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Review

Physiological basis of temperature-dependent biogeography: trade-offs in muscle design and performance in polar ectotherms

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Summary

Polar, especially Antarctic, oceans host ectothermic fish and invertebrates characterized by low-to-moderate levels of motor activity; maximum performance is reduced compared with that in warmer habitats. The present review attempts to identify the trade-offs involved in adaptation to cold in the light of progress in the physiology of thermal tolerance. Recent evidence suggests that oxygen limitations and a decrease in aerobic scope are the first indications of tolerance limits at both low and high temperature extremes. The cold-induced reduction in aerobic capacity is compensated for at the cellular level by elevated mitochondrial densities, accompanied by molecular and membrane adjustments for the maintenance of muscle function. Particularly in the muscle of pelagic Antarctic fish, among notothenioids, the mitochondrial volume densities are among the highest known for vertebrates and are associated with cold compensation of aerobic metabolic pathways, a reduction in anaerobic scope, rapid recovery from exhaustive exercise and enhanced lipid stores as well as a preference for lipid catabolism characterized by high energy efficiency at high levels of ambient oxygen supply. Significant anaerobic capacity is still found at the very low end of the activity spectrum, e.g. among benthic eelpout (Zoarcideae).

In contrast to the cold-adapted eurytherms of the Arctic, polar (especially Antarctic) stenotherms minimize standard metabolic rate and, as a precondition, the aerobic capacity per milligram of mitochondrial protein, thereby minimizing oxygen demand. Cost reductions are supported by the downregulation of the cost and flexibility of acid–base regulation. At maintained factorial scopes, the reduction in standard metabolic rate will cause net aerobic scope to be lower than in temperate species. Loss of contractile myofilaments and, thereby, force results from space constraints due to excessive mitochondrial proliferation. On a continuum between low and moderately high levels of muscular activity, polar fish have developed characteristics of aerobic metabolism equivalent to those of high-performance swimmers in warmer waters. However, they only reach low performance levels despite taking aerobic design to an extreme.

Key words: polar, ectotherm, thermal tolerance, muscle, performance, oxygen availability, temperature.

Thermal tolerance and muscular performance

The special features of muscular activity in the cold and their biochemical and physiological background in fish have attracted considerable attention. Much of this work has focused on the fish group Notothenioideae, which dominates the fish fauna in Antarctic seas. Other fish groups and invertebrates have rarely been considered (for reviews, see van Dijk et al., 1998; Peck, 2002). Some work on whole-animal respiration and on metabolic rate during rest or, less frequently, exercise has also been carried out in Arctic fish (e.g. Bushnell et al., 1994; Steffensen et al., 1994; Hop and Graham, 1995; Zimmermann and Hubold, 1998). This review analyses the special characteristics of exercise metabolism in the cold from a more general point of view by addressing the question of how tissue and whole-organism function are influenced by mean temperature and by regular temperature fluctuations and, accordingly, what the selective forces are that cause temperature-dependent trade-offs in tissue and cell design. All this relates to the question of how animals maintain function at different temperatures (e.g. Guderley, 1990) and how they are able to adjust their thermal tolerance windows (Pörtner et al., 2000; Pörtner, 2001, 2002a). Interpreting the mechanisms of temperature adaptation in the light of how they contribute to adjusting and limiting both cold and heat tolerance is important in the light of global warming (e.g. Wood and MacDonald, 1997; Pörtner et al., 2001) and associated shifts in the geographical distribution or

physiological performance of ectothermic animals (Pörtner et al., 2001).

The present analysis was undertaken to demonstrate that the currently emerging unifying picture of an oxygen limitation of thermal tolerance in animals (Pörtner, 2001, 2002a) helps to develop our knowledge of muscle function at various temperatures towards a whole-animal understanding. The comparisons of polar (especially Antarctic) stenotherms with eurytherms (temperate and Arctic) and even endotherms is appropriate for testing some of the hypotheses developed. Because of the many gaps in our understanding of the trade-offs involved in adaptation to various climates and temperature regimes in a latitudinal cline (see Pörtner et al., 2000; Pörtner, 2001, 2002a), this review must necessarily remain conceptual, and the hypothetical relationships proposed should contribute to ideas for future experimental work.

Recent work carried out in marine invertebrates and fish has demonstrated that, in accordance with Shelford's law of tolerance, the onset of a decrease in whole-animal aerobic scope characterises thermal limitation at low and high pejus thresholds (T_p, pejus=getting worse) (see Frederich and Pörtner, 2000; Pörtner, 2001). Towards temperature extremes, the decrease in aerobic scope is indicated by falling oxygen levels in the body fluids and by the progressive limitation of the functional capacity of circulatory and ventilatory systems to ensure oxygen supply. According to a previous model (Pörtner et al., 2000; Pörtner, 2001), the aerobic capacity of muscle mitochondria may become limiting for ventilation and circulation at low temperatures, whereas at high temperatures, excessive oxygen demand causes an uncompensated decrease in oxygen levels in the body fluids. Further cooling or warming beyond pejus limits leads to low or high critical threshold temperatures (T_c) at which aerobic scope disappears and the transition to an anaerobic mode of mitochondrial metabolism and a progressive decline in cellular energy levels occur (Pörtner et al., 2000; Pörtner, 2001) (Fig. 1).

At extreme temperatures, oxygen limitation will contribute to oxidative stress and, eventually, to the denaturation of molecular functions. Time-limited passive survival is supported by anaerobic metabolism or by the protection of molecular functions by heat-shock proteins and antioxidative defences. In accordance with a hierarchy of thermal tolerance ranging from the systemic to the cellular and molecular levels, capacity limitations at a high level of organisational complexity, i.e. the integrated function of the oxygen delivery system, define the onset of thermal limitation, which then transfers to lower hierarchical levels and contributes to cellular and molecular disturbances (for reviews, see Pörtner, 2001, 2002a).

Thermal limits differ among ectothermic species depending on latitude or seasonal temperature acclimatisation and are therefore related to geographical distribution. The tolerance window is narrow, especially in polar areas, most notably in the Southern Ocean. Nevertheless, despite constant water temperatures between –1.9 and +1 °C, this window is not the same for all Antarctic species (for a review, see Pörtner et al., 2000). The capacities for ventilation and circulation are higher in mobile fish or octopods than, for example, in sessile bivalves, and this probably relates to the higher pejus (see above) and critical temperatures found in more mobile compared with sessile epifauna species.

The finding of an oxygen-limited thermal tolerance is in line with the concept of symmorphosis (Taylor and Weibel, 1981), which implies that excess capacity of any component of the oxygen delivery system is avoided in an evolutionary context. In the context of thermal adaptation and limitation, this means that oxygen delivery systems (and, possibly, other systems) are set to a minimum level of functional capacity – in the case of oxygen delivery, just sufficient to meet maximum oxygen demand between the average highs and lows of environmental temperatures. Accordingly, the processes and limits of thermal tolerance are linked to the aerobic scope and aerobic capacity of the whole animal in

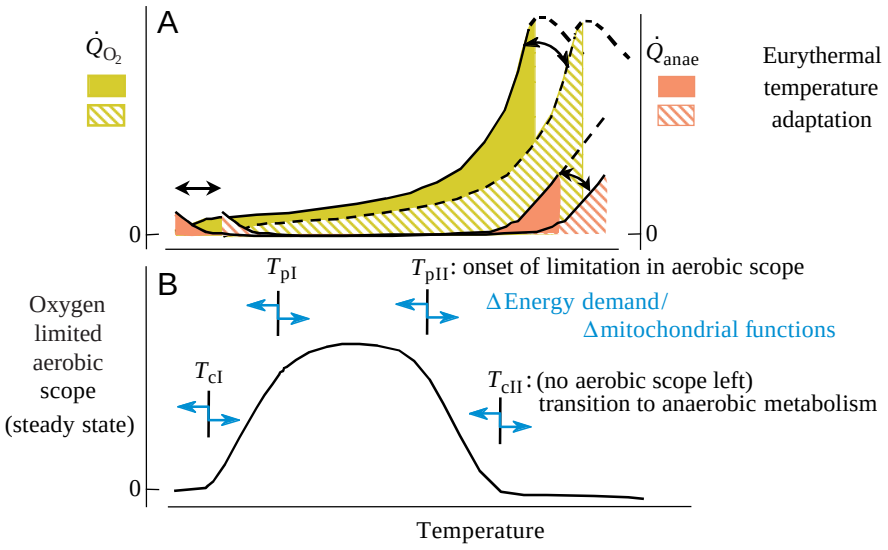

Fig. 1. Simplified model of thermal tolerance and eurythermal temperature adaptation in animals (modified after Pörtner, 2001a) considering (A) contributions to metabolic heat production ($\dot{Q}$) by oxygen consumption ($\dot{Q}_{O_2}$) and anaerobic metabolism ($\dot{Q}_{anae}$). The model is based predominantly on data for water-breathers. Mechanisms shifting the respective tolerance thresholds include a change in overall mitochondrial functional capacity (B), which causes a shift in both lower (I) and upper (II) pejus temperature (T_p, the onset of a decrease in aerobic scope) and critical temperature (T_c, the onset of anaerobic metabolism). Reductions in oxygen demand and anaerobic heat production are expected to result from a decrease in mitochondrial densities and capacities during warming (indicated by arrows in A).

addition to parallel adjustments at the molecular or membrane levels. The crucial mechanism(s) of thermal limitation and adaptation should link low and high tolerance limits, i.e. changes occurring during acclimation to high temperatures should reverse the processes involved in acclimation to low temperature. Since thermal tolerance limits are probably set at the highest levels of organismic complexity (Pörtner, 2001, 2002a), the crucial mechanisms of temperature adaptation should be visible in all tissues supporting the functional coordination of the organismic entity.

Following the treatment of thermal tolerance, adaptation to changing temperatures involves escaping the threat of temperature-induced hypoxia (Pörtner et al., 1998, 2000; Pörtner, 2001). Accordingly, thermal adaptation will also affect performance levels, which depend on the maintenance of aerobic and, also, anaerobic scope. Performance increases with temperature to a maximum and then decreases at higher temperatures, yielding a species-specific bell-shaped curve that shifts depending on thermal adaptation (Beamish, 1978). The temperature of optimum performance is expected to correspond to the preferred temperature of the fish, maximum performance occurring when acclimation and exposure temperature are identical (Beamish, 1978; Schurmann and Steffensen, 1997). However, this can only be true within the thermal tolerance window when full aerobic scope becomes available to the locomotory muscles (see above). Accordingly, temperature-dependent performance limitations set in at an organismic level, i.e. the capacity of circulatory and ventilatory muscle tissues to support the take-up and provision of substrates and oxygen to the tissues.

Taking this integrative view of thermal tolerance, low temperatures in particular appear to be a major constraint limiting the scope of cellular functional capacity, including muscular activity levels, with ultimate consequences for lifestyle and population dynamics. As the narrowest tolerance windows are found at low standard metabolic rates (SMRs) in Antarctic stenotherms and correlate with a reduced aerobic scope for exercise, the question arises of whether the elevated SMRs observed in cold-adapted populations of eurythermic animals (Pörtner et al., 2000) will extend to cold-compensated metabolic scopes and activity levels. In this context, the treatment of cold adjustments needs to consider not only the physiology of stenotherms and eurytherms but also the various short-term to evolutionary time scales involved; i.e. between seasonal cold acclimatisation *via* cold adaptation in eurytherms at high latitudes (especially in the Northern hemisphere) (see Pörtner, 2001) to permanent cold adaptation in polar areas. Compared with Antarctic seas, the much younger thermal history of Arctic fauna and the lesser degree of isolation of the Arctic from adjacent seas require consideration. Accordingly, species or species subpopulations (as in the case of the Atlantic cod *Gadus morhua*) in the Arctic may still be found in transition to life in the permanent cold, while those in the Antarctic have developed features of permanent cold adaptation over millions of years.

Temperature adaptation, aerobic scope and whole-animal performance

In a simplified view, the aerobic scope for exercise of the whole animal is reflected in the level of mitochondrial density and/or capacity in the musculature. On the one hand, this capacity can be made available only if blood and cellular P_{O_2} are kept high, which requires sufficient ventilation and circulation. On the other hand, the limited capacity of mitochondria to produce energy in the cold is likely to contribute to the loss of function and scope, e.g. in circulatory, ventilatory and locomotory muscles (Frederich and Pörtner, 2000; Pörtner, 2001). Limitations in the availability of aerobic energy may therefore be the key to understanding why an increase in overall mitochondrial aerobic capacity occurs during adaptation to cold (Pörtner et al., 1998, 2000; Pörtner, 2001, 2002b) (Fig. 1). The interpretation that temperature-induced changes in mitochondrial densities and functional capacities cause a shift in oxygen-limited thermal tolerance windows casts new light on the primary role of these processes. In fish, such changes are observed in slow oxidative and fast glycolytic muscle and also include a proliferation of aerobic (red) muscle fibres (Johnston and Maitland, 1980; Sidell, 1980; Tyler and Sidell, 1984; Egginton and Sidell, 1989; Sidell and Moerland, 1989; Guderley and Johnston, 1996; St-Pierre et al., 1998; Guderley, 1998; Pörtner et al., 1998, 2000). In temperate eurytherms, such changes are reversed during seasonal warming.

Although the rises in mitochondrial density and capacity in the cold are probably cost-determined (Pörtner et al., 2000; Pörtner, 2001), the mechanistic stimulus for cold-induced mitochondrial proliferation remains unclear because energy deficiency and hypoxia occur at both ends of the thermal tolerance window and mitochondrial density decreases during warm acclimation, thereby reducing the excessive oxygen demand associated with excess mitochondrial capacity (see below). The key role of mitochondria does not neglect that integrated modifications in lipid saturation, kinetic properties of metabolic enzymes, contractile proteins and transmembrane transporters are required to contribute to the optimization of higher functions within the window of thermal tolerance (see Johnston, 1990; Hazel, 1995; Storelli et al., 1998; Pörtner et al., 1998). These functions include the integration of muscular and nervous systems operative in ventilation and circulation which maintain contractility and contractile frequency such that aerobic scope is retained to support metabolic flexibility and locomotor activity in the cold.

In addition to the cost of mitochondrial biosynthesis and degradation, proton leakage accounts for the cost of mitochondrial maintenance (Pörtner et al., 1998). Mitochondrial proton leakage rates in the resting cell make up a consistently large fraction of SMR in ecto- and endotherms, 25 % of baseline metabolic rate in rat hepatocytes, 50 % in skeletal muscle and 20–30 % in the whole animal (Brand, 1990; Brand et al., 1994; Rolfe and Brand, 1996; Brookes et al., 1998). Higher mitochondrial densities as a consequence of cold adaptation should therefore indicate elevated oxygen demand

under resting conditions or cold-compensated SMR and aerobic scope. This may not, however, be observed in polar stenotherms (see below). Nonetheless, while enhancing mitochondrial capacity in the cold eliminates the capacity limitations of ventilation and circulation, a reduction in mitochondrial capacity in the warm reduces the oxygen demand of mitochondrial maintenance, thereby allowing the upper T_c to shift to higher values (see Pörtner, 2001, 2002a,b) and, as a trade-off, leaving enough aerobic energy for ventilation and circulation to maintain aerobic scope. These observations immediately suggest that mitochondria are more efficient in the warm. They will then leave more cellular space for enhancing contractile apparatus and, thus, capacity and performance levels than is possible in the cold.

Metabolic scopes and performance in cold-acclimated versus *cold-adapted eurytherms*

The questions frequently asked in this context are to what extent the capacity of locomotory muscular activity is fully compensated in the cold and whether it reaches as high as at warmer temperatures (for a general treatment of the effects of temperature on muscular function in teleost fish, see Sidell and Moerland, 1989). In temperate-zone eurythermal fish, cold acclimation in goldfish and striped bass (Fry and Hart, 1948; Rome et al., 1984; Sisson and Sidell, 1987) does not lead to complete compensation of performance levels since warm-acclimated fish are able to maintain higher swimming speeds than cold-acclimated animals at their respective acclimation temperature. The same conclusion is true for Atlantic cod (*Gadus morhua*) from the North Sea: fish acclimated to 15 °C reached higher swimming speeds than at 5 °C (Schurmann and Steffensen, 1997). One reason may be reduced force generation per muscle cross-sectional area (see Rome, 1995) at cold temperature, which requires the recruitment of a larger number of fibres for a similar power output to that in warm-acclimated fish (for comparison, 1.5–2 times more fibres would be recruited in carp *Cyprinus carpio* or scup *Stenotomus chrysops* to compensate for a 10 °C fall in temperature) (see Rome, 1995). Recruitment of a larger number of muscle fibres for the same performance level will contribute to the enhanced expression of slow oxidative fibres in the cold (see Sidell and Moerland, 1989). Nonetheless, using more fibres for the same power output indicates that maximum limits are reached earlier than in warm waters causing, on average, lower performance levels in cold- compared with warm-acclimated fish (see above) (see Johnston and Altringham, 1985; Johnston, 1989; Sidell and Moerland, 1989; Johnson and Johnston, 1991; Johnson et al., 1996; Johnston and Ball, 1997; Guderley, 1998). For compensatory molecular changes involved, see Gauvry et al. (2000).

In the muscle of cold-acclimated eurytherms (Guderley and Johnston, 1996; Guderley, 1998) or in cold-adapted populations of eurytherms analysed during the summer at high latitudes (Tschischka et al., 2000; Sommer and Pörtner, 2002), both a rise in mitochondrial density and a significant, approximately twofold, rise in mitochondrial capacity (per milligram of mitochondrial protein) have been observed. According to the scenario developed above, these processes increase the costs of mitochondrial maintenance and cause a rise in SMR. Such metabolic cold compensation at the whole-animal level is actually seen in temperate ectotherms during winter cold (with activity maintained, no dormancy involved) or in eurythermal, especially Northern hemisphere, populations along a latitudinal cline (for further climate-related and evolutionary background, see Pörtner, 2001, 2002a).

In a variety of physiologically distinct populations of Atlantic cod (*Gadus morhua*) between the North Sea and the Barents Sea, the northernmost subpopulations studied (northeastern Arctic cod from the Barents Sea or cod from the White Sea) display higher, i.e. cold-compensated, SMRs (if acclimated and analysed at identical temperatures) than their temperate conspecifics (Pörtner et al., 2001; T. Fischer, R. Knust and H. O. Pörtner, unpublished results). The increment between populations appears to be larger than the temperature-specific metabolic increment observed, for example, in cold-acclimated North Sea specimens between 12 and 4 °C. A similar pattern is found in invertebrate eurytherms such as *Arenicola marina* (Pörtner et al., 2000; Sommer and Pörtner, 2002). However, very few data indicate whether cold compensation also extends to the levels of maximum metabolic rate and factorial and net metabolic scope. Because of their very similar modes of life, swimming behaviour and morphology, cod populations and species from various latitudes provide a unique basis for such analyses.

At acclimation temperatures between 15 and 4 °C, cod maintain factorial scopes of between 2 and 5 (Bushnell et al., 1994; Claireaux et al., 1995; Schurmann and Steffensen, 1997). Scope increased from 2.6 to 3.5–4.1 with some decrease in acclimated SMR from 15 to 10 or 5 °C in the North Sea cod population (Schurmann and Steffensen, 1997) (Fig. 2). Net metabolic scope during exercise fell only slightly (Schurmann and Steffensen, 1997). In conclusion, net metabolic scope appears to be partially compensated in exercising cold-acclimated North Sea cod on the basis of cold-compensated SMR (see above) between 15 and 5 °C.

As a consequence of enhanced red fibre densities, striped bass (*Morone saxatilis*) acclimated to 9 °C reached higher critical swimming velocities during acute exposure to 15 °C (2.5 compared with 1.9 $BL\,s^{-1}$ at 9 °C, where *BL* is body length) than fish acclimated to 25 °C and brought to 15 °C (1.8 compared with 2.8 $BL\,s^{-1}$ at 25 °C) (Sisson and Sidell, 1987). The data of Claireaux et al. (1995) indicate, however, that cod acutely exposed to higher temperatures reach a metabolic ceiling during exercise that is not very different from the maximum rate seen at the lower, acclimated temperature. According to the modelled depiction in Fig. 2B, this implies a drop in maximum sustainable activity (critical velocity) at acutely elevated temperature in cod. However, this does not appear to be a general pattern (Sisson and Sidell, 1987; Taylor et al., 1997). In cod of the same population, longer-term acclimation to the warmer temperature may be required for improved performance to U_{crit} levels beyond those reached in

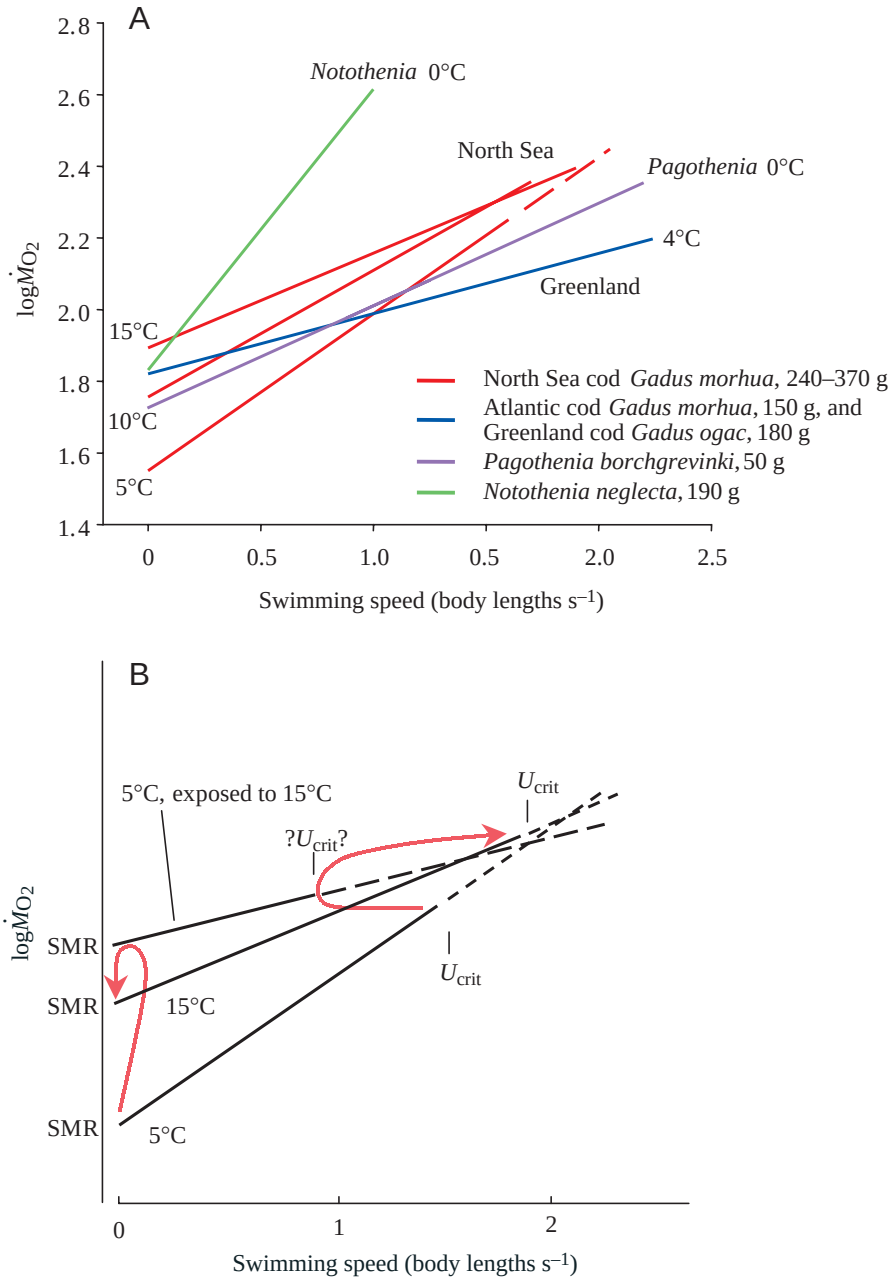

Fig. 2. Patterns of changes in rates of oxygen consumption ($\dot{M}_{O_2}$, mg h^{-1} kg^{-1}, red lines) with increasing exercise levels in Atlantic cod (*Gadus morhua*) and Greenland cod (*Gadus ogac*) from various latitudes based on data for North Sea cod (Schurmann and Steffensen, 1997) and for the Greenland populations of both species (Bushnell et al., 1994). Solid lines depict the actual performance range up to the critical swimming velocity (U_{crit}). Dashed lines depict extrapolated costs at higher swimming speeds (not reached) for a comparison between acclimation temperatures. For comparison, results at 0 °C for Antarctic stenotherms (*Notothenia neglecta* and *Pagothenia borchgrevinki*) are included (Johnston et al., 1991; Forster et al., 1987). (A) In accordance with a compensatory increase in mitochondrial densities and overall aerobic metabolic capacities (see text), the intersection of extrapolated lines suggests that cold-acclimation at 10 or 5 °C compared with 15 °C causes a larger metabolic increment for the same exercise level in cod (as well as a rise in temperature-specific standard metabolic rate, SMR; not shown, see text). Metabolic cold adaptation observed in northern cod populations (at 4 °C) also elevates SMR. However, the metabolic increment with rising swimming speed reflects enhanced energy efficiency and a somewhat higher U_{crit}. (B) A model of the transitions between acute warming and long-term warm acclimation (from 5 to 15 °C) based on data for cod by Claireaux et al. (1995) and Schurmann and Steffensen (1997). The reduction in baseline oxygen demand associated with a warm-induced decrease in mitochondrial densities should contribute to the increase in U_{crit} observed during long-term warm acclimation. For further explanation, see the text.

cold-acclimated specimens (see Beamish, 1978; Schurmann and Steffensen, 1997) (Fig. 2B).

For comparison, cold-adapted Atlantic cod collected from a population close to Greenland as well as Greenland cod (*Gadus ogac*), both acclimated and studied at 4 °C, displayed temperature-specific SMRs even higher than North Sea cod acclimated to and analysed at 5 °C. With a value of 2.1, factorial scope in the polar cod was below that of North Sea cod acclimated to low temperature (Fig. 2) (Bushnell et al., 1994). Nonetheless, the northern cod reached somewhat higher critical swimming velocities with a lower metabolic increment and similar or slightly lower maximum metabolic rates than their southern conspecifics or confamilials (in the case of *G. ogac*) at 5 °C.

First, these findings would indicate metabolic cold compensation of SMR to a higher degree in northern cod than in North Sea cod, a finding in line with recent comparisons of SMR in Barents Sea (northeastern Arctic) cod and Norwegian coastal or North Sea cod acclimated to the same temperature (T. Fischer, R. Knust and H. O. Pörtner, unpublished results; see above). Second, maximum metabolic rates may also be cold-compensated in northern cod. However, measured values remain close to levels seen in cold-acclimated North Sea cod despite higher SMRs (Fig. 2A). The slope of the metabolic increment seen during exercise is reduced in northern cod, reflecting an increased energy efficiency that may be a result of the long-term cold adaptation process in these populations. The increased slopes observed for cold-acclimated North Sea

cod in Fig. 2B suggest that, possibly as a consequence of cold-compensated SMR, North Sea cod at 5 °C would reach the same extrapolated activity levels (point of intersection) at higher net costs of swimming than those acclimated to 15 °C. A larger metabolic increment with increasing swimming speed in cold-acclimated cod from the same population is also visible in the data of Webber et al. (1998). Over the range of temperatures studied, changing water properties would not explain such a significant increase in the cost of swimming (e.g. Rome et al., 1990). The elimination of this cost increment in Arctic cod instead indicates that, at low to medium activity levels, complete cold compensation of performance may be possible during long-term cold adaptation, as has also been concluded for Antarctic fish (see below).

As a result of elevated SMRs, factorial scopes appear to be reduced in cold-adapted northern cod populations to levels lower than in cold-acclimated temperate cod and even more so than in Antarctic fish (see below). Similarly, among benthic zoarcids, a comparison of a cold-acclimated eurythermal temperate (*Zoarces viviparus*) and an Antarctic species *(Pachycara brachycephalum*) revealed a somewhat lower factorial scope (2.9 *versus* 6.6, estimated from the oxygen demand of the recovery processes) in the North Sea than in the Antarctic eelpout (Hardewig et al., 1998). These comparisons clearly show that factorial scope is influenced by metabolic cold-acclimation *versus* adaptation.

As a corollary, the few data available for northern populations indicate that cold-adapted eurytherms, at the expense of elevated SMR, keep performance levels similarly high in the cold as their southern conspecifics at warmer temperatures, possibly even higher the more eurythermal they are. This contrasts with the situation in Antarctic stenotherms (see below). Many more species need to be investigated to determine whether this pattern reflects a unifying principle of long-term eurythermal cold adaptation to Arctic and sub-Arctic environments. Also, very little is known about the specific cellular biochemistry of cold adaptation (compared with acclimation) in eurytherms in a latitudinal cline in contrast to the large body of knowledge existing for polar stenotherms, especially from the Antarctic. From a wider perspective, the earlier use of maximum aerobic capacity at lower performance levels in the cold would explain why high-performance species observed in temperate to tropical areas among salmonids or scombrids do not exist in polar areas. As adequately stated by Clarke (1998), there are no polar tuna.

Metabolic scopes and performance in polar stenotherms

Antarctic fish display continuous exercise at moderate levels of 1.4–2.6 $BL\,s^{-1}$, which have been interpreted as adequately cold-compensated compared with fish of similar lifestyle and body size in temperate and tropical waters (van Dijk et al., 1998; Peck, 2002; cf. Wardle, 1980). With respect to performance levels in Antarctic fish, some uncertainty arises during interpretation of the data because of the predominance of one fish family in the Antarctic, the Notothenioidea, which makes it difficult to distinguish true patterns of cold adaptation from the special features of this group. In this case, low performance levels might arise from a benthic ancestor of all notothenioids, characterised by labriform locomotion. Such an origin would also explain the absence of a swim bladder; pelagic descendants use reduced ossification and extensive lipid deposits as a secondary means of adjusting buoyancy (Kock, 1992; Eastman, 1993). However, Peck (2002) reviewed the invertebrate literature and concluded that the levels of activity (walking and digging in limpets, anemones and bivalves) in pelagic and benthic invertebrate taxa are reduced compared with those in temperate taxa with little evidence for temperature compensation. As in fish, performance at the high end appears to be cold-restricted in invertebrates, although data on the invertebrates with the highest performance rates, muscular squid, are not yet available (see Pörtner, 2002b, and below).

Factorial metabolic scopes during exercise of between 3.9 and 5.7 have been measured in *Pagothenia borchgrevinki* and *Notothenia coriiceps*, values similar to those of temperate or tropical species with a similar lifestyle (Forster et al., 1987; Johnston et al., 1991; for spontaneous scopes, see Zimmermann and Hubold, 1998). Higher factorial scopes in Antarctic stenotherms than in cold eurythermal species (see above) clearly appear to be a consequence of low SMRs, which are 4–9 times lower in polar fish than in other species at 20 °C (Clarke and Johnston, 1999). Nonetheless, the metabolic rates reached during exercise are well within the range seen in cod from temperate to Arctic latitudes (Fig. 2A). Accordingly, overall comparisons suggest that significant cold compensation of standard metabolic rate, in contrast to findings in cold eurythermal fish and invertebrates (see above), does not occur in fish and invertebrates from the permanent cold of polar areas and the deep sea (e.g. Clarke and Johnston, 1999; Peck and Conway, 2000) and may, thus, be small. It must be emphasized that these global statements are based on among-species comparisons and fail to pick up more subtle differences among closely related species or among species subpopulations in a latitudinal cline.

Nonetheless, the global trend observed matches a model presented previously by Pörtner et al. (2000) (cf. Pörtner, 2002b) that predicts that metabolic cold compensation should be greater in winter-acclimated (with activity maintained) or cold-adapted eurytherms than in stenotherms (Fig. 3). Antarctic species in particular were able to minimize the metabolic costs of cold adaptation, but at the expense of being obligatory stenotherms. Significant metabolic cold adaptation, although not to the full extent originally postulated by Wohlschlag (1964), may still be found if more closely related species are compared. For example, cold-adapted Antarctic eelpout (*Pachycara brachycephalum*) displayed the same metabolic rate as cold-acclimated North Sea or Baltic eelpout (*Zoarces viviparus*), both values being greater than that of warm-acclimated *Zoarces viviparus* at low temperatures (van Dijk et al., 1999).

Despite a secondary reduction in SMR during the transition from eurythermal to stenothermal cold (Pörtner et al., 1998),

Antarctic species display features of metabolic cold adaptation in their musculature: high mitochondrial densities compared with temperate species at warm acclimation temperatures are still found in cold stenothermal ectotherms, e.g. in slow fibres of notothenioid fish muscle (Johnston, 1987; Dunn et al., 1989). In white muscle of the Antarctic notothenioid *Gobionotothen gibberifrons*, cytochrome *c* oxidase (COX) activities were five times higher than in a temperate-zone fish with a similar lifestyle (Crockett and Sidell, 1990), whereas citrate synthase activities were increased by only 1.4- to 2.8-fold in *Trematomus newnesi* and *G. gibberifrons* above the levels found in temperate species. Mitochondrial proliferation involves enhanced expression of aerobic enzymes such as cytochrome oxidase, measured by the accumulated message (RNA) for this process. However, more message was found in eurythermal fish (eelpout *Zoarces viviparus*) after cold-acclimation than in cold-adapted stenothermal Antarctic eelpout *Pachycara brachycephalum* (Hardewig et al., 1999), a finding in line with the suggested secondary downregulation of cold compensation.

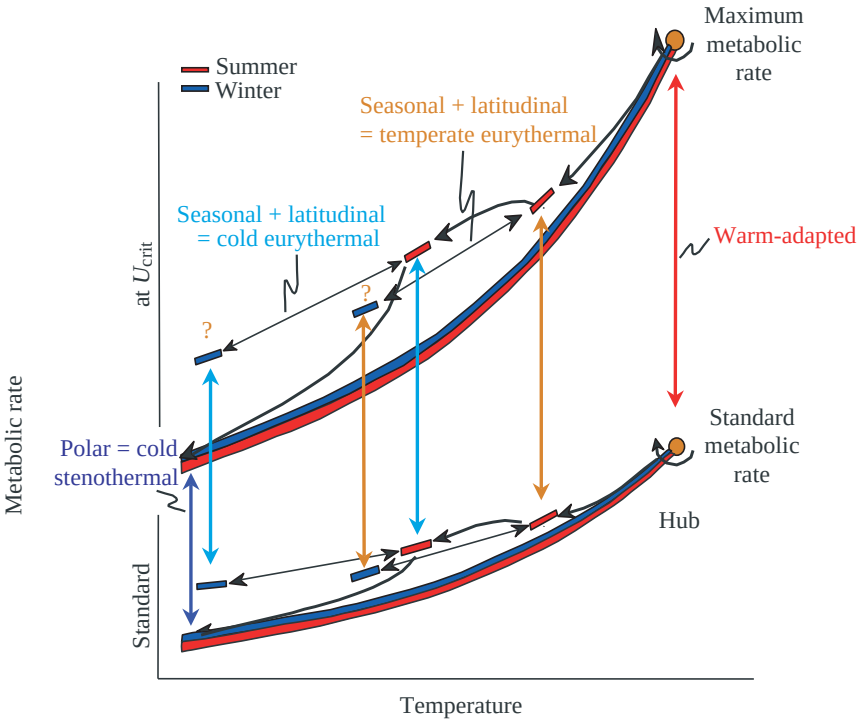

Fig. 3. Temperature-compensated aerobic scopes depending on standard metabolic rates (SMRs) in warm-adapted fish or other ectotherms compared with cold-compensated SMRs in eurytherms (Northern hemisphere) and reduced SMRs in Antarctic (and possibly Arctic) polar stenotherms. As a consequence of metabolic adjustments to cold, active metabolic rates at maximum aerobic activity (given as the metabolic rate at the critical swimming speed, U_{crit}) may be cold-compensated in eurytherms, whereas lower rates may result for those Antarctic stenotherms with low SMRs (see, however, Fig. 2A and the text for a balanced view of these patterns). The low SMR in polar stenotherms despite high mitochondrial densities suggests that capacities are downregulated to levels expected from the Q_{10} relationship, possibly because of high Arrhenius activation energies (see text). For each of the four groups (warm-adapted *versus* temperate eurythermal *versus* cold eurythermal *versus* polar), straight vertical arrows depict the relationship between standard and maximum aerobic metabolic rate. The warm-water situation is interpreted to be the original situation (on evolutionary time scales) and, accordingly, to represent 'the hub of metabolic cold adaptation' according to Pörtner et al. (2000).

In contrast to eurythermal cold-acclimation or adaptation (see above), the aerobic capacity per milligram of mitochondrial protein in Antarctic fish and invertebrates is not evidently cold-compensated (Johnston et al., 1998; Pörtner et al., 2000), and the surface area of mitochondrial cristae in Antarctic red-blooded fish (36–37 $m^2\,m^{-3}$), a measure of membrane folding, is not significantly different from those of temperate and tropical perciform fish with similar lifestyles (Archer and Johnston, 1991; Johnston et al., 1998), but is significantly lower than the highest values reported, which are for tuna red muscle (63–70 $m^2\,cm^{-3}$) and hummingbird flight muscle (58 $m^2\,cm^{-3}$) (Moyes et al., 1992; Suarez et al., 1991). This surface area is traditionally interpreted to correlate with aerobic capacity, but recent evidence suggests that this correlation is less tight in icefish (O'Brien and Sidell, 2000). For comparison, a theoretical limit of 83 $m^2\,cm^{-3}$ was suggested by Srere (1985), with limited space left for Krebs cycle enzymes. The respective values for invertebrates are not known.

In Antarctic fish, skeletal muscle mitochondrial volume density is at least 29–33 %, whereas in Mediterranean fish with similar activity the value is 8–13 % (Johnston et al., 1998). The highest mitochondrial densities found in the cold were 56 % for the pelagic notothenioid *Pleuragramma antarcticum* and above 50 % for icefish (Johnston, 1987; Dunn et al., 1989; Johnston et al., 1998). Species lacking haemoglobin tend to have higher densities than red-blooded species in association with a lower density of lipid droplets (Johnston, 1987; Dunn et al., 1989) (see below). In warm waters, even very active fish do not have values above 40 %, with some influence of body size; 46 % was reported for small anchovies and 29 % for tuna (Johnston, 1982; Moyes et al., 1992). The value for hummingbird flight muscle is 35 % (Suarez et al., 1991).

As a corollary, despite extreme cold-induced mitochondrial proliferation in pelagic Antarctic fish, full performance compensation is only possible for low to moderate activity levels. Mitochondrial volumes in the red muscles of Antarctic fish (Johnston et al., 1988, 1998; Archer and Johnston, 1991) are elevated even beyond those seen in temperate or warm-blooded species. In contrast to cold eurytherms, SMR is only cold-compensated to a non-significant degree in Antarctic

ectotherms, such that factorial scope is higher than, for example, in northern cod. It appears that, in a trade-off between the space adopted by myofilaments, oxidative metabolism and SMR, the requirement for more mitochondrial volume for the same functional capacity in the cold is a major constraint on the maximum scope for activity, linked to lower levels of muscular force per muscle cross-sectional area. This affects all the musculature, including the circulatory muscles, such that the space constraints within cardiomyocytes and the limits on the size of the heart in relation to body mass in icefish (Tota et al., 1991; Axelsson et al., 1998; O'Brien and Sidell, 2000) will also reflect the limitation of aerobic scope for the whole organism to within the borders of the narrow thermal tolerance window.

According to Pörtner et al. (2000), the reduction in SMR during stenothermal, in contrast to eurythermal, cold may be a secondary situation linked to the reduced mitochondrial capacities and increased Arrhenius activation energies (E_a) of mitochondrial oxygen demand, particularly proton leakage, and of flux-regulating enzymes in metabolism such as isocitrate dehydrogenase. A high kinetic barrier may support a low metabolic flux in cold stenotherms despite mitochondrial proliferation. High E_a values in Antarctic species reflect a high temperature-dependence of metabolism and, in consequence, reduced heat tolerance, i.e. cold stenothermy of the whole organism. In contrast, the E_a value of overall metabolism appears to be reduced in active cold-acclimated eurytherms, with the consequence that SMR is elevated (see Pörtner, 2001, 2002a).

The finding of excess aerobic design for low-activity lifestyles would also explain why anaerobic capacity is, on average, reduced in polar fish. In general, oxidative enzymes and creatine kinase, adenylate kinase and AMP deaminase show relatively high degrees of cold compensation in notothenioids, while glycolytic enzymes do not (Dunn and Johnston, 1986; Johnston, 1987; Crockett and Sidell, 1990), with some variability in enzyme levels depending on mode of life (Dunn et al., 1989). Cold-compensated anaerobic capacity was, however, found in some, but not all, temperate freshwater fish (see van Dijk et al., 1998). In contrast to findings of low lactate levels in fatigued notothenioids, a recent comparison of cold-acclimated temperate (North Sea) with Antarctic zoarcids (*Zoarces viviparus versus Pachycara brachycephalum*) led to the conclusion that a similar anaerobic capacity is expressed in both species in the cold (Hardewig et al., 1998). These benthic sluggish fish formed similar amounts of lactate (11.5 μmol g^{-1} muscle tissue) as flounder acclimated to 11 °C (Milligan and Wood, 1987). On the basis of these data, a low glycolytic capacity does not appear to be a general phenomenon in Antarctic fish. The conclusion arises that cold compensation of anaerobic pathways is, in principle, possible, but this possibility is not expressed in the notothenioid fish family (Egginton and Davison, 1998) which, in contrast to strictly benthic zoarcids, tend to have a more active mode of life and, therefore, tend to express a more aerobic mode of metabolism.

Low SMRs and similar factorial scopes as in moderately active temperate species should cause the absolute metabolic increment to be lower in Antarctic fish compared with temperate or eurythermal species. Peck (1998, 2002) emphasized that the four- to ninefold lower absolute metabolic scopes in Antarctic fish and invertebrates compared with temperate species at 20 °C also contribute to extended periods of post-prandial metabolic increments (specific dynamic action, SDA) when polar and temperate species have the same meal size. Again, factorial increments of SDA above SMR are similar in polar, temperate and tropical environments (Johnston and Battram, 1993; Peck, 1998), corroborating the observation that, despite similar factorial scopes, absolute metabolic scopes are reduced in the permanent cold because of low SMRs. The general validity of this statement still needs to be established because SMRs and metabolic scopes in the few Antarctic fish species analysed during exercise in swim tunnels are well within, and even beyond, the range found for moderately active, temperate and cold-adapted Arctic eurytherms such as cod (Fig. 2A). Nonetheless, the patterns seen within the closely related gadids suggest different modes of cold-acclimation and adaptation in eurythermal compared with stenothermal fish species (see above).

Similarities of cold- and high-performance adaptations

The factorial scopes of Antarctic fish, although higher than in cold-adapted Arctic gadids, appear somewhat low compared with those seen in active temperate and tropical fish, with values between 6 and 12 in salmonids, bass or mackerel (Bennett, 1978; O'Dor and Webber, 1991; Brill, 1996; Korsmeyer et al., 1996; Pörtner and Zielinski, 1998). Among polar fish, only an Arctic cryopelagic species, *Boreogadus saida*, reaches the latter range, with a factorial scope of up to 8.4 (Zimmermann and Hubold, 1998). However, taking a high factorial scope as an indicator of high performance may be misleading. First, the factorial scope does not closely reflect performance capacities unless the effect of cold-acclimation or cold-adaptation on both SMR and absolute metabolic scope are taken into account (see above). Second, trends similar to those during seasonal cold-acclimation and longer-term (especially eurythermal) cold-adaptation, i.e. elevated SMR combined with low factorial aerobic scopes, are observed in high-performance scombrids (tuna) and in squid. As a benefit of a high SMR, recovery from excessive anaerobic exercise in tuna (Brill, 1996) and in squid (Pörtner et al., 1993) is rapid.

In similar ways, cold-adaptation seems to favour rapid recovery. Starting from similar levels of anaerobic disturbance, enhanced recovery rates were observed in cold-adapted Antarctic compared with cold-acclimated eurythermal eelpout (Hardewig et al., 1998). Moreover, starting from elevated SMRs, a lower increment in metabolic rate, i.e. enhanced aerobic efficiency at high swimming speed, characterizes cold-adapted Northern compared with cold-acclimated temperate cod populations (see above). This resembles the effects of long-term exercise training. Less circulatory work results,

thereby contributing to cost reductions during exercise. Despite elevated factorial scopes and low SMRs compared with eurythermal fish, features of high-performance metabolism are still present in Antarctic fish (Fig. 4), as in both seasonal and permanent cold, more mitochondrial volume or mitochondrial functional components are needed for the same level of functional performance than in the warm.

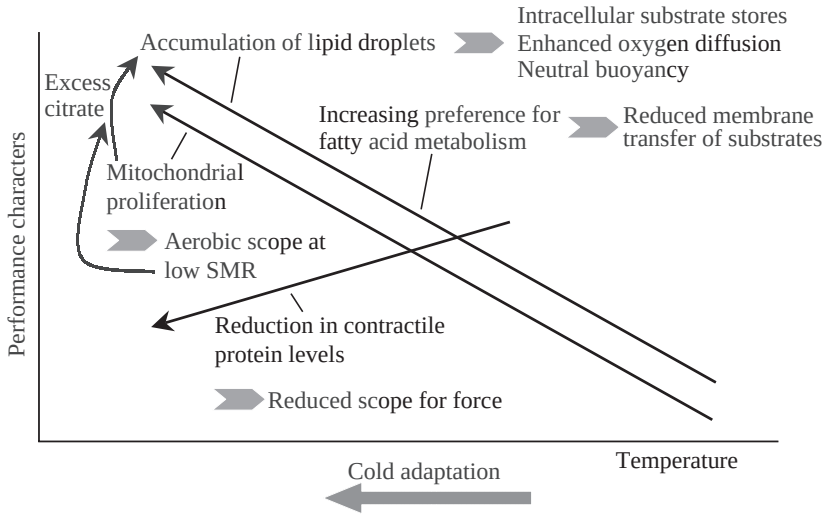

Fig. 4. High performance characteristics are required for moderate activity levels in Antarctic fish muscle. The trend from carbohydrate to lipid metabolism and the associated accumulation of intracellular lipid, favoured by high mitochondrial densities and, most likely, excess citrate levels at low standard metabolic rates (SMRs), contribute to energy savings in the cold by reducing energy-dependent membrane transport and neutral buoyancy. Low SMRs correlate with extreme stenothermy in the cold (see text).

The role of lipids and lipid metabolism

Another important aspect of cold-acclimation and cold-adaptation is that the development of an enhanced mitochondrial density goes hand in hand with a shift from carbohydrate to lipid catabolism, the preferred use of lipids by mitochondria and, as a precondition, enhanced whole-body and intracellular storage of lipids (Fig. 4). The trend to accumulate lipids seen in rainbow trout slow muscle fibres during seasonal cold (Egginton et al., 2000) goes hand in hand with a trend to increase the capacity of β-oxidation, especially in red muscle (Guderley and Gawlicka, 1992). Such a shift from the glycogenolytic pathway, which is structurally associated with muscular fibrils, to the β-oxidation pathway located in the mitochondrial matrix appears to be a logical consequence of increased mitochondrial and decreased myofibrillar cell volume fractions in the cold. Preferred use of lipids by cold mitochondria is seen in striped bass, for example, with twofold higher rates observed at cold (5 °C) compared with warm (25 °C) acclimation and measurement temperatures. As a consequence of high mitochondrial densities, Antarctic fish also display elevated capacities of mitochondrial β-oxidation, indicated by activities of 3-hydroxy-acyl-CoA dehydrogenase and carnitine palmitoyltransferase (CPT) (for a review, see Sidell and Moerland, 1989). Rate-limiting control of lipid catabolism may be exerted by transfer of acyl derivatives across the mitochondrial membrane supported by CPT rather than by β-oxidation (Driedzic and Hochachka, 1978; Weber and Hamann, 1996). In the lipid stored by notothenioids (whole animal), monoenoic fatty acids such as 18:1 predominate (Hagen et al., 2000), a finding in line with the preferred use of this fatty acid by the mitochondria of notothenioid fish (Sidell et al., 1995). Overall, the high volume density of mitochondria combined with cold-compensated and preferred lipid metabolism in cold-acclimated temperate and permanently cold-adapted polar fish (Johnston and Harrison, 1985; Sidell, 1991, 1998; Sidell et al., 1995) again resemble metabolic features seen in high-performance scombrid fish such as mackerel or tuna and also in mammals.

In notothenioids, intracellular lipid content in oxidative muscle ranges between approximately 9 % of dry mass in the demersal species *Gobionotothen nudifrons*, 37 % in the cryopelagic *Pagothenia borchgrevinki* (Sidell et al., 1995) and 45.6 % in *Pleuragramma antarcticum* (Hubold, 1985). Much of this lipid is accumulated, especially in the pelagic food chain, by feeding on lipid-rich pelagic zooplankton (see Clarke and Peck, 1991). Although the largest lipid contents have been found in pelagic fish, selective pressure towards lipid storage and aerobic lipid metabolism in the cold appears to prevail even at low activity levels. This is emphasized by a comparison of benthic North Sea and Antarctic zoarcids fed on the same shrimp (*Crangon crangon*). North Sea eelpout (*Zoarces viviparus*) accumulated only 10 % lipids per body dry mass, whereas Antarctic eelpout (*Pachycara brachycephalum*) accumulated 33 % (Brodte, 2001), suggesting that this process is triggered by cold temperatures. These findings also corroborate the conclusion that, in the Antarctic ecosystem, lipid accumulation in fish cannot be explained simply by the uptake of lipid-rich zooplankton. Cold-adapted metabolism in predators and their prey instead follows the same rules, leading to an overall accumulation of lipid in the food chain.

Lipid storage in fish is in the form of triglycerides. In a comparison of ^{31}P nuclear magnetic resonance (NMR) spectra of the muscle tissue of the two species (Bock et al., 2001), the levels of high-energy phosphates and inorganic phosphate did not appear very different, but high levels of glycerophosphatidylethanolamine (GPE), glycerophosphatidylcholine (GPC), phosphatidylethanolamine (PE) and phosphatidylcholine (PC), in addition to an unknown phosphate compound (*X*) in the phosphodiester region, were visible in Antarctic eelpout *Pachycara brachycephalum* but not in North Sea eelpout *Zoarces viviparus* (Fig. 5). This indicates a higher free phospholipid content, possibly associated with higher membrane contents and metabolic rate used to preserve

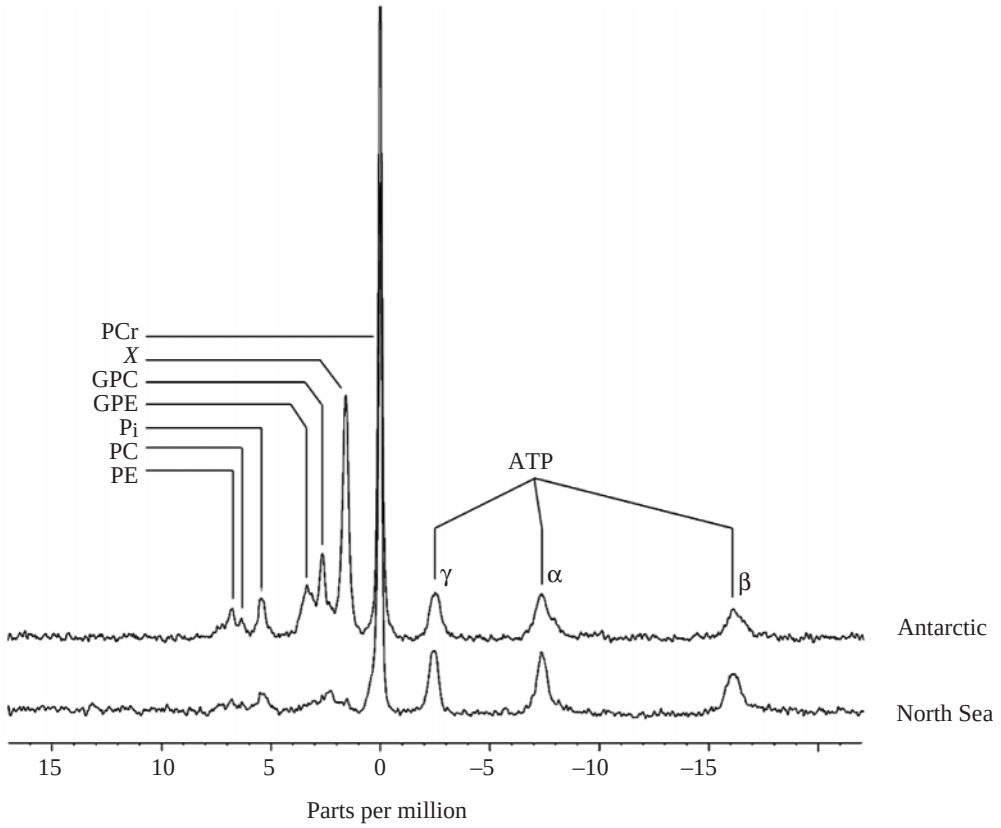

Fig. 5. The phosphorus spectra of Antarctic and temperate eelpout (*Pachycara brachycephalum*) reflect the higher concentration of phospholipids in the Antarctic species. GPC, glycerophosphatidylcholine; GPE, glycerophosphatidylethanolamine; PC, phosphatidylcholine; PCr, phosphocreatine; PE, phosphatidylethanolamine; P_i, inorganic phosphate; *X*, unassigned phosphodiester (adapted from Bock et al., 2001).

membrane fluidity (Storelli et al., 1998). The high phosphodiester content will also reflect the higher density of mitochondria and their membranes in Antarctic fish. Mitochondria predominantly possess phosphatidylcholine, phosphatidylethanolamine and, to a lesser extent, cardiolipin, with decreasing concentrations of phosphatidylcholine, a general phenomenon in cold-adapted membranes (Wodtke, 1978; van den Thillart and de Bruin, 1981; Storelli et al., 1998).

A rise in cellular/organellar lipid contents and a reduction in inter-mitochondrial distances cause a parallel rise in cellular oxygen solubility and diffusibility (Tyler and Sidell, 1984; Egginton and Sidell, 1989; Londraville and Sidell, 1990), which appears beneficial during both cold and performance adaptations by supporting oxygen supply. Otherwise, a drop in the Krogh diffusion constant (K_{O_2}) of 1.6 % °C^{-1} would occur during cooling, which comprises a drop in diffusion coefficient D_{O_2} of 3 % °C^{-1} as well as an increase in oxygen solubility of approximately 1.4 % °C^{-1}. A 42 % lower Krogh constant would result for the cytosol of an Antarctic fish (at –1.8 °C) compared with that of a temperate fish at 25 °C (Sidell, 1998). Work carried out on striped bass unequivocally demonstrates that this drop in K_{O_2} is not only compensated for during lipid accumulation but that, instead of being reduced, K_{O_2} virtually doubles between 25 and 5 °C as a result of a more than 10-fold increase in the fractional cell volume filled with lipid and the associated doubling of cellular oxygen solubility. The diffusion coefficient D_{O_2} is more-or-less maintained because of structural changes in the cell. With membranes being the preferred pathways of oxygen diffusion, Sidell (1998) convincingly argued that the enhanced mitochondrial density, together with the increasing level of lipid unsaturation, would support oxygen flux into the centre of the cell.

The question arises of whether lipid accumulation is adaptive and driven by limitations in aerobic scope (as concluded for mitochondrial proliferation; see above) or whether it is a beneficial by-product of an increased mitochondrial proliferation and the associated shift to lipid metabolism. The latter appears to be the case. The scenario of a secondary drop in SMR during the evolution of Antarctic fauna probably occurred at elevated mitochondrial densities and enhanced lipid contents in aerobic muscles, as seen in cold-acclimated eurytherms. At low metabolic rates and high oxygen solubility in body fluids and cellular lipid depots, excess rather than too little oxygen becomes available, supporting energy savings by a reduction in the P_{O_2} gradients needed for oxygen flux and, thus, in ventilatory and circulatory effort. Loss of haemoglobin and myoglobin function in icefish corroborate this view by indicating excess oxygen availability not only linked to excess oxygen availability from cold ambient water and *via* cold body and cell fluids but also *via*

the accumulation of lipids in the cold. Excess oxygen availability in the cold and diffusion/solubility limitations lower than in warm-acclimated fish are also indicated by a trend for the development of red muscle fibre hypertrophy, as seen in cold-acclimated striped bass (Egginton and Sidell, 1989). In Antarctic fish, both slow oxidative and fast glycolytic muscle fibres are larger, with reduced capillary density for a maintained intracellular P_{O_2} profile, compared with those of temperate and tropical species with a similar mode of life (Johnston, 1987, 1989; Johnston et al., 1988; Dunn et al., 1989; Egginton, 1999).

An increased mitochondrial capacity for lipid oxidation in itself does not explain why and how lipids are accumulated in tissues and organisms in the cold. It has been hypothesized that tissues with elevated mitochondrial contents maintain a higher adenylate energy charge with low levels of free ADP, AMP and inorganic phosphate, thus minimizing stimulation of glycolysis and favouring the use of non-carbohydrate substrates (Holloszy and Coyle, 1984). In Antarctic species, such trends would be supported by a low-energy-turnover lifestyle with infrequent activity bouts and rare use of anaerobic metabolism.

Again, the reasons and mechanisms for enhanced cellular lipid accumulation may be similar in high-performance skeletal and heart muscle to those in animals acclimating to cold temperatures and are emphasized at high SMRs. Lipogenesis in male gulf killifish, *Fundulus grandis*, was stimulated by cold temperature and led to lipid accumulation during cold exposure in autumn (Weld and Meier, 1985). Hepatocytes from cold-acclimated rainbow trout exhibited significantly higher rates of fatty acid and sterol synthesis (measured as tritium incorporation) (Hazel and Sellner, 1979) at assay temperatures of 15 and 20 °C than did hepatocytes from warm-acclimated trout. Enhanced mitochondrial densities, seen particularly in pelagic species, probably support the shift towards enhanced fatty acid synthesis in liver and muscle. Put simply, enhanced mean cellular levels of mitochondrial intermediates such as citrate are a logical consequence of high mitochondrial volume fractions. Excess citrate is exported from the mitochondria into the cytosol to initiate lipogenesis. This occurs particularly at resting metabolic rates, when other cellular costs are reduced (Goodridge, 1985). This scenario matches the situation in Antarctic fish and invertebrates, in which long periods of resting metabolic rate prevail. Low levels of muscular exercise at high mitochondrial densities will further increase the fraction of time available for net lipogenesis. All these factors probably contribute to the extraordinary levels of lipid seen in pelagic Antarctic ectotherms.

Last but not least, a trend to use fatty acids as substrates transported to and synthesized within muscle cells may be enforced by greatly reduced rates of energy-dependent transport across cellular membranes in polar cold mirrored by low Na^+/K^+-ATPase activity (see Pörtner et al., 1998) as well as reduced capacities for temperature-dependent acid–base regulation (Pörtner and Sartoris, 1999; van Dijk et al., 1997). Fatty acids are bound to an albumin-like protein to be transferred through the interstitial fluid. Diffusive transfer through the membrane does not appear to be energy-dependent, but driven by a concentration gradient. As in endurance-adapted species, a larger fraction of cellular energy demand in cold-adapted muscle will be obtained from intramuscular substrate stores and a smaller fraction from blood-borne substrates (Johnston and Moon, 1980a,b; Weber and Haman, 1996). Protein stores will be reduced as a result of enhanced mitochondrial densities, so cellular lipid stores, with their high energy density, would appear most suitable to replace protein as a substrate. However, lipid transport in fish is largely unexplored, especially with respect to its thermal sensitivity.

Conclusions and perspectives

With the limited data available, particularly for Arctic eurytherms, the preliminary conclusion arises that one of the benefits of long-term adaptation to the permanent cold of polar areas may be the reduction in SMR at maintained factorial, but possibly reduced absolute, metabolic scopes. In Antarctic invertebrates and fish in particular, this trend goes hand in hand with low activity levels and energy-saving lifestyles. In contrast, Arctic eurytherms, especially cold-adapted subpopulations of otherwise temperate species, as exemplified for cod, may not (yet) be as performance-limited as their more stenothermal Antarctic counterparts. With permanently elevated SMRs and low factorial scopes, maximum metabolic rates in cold-adapted Arctic eurytherms may be more-or-less

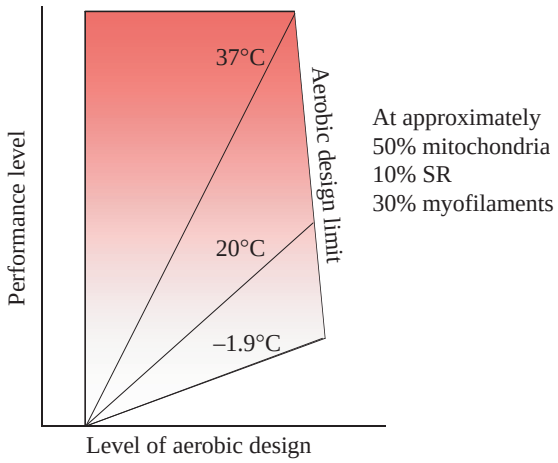

Fig. 6. Simple qualitative model of tissue design limits for aerobic metabolism and performance levels as a function of body temperature. In a trade-off between the cellular space required by mitochondria, sarcoplasmic reticulum (SR) and myofilaments, the increased mitochondrial energy production at warm temperatures allows muscular power output at high body temperatures to be maximized. Design limits for aerobic metabolism are reached at lower performance levels in Antarctic fish. The model takes account of the higher mitochondrial densities seen in Antarctic fish than in mammal and bird muscles (see text).

cold-compensated. However, energy use during exercise may be more efficient, leading to a lower metabolic increment with increased performance levels than in cold-acclimated southern populations of the same species. This appears as a benefit resulting from the process of irreversible cold-adaptation compared with cold-acclimatisation. According to this rationale, low SMRs in both Antarctic (and Arctic) stenothermal fish and invertebrates probably represent a secondary situation that arose from a more eurythermal ancestor pre-adapted to cold temperatures in a latitudinal cline. The detailed biochemical background of these whole-animal patterns needs to be elaborated.

Reduced motor activity combined with enhanced levels of aerobic machinery, particularly in pelagic Antarctic fishes, and a cold-induced shift to aerobic metabolism probably explain the reduced glycolytic capacity compared with the high end of the activity spectrum in warm-water fish such as scombrids or salmonids. In parallel with the maximization of aerobic design, lipid accumulation occurs in the cold; this is not driven by oxygen limitations but rather appears as a secondary benefit from enhanced mitochondrial densities supported by low-activity lifestyles and low metabolic rates in the cold.

The patterns observed support the overall conclusion that warm-bodied animals achieve a higher power output with lower mitochondrial density. For this reason, cold-adapted animals develop characteristics of cellular design and biochemistry typical of high-performance species in the warm. These are maximized in Antarctic pelagic fish, with extreme mitochondrial densities and huge lipid depots but a lack of cold-compensated mitochondrial capacities compared with Arctic eurytherms. The attainment of a lower muscular performance with maximized aerobic design characterizes the trade-offs and constraints involved in adaptation to the permanent cold. As a by-product of these considerations, extrapolations to the warm end of the activity spectrum immediately suggest that aerobic muscle with maximized aerobic power output is best designed close to mammalian and bird body temperatures (Fig. 6).

While previous work has focused on the degree of cold compensation of SMR, with the general conclusion that minimisation of the degree of metabolic cold-adaptation and cold stenothermy go hand in hand (Pörtner et al., 2000), future work must consider how far metabolic cold-compensation extends to maximum metabolic rates and absolute metabolic scopes. Closely related species need to be preferentially investigated to minimize the risk of otherwise misleading conclusions from global comparisons. Fig. 2 demonstrates that maximum metabolic rates in notothenioid fish match or even, in the case of *Notothenia neglecta*, exceed those in cold-acclimated Atlantic cod. This suggests cold-compensation of metabolic scope at relatively low SMRs in more active Antarctic fish; however, such a generalized conclusion is not (yet) possible. Future work must also focus on the close interactions between cellular biochemistry, whole-animal performance and developments of the natural temperature regime and its oscillations over time to provide a more comprehensive picture of eurythermal *versus* stenothermal adaptation to cold.

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