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Commentary

Environment, antecedents and climate change: lessons from the study of temperature physiology and river migration of salmonids

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Summary

Animal distributions are shaped by the environment and antecedents. Here I show how the temperature dependence of aerobic scope (the difference between maximum and minimum rates of oxygen uptake) is a useful tool to examine the fundamental temperature niches of salmonids and perhaps other fishes. Although the concept of aerobic scope has been recognized for over half a century, only recently has sufficient evidence accumulated to provide a mechanistic explanation for the optimal temperature of salmonids. Evidence suggests that heart rate is the primary driver in supplying more oxygen to tissues as demand increases exponentially with temperature. By contrast, capacity functions (i.e. cardiac stroke volume, tissue oxygen extraction and haemoglobin concentration) are exploited only secondarily if at all, with increasing temperature, and then perhaps only at a temperature nearing that which is lethal to resting fish. Ultimately, however, heart rate apparently becomes a weak partner for the cardiorespiratory oxygen cascade when temperature increases above the optimum for aerobic scope. Thus, the upper limit for heart rate may emerge as a valuable, but simple predictor of optimal temperature in active animals, opening the possibility of using biotelemetry of heart rate in field situations to explore properly the full interplay of environmental factors on aerobic scope. An example of an ecological application of these physiological discoveries is provided using the upriver migration of adult sockeye salmon, which have a remarkable fidelity to their spawning areas and appear to have an optimum temperature for aerobic scope that corresponds to the river temperatures experienced by their antecedents. Unfortunately, there is evidence that this potential adaptation is incompatible with the rapid increase in river temperature presently experienced by salmon as a result of climate change. By limiting aerobic scope, river temperatures in excess of the optimum for aerobic scope directly impact upriver spawning migration and hence lifetime fecundity. Thus, use of aerobic scope holds promise for scientists who wish to make predictions on how climate change may influence animal distributions.

Key words: thermal niches, optimal temperature, aerobic scope, oxygen uptake, metabolic rate, cardiac output, heart rate, tissue oxygen extraction, oxygen partial pressure, biotelemetry, lifetime fecundity, climate change.

Introduction

The study of the physiological and biochemical mechanisms that set the limits for environmental tolerance, and which in many ways distinguish species, is an active area of investigation that has gained importance in the current era of climate change. This article is focused on the physiological mechanisms that become critical when fishes, particularly salmonids, approach their upper temperature limits. Furthermore, to address the need for examples of how large-scale environmental records of climate are translated at the scale of the organism (Helmuth, 2009), this mechanistic understanding is applied to the river migration of an adult Pacific salmon species.

My focus on predominantly one group of fishes (the salmonids) and on one environmental variable (temperature) is for two reasons. First, this is where data are most abundant. Second, a case study of temperature tolerance among fishes is likely to prove extremely fruitful in addressing the more general and important question of animal resilience and adaptability to environmental change. This is because fishes have evolved around species-specific niches, living in almost every conceivable aquatic habitat and representing almost half of the earth's vertebrate species. However, no single fish species tolerates the entire temperature range exploited by fishes (from –2°C in Antarctica to +42°C in Lake Magadi, Kenya). Similarly, ~43% of all fish species live in freshwater rather than the vastly more abundant saline habitats [>99% of the available aquatic habitat (Nelson, 2006)]. Although the foundation for the thermal distributions that we see today may seem to reflect an absence of the requisite genomic machinery, a more circumspect view may be need. For example, Antarctic fishes, which have lived in a thermally stable environment for many thousands of years, are now known to be able to thermally acclimate to temperatures previously thought to be lethal and well above those found in their present ecological niche (Franklin et al., 2007). Thus, observing a stenothermal existence does not necessarily mean insufficient phenotypic plasticity to tolerate a broader temperature range.

Temperature and aerobic scope

Temperature has a central role in shaping the distribution of animals. In explaining latitudinal and longitudinal limits of biomes, Shelford's law of tolerances envisaged a centre of animal abundance bounded by 'toleration' of environmental 'controlling factors' (Fig. 1A). Clearly, the poleward shift in fish distributions with the progressive warming of aquatic habitats (Brander et al., 2003; Brander, 2007; Pörtner and Knust, 2007; Dulvy et al., 2008)

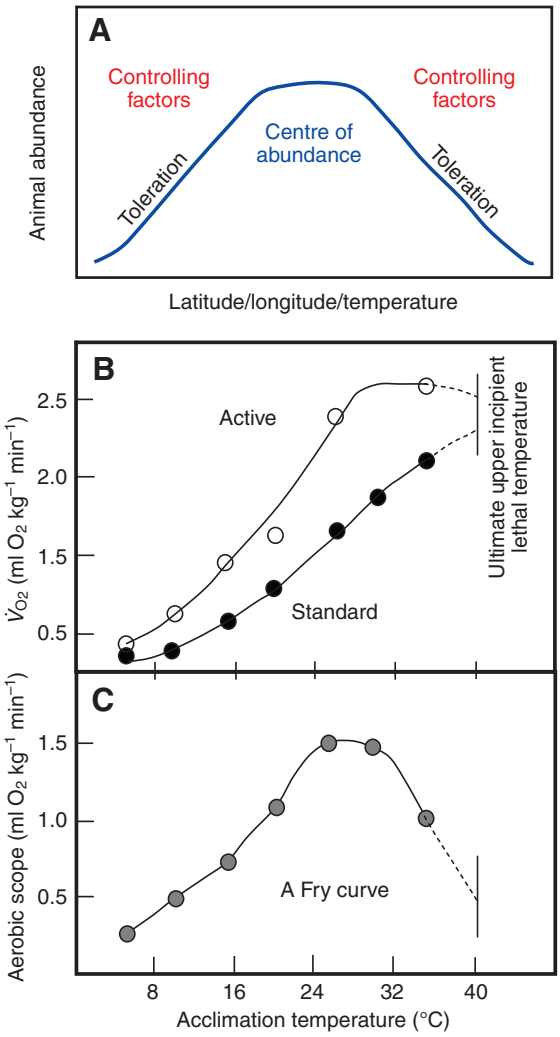

Fig. 1. The controlling and limiting effects of temperature on animal distributions, metabolic rate and scope for activity. (A) A schematic representation of Shelford's law of tolerances (Shelford, 1931). (B) Measurements of standard and active metabolic rates for goldfish as a function of temperature approaching their upper incipient lethal temperature. (C) Aerobic scope (or scope for activity) as a function of temperature, which is the difference between the measurements of standard and active metabolic rates shown in B (Fry, 1947).

represents a more insidious manifestation of the anthropogenic-driven change in animal distribution that Shelford characterised nearly 80 years ago (Shelford, 1931).

Temperature tolerance at the whole animal level was first given a mechanistic explanation for fishes by Fry (Fry, 1947), who showed that temperature both controlled *and* limited their metabolic rate. To illustrate his ideas, he used scope for activity, which is now termed aerobic or metabolic scope, i.e. the difference between standard and active metabolic rates (Fig. 1B,C). In doing so, Fry recognized that the predictive value of knowing the temperature dependence of aerobic scope was considerably greater than that of knowing a temperature tolerance range (e.g. critical maximum and minimum temperatures; CT_{max} and CT_{min}). Indeed, the aerobic scope concept is now being used broadly to examine the impacts of the aquatic warming trends and other environmental climate changes on marine ectotherms (Pörtner, 2001; Pörtner, 2002; Mark et al., 2002; Pörtner and Knust, 2007; Pörtner and Farrell, 2008), illustrating an importance well beyond fishes. Even so, and as shown in the following, our understanding of the proximate causes that limit a fish's aerobic scope beyond its optimal temperature range remains formative.

The Fry curve for aerobic scope

Aerobic scope is derived from measurements of a fish's minimum and maximum rates of oxygen uptake ($\dot{V}_{O_2}$) as a function of temperature (Fig. 1B). The difference between these two rates is aerobic scope, which takes the form of a bell-shaped curve as a function of temperature – a 'Fry curve' for aerobic scope (Fig. 1C). Simplistically, a Fry curve represents an animal's capacity for activity as a function of temperature.

Minimum $\dot{V}_{O_2}$ (standard or basal metabolic rate) represents the metabolic cost to support an animal's existence in a non-feeding, non-reproducing and non-motile state. Minimum $\dot{V}_{O_2}$ is directly affected by body temperature [thermodynamics (Krogh, 1914)], typically doubling or tripling with a 10°C acute increase in temperature (termed a Q_{10} effect; Fig. 1B). Minimum $\dot{V}_{O_2}$ also varies among species (a genetic basis) and with body size [scaling (Schmidt-Nielsen, 1984)].

Clearly, life beyond short-term existence requires a capacity to increase $\dot{V}_{O_2}$ above this minimum level. Energy expenditure for feeding, growth, reproduction and locomotion (used for foraging as well as escape from predators and unfavourable environments) needs an active $\dot{V}_{O_2}$. In terms of the temperature dependence of active $\dot{V}_{O_2}$, Fry (Fry, 1947; Fry and Hart, 1948) made the crucial observation that maximum $\dot{V}_{O_2}$ of exercising goldfish (*Carassius auratus*) failed to continue increasing with temperature beyond an optimal temperature (T_{opt}). By contrast, standard $\dot{V}_{O_2}$ of resting fish continued its exponential increase until temperature approached a lethal level (Fig. 1B). Thus, the T_{opt} for aerobic scope is created by the failure of maximum $\dot{V}_{O_2}$ to continue increasing with temperature. Consequently, because activities such as growth depend on aerobic scope, it is not surprisingly that growth rate as a function of temperature has a similar bell-shaped, species-specific curve for fishes (Fig. 2B) (Brett, 1971). In fact, fish must eat more just to deal with the exponential increase in standard $\dot{V}_{O_2}$. Like minimum $\dot{V}_{O_2}$, active $\dot{V}_{O_2}$ is also species-specific and varies with body size.

At a critical temperature (T_{crit}), aerobic scope is zero and aerobic activity becomes impossible. Thus, a thermal niche for existence in a resting state is bounded by the upper and lower T_{crit} values (which correspond closely to the CT_{max} and CT_{min} values determined using other methods). However, existence without an aerobic scope is necessarily short-lived in nature because, besides being an easy target for predators, starvation is just a matter of time. Consequently, an animal's functional thermal niche is narrower than that bounded by T_{crit}.

Fry curves are species specific. Differences result from their position on the temperature scale (temperature niches), being centred near 27°C for goldfish and at cooler temperatures (<20°C) for most salmonids (Fig. 2A). There are also species differences in standard and active $\dot{V}_{O_2}$. Athletic species such as salmonids have a high aerobic scope, but this does not necessarily translate into a larger thermal niche. For example, generalists such as goldfish (Fig. 2A) and *Fundulus heteroclitus* (Fangue et al., 2006) have a low aerobic scope and a broader thermal niche (eurythermal) compared with salmonids.

Scaling up of laboratory-derived aerobic scope data to ecology and biogeography will not necessarily be a simple task because other environmental factors reduce aerobic scope and narrow an

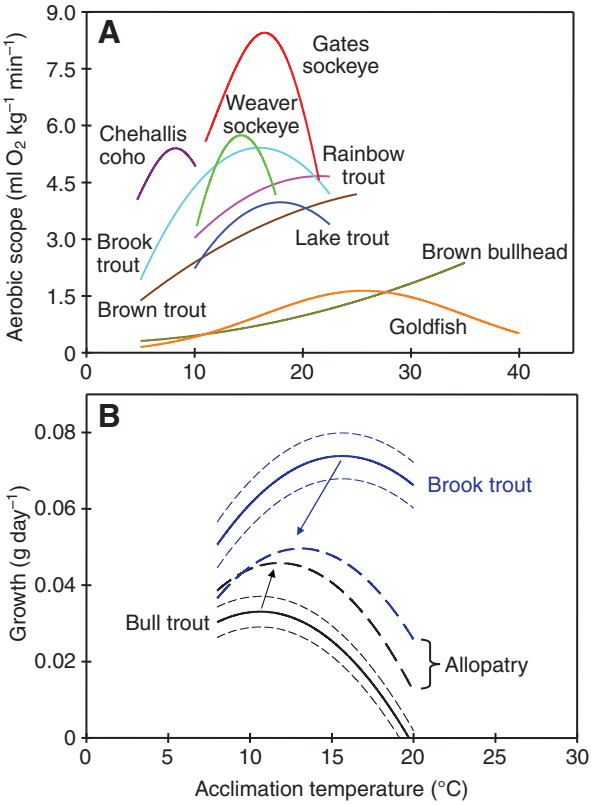

Fig. 2. The influence of temperature on aerobic scope and growth rate. (A) Fry curves for a range of salmonids and other species (Fry, 1947; Fry, 1948; Fry and Hart, 1948; Lee et al., 2003). (B) Growth rates of brook trout and bull trout grown either separately (solid lines with the accompanying dashed lines showing the 95% confidence limits) or together (long dashed lines grouped by allopatry) (McMahon et al., 2007).

animal's functional thermal niche (Fry, 1947; Fry, 1971; Brett, 1971; Pörtner and Farrell, 2008; Munday et al., 2009). For example, aquatic hypoxia, independent of temperature, can reduce aerobic scope (Graham, 1949; Gibson and Fry, 1954; Fry, 1971; Brett, 1971) to the extent that feeding and growth are halted, and development and reproduction are delayed (see Richards et al., 2009). Therefore, both hypoxia and hypercapnia are likely to constrain the breadth and height of a Fry curve (Pörtner and Farrell, 2008). Furthermore, the aerobic scope for a developing fish may not reach its full potential until the cardiorespiratory system is fully developed. Therefore, a family of Fry curves may exist for different life stages. Behaviour adds further complexity. For example, inter-specific competition can shift the T_{opt} for growth (Fig. 2B), as seen in brook trout (*Salvelinus fontinalis*) when growth was suppressed while competing with bull trout (*Salvelinus confluentus*), but not *vice versa* (McMahon et al., 2007).

An important index that can be derived from a Fry curve is the thermal window, the temperature difference between T_{opt} and T_{crit}. This thermal window is an index of a species' resilience to temperature change. In salmonids, the thermal window for the collapse of aerobic scope with warming is just 6–7°C (Fry, 1947; Farrell et al., 2008), which is a relatively small safety margin in the context of global warming scenarios. Tropical species apparently have narrow thermal windows too (Hoegh-Guldberg et al., 2007; Tewksbury et al., 2008) and live close to their T_{crit}. For example, cardinalfishes (*Ostorhinchus doederleini* and *O. cyanosoma*) were found to lose nearly 50% of their aerobic scope with only a 2°C increase above the average summer temperature (Nilsson et al., 2009), and an increase of 3°C compromised growth of spiny-damselfish (*Acanthochromis polyacanthus*) (Munday et al., 2008). However, the collapse of aerobic scope at warm temperatures was less evident (Fig. 2A) for the bullhead (*Ameiurus nebulasa*) and brown trout (*Salmo trutta*), suggesting that other factors may set thermal tolerance.

The rise and fall of aerobic scope in salmonids

As temperature increases, exponentially more oxygen must be delivered to tissues, which is the task of the cardiorespiratory system. Since maximum $\dot{V}_{O_2}$ fails to increase beyond T_{opt}, the decline in aerobic scope beyond T_{opt} (i.e. the downward trend of a Fry curve) therefore reflects the inability of the *maximum* cardiorespiratory capability to keep pace with these increasing tissue oxygen demands. By contrast, T_{crit} corresponds with a failure of the *resting* cardiorespiratory capability to keep pace with increasing tissue oxygen demands. The resultant mismatch between oxygen supply and oxygen demand forces animals to progressively switch to anaerobic metabolism to survive (Pörtner, 2001; Frederich and Pörtner, 2000), perhaps causing an acceleration of cardiorespiratory collapse (Farrell et al., 2008) and the rightward skew often seen in Fry curves.

At present, cardiorespiratory information pertaining to the collapse of aerobic scope during warming is most abundant for salmonids. The data are examined below within the context of the cardiorespiratory oxygen cascade in order to explore why active $\dot{V}_{O_2}$ does not increase beyond T_{opt} and why minimum $\dot{V}_{O_2}$ collapses at T_{crit}.

Active $\dot{V}_{O_2}$ and the cardiorespiratory oxygen cascade

The cardiorespiratory oxygen cascade conceptualizes the movement of oxygen down its partial pressure gradient from a respiratory medium to tissues. Hence, $\dot{V}_{O_2}$ corresponds to the oxygen flux per unit time through this cascade and oxygen diffusion rates are proportional to the relevant oxygen partial pressure (P_{O_2}) gradients. For fish, oxygen diffuses from water across gill secondary lamellae and binds to haemoglobin (Hb) in red blood cells, which are transported by the circulatory system to tissues where oxygen diffuses across the capillary wall and into the cell to be used in mitochondrial respiration (Fig. 3).

A countercurrent arrangement of blood and water flow at the secondary lamellae ensures that the arterial blood leaving the gills has a P_{O_2} (Pa_{O_2}) close to ambient water, and its Hb is almost fully saturated, i.e. the oxygen content of arterial blood (Ca_{O_2}) is near maximal. Convection of oxygen to tissues by the arterial system is quantified as the product of Ca_{O_2} and cardiac output. Thus, increasing cardiac output is the only means to internally transport more oxygen to the tissues, unless stored red blood cells are released from the spleen to increase Hb concentration [Hb] and hence Ca_{O_2} (see Gallaugher and Farrell, 1998). Once in tissue capillaries, factors such as the architecture of the capillaries, the presence of myoglobin and lipid droplets in the cytoplasm and the actual location of mitochondria within the cell significantly influence the rate of diffusion of oxygen from the red blood cell to the mitochondria.

In a resting fish, increasing tissue oxygen delivery with increasing temperature could simply recruit mechanisms that are normally used during exercise. When salmonids exercise at a constant temperature, there are increases in gill ventilation (to deliver more water), cardiac output (to transport more oxygen to

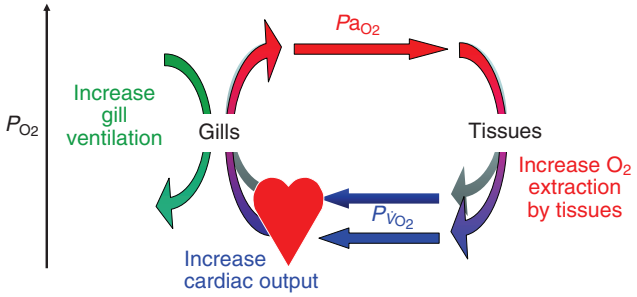

Fig. 3. A schematic diagram representing the oxygen cascade for a fish during rest (shaded lines and arrows) and swimming (dark lines and arrows). The oxygen partial pressure is an arbitrary scale (see text for details).

the tissues) and tissue oxygen extraction from blood (Stevens et al., 1967; Kiceniuk and Jones, 1977). Increased tissue oxygen extraction can contribute almost as much to the increased $\dot{V}_{O_2}$ as cardiac output because resting fish remove only about one third of the arterial oxygen and so venous oxygen content (Cv_{O_2}) and venous blood P_{O_2} (Pv_{O_2}) can decrease considerably during exercise (Fig. 3). While all of these exercise-induced cardiorespiratory changes are possible during warming, as shown below, not all of them occur when resting fish are warmed up to T_{crit}.

When an exercising fish is warmed, it is more a matter of how much the warming increases the rate and force of muscle contraction to enhance maximum cardiorespiratory capacity. In addition, oxygen diffuses at a faster rate, potentially allowing a lower Pv_{O_2}. Furthermore, the temperature sensitivity of the Hb–oxygen binding curve (e.g. Clark et al., 2008a) is such that a rightward shift with warming increases the Pa_{O_2} of fully saturated arterial blood. This also promotes a faster unloading of oxygen at the tissues. In fact, Cv_{O_2} could decrease during warming without a decrease in Pv_{O_2} (this direct temperature effect is in addition to a similar benefit from the Root- or Bohr-shifts as tissues release more carbon dioxide and H^+ during exercise).

Some fairly simple theoretical predictions can be made using this conceptual framework, against which existing cardiorespiratory data on warming in fishes can be compared. The analysis is further simplified by asking where the potential limitation might exist (gills, circulatory system or tissues), and by focusing on underlying mechanisms (at near T_{crit} for resting fish and at T_{opt} for exercising fish).

Changes in cardiorespiratory variables with acute warming in association with T_{opt} in exercising salmonids and T_{crit} in resting salmonids

A limitation at the gills?

Oxygen is poorly soluble in water. Compounding this, its solubility in water decreases ~2% per degree centigrade. Therefore, gill ventilation must compensate for the decreased oxygen availability and the lower Hb–oxygen affinity, as well as increased tissue oxygen demand as temperature increases. Therefore, a decrease in Pa_{O_2} during warming would indicate a clear problem associated with gill oxygen delivery and transfer. However, the data for salmonids are inconsistent on this matter.

When exercising adult sockeye salmon (*Oncorhynchus nerka*) were warmed to a temperature well above T_{opt}, Pa_{O_2} was maintained (Steinhausen et al., 2008). Similar results were found in resting Chinook salmon (*O. tshawytscha*) warmed up to T_{crit} (Clark et al., 2008a). In fact, Pa_{O_2} actually increased in resting sockeye salmon warm to T_{crit} (Steinhausen et al., 2008).

Interpreting Ca_{O_2} data during warming is more complex because of potential pH and temperature effects on the Hb–oxygen affinity curve, and because warming has variable effects on blood [Hb] (Taylor et al., 1997; Farrell, 1997; Sandblom and Axelsson, 2007). Even so, Ca_{O_2} was maintained in resting sockeye salmon warmed to T_{crit} as well as in exercising sockeye salmon warmed above T_{opt} (Steinhausen et al., 2008). By contrast, Ca_{O_2} decreased at T_{crit} in resting rainbow trout (*O. mykiss*) (Heath and Hughes, 1973) and in resting Chinook salmon (Clark et al., 2008a). The modest decrease in Ca_{O_2}, in the absence of an effect on Pa_{O_2}, in resting Chinook salmon probably reflects a decrease in Hb–oxygen affinity rather than a limitation on oxygen diffusion at the gills.

A limitation in the circulatory system?

If a circulatory limitation exists for exercising salmonids during warming, increases in cardiac output should cease once T_{opt} is reached. Indeed, maximum cardiac output in exercising sockeye salmon (Brett, 1971; Steinhausen et al., 2008) and rainbow trout (Taylor et al., 1996) reached a maximum value at a temperature well below T_{crit}, as did $\dot{V}_{O_2}$. Thus, ultimately as warming approaches T_{opt} the potential to increase maximum cardiac output (as revealed by exercising fish) fails to keep up with the required increase in cardiac output in a resting fish (Fig. 4). As a result, because scope for cardiac output does not increase above T_{opt} (Fig. 5), swimming effort either declines or stops.

For resting salmonids, the cardiac limitation at T_{crit} is even more obvious. Cardiac arrhythmias and bradycardia often develop at T_{crit} (Heath and Hughes, 1973; Clark et al., 2008a), although their physiological basis has not been studied. Thus, experimental evidence points unequivocally towards a cardiac limitation both at T_{opt} in exercising salmonids and at T_{crit} in resting salmonids. Further insight into the mechanistic basis of the cardiac response to warming and its limitations comes from an analysis of heart rate (the rate function) and cardiac stroke volume (the capacity function).

The importance of increased heart rate during acute warming is extremely clear. Warming increases cardiac output solely by increasing heart rate. This is true for both resting and exercising salmonids (Sandblom and Axelsson, 2007; Clark et al., 2008a; Steinhausen et al., 2008), presumably through a direct temperature effect on the cardiac pacemaker rate (Randall, 1970). However, because fish have a maximum heart rate (Farrell, 1991) and heart rate is already elevated by the exercise, the maximum heart rate must be reached at a temperature well below that for resting fish (Steinhausen et al., 2008). In fact, the scope for heart rate plummets from its maximum at T_{opt} to zero near T_{crit} (Fig. 5). Fred Fry made a similar observation for heart rate in *Salvelinus fontinalis* alevins (Fig. 6A) (Fry, 1947) and commented that this might reflect the T_{opt} for the activity of an organ (i.e. the heart)! We now know that Fry's assertion was correct because the T_{opt} for the maximum performance of isolated rainbow trout hearts is well below T_{crit} (Fig. 6B).

In contrast to heart rate, cardiac stroke volume appears to be thermally insensitive to warming. This is true for resting and exercising salmonids (Sandblom and Axelsson, 2007; Clark et al., 2008a; Steinhausen et al., 2008), but it is an especially surprising result for resting fish. In fact, it seems paradoxical, given that cardiac stroke volume can triple during swimming at constant temperature (Stevens et al., 1967; Brett, 1971; Kiceniuk and Jones, 1977; Farrell and Jones, 1992; Thorarensen et al., 1996; Gallaugher

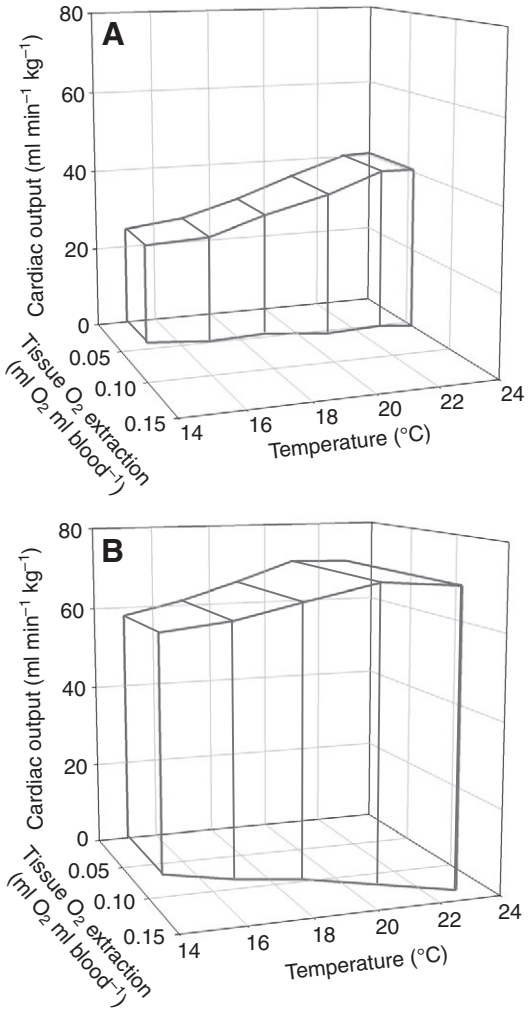

Fig. 4. Cardiac output and tissue oxygen extraction (Ca_{O_2}–Cv_{O_2}) for 12°C-acclimated sockeye salmon either (A) at rest, or (B) swimming continuously at about 70% of maximum swimming speed, while the temperature was acutely increased at 2°C h^{-1} and held at the temperature for 1 h while cardiorespiratory measurements were made. All resting fish completed the temperature challenge and recovered, but above 19°C swimming fish began to stop swimming and so progressively fewer are represented at higher temperatures. The *x*–*y* surface at each temperature represents oxygen uptake (i.e. the product of cardiac output and tissue oxygen extraction), which clearly increases with temperature in resting but not swimming fish above their optimum temperature of around 15°C. Changes in cardiac output with temperature are a result of increased heart rate (see text) (Steinhausen et al., 2008).

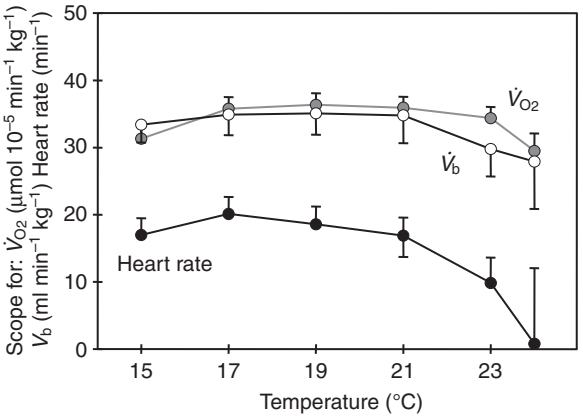

Fig. 5. Changes in scope for oxygen uptake ($\dot{V}_{O_2}$), cardiac output ($\dot{V}_b$) and heart rate (f_H) in swimming sockeye salmon during acute warming. Note that although all fish continued swimming in temperatures up to and including 19°C, some fish stopped swimming at higher temperatures and so the data are only for those that continued to swim (Steinhausen et al., 2008).

et al., 2001), that this additional capacity for increasing cardiac stroke volume is not exploited by resting fish when they are warmed to T_{crit} (Fig. 4). So why is this?

The difficulty may revolve around the fact that cardiac end-systolic volume is essentially zero in salmonids (Franklin and Davie, 1992). This means that, unless venous return and end-diastolic volume are increased first, an increase in cardiac contractility cannot increase cardiac stroke volume appreciably (Sandblom and Axelsson, 2007). Furthermore, there are indications that during warming inadequate venous return may limit cardiac stroke volume in the first instance. In resting rainbow trout warmed from 10 to 13°C, cardiac stroke volume was maintained when heart rate increased because venous blood pressure and mean circulatory filling pressure also increased (Sandblom and Axelsson, 2007). However, with further warming to 16°C, which is near T_{opt}, venous blood pressure was unchanged and cardiac stroke volume decreased when heart rate increased further. Although a complete systolic emptying of the ventricle may be a disadvantage with regard to the capacity to increase cardiac stroke volume during warming, it may be more important in ensuring a completely 'fresh' supply of oxygen enters the lumen of the heart with each heart beat given oxygen diffusion to the myocardium is driven by a low $P_{v_{O_2}}$ (see Farrell, 2002).

The increase in cardiac stroke volume when salmonids swim at a constant temperature is supported by an increase in venous blood pressure (Kiceniuk and Jones, 1977) and by contraction of locomotory muscles aiding venous return (Farrell et al., 1988). There are several potential reasons why warming does not increase cardiac stroke volume any further. There could be physical upper limits to venous return and end-diastolic volume. Also, increasing heart rate during warming reduces cardiac filling time and creates a negative frequency effect on cardiac contraction, both of which could constrain cardiac stroke volume (Farrell, 2007). In addition, at a time when the heart is working maximally, its extracellular environment (the venous blood) becomes acidemic and hyperkalemic, and has a low $P_{v_{O_2}}$ (Steinhausen et al., 2008). Although the negative inotropic effects of these extracellular changes were prevented by adrenergic stimulation of the heart (Driedzic and Gesser, 1994; Nielsen and Gesser, 2001; Hanson et al., 2006), this adrenergic protection was greatly reduced at 18°C compared with 10°C in rainbow trout (Hanson and Farrell, 2007).

A limitation at the tissues?

The rate and degree of oxygen diffusion from capillaries to tissues is influenced by several factors besides the P_{O_2} gradient. These include tissue capillary density, the intracellular mitochondrial location, regional blood flow and red blood cell capillary contact time. Taylor et al. (Taylor et al., 1997) suggested that regional oxygen delivery by convective transport in exercising rainbow trout is determined mainly by changes in cardiac output as temperature changes, i.e. active peripheral redistribution of blood flow is modest. Even so, red muscle blood flow during aerobic swimming

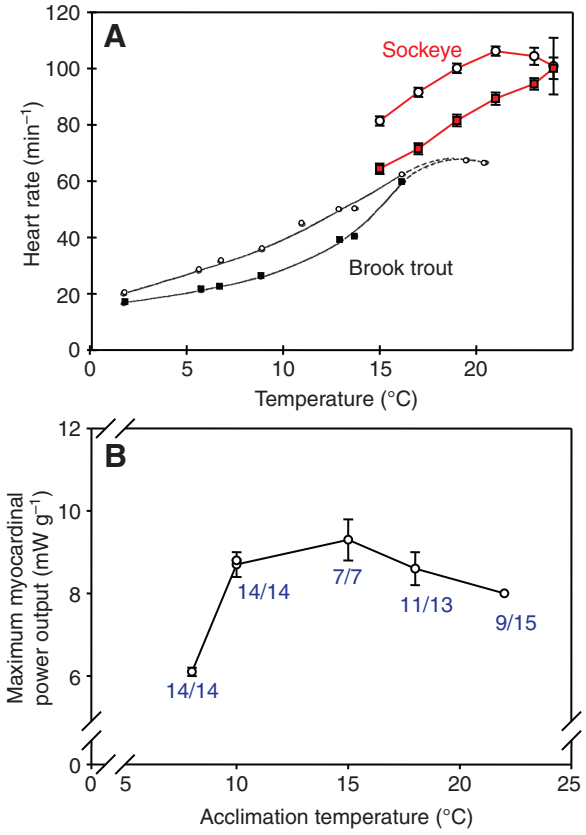

Fig. 6. (A) A comparison of heart rates measured in brook trout alevins (Fry, 1947) and adult sockeye salmon (Steinhausen et al., 2008) to illustrate the convergence of heart rate in resting (lower lines) and active (upper lines) fish such that there is no scope for heart rate at T_{crit}. (B) A composite of the maximum cardiac performance for isolated perfused rainbow trout hearts acclimated to different temperature to illustrate that there is a peak performance around 15°C for the heart. Beyond this temperature, an increasing number of preparations would fail as indicated by the ratio of successful/attempted preparations besides each data point (Farrell et al., 1988; Keen and Farrell, 1994; Farrell et al., 1996).

was lower at 18°C than at 11°C (Taylor et al., 1997). In addition, the basal oxygen requirement of white (fast glycolytic) muscle in fish increases during warming because it accounts for >50% of body mass and receives 28–50% of routine cardiac output in resting rainbow trout (Randall and Daxboeck, 1982; Bushnell et al., 1992). Indeed, the finding that blood flow to white muscle increased from 40% to 75% of cardiac output at 6°C *versus* 18°C in resting rainbow trout (Barron et al., 1987) clearly reflects a significant elevation of white muscle oxygen demand relative to whole animal $\dot{V}_{O_2}$. White muscle also has a low capillary density (Egginton, 2000), which increases the likelihood of a diffusion limitation developing for oxygen diffusion.

Further insight into potential limitations on tissue oxygen removal during warming is evident from measurements of Cv_{O_2} and Pv_{O_2}. For example, Pv_{O_2} and Cv_{O_2} could not decrease if there was a diffusion limitation. In fact, a decrease in Cv_{O_2} is a very important mechanism for increasing tissue oxygen extraction during swimming at constant temperature (Fig. 4). However, for resting sockeye salmon, warming actually increased Pv_{O_2} and Cv_{O_2}, and tissue oxygen extraction (Fig. 4) remained unchanged (Steinhausen et al., 2008). Similarly, Pv_{O_2} was temperature insensitive in resting Chinook salmon, except at 25°C when there was acidemia and Cv_{O_2} decreased (Clark et al., 2008a). When exercising sockeye salmon were warmed, Pv_{O_2} again remained temperature insensitive, albeit it at a lower level compared with resting fish (Steinhausen et al., 2008). This consistent temperature insensitivity of Pv_{O_2} points to a diffusion limitation for oxygen unloading (see Farrell, 2002; Farrell and Clutterham, 2003). Why in resting fish warming does not decrease Pv_{O_2} to the level seen with swimming at a constant temperature is unclear.

In resting salmonids, the decrease in Cv_{O_2} just prior to T_{crit} may reflect a desperate situation created by inadequate tissue perfusion. The ability of fish to recover from warming may be informative in this regard. For example, when sockeye salmon and Chinook were incrementally warmed at 2–4°C h^{-1} and kept at a constant temperature for 1 h between temperature steps, the fish recovered well at the control temperature and within 1–2 h, especially if the heat stress was terminated before cardiac arrhythmias developed (Steinhausen et al., 2008; Clark et al., 2008a). In these experiments, sockeye salmon maintained Cv_{O_2} and Chinook salmon decreased Cv_{O_2} only in association with acidemia at 24°C. By contrast, when 'opportunistic' blood samples were taken from resting rainbow trout during continuous warming (1.5°C h^{-1}), all but one fish died and venous blood became depleted of oxygen (Heath and Hughes, 1973).

What emerges from the above is that the heart becomes a weak link for the cardiorespiratory oxygen cascade when exercising salmonids are warmed above T_{opt}. Although a direct temperature effect on the cardiac pacemaker rate appears to be the predominant mechanism for improving tissue oxygen transport, a crucial limitation is reached when this rate function reaches its maximum. This apparently occurs at T_{opt} for exercising fish and at T_{crit} for resting fish. What follows during warming is a sequela of events: a decrease in scope for heart rate preceding that for cardiac output, which precedes that for aerobic scope (Fig. 5). It is also evident that during warming the contributions of several capacity functions ([Hb], tissue oxygen extraction and cardiac stroke volume) are only small and variable. Why this excess capacity is not exploited when resting fish are warmed is particularly perplexing and warrants further study.

Beyond salmon

The details provided above for salmonids apparently apply more broadly to other fishes. For example, warming of three species showed that like rainbow trout: (1) cardiac output increases predominantly through increased heart rate, (2) routine heart rate shows a plateau or collapse before T_{crit} that is species specific, and (3) cardiac stroke volume is temperature insensitive (Fig. 7) (Sandblom and Axelsson, 2007 and references therein). In addition, the temperature dependence of Hb–oxygen affinity and the variable effects of warming on [Hb] are well known among fishes (Cech et al., 1976; Gallaugher and Farrell, 1998; Gollock et al., 2006), and a direct temperature effect on the spontaneous pacemaker rate is recognised for plaice (*Pleuronectes platessa*) (Harper et al., 1995). Furthermore, in resting Atlantic cod (*Gadus morhua*), although heart rate and cardiac output both collapsed before CT_{max}, heart rate reached a plateau before cardiac output and $\dot{V}_{O_2}$ (at 18°C *versus* at 20°C) (Gollock et al., 2006).

The effects of acute warming have been thoroughly studied in winter flounder (*Pseudopleuronectes americanus*) seasonally acclimated between 5°C and 18°C (Cech et al., 1975; Cech et al., 1976). After a 5°C warming at each acclimation temperature, an increase in $\dot{V}_{O_2}$ (67–83% per 5°C increment) was always accompanied by a nearly equivalent increase heart rate (54–77%

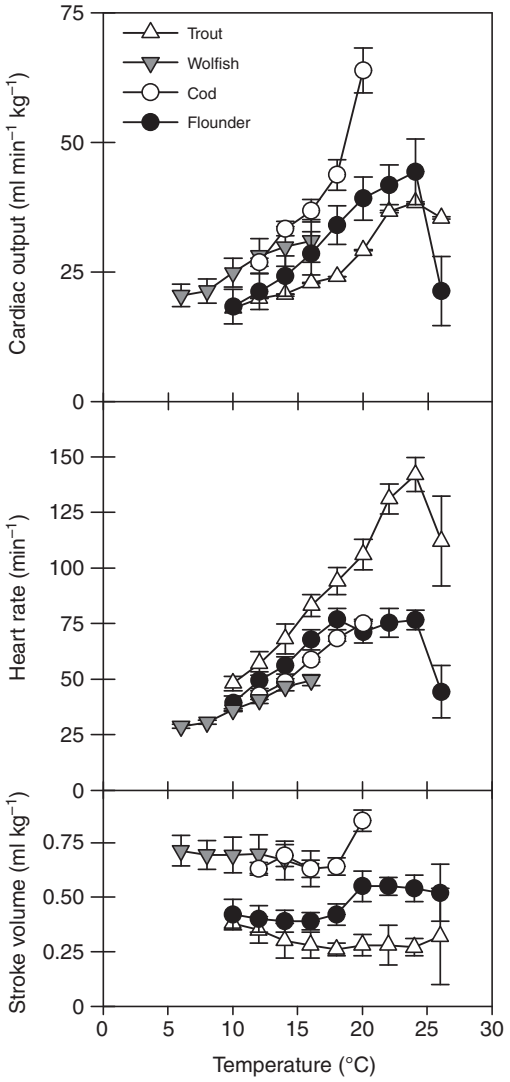

Fig. 7. Changes in cardiorespiratory variables in resting fishes during acute warming: a comparison of wolffish, winter flounder and Atlantic cod with rainbow trout. (Data kindly supplied by Dr Kurt Gamperl: wolffish – N. Joaquim and A. K. Gamperl, unpublished; trout – A. K. Gamperl, unpublished; Atlantic cod – L. H. Petersen and A. K. Gamperl, unpublished; flounder – P. C. Mendonca and A. K. Gamperl, unpublished.)

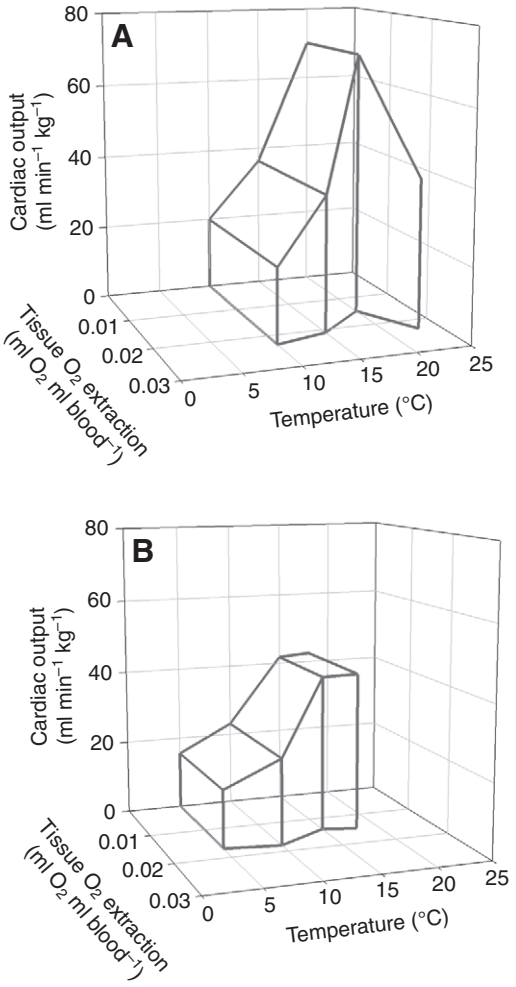

Fig. 8. Cardiac output ($\dot{V}_b$) and tissue oxygen extraction (Ca_{O_2}–Cv_{O_2}) for winter flounder either (A) seasonally acclimated to a temperature, or (B) acutely warmed by 5°C increments from the acclimation temperature. The x-y surface at each temperature represents oxygen uptake (the product of $\dot{V}_b$ and Ca_{O_2}), which clearly increases with temperature and either reaches a plateau between acclimation temperatures of 15 and 18°C, or collapses with an acute increase to 23°C. The greatest contributor to increases in $\dot{V}_{O_2}$ is almost always $\dot{V}_b$, which is a result of increased heart rate (see text) (Cech et al., 1975; Cech et al., 1976).

per 5°C increment). However, with warming from 18°C to a near-lethal temperature, cardiac output and cardiac stroke volume collapsed even though heart rate increased (Fig. 8). Ca_{O_2}, Pa_{O_2}, Cv_{O_2} and Pv_{O_2} were all maintained, except for 5°C- and 18°C-acclimated fish when tissue oxygen extraction increased (Fig. 8).

Heart rate may be a limiting factor during warming in decapod crustaceans as well. Heart rate is reported to reach a plateau near T_{crit} in various crab species: the spider crab [*Maja squinado* (Frederich and Pörtner, 2000)], the rock crab [*Cancer irroratus* (Frederich et al., 2009)] and the kelp crab [*Taliepus dentatus* (Storch et al., 2009)]. Cardiac stroke volume was also temperature insensitive in the kelp crab. Therefore, the upper limit for heart rate may emerge as a valuable, yet simple predictor of T_{opt} in active animals and T_{crit} in resting animals. If this is the case, biotelemetry of heart rate could easily extend this work to field situations (Clark et al., 2008b; Clark et al., 2009), allowing the full interplay of environmental factors on aerobic scope to be properly explored. Accompanying such fieldwork is the need to better understand the control of heart rate at high temperature and to determine if the heart is operating at its maximum pacemaker rate.

Temperature and the river migration of sockeye salmon

Beyond direct temperature reactions (i.e. acute effects occurring in minutes to hours considered above), two other time scales can be applied to temperature effects. Thermal adaptation spans generations and occurs at the population level through natural selection acting on individual variability. The study of heritable factors related to thermal tolerance is in its infancy. Thermal acclimation (or thermal compensation), however, occurs when an individual undertakes physiological and biochemical adjustments over days to weeks [or perhaps months for Antarctic fishes at near freezing temperatures (Franklin et al., 2007)]. Here, a new

phenotype emerges from an existing genome as an animal acclimates to a new thermal environment. Given the potential for thermal acclimation and adaptation, the obvious question becomes: Do the acute responses to temperature in fishes have any ecological or evolutionary relevance? In the specific case of adult sockeye salmon that return to the Fraser River, BC, Canada to spawn, the answer is categorically yes. During this return migration, sockeye salmon can experience large and rapid temperature changes when they make daily vertical ocean movements prior to river entry and exploit deeper, cool water in lakes (Fig. 9).

Adult sockeye salmon return migrations also provide a fascinating insight into something that is normally difficult to witness, an ecological significance for T_{opt} and T_{crit}. The linkage between aerobic scope and lifetime fecundity is obvious for sockeye salmon because their entire lifetime fitness hinges on a single, precise spawning date that is preceded by an energetic upstream migration lasting up to several weeks. Therefore, to spawn, they are committed to an upriver migration that periodically may require their full aerobic scope, with only a sensory imprint for navigation, while developing gonads, without feeding and without prior experience of the temperature conditions en route (Hinch et al., 2006). Consequently, if a warm river temperature reduces aerobic scope, sockeye salmon do not have an option of postponing reproduction as other fishes might do. In fact, with just 4–6 weeks to live after entering the river, even a slower migration could reduce lifetime fecundity.

Using Weaver Creek sockeye salmon as an example and considering only aerobic swimming, upstream migration should be favoured at 14.3°C (their T_{opt} for aerobic scope) but impossible at 20.4°C (their T_{crit}) (Lee et al., 2003). As predicted, when adult Weaver Creek sockeye salmon were intercepted in 2004, implanted with biotelemetry devices and released back to the river to follow their subsequent progress, migration success was inversely related to river temperature above T_{opt}. In fact, migration success was only 0–11% when river temperature was near T_{crit} (at 18–21°C), but increased to 77% when the river seasonally cooled to 14°C and near their T_{opt} (Farrell et al., 2008). This result suggests that a warm river temperature limited aerobic scope, and impaired upriver migration and lifetime fecundity. These warm river temperatures experienced by Weaver Creek sockeye salmon in 2004, which turned out to be record highs, contributed to a catastrophic 70% loss of the migrating population!

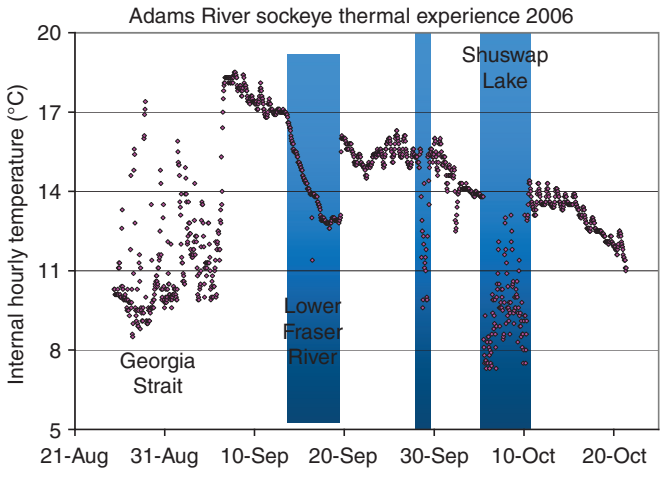

Fig. 9. Hourly temperature recordings from an I-button temperature logger that was recovered from an Adams River sockeye salmon after implantation in the peritoneal cavity in the Georgia Strait (ocean conditions) and a 40-day migration through the Fraser River watershed to its spawning area near the Shuswap Lake, BC, Canada. The highlighted areas represent periods where the fish behaviourally sought out water that was cooler than either the mainstem river or at the surface of lakes. The general downward trend over time represents seasonal cooling of the watershed, and daily oscillations in temperature can be resolved in the shallow spawning streams towards the end of the trace. (Data kindly supplied by David Patterson.)

Thermal acclimation

Warm acclimation alters thermal tolerance (Fry et al., 1942), increasing T_{opt}, T_{crit} and maximum aerobic scope (Fry and Hart, 1948). Warm acclimation, in addition to permitting a higher maximum heart rate, also decreases routine heart rate at the level of the pacemaker. This acclimatory change then provides compensation for the limitation that maximum heart rate imposes on aerobic scope by restoring the scope for heart rate either fully (Harper et al., 1995) or partially (Farrell, 1997). However, the benefits of temperature acclimation for specialists like salmon are small compared with temperature generalist. For example, CT_{max} for salmon increases by only 2°C over a 15°C acclimation temperature range *versus* an increase in CT_{max} of 10°C for goldfish over a 30°C acclimation range (Brett, 1956). In fact, routine and maximum heart rate in 22°C-acclimated sockeye salmon [86 beats min^{-1} and 106 beats min^{-1}, respectively (Brett, 1971)] are barely different for a 14°C-acclimated fish acutely warmed to 22°C [90 beats min^{-1} and 106 beats min^{-1} (Steinhausen et al., 2008)]. Other documented responses to warm acclimation, such as the decrease in cardiac mass (Gamperl and Farrell, 2004) and decrease in capillary density the red (slow aerobic) muscle of rainbow trout (Taylor et al., 1996; Egginton, 2000), even seem counterproductive. Conversely, compensatory decreases in gill epithelial thickness, as seen for other species (Taylor et al., 1997), would be beneficial.

Antecedents and concluding remarks

Like a salmon down on the Fraser, swimmin' with their battered fins,
Searchin' for their childhood home,
A patch of gravel they knew as their own.
Excerpt from 'The Ballad of Old Tom Jones' *by Barney Bentall*

The genomic information passed down by antecedents determines an individual's potential for survival, growth and reproduction. The antecedents of present day Fraser River salmon have passed on their environmental experiences through natural selection for over ~10,000 years since their post-glacial invasion. However, we have only ~60 years of reliable archival records of the river temperatures experienced during recent salmon migrations (Farrell et al., 2008). Nevertheless, remarkably the historic mean and median river migration temperature for Weaver Creek sockeye salmon is 14.5°C (their T_{opt} is 14.3°C). This observation, combined with the fact that the thermal window between T_{opt} and T_{crit} is only 7.3°C and that thermal acclimation provides little benefit to CT_{max}, suggests that their T_{opt} is potentially a product of natural selection. If this is the case, one has to question whether or not natural selection among sockeye salmon can accommodate the rapid warming trend already evident for the Fraser River (peak summer temperature has increased 1.8°C in the past 60 years).

If the salmonid genome is too inflexible to adapt to a new T_{opt}, perhaps the genetic determinants of the spawning date are more flexible. Dangerously high temperatures could then be avoided by

migrating when the river is seasonally cooler (see Keefer et al., 2008), but this may result in a fish encountering other unfavourable conditions such as faster river flows earlier in the year and an inevitable run-on-effect on the timing of larval emergence. Alternatively, warm water could be avoided behaviourally if opportunities exist. Behavioural temperature preferences are certainly shown by adult salmon during migration, which include seeking water cooler than their T_{opt} (Fig. 9) to lower $\dot{V}_{O_2}$ and perhaps slow energy depletion, suggest they likely know which temperature conditions are best for them. However, opportunities to seek cool refuges are very limited in the Fraser River (Donaldson et al., 2009). Without such behavioural responses, the warmer than normal river temperatures may force Pacific salmon near the southern limit of their geographic distribution to follow the fate of other species, a heart-breaking (Wang and Overgaard, 2006) northward shift in their distribution. The response of tropical coral reef fish species to climate change could be equally dramatic.

In closing, the best, albeit limited data set for a single animal group appears to provide a mechanistic understanding for the Fry curve. Heart rate, which is the main driver for the increase in $\dot{V}_{O_2}$ during warming, reaches its maximum rate at T_{opt} and becomes a weak link for the cardiorespiratory oxygen cascade. Shelford (Shelford, 1931) recognized that 'Animals are better short-period indicators (of environmental change) than plants' because animals can potentially move away from unfavourable environments. However, this behavioural response requires an aerobic scope, which is both controlled and limited by temperature. Future study on aerobic scope will continue to inform us of an animal's fundamental thermal niche. By contrast, a continued focus on temperature tolerances for resting animals will only inform us of thermal niche for existence and perhaps create needless worry about the precise techniques for such measurements (Chown et al., 2009).

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Glossary

Aerobic scope	the difference between maximum and minimum (standard or basal) oxygen uptake under a given set of test conditions
Ca_{O_2}	concentration of oxygen in arterial blood
Cv_{O_2}	concentration of oxygen in venous blood
CT_{max}	the critical thermal maximum that a fish can tolerate
CT_{min}	the critical thermal minimum that a fish can tolerate
Fry curve	the relationship between aerobic scope and temperature
Hb	haemoglobin
Pa_{O_2}	partial pressure of oxygen in arterial blood
P_{O_2}	partial pressure of oxygen
Pv_{O_2}	partial pressure of oxygen in venous blood
T_{crit}	the temperature at which a fish has no aerobic scope
T_{opt}	the temperature at which a fish has maximum aerobic scope
$\dot{V}_{O_2}$	rate of oxygen uptake

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