Antarctic fish can compensate for rising temperatures: thermal acclimation of cardiac performance in *Pagothenia borchgrevinki*

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Summary

Antarctic fish *Pagothenia borchgrevinki* in McMurdo Sound, Antarctica, inhabit one of the coldest and most thermally stable of all environments. Sea temperatures under the sea ice in this region remain a fairly constant –1.86°C year round. This study examined the thermal plasticity of cardiac function in *P. borchgrevinki* to determine whether specialisation to stable low temperatures has led to the loss of the ability to acclimate physiological function. Fish were acclimated to –1°C and 4°C for 4–5 weeks and cardiac output was measured at rest and after exhaustive exercise in fish acutely transferred from their acclimation temperature to –1, 2, 4, 6 and 8°C. In the –1°C acclimated fish, the factorial scope for cardiac output was greatest at –1°C and decreased with increasing temperature. Increases in cardiac output with exercise in the –1°C acclimated fish was achieved by increases in both heart rate and stroke volume. With acclimation to 4°C, resting cardiac output was thermally independent across the test temperatures; furthermore, factorial scope for cardiac output was maintained at 4, 6 and 8°C, demonstrating thermal compensation of cardiac function at the higher temperatures. This was at the expense of cardiac function at –1°C, where there was a significant decrease in factorial scope for cardiac output in the 4°C acclimated fish. Increases in cardiac output with exercise in the 4°C acclimated fish at the higher temperatures was achieved by changes in heart rate alone, with stroke volume not varying between rest and exercise. The thermal compensation of cardiac function in *P. borchgrevinki* at higher temperatures was the result of a change in pumping strategy from a mixed inotropic/chronotropic modulated heart in –1°C acclimated fish at low temperatures to a purely chronotropic modulated heart in the 4°C acclimated fish at higher temperatures. In spite of living in a highly stenothermal cold environment, *P. borchgrevinki* demonstrated the capacity to thermally acclimate cardiac function to elevated temperatures, thereby allowing the maintenance of factorial scope and the support of aerobic swimming at higher temperatures.

Key words: fish, Antarctica, cardiac output, heart, specialist, acclimation, thermal plasticity, chronotropic.

Introduction

Rising water temperatures as a result of global warming are forecast to have detrimental effects on many marine and freshwater organisms (Wood and McDonald, 1996), and particularly on thermal specialists (stenotherms) that lack the ability to physiologically compensate for a rise in water temperature. Ectothermic organisms found in the cold and stable waters of the Southern Ocean close to the Antarctic continent are often regarded as the archetypal thermal specialists. They experience body temperatures close to the freezing point of seawater (–1.9°C), and annual temperature fluctuations are less than 1°C (Clarke and Johnston, 1996; Hunt et al., 2003). The finding that small acute increases in water temperature dramatically reduced the maximum sustainable swimming performance (U_{crit}) of a cryopelagic Antarctic fish, *Pagothenia borchgrevinki* (Wilson et al., 2002) lends support for the concerns raised about the impact of global warming. The respiratory factorial scope (the difference between ventilation rates at rest and at U_{crit}) in these fish decreased with increasing temperatures, such that at 6°C there was no difference between resting and maximal ventilation rates and, hence, no scope for the increased oxygen requirements during exercise (Wilson et al., 2002).

Aerobic swimming performance of fish is closely associated with the functioning of the cardiovascular system and the ability of the heart to provide an adequate supply of oxygen and metabolites to the working muscles. During swimming, fish augment cardiac output by a combination of increases in heart rate and stroke volume, thereby increasing oxygen delivery to the tissues (Axelsson, 1988; Farrell, 1991; Shiels et al., 2002). The relative contributions of stroke volume and heart rate to increases in cardiac output vary among species. Some fish increase cardiac output predominantly by an elevation in heart rate (e.g. tuna), while others mainly employ increases in stroke

volume [e.g. rainbow trout (Farrell, 1991; Franklin and Davie, 1992)].

Cardiac function in fish is also profoundly influenced by changes in temperature (Taylor et al., 1996; Farrell et al., 1996; Farrell, 2002). An acute rise in water temperature increases resting cardiac output in fish, chiefly as a result of increased heart rate (Gollock et al., 2006). Increases in heart rate and cardiac output with acute increases in temperature may be the consequence of the direct thermal effects on the intrinsic rate of physiological processes. Additionally, heart rate may increase to compensate for the reduction in oxygen carrying capacity at higher temperatures, thereby ensuring constant oxygen delivery to the tissues (Taylor et al., 1996; Taylor et al., 1997). In fish, cardiac scope decreases with an acute increase in water temperature, resulting in a decrease in U_{crit} (Farrell, 1997). Generally, acute increases in temperature elevate both resting and maximal cardiac output. However, if the thermal dependence of resting cardiac output is greater than that of maximal cardiac output, cardiac scope will be reduced at higher temperatures (Farrell, 2002).

Phenotypic flexibility in response to thermal fluctuation [acclimatisation, acclimation (Piersma and Drent, 2003)] provides a physiological strategy that allows animals to compensate for environmental temperature change (Wilson and Franklin, 2002). Acclimation or acclimatisation allows ectotherms to maintain physiological function and performance across a wide thermal range, and it is often associated with species that experience pronounced seasonal changes in temperature (Huey and Hertz, 1984; Guderley and St-Pierre, 2002). As such, phenotypic flexibility is not predicted to be an attribute characteristic of stenothermal fish (Somero and DeVries, 1967) living in the cold and highly stable waters of the Southern Ocean, and indeed it is currently believed that there has been a trade-off between thermal tolerance and the ability to live in extreme cold waters (Pörtner et al., 2000). Hence, it was surprising that *P. borchgrevinki* was found to thermally acclimate sustained swimming performance (Seebacher et al., 2005). *P. borchgrevinki* exposed to 4°C for 4–5 weeks compensated their aerobic swimming performance such that after acclimation, U_{crit} at 6°C was not significantly different from that at –1°C in cold (–1°C) acclimated fish. The ability to maintain their swimming performance (i.e. U_{crit}) at 6°C would be expected to be accompanied by changes in their cardiovascular system that allowed for a maintenance of factorial scope for cardiac output. Therefore, the aim of this study was to assess the mechanisms allowing warm acclimated *P. borchgrevinki* to maintain factorial scope for cardiac output, and hence aerobic scope and U_{crit}, at higher temperatures.

Materials and methods
Experimental animals

Pagothenia borchgrevinki Boulenger 1902 were captured on baited lines from holes drilled through 2 m of annual sea ice in McMurdo Sound, Antarctica. Fish were transported to an aquarium facility at Scott Base on Ross Island and maintained in a flow through system. Fish were randomly divided into identical, temperature controlled tanks at –1°C and 4°C (acclimation groups) and maintained at these temperatures for 4–5 weeks before experimentation.

Surgery

Fish were anaesthetised in MS222 (1:5000) and transferred to a surgical sling. To maintain anaesthesia during surgery, the gills were irrigated with seawater containing 1:10 000 MS222. The ventral aorta was exposed *via* a small mid-ventral incision and a single-crystal Doppler flow probe placed around the vessel, just distal to the pericardium. The Doppler crystals were incorporated into cuffs (1–2 mm diameter) that were custom made from Perspex™. The Perspex™ provided a more secure fit around ventral aorta than the silicone cuffs provided by Iowa Doppler Products. The incision was closed with silk sutures (Ethicon 4-0) and the leads from the Doppler flow probe secured to the body wall. This procedure took approximately 10 min to complete. Fish were returned to tanks at their acclimation temperatures and allowed to recover for 24 h before recording and experimentation.

Experimental setup

For each acclimation group, the cardiac performance of eight fish was examined (body mass: –1°C, 88.9±15.1 g; 4°C, 91.1±11.8 g; means ± s.d.). Individual fish were transferred to an experimental tank (70 cm×40 cm×20 cm length×width×height) and the leads from the flow probe connected to a Doppler flowmeter (Iowa University, Iowa City, IA, USA, Model 545C-4). Signals from the flowmeter were directed to a computerised recording system (Powerlab, ADInstruments, Sidney, NSW, Australia) sampling at 40 Hz and displayed on a Toshiba laptop computer running Chart software (ADInstruments). Heart rate (f_H) was determined from the pulsatile flow signals using Chart.

Calibration of the Doppler flow probes was undertaken *in situ* at the completion of the experimental treatments. Fish were euthanased with an overdose of MS222 and a blood sample (approx. 1 ml) taken from the caudal vessels with a heparinised syringe (21-gauge needle). The heart was exposed and a polyethylene cannula inserted into the bulbous arteriosus and tied firmly in place. A peristaltic pump (Gilson Minipuls, Villers-le-Bel, France), passed blood diluted with saline (to a haematocrit of approx. 8%) through the cannula and ventral aorta at known flow rates (for details, see Axelsson et al., 1992).

Experimental protocol

Fish with implanted flow probes were transferred from the acclimation tanks to the experimental tank at their acclimation temperature (–1°C or 4°C) and left undisturbed for 4 h. Previous work had shown that this was sufficient time for cardiovascular parameters to return to resting levels (Lowe et al., 2005). Cardiac output ($\dot{Q}$) was continuously recorded at rest and after exhaustive exercise, which was achieved by chasing and tapping the tail of the fish with a spatula for 5 min. Measurements were taken at rest, and for 30 min after exhaustive exercise. Measurements were not taken during the forced exercise period as the signal from the flow probe was erratic. Fish were transferred from their holding tanks into the experimental chamber and initially tested at their acclimation temperature. They were then randomly tested at –1, 2, 4, 6 and 8°C, allowing 6 h recovery (at their acclimation temperature) between test temperatures. Temperature was increased or decreased in the experimental chamber at a rate of 2°C h^{-1}.

Analysis

Measurements of cardiac function (cardiac output and heart rate) were made during rest, exercise and recovery. Cardiac function was measured every 4–5 min, taking mean values for blocks of 30–60 s. Shorter sampling periods (30 s) were required immediately after exercise as cardiac function changed rapidly during this period. Stroke volume (V_S) was calculated from $\dot{Q}/f_H$. Factorial scope for cardiac output, heart rate and stroke volume was calculated by dividing maximal values (immediately after exercise) by resting values.

The effect of exercise and recovery time on $\dot{Q}$, V_S and f_H were analysed by a one-way repeated-measures ANOVA. The effect of exercise and test temperature on $\dot{Q}$, V_S and f_H were analysed with a two-way repeated-measures ANOVA. The effect of acclimation temperature and test temperature on $\dot{Q}$, V_S and f_H were analysed with a two-way repeated-measures ANOVA. Pairwise comparisons were analysed using t-tests. Results were considered significant at $P<0.05$. All results are presented as means ± s.e.m.

Results

Pagothenia borchgrevinki swam intensively and in bursts during the 5 min forced exercise period. The cardiac output of –1°C and 4°C acclimated *P. borchgrevinki* increased significantly from resting levels with exercise at all temperatures tested: –1, 2, 4, 6 and 8°C (one-way repeated-measures ANOVAs, all $P<0.05$). Fig. 1 shows the changes in cardiac output with exercise for –1°C and 4°C acclimated *P. borchgrevinki* at –1, 4 and 8°C. Cardiac output was at its peak at the end of the 5 min intensive exercise period and slowly decreased during the 30 min recorded recovery period (Fig. 1A–C).

In *P. borchgrevinki* acclimated to –1°C, there was a significant increase in resting cardiac output with increasing temperature (Fig. 2A; $F_{7,4}=10.50$, $P<0.001$; $Q_{10[-1°C\ to\ 8°C]}=$ 1.62). At –1°C, resting cardiac output of –1°C acclimated *P. borchgrevinki* was 22.2±2.9 ml min^{-1} kg^{-1} body mass and, upon exposure to 8°C, resting cardiac output increased by 54% to 34.2±2.9 ml min^{-1} kg^{-1} body mass. In contrast to the –1°C acclimated *P. borchgrevinki*, the exposure of the 4°C acclimated fish to an acute temperature change, resulted in higher resting cardiac outputs at lower temperatures (Fig. 2B; $F_{7,4}=5.26$, $P<0.01$). After acclimation to 4°C, resting cardiac output at 4°C was not significantly different from resting cardiac output of the –1°C acclimated fish at –1°C, demonstrating compensation of resting cardiac output with acclimation at higher temperatures. An acute change in temperature had no significant effect upon maximal cardiac output across the test temperatures in either the –1°C or 4°C acclimated *P. borchgrevinki* (Fig. 2A,B). Maximal cardiac outputs of the –1°C and 4°C acclimated fish at –1°C and 4°C, respectively, were also not significantly different.

Temperature had a highly significant effect upon both resting and maximal heart rates of the –1°C acclimated *P.*
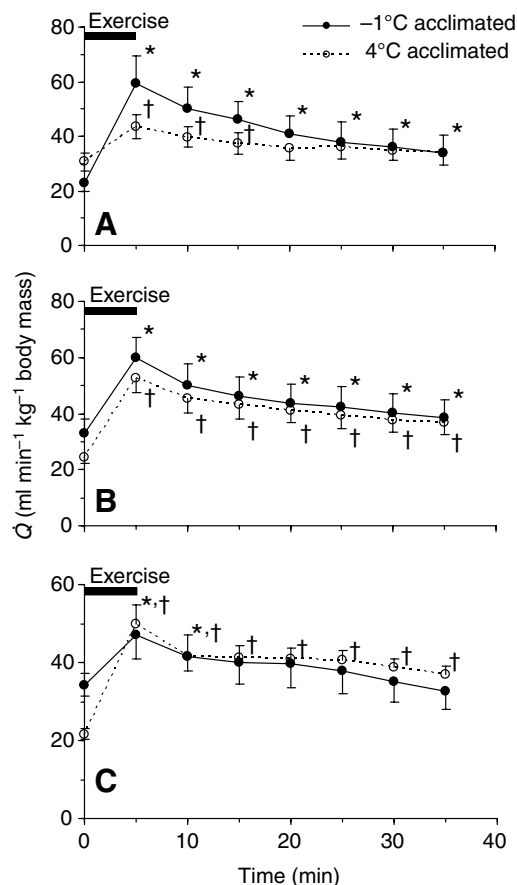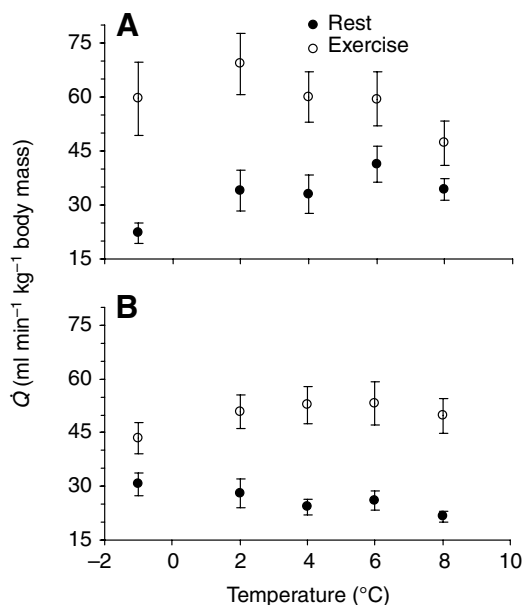

Fig. 1. The effect of 5 min exercise on cardiac output at –1°C (A), 4°C (B) and 8°C (C) in *Pagothenia borchgrevinki* acclimated to –1°C and 4°C for 4–5 weeks. There was a significant increase in cardiac output at all temperatures for both acclimation groups. Values are means ± s.e.m. ($N=8$). *Significant difference in cardiac output from resting levels (time=0 min) for –1°C acclimated fish; †significant difference in cardiac output from resting levels (time=0 min) for 4°C acclimated fish.

Fig. 2. The effect of an acute increase in temperature on resting and maximum cardiac output (ml min^{-1} kg^{-1} body mass) in *Pagothenia borchgrevinki* acclimated to –1°C (A) and 4°C (B). Values are means ± s.e.m. ($N=8$).
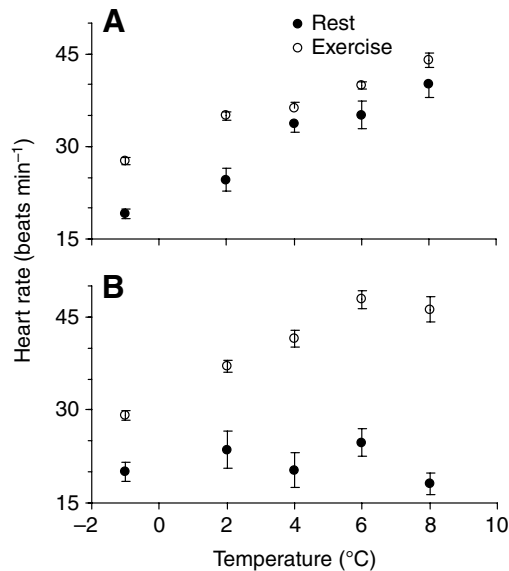
Fig. 3. The effect of an acute increase in temperature on heart rate (beats min^{-1}) at rest and during maximum cardiac output in *Pagothenia borchgrevinki* acclimated to –1°C (A) and 4°C (B). Values are means ± s.e.m. (*N*=8).
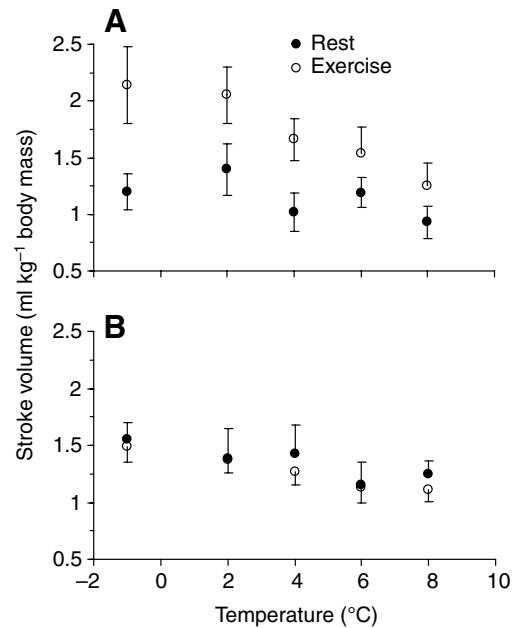

Fig. 4. The effect of an acute increase in temperature on stroke volume (ml kg^{-1} body mass) at rest and during maximum cardiac output in *Pagothenia borchgrevinki* acclimated to –1°C (A) and 4°C (B). Values are means ± s.e.m. (*N*=8).

borchgrevinki (Fig. 3A). With an increase in temperature from –1°C to 8°C, resting heart rate doubled from 19.1±0.9 to 40.1±2.1 beats min^{-1} ($F_{7,4}$=4.96, P<0.01; $Q_{10[-1°C to 8°C]}$=2.28). There was a significant effect of exercise on heart rate across test temperatures in the –1°C acclimated fish ($F_{1,4}$=7.09, P<0.01). Maximal heart rate of the –1°C acclimated *P. borchgrevinki* was 27.6±0.7 beats min^{-1} at –1°C and at 8°C heart rates reached 44.0±1.3 beats min^{-1} after 5 min of exercise, representing a Q_{10}=1.68 (Fig. 3A; $F_{7,4}$=20.72, P<0.001). The differences in Q_{10} values between resting and exercised *P. borchgrevinki* meant that as temperature increased, the ability to increase heart rate from resting levels decreased (Fig. 3A).

There was a significant effect of exercise on heart rate across test temperatures in the 4°C acclimated fish ($F_{1,4}$=5.58, P<0.01). Resting heart rates of the 4°C acclimated *P. borchgrevinki* were thermally independent between –1°C and 8°C (Fig. 3B). However, maximal heart rates were significantly affected by temperature (Fig. 3B; $F_{7,4}$=66.9, P<0.001). At –1°C, heart rate reached a maximum of 29.1±0.7 beats min^{-1} with exercise, whereas at 8°C, the maximum recorded heart rate was 46.2±2.0 beats min^{-1} ($Q_{10[-1°C to 8°C]}$=1.68). Therefore in the 4°C acclimated fish, as temperature increased, so did scope for heart rate (Fig. 3B). Resting heart rates of the –1°C and 4°C acclimated fish at –1°C and 4°C, respectively (–1°C 19.0±0.9 beats min^{-1}; 4°C 20.8±3.2 beats min^{-1}) were not significantly different from each other; however, the maximal heart rate of the 4°C acclimated fish at 4°C (41.5± 1.3 beats min^{-1}) was significantly higher (P<0.001) than the maximal heart rate of the –1°C acclimated fish at –1°C (27.6±0.7 beats min^{-1}).

There was a significant increase in stroke volume with exercise across test temperatures in the –1°C acclimated *P. borchgrevinki* (Fig. 4A; $F_{1,4}$=4.99, P<0.01). There was no effect of temperature on stroke volume of –1°C acclimated fish

at rest; however, with exercise there was a significant decrease in stroke volume with increasing temperature (Fig. 4A; $F_{7,4}$=4.69, P<0.01). Stroke volume during exercise decreased from 2.14±0.34 ml kg^{-1} at –1°C, to 1.25±0.20 ml kg^{-1} at 8°C. In the 4°C acclimated *P. borchgrevinki*, exercise had no effect on stroke volume across test temperatures (Fig. 4B; $F_{1,4}$=0.361, P=0.83). Acute increases in water temperature between –1°C and 8°C resulted in a small decrease in stroke volume at rest and during/after exercise in the 4°C acclimated fish (Fig. 4B).

At an ambient temperature of –1°C, *P. borchgrevinki* acclimated to –1°C had a significantly greater cardiac scope than fish acclimated to 4°C (P<0.01). In the –1°C acclimated fish at –1°C, cardiac output increased with exercise from 22.2±2.92 to 59.5±10.1 ml min^{-1} kg^{-1} body mass, representing a factorial cardiac scope of 2.62 (Fig. 2A and Fig. 5A). In contrast, the cardiac output of 4°C acclimated *P. borchgrevinki* exercised at –1°C had a factorial scope of 1.43, with cardiac output only increasing from 30.6±3.1 to 43.8±4.4 ml min^{-1} kg^{-1} (Fig. 2B and Fig. 5A).

There was a significant difference in the factorial scope for cardiac output between the –1 and 4°C acclimated fish across the test temperatures ($F_{1,4}$=17.53, P<0.001). In the –1°C acclimated *P. borchgrevinki* there was a significant decrease in factorial scope for cardiac output with an increase in temperature (Fig. 5A; $F_{7,4}$=12.9, P<0.001). At 8°C, the factorial scope of the –1°C acclimated fish had decreased to 1.38±0.16. In a complete reversal of the pattern seen in the –1°C acclimated fish, the factorial scope for cardiac output of the 4°C acclimated *P. borchgrevinki* significantly increased with increasing temperature (Fig. 5A; $F_{7,4}$=5.93, P<0.01), and scope was 2.32±0.21 at 8°C. This was due to the thermal independence of
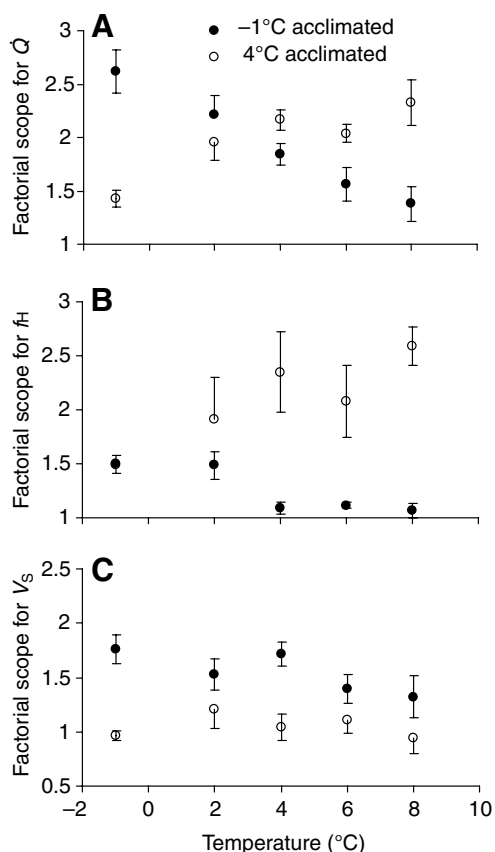
Fig. 5. Effect of acclimation temperature in *Pagothenia borchgrevinki* on factorial scope for (A) cardiac output, (B) heart rate and (C) stroke volume. Factorial scope was calculated as maximum level during exercise divided by resting level. Values are means ± s.e.m. (*N*=8).

resting cardiac output across the test temperatures in the 4°C acclimated fish (Fig. 2B).

In the –1°C acclimated fish, the increase in cardiac output with exercise at –1°C was due to increases in both heart rate and stroke volume (Fig. 3A, Fig. 4A, Fig. 5B,C). With exercise at –1°C, heart rate increased from 19.1±0.9 to 27.6± 0.7 beats min^{-1} (factorial scope for heart rate=1.50±0.08) and stroke volume increased from 1.20±0.16 to 2.14± 0.34 ml kg^{-1} body mass (factorial scope for stroke volume= 1.76±0.13) (Fig. 3A, Fig. 4A). The decrease in factorial scope for cardiac output of the –1°C acclimated fish at the higher ambient temperatures could be attributed to a significant decrease in factorial scope for heart rate combined with a drop in factorial scope for stroke volume (Fig. 5A–C).

There was a significant difference in the factorial scope for heart rate between the –1 and 4°C acclimated fish across the test temperatures ($F_{1,4}$=13.35, P<0.001) but not in factorial scope for stroke volume ($F_{1,4}$=1.72, P=0.159). The maintenance of factorial scope for cardiac output in the 4°C acclimated fish between 2–8°C was due to changes in heart rate alone (Fig. 5A–C). Stroke volume did not change with exercise and hence did not contribute to scope for cardiac output in 4°C acclimated *P. borchgrevinki* (Fig. 4B, Fig. 5C). At –1°C, the decrease in factorial scope for cardiac output of the 4°C

acclimated fish could be attributed to a decrease in factorial scope for heart rate (Fig. 5A,B).

Discussion

The cardiovascular system of *P. borchgrevinki* acclimated to –1°C is highly sensitive to acute changes in water temperature. This is in agreement with a recent study that reported a significant effect of temperature on resting heart rate and ventral aortic pressure in *P. borchgrevinki* (Lowe et al., 2005), but is in contrast to another, which found that heart rate was thermally independent between –1 and 3°C (Franklin et al., 2001). The different thermal responses of the cardiovascular system of *P. borchgrevinki* are possibly due to the rate and magnitude of the temperature increases, with the temperature increase in the present study and in Lowe et al. (Lowe et al., 2005) being considerably larger and more rapid than that of Franklin et al. (Franklin et al., 2001). In the present study, subjecting the –1°C acclimated fish to acute increases in temperature up to 8°C had a positive chronotropic effect on the heart at both rest and with exercise. Stroke volume, however, remained unaffected at rest by an increase in temperature, and with exercise there was a negative influence on stroke volume, with a marked reduction being observed at high temperatures. In general, acute increases in temperature have a positive chronotropic and negative intropic effect on the myocardium of fish, resulting in a reduction of maximum cardiac power output (Farrell et al., 1996; Gamperl and Farrell, 2004). While there was no significant decrease in maximum cardiac output at high temperatures, factorial scope for cardiac output was severely compromised. In the –1°C acclimated fish, the factorial scope for cardiac output was greatest at –1°C with increase in cardiac output at –1°C after exercise being the result of near equal increases in both heart rate and stroke volume. When exposed to water temperatures of 4–8°C there was a marked reduction in scope for cardiac output, and at 8°C maximal cardiac output was only 10% higher than resting cardiac output. At the higher temperatures, there was virtually no scope for heart rate, with maximal heart rates with exercise being close to resting heart rates. Only stroke volume contributed significantly to the increase in cardiac output at the high temperatures. The observed detrimental affects of an increase in temperature on scope for cardiac output in *P. borchgrevinki* would contribute to the reduction in sustained swimming performance of this fish at higher temperatures, and its narrow thermal swimming performance breadth (Wilson et al., 2002). The reduction in swimming performance would also result from a decrease in O$_2$ binding affinity and O$_2$ carrying capacity at higher temperatures as a consequence of a right shift of the oxygen equilibrium curve (Farrell, 2002; Taylor et al., 1996; Taylor et al., 1997).

Farrell suggested that cardiac oxygen supply in fish may become limited at high temperatures due to lowered venous oxygen levels (Farrell, 2002). The lower oxygen levels would become insufficient to meet cardiac oxygen demand, resulting in a reduction in the ability of the myocardium to maintain cardiac scope. While this may be a factor in compromising cardiac function in *P. borchgrevinki* at high acute temperatures, maximum cardiac output was maintained from –1°C to 8°C in

–1°C acclimated fish, and rather it was the inability of *P. borchgrevinki* to regulate resting cardiac output *via* heart rate that resulted in a reduction in cardiac scope. The increase in cardiac output with an increase in temperature under resting conditions is most likely due to the thermal dependence of metabolism and the requirement to increase oxygen delivery to the tissues at higher temperatures (Wilson et al., 2002).

When exposed to elevated temperatures (4°C) for a prolonged period *P. borchgrevinki* fully compensated for the negative effects of high temperatures, and sustained swimming performance was restored (Seebacher et al., 2005). With acclimation to 4°C, scope for cardiac output was maintained at 4, 6 and 8°C, resulting in the thermal compensation of cardiac function at the higher temperatures. This however, was at the expense of cardiac performance at –1°C, where there was a significant decrease in cardiac scope. This reduction in scope for cardiac output of the 4°C acclimated fish at –1°C was attributed to a lack of scope for stroke volume; that is, stroke volume did not change with exercise.

While increases in cardiac output with exercise in the –1°C acclimated fish at 1°C were achieved by increases in both heart rate and stroke volume, increases in cardiac output with exercise in the 4°C acclimated fish were achieved by changes in heart rate alone, with stroke volume not varying between rest and exercise at any test temperatures. This striking difference between thermal acclimation groups represents a change in pumping strategy from a mixed inotropic/chronotropic modulated heart in –1°C acclimated *P. borchgrevinki* to a purely chronotropic modulated heart in the 4°C acclimated fish.

Cardiac plasticity in response to thermal acclimation has been reported for a number of fish species (Gamperl and Farrell, 2004), although the marked change in pumping strategy seen in *P. borchgrevinki* to chronic temperature exposure has not been noted previously. Thermal compensation of maximum cardiac output and maximum power output occurs in rainbow trout with Q_{10} values of 1.2–1.4 between 5°C and 18°C (Graham and Farrell, 1989; Keen and Farrell, 1994). Q_{10} values for factorial scope of cardiac output in *P. borchgrevinki* are also reduced with acclimation, having equivalent scopes when tested at their acclimation temperatures, indicating thermal compensation. Increases in ventricular muscle mass occur with cold acclimation in rainbow trout, which offsets the decrease in contractility at lower temperatures, thereby maintaining stroke volume (Gamperl and Farrell, 2004). The intrinsic cardiac pacemaker rate is reset with cold acclimation in rainbow trout and involves changes to membrane ion channel function and density (Vornanen et al., 2002). This mechanism warrants investigation in *P. borchgrevinki*, given the change in pumping strategy to a chronotropic driven heart with acclimation to 4°C.

The maintenance of cardiac function in *P. borchgrevinki* with acclimation to 4°C could result from thermal compensation of metabolism at the higher temperature and a corresponding reduction in oxygen demand. This is likely, as resting cardiac output was reduced at 4°C acclimated fish compared with –1°C acclimated fish. The stenothermal gadoid, *Lota lota*, can survive high summer water temperatures because it is able to downregulate its aerobic metabolism (Hardewig et al., 2004). Surprisingly, the thermal acclimation of resting metabolic rate

has not been studied in *P. borchgrevinki*, although metabolic capacities (i.e. lactate dehydrogenase and cytochrome *c* oxidase activities) increased with warm acclimation (Seebacher et al., 2005).

In Antarctic fish, the cholinergic system strongly regulates heart rate and the cholinergic tone increased with an acute increase in temperature (Axelsson et al., 1992; Axelsson et al., 1994; Franklin et al., 2001; Lowe et al., 2005). Thermal acclimation has been shown to alter the extrinsic modulation of the heart affecting the relative contributions from the cholinergic and adrenergic systems. Wood et al. found that cholinergic tone increased with cold acclimation in rainbow trout, and that adrenergic tone was more important at higher temperatures (Wood et al., 1979). Again, this aspect of cardiac control with thermal acclimation warrants further investigation in *P. borchgrevinki*.

The thermal sensitivity of factorial scope for cardiac output provides an excellent indicator of cardiac performance in response to temperature (Farrell, 2002). The greatest impact of a change in temperature can be seen on scope for cardiac output in *P. borchgrevinki*. An acute increase in temperature negatively affected scope for cardiac output in the –1°C acclimated fish, and conversely a decrease in temperature had a deleterious effect on scope in the 4°C acclimated fish. Investigating only the acute effects of temperature on resting and/or maximal cardiac performance would have failed to detect significant negative effects on cardiac performance unless at extreme temperatures, where cardiac function would be severely compromised and arrhythmias often result (Farrell, 2002; Gollock et al., 2006).

The compensation of cardiac and swimming performance (Seebacher et al., 2005) in *P. borchgrevinki* with thermal acclimation is contrary to the general dogma that Antarctic fish are stenothermal and highly specialised to the very stable and cold waters of the Antarctic (Somero et al., 1996). Evolutionary theory predicts that specialisation to a narrow set of environmental conditions will result in a decrease in capacity for an organism's phenotype to change in response to environmental change and variability (Huey and Hertz, 1984). Antarctic fish from high latitudes are often regarded as the archetypal thermal specialists (Somero and DeVries, 1967; Wilson et al., 2002; Johnston, 2003). However, the ability of *P. borchgrevinki* to thermally acclimate and in so doing increase performance breadth over a range of temperatures not experienced on an annual basis, calls for a re-evaluation of the basis of thermal specialisation in Antarctic fish. This is perhaps not surprising given that *P. borchgrevinki* evolved from an ancestral stock of temperate fishes (Eastman, 1993), and that despite the present cold and stable temperatures of the Southern Ocean, fishes from the Antarctic waters have been exposed to episodic periods of warming and cooling over geological time (Clarke and Johnston, 1996). This temperature variation provides the necessary driver for thermal plasticity.

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