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# Why does metabolism scale with temperature?

A. CLARKE† and K. P. P. FRASER

*British Antarctic Survey, High Cross, Madingley Road, Cambridge CB3 0ET, UK*

## Introduction

At a fundamental level, all of ecology is underpinned by the laws of thermodynamics, the conservation of matter and the general physical principles that dictate how gases and liquids move. Taken altogether, this indicates that there is nothing special about the way organisms function; physiology and ecology are simply physics and chemistry writ complex.

In a stimulating review, Lawton (1999) argued that ecology has no laws comparable to physics, but does have what he called widely observable tendencies. These provide a structure for ecology, but they cannot be deduced from first principles. The complication here is scale. At small scales organisms do things that can be analysed, understood and relatively easily predicted. At very large scales (typically continental or global) patterns emerge that can be described statistically; explanations for these global patterns are proving more elusive and this is the challenge of the rapidly developing field of macroecology (Brown 1995). At intermediate scales we are faced with a mixture of contingent case studies and weak generalizations (Lawton 1999). The emerging field of complexity has much to offer here (Maurer 1999), but as yet its impact on mainstream ecology has been small.

Organism size has long been recognized as a major factor in ecology and in the 1980s a number of attempts were made to use allometric or scaling techniques to provide a theoretical framework for understanding ecology (Peters 1983; Calder 1984; Schmidt-Nielsen 1984). Despite the stronger claims that allometry provided a theory of size (Peters 1983), the field of scaling has not provided the conceptual framework that is needed, although it has supplied useful analytical tools for studies of energy flow through aquatic food webs (Kerr & Dickie 2001). The problem we face is that a statistical description of phenomena is of limited use if we do not also have a sound understanding of the underlying processes that produce the patterns we observe and describe.

Recently Brown, West and a series of colleagues have looked again at scaling properties in an attempt to build ecological laws from first principles. This research programme has had three main phases to date:

1. To explain the size scaling properties of organismal physiology through the fractal-like design of exchange surfaces and distribution networks in animals and plants (West, Brown & Enquist 1997; West *et al*. 1999).
2. To include the effect of temperature by incorporation of the temperature-dependence of biochemical reaction rate through the Boltzmann factor, which they termed the Universal Temperature Dependence (UTD) of metabolism (Gillooly *et al*. 2001).
3. To extend this model of metabolism to other aspects of energetics such as development (Gillooly *et al*. 2002) as well as macroecological features such as global patterns in diversity (Allen, Brown & Gillooly 2002).

The combination of the mass scaling and temperature components led Gillooly *et al*. (2001) to propose a simple equation describing the variation of metabolic rate ($Q$) of all organisms:

$$Q = b_0 M^{3/4} e^{-E/kT},$$

where $M$ is body mass, $T$ is absolute temperature, $k$ is Boltzmann's constant, $E$ is the activation energy of metabolism (defined as the average activation energy for the rate-limiting enzyme-catalysed biochemical reactions of metabolism) and $b_0$ is a scaling constant independent of $M$ and $T$.

This equation has attracted a great deal of attention from ecologists, and the fractal explanation of the widely described $^3/_4$ mass scaling property of metabolism has been questioned (see for example Dodds, Rothman & Weitz 2001). The formulation of the relationship between metabolic rate and temperature is identical mathematically to the treatment of the thermal behaviour of reaction rate and the equilibrium constant by the early physical chemists (Arrhenius 1889, 1915; van't Hoff 1896). The proposition of the UTD is that the metabolic rate of organisms is driven directly by the kinetic energy of the cell; in other words a higher temperature leads automatically to a higher metabolic rate, whether this is an acute temperature change within an individual organism or in different species which have adapted to divergent thermal environments over evolutionary time. In this mechanistic form, the UTD thus makes no provision for acclimatization or evolutionary adaptation. This could theoretically be achieved to some extent by variations in



†Author to whom correspondence should be addressed. E-mail: accl@bas.ac.uk

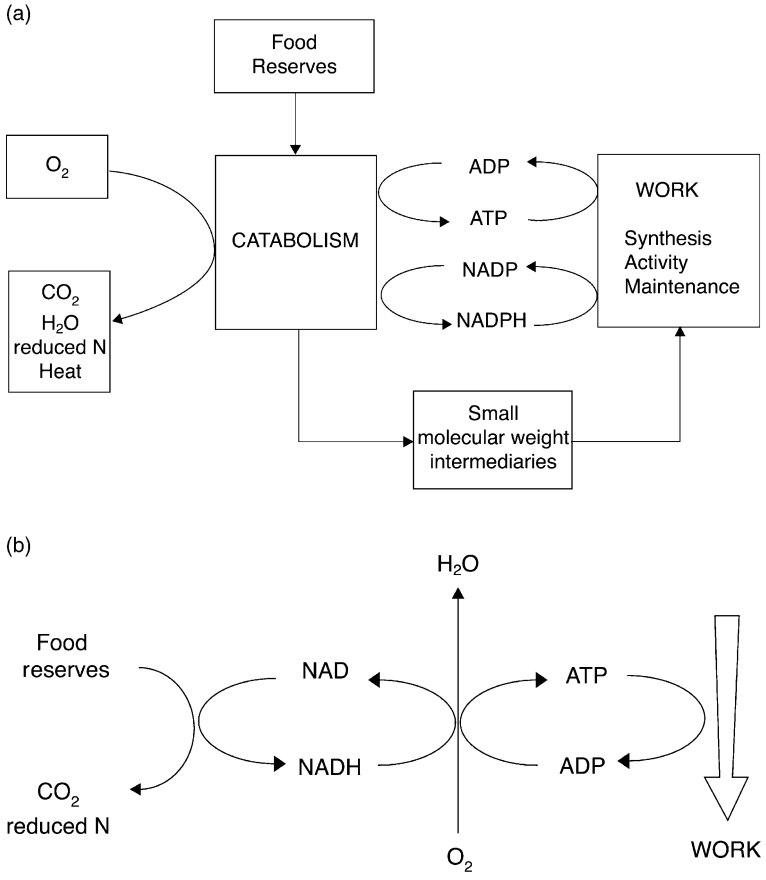


**Fig. 1.** A conceptual model of metabolism. (a) Schematic diagram of the relationship between catabolic processes generating ATP, reducing power and a range of small molecular weight intermediates to provide carbon skeletons, and anabolic processes plus maintenance. (b) A further simplification to emphasize the role of physiological work, and not temperature, in regulating the rate at which ATP is synthesized, and hence oxygen and food or reserves utilized. Diagrams modified from Clarke (1991, 1993).

*E* (the 'activation energy of metabolism'), and Gillooly *et al*. (2001) acknowledge that the residual variance about the fitted UTD model is probably related to differences in ecology between species.

The key assumption underlying the UTD, that metabolic rate is driven mechanistically by temperature, is incompatible with what we know of cellular physiology and the molecular mechanisms of evolutionary adaptation to temperature (Clarke 2004). Nevertheless resting metabolic rate in ectotherms is widely (though not universally) observed to co-vary with environmental temperature, and this requires explanation. Here we present an alternative explanation for the relationship between resting metabolic rate and environmental temperature in ectotherms.

## Metabolic rate

A glance at any biochemistry textbook will indicate the intense complexity of cellular metabolism. Complexity theory is now providing a glimpse at the underlying structure of metabolism (Jeong *et al*. 2000; Wagner & Fell 2001) but in energetic terms there is a common theme in that all these reactions involve water either as reactant or product (Clarke 2003a) and most require ATP. The production of ATP from glycolysis and the tricarboxylic acid (TCA) cycle is thus the central process in cellular metabolism.

Metabolic rate is a measure of the power utilization of an organism, and is traditionally measured as the rate of oxygen consumption. This is a reasonable approximation for in most organisms ATP is generated aerobically using oxygen as the final electron acceptor. Some organisms do generate ATP anaerobically, and this can be important in low oxygen environments, or when large amounts of ATP are needed rapidly such as during intense muscular activity. Typically the accumulation of anaerobic end products is temporary and these are cleared later when oxygen is available once more. The stoichiometry of oxygen utilized to ATP generated is, however, not constant for it depends on the metabolic substrate being oxidized.

A critical factor for any ecological discussion of metabolic rate is that the generation of ATP, and hence uptake of oxygen, does not proceed regardless; it is under complex and subtle feedback control. The most important control stems from the concentration of ATP itself. When ATP concentration is high, synthesis of ATP from ADP slows, and when ATP concentration drops because it is being utilized rapidly, synthesis of ATP is stimulated. There are also many other feedback mechanisms, such as inhibition of glycolysis by increase in the concentration of citric acid cycle intermediates.

This leads to a simple ecological picture of metabolism (Fig. 1) which emphasizes that the synthesis of ATP, and hence the utilization of oxygen and metabolic substrate (food or reserves), is driven by demand for ATP. Synthesis of new macromolecules also requires a supply of reducing power (typically NADPH) and a limited suite of small molecular weight intermediaries to provide carbon skeletons (Fig. 1a), but in simply ecological terms the synthesis of ATP is driven by the demand for physiological work (Fig. 1b). This control of ATP synthesis (and hence oxygen consumption) is central to any ecological discussion of metabolic rate. In ectotherms there is no mechanism for a purely temperature-driven synthesis of ATP, which would anyway be needlessly wasteful of valuable resources: once ATP concentration has reached a high level, ATP synthesis stops. Since any living organism continues to require oxygen, the ecological question thus becomes, what is the ATP needed for?

## Resting metabolism

It has long been recognized that there are a number of ATP-requiring processes which are essential for the cell to remain alive. These are often grouped together as basal metabolism. A simple functional definition of basal metabolism is shown in Fig. 2(a). Here basal metabolism is defined as the metabolic rate of an organism whose food intake is such that there is no net change in body mass. Higher food intake results in

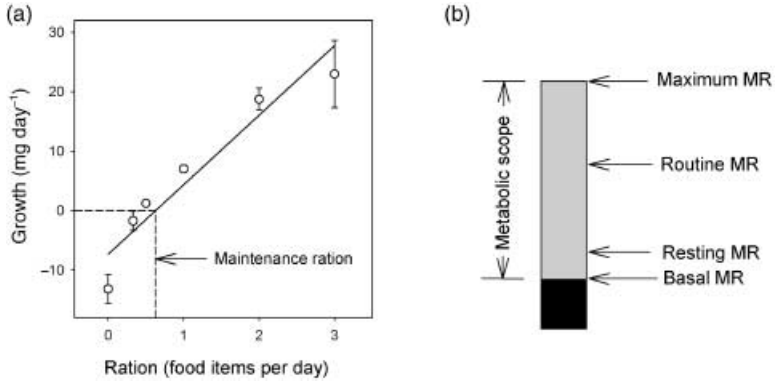


**Fig. 2.** A definition of basal metabolism. (a) The relationship between growth rate and ration, illustrating a functional definition of maintenance ration. Maintenance metabolism (basal metabolic rate) is defined operationally as the resting metabolic rate of an organism fed at maintenance levels. Data for larval *Sepia officinalis* (Clarke *et al*. 1989). (b) Diagrammatic representation of relationship between basal metabolic rate and active respiration, showing the definition of metabolic scope. This diagram also emphasizes that although resting metabolic rate is the best practical estimate we can make of basal metabolic rate, the two are not identical. MR = metabolic rate.

positive growth, a lower food intake in loss of mass as tissue is utilized to provide energy. The balance point is that food intake which just maintains body mass (hence the alternative term of maintenance metabolism), and this has been determined experimentally in many organisms by measuring growth rate as a function of food intake. This functional definition of basal metabolism reflects the early history of energetics, for which studies of domesticated mammals and birds were central (Brody 1945; Blaxter 1967). This approach is, however, neither suitable nor perhaps meaningful for many invertebrate organisms, particularly during larval development. A pragmatic alternative is to estimate basal metabolism by the oxygen consumption of an inactive, postabsorbtive, non-growing and non-reproducing individual. This is often termed resting metabolism and in many cases provides our best practical estimate of basal metabolism (Fig. 2b). Fish physiologists have introduced a related (but not identical) concept of standard metabolism, which is the lowest metabolic rate sustained for short periods; this is typically lower than maintenance or resting metabolism (Brett & Groves 1979).

Although the concept of basal metabolism has a long history, it is only recently that we have started to gain an understanding of what cellular processes are involved. Key insights have come from studies of isolated mammalian cell lines and bacterial cultures. These data indicate that a significant proportion of basal metabolism is made up from protein synthesis and work to maintain potential energy gradients (principally ionic) across membranes (Table 1). Particularly important is the proton gradient across the inner mitochondrial membrane, which is the driving force for ATP synthesis, and counteracting the passive leakage of protons back across this membrane is an important component of basal metabolism. Indeed some ectotherms are likely to spend more energy in their lifetime counteracting mitochondrial proton leak than they invest in either growth or reproduction.

The relative proportions of the different components of basal metabolism are unlikely to be the same in all tissues of all organisms. For any given tissue the absolute rate of basal metabolism, and the relative proportions of the various component processes, will differ depending on factors such as mitochondrial density, ribosomal concentration, requirement for ion gradients, protein synthesis and so on (Preedy *et al*. 1988). Thus the basal metabolic rate of a gram of liver, brain or muscle will vary. Furthermore, the basal metabolic rate of a given organism will depend on the relative proportion of tissues with differing inherent rates of basal metabolism (Daan, Masman & Groenewold 1990) and it will also vary during ontogenetic development. This variation in the proportion of different tissues may explain differences in basal metabolism between various lineages (Clarke & Johnston 1999). There are, however, also differences in basal metabolism within lineages, associated with ecology or lifestyle. Thus within fish, more active species tend to have higher basal metabolic rates (Morris & North 1984; Zimmerman & Hubold 1998) and in insects winged forms have a higher standard metabolic rate than flightless species (Addo-Bediako, Chown & Gaston 2002). These differences may be caused for example, by variation in mitochondrial concentration or membrane proliferation, but also by higher costs of cardiovascular work or muscle tonus.

Resting metabolism thus comprises a complex suite of very different processes (Table 1), though all require

**Table 1.** The major components of basal metabolic rate. Data are for rat tissue, which is the best studied system, but the relative proportions of the component processes are likely to vary across taxa. From Rolfe & Brown (1997)

| Process | Contribution to whole-organism basal metabolic rate (%) |
|---|---|
| Non-mitochondrial oxidative processes | 10 |
| Mitochondrial proton leak | 20 |
| $Na^+$ $K^+$-ATPase | 14 |
| Protein synthesis | 2–25 |
| Gluconeogenesis | 7 |
| Others* | 6–35 |

*Others includes $Ca^2$-ATPase (3–4%), nucleic acid turnover (1–5%), signal transduction (probably <3%), urea synthesis (2%), substrate cycling (unknown but possibly as much as 25%) and protein degradation.

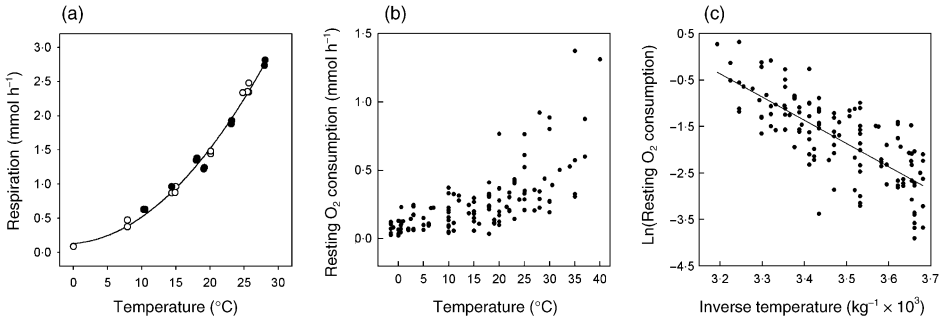


**Fig. 3.** The relationship between temperature and metabolic rate. (a) The relationship determined by Ege & Krogh (1914) for a single goldfish, *Carassius auratus*. The open circles show resting metabolism, and the closed data show oxygen consumption of the same individual under mild anaesthesia. The data have been plotted from Table 1 in Ege & Krogh (1914), following conversion from mass-specific representation and into SI units. This represents the first, and classical, study of the acute, thermodynamic, response of resting metabolic rate to temperature in any ectotherm. (b) The relationship for 68 species of teleost fish, with resting metabolic rate (MR) plotted for the temperature at which the fish actually live. This is the evolutionary, between-species relationship. Redrawn from Clarke & Johnston (1999). (c) The same relationship, plotted in Arrhenius form (natural logarithm of the rate as a function of inverse temperature) which linearizes the relationship.

ATP and hence oxygen consumption. Since the absolute rates, and relative importance, of these processes vary from tissue to tissue, and hence from organism to organism, the important question is, how do these rates compare in organisms living at different temperatures?

## Resting metabolic rate and temperature

Variations in the metabolic rate of organisms with body size and temperature have long been known, with the classic work in this field being that of Hemmingsen (1950, 1960). Comparison of endotherms with ectotherms living over a range of temperatures presented a conceptual problem for this analysis, and the pragmatic approach taken was either to limit observations to those made at a particular temperature, or to 'correct' the data for ectotherms to a common temperature, using an assumed value for the temperature sensitivity of basal metabolism. Although the tradition of plotting resting metabolic rate as a function of the temperature at which the organism was actually living goes back to the seminal work of August Krogh (Ege & Krogh 1914; Krogh 1916), it is only relatively recently that this approach has been taken in between-species studies. The monotonic positive nonlinear relationship between resting metabolic rate and temperature (Fig. 3a,b) is typical of all studies of marine organisms to date (Ivleva 1980; Ikeda 1985; Clarke & Johnston 1999; Peck & Conway 2000; Ikeda *et al*. 2001). There are almost no comparable studies of terrestrial organisms over the same scale, a notable exception being that of Addo-Bediako *et al*. (2002). This study of standard metabolism in 346 species of insect found a weak but significant negative relationship with habitat temperature. This different pattern may reflect a fundamental difference between the physiology of marine and terrestrial ectotherms, or the more complex thermal environment inhabited by insects. The generality of this result urgently needs to be tested for other groups, and for the rest of this paper we will concentrate on the marine environment where the signal is stronger and clearer.

Two features of the relationship between metabolic rate and temperature are of interest. The first is that there is a qualitative similarity between the within-species (Fig. 3a) and the between-species relationships (Fig. 3b). The second is that the between-species variance also increases with temperature. There are three possible classes of explanation for the relationship between resting metabolic rate and temperature:

1. Deterministic: temperature dictates resting metabolic rate in a mechanistic manner. The most recent such explanation is the universal temperature dependence (UTD) of metabolism proposed by Gillooly *et al*. (2001). In its strictest form (what might be termed the hard UTD hypothesis: Clarke 2004) metabolic rate is assumed to be dictated mechanistically by temperature, driven by the kinetic energy of cellular constituents. This mechanism applies both to an individual organism subject to an acute temperature change, and to organisms adapted over evolutionary time to live at different times (see discussion in Clarke 2004).
2. Indirect: temperature dictates the overall slope of the relationship between resting metabolic rate and temperature in organisms adapted evolutionarily to live at different temperatures, but its influence is indirect, through a combination of energetic trade-offs and evolutionary temperature adaptation. This is the evolutionary trade-off hypothesis of Clarke (1993, 2004).
3. No effect: temperature has no influence on resting metabolic rate and the relationship is epiphenomenal, being the result of selection on other factors entirely. Nobody has proposed this but the possibility needs formal consideration (although it is very difficult to know how to do so other than by elimination of all other hypothesis involving direct or indirect temperature effects).

## Does temperature determine resting metabolic rate directly?

Gillooly *et al*. (2001) propose that the higher kinetic energy of cellular components at higher temperatures leads directly to higher resting metabolic rate. Gillooly *et al*. (2001) do acknowledge that there is residual variance about the UTD relationship, and that this is related to ecological factors. The essence of the UTD relationship is, however, a mechanistic relationship between resting metabolic rate and temperature. In other words, an organism living at a higher temperature has no option but to synthesize more ATP and consume more oxygen.

There are several problems with a purely deterministic relationship between temperature and resting metabolic rate. The first is the evolutionary question why a higher temperature should drive an organism to produce more ATP, and hence utilize hard-won food or reserves, for no good ecological reason. There are clear ecological benefits to be gained from a high resting metabolic rate (notably the ability to generate a higher absolute aerobic scope and thereby allow a more active lifestyle: Clarke 2004). The evolution of such a high resting metabolic rate is, however, the result of selection and involves evolutionary trade-offs; it is not the result of a simple mechanistic response to the higher temperature. Evidence for the latter comes from the existence at the same environmental temperature of organisms with both high and low resting metabolic rates (Fig. 3b).

The most critical problem for the mechanistic explanation, however, centres on the evolutionary adaptation of enzymes to temperature. The combination of the Stefan–Boltzmann distribution of energy in a population of molecules together with the concept of activation energy introduced by Arrhenius (1889, 1915) provides a powerful mechanistic explanation of the thermal dependence of reaction rate of simple mixtures in dilute solution under equilibrium conditions, and where nothing in the system changes apart from temperature. We now know, however, that the rate-limiting step in enzyme catalysis is not the breaking and formation of covalent bonds to form product (which tends to be relatively fast because of the structure and ionic environment of the active site); rather it is the binding and release of ligands and products. These tend to be relatively slow processes, and the contribution of cellular enthalpy to the overall free energy change involved is not constant across the temperature range inhabited by organisms (Hochachka & Somero 2002).

In simple terms, homologous enzymes from organisms living at different temperatures tend to exhibit differences in primary structure away from the active site which render catalytic activity relatively independent of temperature (Somero 1995). Furthermore, the variation in concentration of reactants and of the kinetic characteristics of the enzyme(s) involved violate the assumptions necessary for the application of statistical thermodynamics to between-species analyses of the thermal behaviour of resting metabolic rate. The outcome of evolutionary temperature adaptation is thus to render key aspects of organismal physiology moderately independent of cellular enthalpy: evolution has worked to overcome the tyranny of Boltzmann. (For the classic work in this area see Graves & Somero 1982; and Holland, McFall-Ngai & Somero 1997; for a review of the field see Hochachka & Somero 2002.) These arguments are presented in more detail in Clarke (2004).

If the activity of key metabolic enzymes can be rendered relatively independent of temperature when organisms adapted to live in different thermal environments are compared, why does the cost of living (basal metabolism) still scale with temperature? The answer lies in the indirect effects of temperature on cellular metabolism.

## The indirect effect of temperature on resting metabolic rate: the evolutionary trade-off hypothesis

Although we can reject a direct mechanistic influence of temperature on resting metabolic rate, it is clear that the two are nevertheless linked in some way (Fig. 3a,b). Recent advances in our understanding of the processes involved in basal metabolic rate are starting to clarify what these links might be.

A key component of basal metabolic rate is protein synthesis. Although it has long been known from incubation studies of isolated enzymes that individual proteins degrade faster at higher temperatures, studies of homologous enzymes isolated from organisms living at different temperatures have shown that enzymes from polar organisms tend to be more thermally sensitive than those from temperate or tropical organisms (Somero 1995; Moerland 1995; Russell 2000; Hochachka & Somero 2002). This might imply a requirement for greater protein turnover in animals living at lower temperatures, and yet the relationship between resting metabolic rate and temperature (Fig. 3a,b) would suggest that absolute rates of protein synthesis (a major contributor to basal metabolic rate: Table 1) are faster in animals living at warmer rather than colder temperatures; that is, precisely the reverse. The resolution of this apparent paradox is that comparative studies of enzyme thermal stability tend to be carried out all at the same temperature, whereas the homologous enzymes are adapted to work optimally at different temperatures. The important factors are enzyme activity and enzyme stability at the temperature at which the organism lives. The rate at which a given enzyme needs to be turned over is thus the result of an evolutionary trade-off between structure, function (kinetics) and stability, the net result being lower absolute rates of resting protein synthesis at low temperatures and higher rates in organisms living at warmer temperatures (Clarke 1993). This hypothesis for the relationship

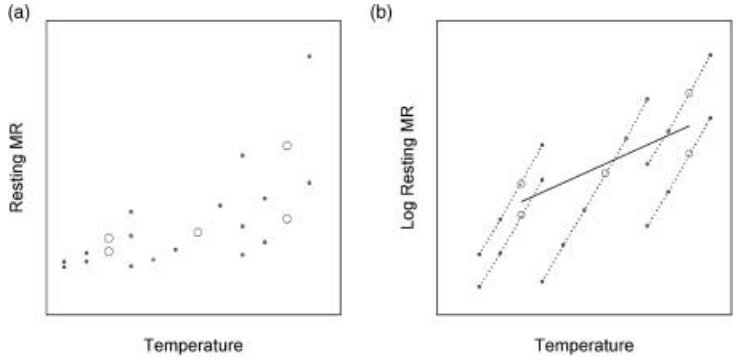


**Fig. 4.** A conceptual model of the relationship between resting metabolic rate (MR) and temperature in ectotherms. Data are shown for five hypothetical organisms, two living at a cool temperature, one at a moderate temperature and two at a high temperature. The closed symbols represent the acute (thermodynamic) effect of temperature on each species. The open symbol represents the resting metabolic rate for each species at its normal living temperature. The across-species relationship is different in slope from the within-species relationships. It is also different in kind, being a statistical population of individual evolutionary optimizations. (a) Linear plot. (b) Logarithmic plot which emphasizes the difference in slope (temperature sensitivity) between the within (dashed lines) and across-species (solid line) relationships.

between protein structure, function and turnover is based on studies of a small number of enzymes, and involves extrapolation to the entire protein complement of the cell. We know effectively nothing of the thermal and kinetic characteristics of most of the cells proteins, and furthermore it is likely that both kinetics and turnover of cellular proteins are influenced by other factors including changes in concentration, cellular milieu or chaperone proteins (Hofmann *et al*. 2002; Clarke 2003b).

The passive leakage of protons across the inner mitochondrial membrane varies with membrane composition and temperature (Hulbert & Else 2000). As with discussion of any physiological response to temperature, however, we cannot generalize from the effect of temperature within one species to the between-species evolutionary relationship. The acute response of a given system to a change in temperature is driven essentially by thermodynamics, whereas in comparing between species much more than temperature changes. Thus in the case of passive proton leak, organisms living at different temperatures will probably have mitochondria that differ in membrane architecture and composition, cristae density and protein concentration. The concentration of mitochondria within tissues may also vary (Archer & Johnston 1991). All of these are likely to influence overall proton leak (Hulbert & Else 2000), and hence we cannot generalize from results obtained from within one species to the evolutionary response across species. To some extent proton leak is under evolutionary control (for example in the evolution of endothermy, and of thermogenic tissues: Rolfe & Brown 1997), and all we can do is look at the end result of the evolutionary process.

Analogous arguments to those above for protein turnover and mitochondrial proton leak can be advanced for the other components of basal metabolic rate, although here the experimental data are almost non-existent. We can, however, generalize to a conceptual model of how and why resting metabolic rate varies with temperature, and this is illustrated in Fig. 4. This model has two key features:

1. The within-species relationship between resting metabolic rate and temperature reflects the acute thermodynamic effect of temperature on the organism. As temperature increases, more ATP is required to fuel processes driven faster by higher cellular kinetic energy, at least until acclimation processes take effect.
2. The resting metabolic rate of any one species at its normal environmental temperature represents an evolutionary optimization for that species, influenced by temperature, ecology and life history. This is the evolutionary trade-off hypothesis (Clarke 1993, 2004).

The complexity of cellular structure and metabolism mean that the acute thermodynamic response to temperature cannot be predicted from first principles. It would appear, however, that this response is determined to a significant extent by the temperature sensitivity of weak bonds, and typical $Q_{10}$ values are 2–3 (Hochachka & Somero 2002). A compilation of within species $Q_{10}$ values for the acute effect of temperature on resting metabolic rate in teleost fish suggested a mean $Q_{10}$ of 2·36 (SE 0·21, $n$ = 14) (Clarke & Johnston 1999).

Because the overall between-species relationship between resting metabolic rate and temperature is the result of species-specific evolutionary optimizations, it too cannot be predicted from first principles. It can only be described statistically. We can, however, ask what evolutionary pressures are likely to influence the resting metabolic rate of a particular species.

The cost of basal metabolic rate must be met from food or reserves, and so it would be expected that environments with chronic or seasonally acute food shortages would select for a reduced basal metabolic rate and an efficient metabolism (Lotka 1922). Where a seasonal abundance of food allows for the synthesis of reserves, this can offset the cost of a higher basal metabolic rate in times of scarcity. Thus in polar regions benthic organisms are characterized by a very low resting metabolic rate and tend not to synthesize over-wintering energy reserves despite the long winter period when feeding is reduced or impossible. In contrast planktonic organisms whose lifestyle involves the extra energetic demands of staying in the water column generally do synthesize lipid reserves for over-wintering (Clarke & Peck 1991).

Although a higher basal metabolic rate is costly, it also brings benefits. In particular it would appear that factorial aerobic scope tends to be broadly constant across the range of physiological temperatures, at least within related lineages (Wilson 1974; Johnston, Clarke

& Ward 1991). This means that a higher basal metabolic rate allows a greater absolute aerobic scope, and hence more active lifestyles (Clarke 2003b). An alternative way of looking at this is that an energetic lifestyle inevitably brings with it a high basal metabolic rate. A higher basal metabolic rate may also allow a more rapid response to an environmental challenge, through having more cellular machinery available.

The basal metabolic rate of an organism thus depends on its ecology, with the level being set by an evolutionary trade-off between costs and benefits. A comparison of teleost fish with similar sedentary lifestyles indicates that even when ecology is controlled, there remains a positive relationship between resting metabolic rate and temperature (Johnston *et al*. 1991). The greater variance in resting metabolic rate at higher temperatures when all lifestyles are included suggests a wider range of evolutionary options at warmer compared with lower temperatures. It is not clear why this is so, though important factors may be temperature related constraints on ATP generation by mitochondria, or on the mechanical power that can be developed by muscle (Clarke 2003b).

One important question is whether the greater basal metabolic rate at a higher temperature, or a more energetic lifestyle, involves a general up-rating of all component processes, or whether some processes increase more with temperature than others. Recent work on the Antarctic Limpet *Nacella concinna* has indicated that at low temperatures, overall absolute rates of protein synthesis are low, but a lower proportion of this protein is retained as growth (K. P. P. Fraser, unpublished data). This suggests that at low temperatures a greater proportion of newly synthesized protein is degraded, perhaps because of folding errors. Although these data are for actively growing animals, they do suggest that the balance of processes comprising basal metabolic rate varies across the physiological temperature range. This is an important area for future research, particularly in respect of protein dynamics and mitochondrial function (Clarke 2003b).

Since each individual species represents a more or less independent separate evolutionary optimization, the overall between-species relationship between resting metabolic rate and temperature can only be described as a statistical population. (The optimizations are only partially independent because of phylogenetic constraints: more closely related species tend to have more similar ecologies and lifestyles than more distantly related organisms.) The energetic arguments above would, however, suggest that the overall slope would be shallower than the acute response (Fig. 4); in other words the within-species $Q_{10} > Q_{10}$ between species. The hard form of the UTD hypothesis, direct regulation of metabolic rate by temperature, would predict that these two $Q_{10}$ values would be identical (Clarke 2004). In the case of teleost fish the prediction of the evolutionary trade-off hypothesis is upheld in that the between-species $Q_{10}$ of 1·83 falls outside the 95% confidence intervals of the mean $Q_{10}$ for within-species studies (Clarke & Johnston 1999). The difference is, however, small and so cannot be regarded as a definitive rejection of direct regulation by temperature (the deterministic or hard UTD hypothesis). Overall, however, our current understanding of the physiological processes comprising basal metabolic rate, and the mechanisms of evolutionary adaptation to temperature, indicate clearly that the relationship between temperature and resting metabolic rate is one of indirect influence.

## Which statistical model?

A plot of resting metabolic rate as a function of temperature (Fig. 3a,b) is markedly non-linear and exhibits increasing variance with temperature. This is a common pattern in macroecology and implies the involvement of more than one causal factor. Ecologists, however, prefer to work with linear relationships, especially where broad generalizations are needed for incorporation in models.

The data for teleost fish can be linearized by any one of several statistical models including logarithmic, exponential and Arrhenius (natural logarithm of metabolic rate as a function of inverse temperature) (Clarke & Johnston 1999). Early studies favoured logarithmic models (e.g. Hemmingsen 1960) but more recently the Arrhenius model has become popular, perhaps because of its basis in statistical thermodynamics. The data for fish are plotted in Arrhenius form in Fig. 3(c). All three models tend to equalize the variance across the temperature range, and they are equally good at explaining variation. There is thus little to choose between them statistically, although the fitted models do give slightly different predictions for thermal sensitivity.

## Conclusion

It has long been known that in marine organisms basal metabolic rate, as estimated by resting metabolic rate, increases with temperature. Although mechanistic explanations have been advanced, namely that temperature drives resting metabolic rate higher at warmer temperatures simply as a result of thermodynamics, energetic and evolutionary arguments suggest this is not so. Rather an organism's resting metabolic rate appears to be set by an evolutionary trade-off between costs, benefits and ecological lifestyle. The resting metabolic rate may thus be regarded as the energetic cost of evolutionary temperature adaptation, in the sense of the energetic demand of maintaining cellular machinery adapted to operate at a particular temperature (Clarke 2003b). While a clear picture of the nature of the relationship between temperature and resting metabolic rate is emerging for marine organisms, this is not yet the case for terrestrial ectotherms. At present we cannot judge whether the more complex thermal environment of many terrestrial habitats has

led to the evolution of a fundamentally different thermal physiology, or whether the nature of the environment simply makes the evolutionary signal harder to detect. A more complete understanding of the component processes of basal metabolic rate, and why these vary with temperature, is important to link physiology to ecology in what has been termed evolutionary physiology (Feder *et al*. 2000).

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