dot{V}O_2$max measured during exercise, but not with $\dot{V}O_2$max elicited by cold exposure (Chappell and Bachman 1995). Hayes (1989b) examined the relationship between resting metabolic rate and maximum aerobic metabolic rate during cold exposure in wild deer mice (*Peromyscus maniculatus*) freshly captured from the field. When effects of body mass and of season were removed, residuals were positively related ($r = 0.318$, $P = 0.0122$). When seasonal effects were not removed, the correlation between residuals (from body mass only) was insignificant.

To obtain an overall indication of the intraspecific correlation based on the data in table 1, we performed a *t*-test on the correlations to test if they were significantly different from zero, and we used Fisher's test for combining independent probabilities (Sokal and Rohlf 1981). Both tests are highly significant ($P < 0.005$), regardless of which of the two nonindependent values for *Spermophilus beldingi* is used. Although the studies reported in table 1 provide support for an intraspecific correlation between resting metabolic rate and maximal aerobic capacity, it should be recognized that the correlations are in general fairly low.

Interspecific Correlations of Aerobic Metabolic Rates

Other evaluations of the aerobic capacity model can be made, based on comparative studies. As indicated by Bennett (1991), comparisons of species of similar body size would be expected to show parallel differences in resting metabolic rate and $\dot{V}O_2$max. This prediction has not been well supported; available data now indicate that the ratio of maximal to resting rate of oxygen consumption can vary from approximately 5- to 30-fold in both reptiles (Bennett 1972, 1978; Ruben 1976; Kamel and Gatten 1983; Garland 1993) and terrestrial mammals (e.g., Taylor et al. 1987; Hinds et al. 1993).

In addition to comparisons of pairs or a few species of similar body size, comparisons based on many species are also available. These interspecific analyses include traditional assessments of relationships of traits across the tips of a phylogeny (i.e., correlations based on species' mean values, with no attempt to account for possible confounding influences of phylogenetic relationships), as well as phylogenetically based comparisons (sensu Felsenstein 1985; Harvey and Pagel 1991; Martins and Garland 1991). Across 17 species of anuran amphibians, Taigen (1983) found a significant positive rank correlation between mass-corrected resting and exercise rates of oxygen consumption. This has generally been interpreted as support for the aerobic capacity model. Likewise, Hinds and Rice-Warner (1992) reported a significant correlation between residual BMR and residual $\dot{V}O_2$max during exercise in 18 species of rodents. They also found a correlation between residual BMR and residual $\dot{V}O_2$max elicited via cold exposure in 39 species of rodents. Bozinovic (1992) found a significant partial correlation (after controlling for body mass) between $\dot{V}O_2$max measured via cold exposure and BMR for 29 species of rodents. The correlations calculated by Taigen (1983), Hinds and Rice-Warner (1992), and Bozinovic (1992) all depended at least in part on data collected with similar methods by the same investigators. In contrast, Koteja (1987) found no correlation between residual resting metabolic rate and exercise $\dot{V}O_2$max for 18 species of wild mammals, using data obtained from a variety of lit-

erature sources. The lack of correlation found by Koteja (1987) may reflect problems with the comparability of metabolic data collected by different methods (cf. Hayes et al. 1992a).

A limitation of conventional correlational studies across the tips of a phylogeny is that the species' mean values for a phenotypic trait may not represent statistically independent data points (Felsenstein 1985); thus, tests of phenotypic correlations will be overly liberal (i.e., Type-I error rates will be higher than indicated by the nominal significance level). Moreover, the correlation across tips of the phylogeny does not necessarily reflect the evolutionary correlation between variables (Martins and Garland 1991). Recent developments in the comparative method make it possible to correct for the lack of independence among species, such that statistically valid correlations are obtained if the phylogeny and evolutionary model are correctly specified (review in Harvey and Pagel 1991). The studies cited in the previous paragraph deserve to be reanalyzed as suitable phylogenetic information becomes available.

At least two interspecific comparative studies of resting and maximal aerobic metabolic rate have used phylogenetically based statistical methods. Walton (1993) found a significant correlation of resting metabolic rate and exercise $\dot{V}O_2$max in 15 species or subspecies of hylid frogs. In contrast, Sparti (1992) found no significant correlation in a study of 13 species of shrews, but she measured $\dot{V}O_2$max elicited via cold exposure, not exercise. Overall, interspecific comparisons provide mixed support for the idea that resting and maximal aerobic metabolic rates have evolved in a positively correlated fashion.

Limitations of Phenotypic and Genetic Correlations

Phenotypic correlations might be more useful in testing the aerobic capacity model if phenotypic correlations were a reasonable proxy for genetic correlations (Cheverud 1988). However, the reliability of phenotypic correlations as predictors of additive genetic correlations is suspect. Understanding why requires an appreciation of the relationship between genetic and phenotypic correlations. The phenotypic correlation of two traits is a function of both their environmental correlation and their genetic correlation. According to Falconer (1989), the relationship for two traits, x and y, is:

$$r_p = h_x h_y r_a + e_x e_y r_e, \tag{3}$$

where r represents a correlation coefficient, h represents a square root of the narrow-sense heritability, e represents a square root of 1 minus the narrow-sense heritability, the subscript p refers to phenotype, the subscript a refers to additive genetic effects, and the subscript e refers to environmental effects (which in this formulation includes "nonadditive" genetic effects, such as dominance and epistasis).

From equation (1), it is evident that when heritabilities are high and consequently environmental effects (e's) are low, the phenotypic correlation will be determined primarily by the additive genetic correlation. When heritabilities are low, the phenotypic correlation will be determined primarily by the environmental correlation. Genetic and phenotypic correlations may differ not only in magnitude but also in sign; however, if genetic and environmental correlations are similar, then all three types of correlation—phenotypic, genetic, and environmental—will be similar (Cheverud 1988; Falconer 1989). Indeed, if selection for positive phenotypic covariances between traits persists for many generations, then negative genetic covariances may eventually result (cf. Burger 1986; Price and Schluter 1991). In general, the complexities of maternal effects and of possible environmental changes make it unlikely that the relationship between phenotypic and genetic correlations can be predicted in any simple way (Arnold 1987; but see Cheverud 1988).

If resting and maximal aerobic metabolism are mechanistically linked, as Bennett and Ruben (1979) have suggested, then both genetic and environmental factors acting during development should affect resting and maximal aerobic metabolic rates in a parallel fashion, such that the traits should be both phenotypically and genetically correlated. Intuitively, one might expect resting and maximal rates of oxygen consumption to be closely coupled physiologically, perhaps because they both share the circulatory and pulmonary systems, which deliver oxygen to the mitochondria, or perhaps because mitochondria have a fixed minimum idling cost (e.g., Else and Hulbert 1981; Hulbert and Else 1981). A discussion of other possible physiological or biochemical mechanisms linking resting and maximal aerobic metabolism is beyond the scope of this article (see Hulbert and Else 1989, 1990; Bennett 1991; Porter and Brand 1993; Ruben 1995). A primary argument against a necessary linkage is that maximal aerobic metabolic rate is primarily muscular in origin, whereas resting metabolic rate is mainly attributable to the visceral organs and the brain. Irrespective of such arguments, all of this must remain highly speculative until we obtain data on genetic and environmental correlations between resting and maximal rates of oxygen consumption in one or more organisms.

Regardless of their outcome, neither tests of the aerobic capacity model based on phenotypic correlations nor tests based on genetic correlations can provide strong support for the aerobic capacity model. The absence of phenotypic correlations between resting and aerobic metabolic rates would argue against the aerobic capacity model, because it would suggest that the assumption that these two traits are ineluctably linked is false. However, the presence of phenotypic correlations is not useful in distinguishing among alternative models. To support the aerobic capacity model, we must show that increased resting metabolism has evolved at least in part because of selection favoring increased aerobic capacity. Alternatives to the aerobic capacity model posit that selection occurred directly on resting metabolic rate, or on some other trait, such as posture or relative brain size, that is supposed to necessarily increase resting metabolism. Phenotypic correlations, even evolutionary correlations (sensu Martins and Garland 1991), between resting metabolic rate and aerobic capacity are consistent with both kinds of models.

Genetic correlations also have limitations for testing models for the evolution of endothermy. The absence of genetic correlations would argue against the model, because genetic correlations would have been necessary for a correlated response to selection; however, the uncertainty about whether

genetic correlations are likely to persist over evolutionary time (Lande 1980; Turelli 1988; Arnold 1992) makes this interpretation problematic. The presence of genetic correlations would also be of questionable utility. Like phenotypic correlations, genetic correlations might support the assumption that resting metabolism and aerobic capacity are ineluctably linked. However, the presence of genetic correlations only tells you that correlated responses to selection are possible, not whether resting metabolism evolved because of direct selection or as a correlated response to selection.

Other Possible Tests of the Aerobic Capacity Model

We have attempted to assess which evolutionary mechanisms are consistent with Bennett and Ruben's (1979) aerobic capacity model for the evolution of endothermy and to elucidate how this historical model can be tested. Our analysis is not intended as a criticism of the model, which we consider to be plausible and insightful. Clearly, however, the model is difficult to test, and researchers testing the model should be aware of the limitations of phenotypic and genetic correlations for doing so.

Bennett (1991) remarked that it may be foolhardy to attempt to determine the types of selection that have acted on unknown organisms in poorly understood environments. Similarly, Hochachka and Somero (1984, p. 374) considered the task of answering questions about the evolution of endothermy to be "empirically, if not logically, impossible." Nonetheless, future attempts to test the aerobic capacity model will undoubtedly occur. As with many questions about evolutionary history, conclusive tests of the aerobic capacity model may be impossible, but the situation is not hopeless. Insight can come from at least six sources (see also Bennett 1972, 1991; Ruben 1976, 1995; Taylor et al. 1987; Block et al. 1993).

First, mechanistic physiological and biochemical studies might be able to demonstrate a necessary link between resting and maximal rates of oxygen consumption. Importantly, evidence for such a linkage would not be inconsistent with those models that postulate selection acting primarily on resting metabolic rate, because they are generally neutral with respect to how aerobic capacity would have evolved. However, failure to find a linkage would argue against the aerobic capacity model. Second, quantitative-genetic analyses could be used to determine whether a positive genetic correlation between resting metabolic rate and aerobic capacity is pervasive in amniotes (cf. Bennett 1991). For example, artificial-selection experiments (e.g., see references in Garland and Carter 1994) for increased $\dot{V}O_2max$ or for increased activity per se could indicate whether correlated increases in resting metabolism occur. (Necessary mechanistic links should be reflected as genetic correlations.) Third, we need population-level studies of natural selection on resting metabolic rate, $\dot{V}O_2max$, and their potential functional correlates, such as aspects of life history. Even though selection may have changed over evolutionary time, the results of such studies would provide insight into how selection may operate on metabolism and its correlates (cf. Hayes 1989a,b; Hayes et al. 1992b). This is largely unstudied (e.g., Brodie 1992; A. F. Bennett pers. comm. 1994; reviews and references in Bennett 1991; Garland and Carter 1994; Garland and Losos 1994).

Fourth, interspecific comparative studies can be used to test for evolutionary correlations (sensu Martins and Garland 1991) between resting and maximal aerobic metabolic rate and other ecological and behavioral traits. Existing studies are inconclusive, and several need to be rerun using phylogenetically based methods. It is important to note, however, that even if an evolutionary correlation between resting and maximal aerobic metabolic rates is found, one cannot distinguish between a selective and a genetic cause of the correlated evolution in the absence of additional information. Additional comparative information on ecological, behavioral, or life history (e.g., Harvey et al. 1991) correlates of metabolic rate could provide additional insight concerning selective factors (Harvey and Pagel 1991; Garland and Adolph 1994; but see Leroi et al. 1994). Methods for distinguishing direct and correlated responses to selection in extinct lineages would be very helpful (cf. Emerson and Arnold 1989; Arnold 1992) but are presently unavailable and indeed may be unattainable (cf. Leroi et al. 1994). Fifth, models integrating behavior, physiology, and biophysics could indicate the costs and benefits of changing metabolism under different ecological conditions, thus shedding light on selective factors that may have affected the evolution of aerobic metabolism. Sixth, paleontological evidence may provide clues about the metabolic status of extinct animals (Barrick and Showers 1994; Hillenius 1994; Ruben 1995). If reliable indicators of metabolic status can be found (cf. Hillenius 1994), then it may be possible to falsify the aerobic capacity model fairly directly. For example, if it could be shown from fossil indicators that high resting metabolic rate and endothermic homeothermy preceded high maximal aerobic capacities (and associated high stamina), then the aerobic capacity model would be falsified.

In summary, we encourage physiologists not to limit tests of the aerobic capacity model to studying phenotypic correlations between resting and maximal aerobic metabolic rate. Testing the aerobic capacity model is difficult, but even if our suggestions for future research do not lead to conclusive tests of models for the evolution of endothermy, they will be useful in understanding the evolution of energy metabolism in general.

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