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RESEARCH ARTICLE

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Effects of changes in water salinity upon exercise and cardiac performance in the European seabass (*Dicentrarchus labrax*)

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Abstract The European seabass is an active euryhaline teleost that migrates and forages in waters of widely differing salinities. Oxygen uptake (M_{O2}) was measured in seabass (average mass and forklength 510 g and 34 cm, respectively) during exercise at incremental swimming speeds in a tunnel respirometer in seawater (SW) at a salinity of 30‰ and temperature of 14°C, and their maximal sustainable (critical) swimming speed (U_{crit}) determined. Cardiac output (Q) was measured via an ultrasound flow probe on their ventral aorta. The fish were then exposed to acute reductions in water salinity, to either SW (control), 10‰, 5‰, or freshwater (FW, 0‰), and their exercise and cardiac performance measured again, 18 h later. Seabass were also acclimated to FW for 3 weeks, and then their exercise performance measured before and at 18 h after acute exposure to SW at 30‰. In SW, seabass exhibited an exponential increase in M_{O2} and Q with increasing swimming speed, to a maximum M_{O2} of 339 ± 17 mg kg^{-1} h^{-1} and maximum Q of 52.0 ± 1.9 ml min^{-1} kg^{-1} (mean ± 1 SEM; $n = 19$). Both M_{O2} and Q exhibited signs of a plateau as the fish approached a U_{crit} of 2.25 ± 0.08 bodylengths s^{-1}. Increases in Q during exercise were almost exclusively due to increased heart rate rather than ventricular stroke volume. There were no significant effects of the changes in salinity upon M_{O2} during exercise, U_{crit} or cardiac performance. This was linked to an exceptional capacity to maintain plasma osmolality and tissue water content unchanged following all salinity challenges. This extraordinary adaptation would allow the seabass to maintain skeletal and cardiac muscle function while migrating through waters of widely differing salinities.

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Introduction

European seabass (Family Moronidae) pursue an active pelagic life-history that comprises widescale migrations. Although predominantly marine as adults, seabass are euryhaline and juveniles often enter estuaries where they migrate and forage through waters of different salinities (Pickett and Pawson 1994). Relatively little is known about the cardio-respiratory, exercise and osmoregulatory physiology of seabass (Jensen et al. 1998; Claireaux and Lagardère 1999; Varsamos et al. 2001, 2002; Axelsson et al. 2002) and, therefore, also about the physiological adaptations that they may have evolved for their migratory euryhaline lifestyle.

Previous studies on fish species that migrate through waters of different salinities have shown a direct link between their ability to perform aerobic exercise and their homeostatic regulation of body-fluid osmolality (Brauner et al. 1992, 1994; McKenzie et al. 2001a, 2001b). In coho salmon (*Oncorhynchus kisutch*) smolts and Adriatic sturgeon (*Acipenser naccarii*) juveniles, acute increases in water salinity (''seawater challenges'') caused accumulation of plasma ions and large increases in plasma osmolality, which were directly related to a significant reduction in their maximum sustainable (critical) swimming speed (U_{crit}) (Brauner et al. 1992, 1994; McKenzie et al. 2001a, 2001b). It has been suggested that this was due to impaired cardiac and skeletal muscle function consequent to ionic imbalances and loss of tissue moisture, plus further strain upon the heart due to haemoconcentration (Brauner et al. 1992, 1994; McKenzie et al. 2001a, 2001b). The effects of acute reductions in salinity have not been studied, but a loss of ions from plasma and tissues, plus increased tissue water

content, would also be expected to impair the function of cardiac and skeletal muscle. Kolok and Sharkey (1997) found that gulf killifish (*Fundulus grandis*) acclimated to freshwater (FW) had a significantly lower U_{crit} than fish maintained in brackish water (BW) at a salinity of 10‰, and attributed this to the osmotic stresses the animals suffered in FW.

Studies investigating effects of salinity change upon ion and water balance in seabass have provided contrasting results. Jensen et al. (1998) acclimated seabass to BW at a salinity of 15‰ and found that acute exposure to either FW or concentrated seawater (SW) at a salinity of 50‰ both caused profound iono-osmotic imbalances that lasted for a number of days. However, Varsamos et al. (2001) found that seabass acclimated to BW at 25‰ had an exceptional ability to regulate osmotic homeostasis, with no changes in plasma ion and osmotic status at 24 h following acute exposure to either BW at 5‰ or SW at 39‰. The ability of seabass to maintain ionic and osmotic homeostasis may, clearly, be of critical importance to their ability to negotiate migratory movements through estuaries, where water salinity may vary quite significantly over short temporal and spatial scales.

In active migratory fish such as salmonids, the capacity for sustained aerobic exercise is believed to be directly related to the ability of the heart to provide blood flow (Farrell 2002; Claireaux et al. 2005). Relatively little is known about cardiac performance during sustained exercise in other teleosts (reviewed by Farrell and Jones 1992; also Kolok et al. 1993; Kolok and Farrell 1994a, 1994b; Korsmeyer et al. 1997a, 1997b). Cardiac output (Q) during swimming is a product of ventricular stroke volume (V_{SH}) and heart rate (f_H) and, in almost all fishes studied to date, increases in V_{SH} make the major contribution to increased Q during exercise, with the tunas and some Antarctic teleosts (Axelsson et al. 1992; Farrell and Jones 1992; Farrell 1996; Thorarensen et al. 1996a; Brill and Bushnell 2001) being considered exceptions to this general rule. This dominant role for inotropic versus chronotropic regulation during exercise has, however, yet to be investigated in the vast majority of teleost fishes.

The current study investigated the performance and energetics of sustained aerobic exercise in seabass, and the associated performance of the heart. It investigated how these were influenced by changes in water salinity, the hypothesis being that maintenance of exercise and cardiac performance following salinity change would be directly dependent upon the regulation of osmotic homeostasis in plasma and tissues.

Materials and methods

Experimental animals

European seabass (*Dicentrarchus labrax*) with a mean (±SEM) mass of 514 ± 23 g and length of 34.4 ± 0.5 cm were obtained from a commercial supplier on the Île de Ré (Charente Maritime, France), where they had been maintained throughout their lives in SW net pens. They were maintained at CREMA in 1-m^2 fibreglass tanks (water volume approximately 400 l) provided with biofiltered SW at a salinity of 30‰ and temperature of 14 ± 0.4°C, for at least 2 weeks prior to any use in experiments. Animals were fed commercial fish feed daily, but were starved for 24 h prior to use. A sub-set of the seabass ($n = 6$) were acclimated for 2 weeks to biofiltered FW (dechlorinated l'Houmeau tapwater, salinity 0‰) at the same temperature. The reduction in salinity was accomplished over 24 h, by flushing the SW in their tank until it had been completely replaced with the FW. The seabass resumed feeding within 48 h of the FW transfer.

Surgical preparation

Bass were anaesthetised with tricaine methane sulphonate (MS-222) at a concentration of 0.1 g l^{-1}, and transferred to an operating table where their gills were irrigated with aerated water containing 0.05 g l^{-1} MS-222. A 2S-type Transonic ultrasound flow probe (resolution 0.1 ml min^{-1}; absolute accuracy ±15%) was placed around the ventral aorta, as described by Axelsson et al. (2002). For surgery on the seabass acclimated to FW, the MS-222 was buffered with $NaHCO_3$ (0.05 g l^{-1}). The animals were allowed 48 h recovery in opaque PVC chambers provided with a flow of water at the appropriate salinity (either SW or FW).

Exercise and cardiac performance

Swimming respirometry was performed with an automated Brett-type swim-tunnel respirometer designed to exercise fish in a non-turbulent water flow with a uniform velocity profile, as described in detail by McKenzie et al. (2001a). Fish were transferred individually to the respirometer and allowed to recover for at least 12 h (overnight) in a current at a speed of 5 cm s^{-1}. At this low current speed, the bass rested on the bottom and maintained position in the flume by very gentle sculling of their pectoral fins and occasional tailbeats. The following day, the seabass were exposed to progressive increments in swimming speed, of 5, 10 and then each 20 cm s^{-1} every 30 min, until fatigue. Fish were considered to be fatigued when they were unable to remove themselves from the posterior screen of the swimming chamber despite gentle encouragement by sudden increases in current velocity.

Measurements of O_2 uptake (M_{O2}, in mg kg^{-1} h^{-1}) at each swimming speed were calculated automatically with the custom-designed data-acquisition system described in McKenzie et al. (2001a), from the decline in water O_2 saturation in the sealed respirometer, the volume of water, and the mass of the fish. Water O_2 saturation was measured with an Orbisphere clarke-type polarographic

oxygen electrode and associated meter (Orbisphere Laboratory, Geneva, Switzerland). For each individual fish, a least squares exponential regression was applied to the relationship between swimming speed and M_{O2}. Note that data for swimming speeds close to fatigue were not included in this analysis, to ensure that swimming performance was sustained by aerobic metabolism and that the relationship between M_{O2} and swimming speed was not, therefore, confounded by contributions to performance from anaerobic metabolic pathways. Extrapolation back to the *y*-intercept, a notional swimming speed of zero, was then employed to correct for the contribution to M_{O2} of locomotor-muscle activity (Brett 1964; Fry 1971). The value thus derived was termed inactive metabolic rate (IMR; McKenzie et al. 2003). The maximum metabolic rate of activity (AMR) was identified during swimming (this occurred at speeds approaching U_{crit}) and net aerobic scope was then calculated as AMR minus IMR (Fry 1971; McKenzie et al. 2003). Each fish's U_{crit} was calculated in cm s^{-1}, and also bodylengths s^{-1} (BL s^{-1}), as described by Brett (1964).

At each swimming speed, Q was measured in ml min^{-1} kg^{-1}, with the signal from the flowprobe displayed on the Transonic amplifier and acquired by a PC with the custom-designed labview software described by Axelsson et al. (2002). The signal was used to calculate f_H in beats min^{-1} and, together with the data for Q, used to calculate V_{SH}, in ml $beat^{-1}$ kg^{-1}, as described by Axelsson et al. (2002). Cardiac scope during exercise was calculated as maximum Q minus "routine" Q. Maximum Q always occurred at swimming speeds approaching U_{crit}. Routine Q was taken as the lowest Q measured when the fish was swimming very gently at a speed of 5 cm s^{-1}, prior to the exercise protocol. Routine f_H and V_{SH} were derived from the measures of routine Q.

Salinity challenges

Following the above exercise protocol in the water to which they had been acclimated, the seabass were exposed to an acute change in water salinity, as follows: SW to SW (control); SW to BW at 10‰; SW to BW at 5‰; SW to FW, or FW to SW. The reductions in salinity were accomplished by flushing the respirometer with dechlorinated tapwater, while the FW to SW transfer was accomplished by flushing with seawater. Water salinity was measured with a salinometer (WTW F216, WTW, Weilheim, Germany).

At 18 h after these changes in water salinity, exercise and cardiac performance were measured once again, exactly as described above. Following this second swim test, animals were rapidly removed from the respirometer and killed with a blow to the head. A blood sample was withdrawn from the caudal vein and centrifuged to obtain plasma, which was stored at −20°C for subsequent measurement of osmolality, with a freezing-point osmometer (13/13DR, Herman Roebling MESSTECHNIK, Berlin). Samples of muscle and ventricle were collected, weighed and then dried to constant weight at 50°C to estimate percentage tissue water content.

Statistical analyses

The effects of the first exercise test upon M_{O2} and cardiac variables were assessed by one-way analysis of variance (ANOVA) for repeated measures. The effects of the salinity challenges upon performance were assessed as their effects upon U_{crit}, IMR, AMR, aerobic scope, and maximum Q, using a two-way ANOVA for repeated measures. The effects of the salinity challenges upon plasma osmolality, and skeletal or cardiac water content, were assessed by one-way ANOVA.

Results

Whereas all of the animals acclimated to SW survived anaesthesia and placement of the Transonic cuff ($n = 19$), the seabass acclimated to FW died when this was attempted, so the procedure was only performed upon 3 fish and the remaining three were exercised without the cuff. Data for cardiac performance are not, therefore, available for fish acclimated to FW.

Exercise and cardiac performance in SW

When all data are pooled for the first swim test in SW, the seabass achieved a U_{crit} of 80.2 ± 4.3 cm s^{-1}, equivalent to 2.25 ± 0.08 BL s^{-1} (mean ± SEM, $n = 19$). As shown in Fig. 1, there was a significant and profound increase in M_{O2} during exercise; the increase was exponential up to 60 cm s^{-1} followed by deviation towards an asymptote as the fish approached U_{crit}. Application of an exponential relationship to the combined raw data between 5 cm s^{-1} and 60 cm s^{-1} revealed a high correlation coefficient, with $R^2 = 0.946$. Mean IMR was 56 ± 7 mg O_2 kg^{-1} h^{-1} and AMR was 339 ± 17 mg O_2 kg^{-1} h^{-1}, such that net aerobic scope was 283 ± 20 mg O_2 kg^{-1} h^{-1}. Thus, the sustained aerobic exercise elicited an approximately fivefold increase in metabolism above IMR.

The mean routine Q of the seabass was 25.0 ± 1.39 ml min^{-1} kg^{-1}; routine f_H was 34.1 ± 2.0 beats min^{-1} and routine V_{SH} was 0.68 ± 0.04 ml $beat^{-1}$ kg^{-1}. The changes in Q during exercise showed a similar response profile to that of M_{O2}, with an exponential increase up to 60 cm s^{-1} followed by an asymptote at higher speeds (Fig. 1). Application of an exponential relationship to the combined raw data between 5 cm s^{-1} and 60 cm s^{-1} revealed a high correlation coefficient, $R^2 = 0.936$. Maximum Q was 52.0 ± 1.9 ml min^{-1} kg^{-1} and net cardiac scope was 30.1 ± 1.6 ml min^{-1} kg^{-1}, so that Q approximately doubled during the exercise protocol. The increase in Q was associated with a significant increase in both f_H and V_{SH} (Fig. 1). As can be seen in

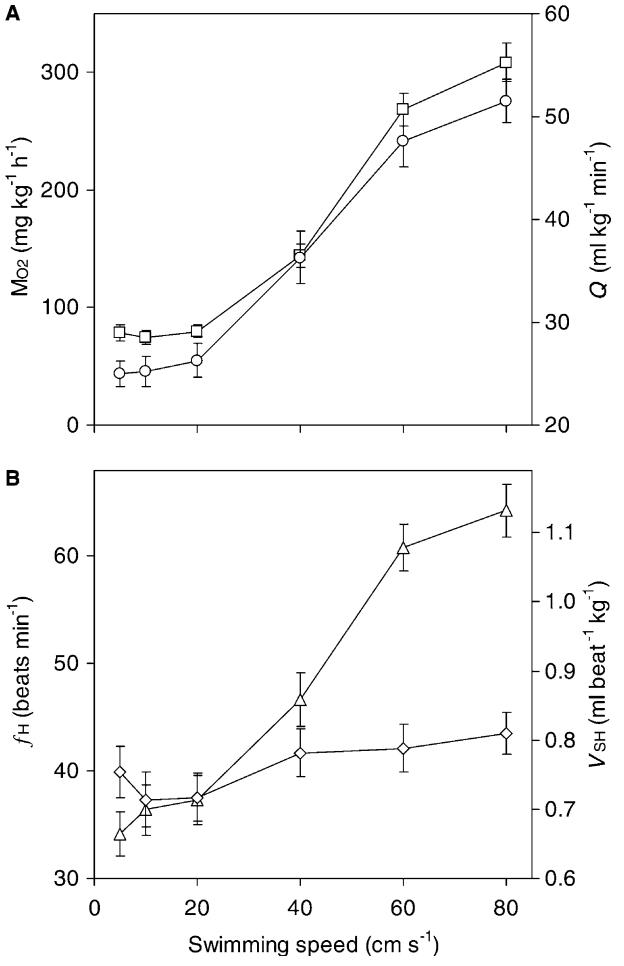

Fig. 1 Metabolic rate and cardiac performance in seabass exercising in seawater (salinity = 30‰) at 14°C. Effects of stepwise increase in swimming speed on **a** oxygen consumption (M_{O2}, *squares*) and cardiac output (Q, *circles*) and **b** heart rate (f_H, *triangles*) and stroke volume (V_{SH}, *diamonds*). Values are mean ± SEM, n = 19

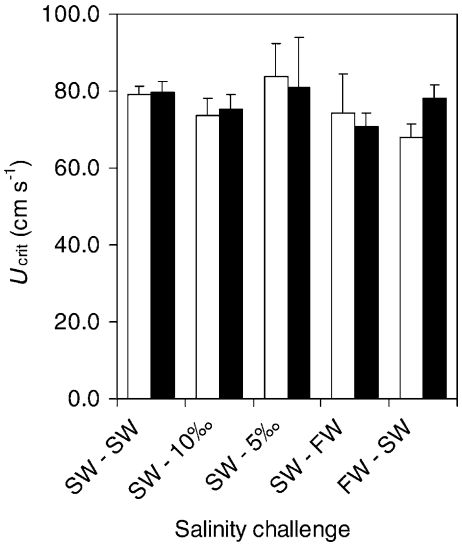

Fig. 2 The effects of acute changes in water salinity on mean (±SEM) critical swimming speed (U_{crit}). *Unfilled columns* are means of the first swim test in either seawater (*SW*) or fresh water (*FW*). *Filled columns* are means of the second swim test 18 h after the salinity challenges. These comprised an acute change from seawater to either seawater (*SW-SW*, n = 6); brackish water at a salinity of 10‰ (*SW-10‰*, n = 4); brackish water at a salinity of 5‰ (*SW-5‰*, n = 6), or to fresh water (*SW-FW*, n = 3); or an acute change from fresh water to seawater (*FW-SW*, n = 3)

Fig. 1, however, the increase in f_H was much more pronounced than the increase in V_{SH}, and showed a response profile that closely mirrored those of both M_{O2} and Q. Indeed, f_H increased exponentially to 60 cm s^{-1} whereas V_{SH} showed no further significant increases after 40 cm s^{-1}, and the increase in f_H accounted for almost 90% of the measured increase in Q.

Effects of the salinity challenges

The seabass exhibited an exceptional tolerance of all the salinity challenges, with no mortalities observed. Indeed, as can be seen in Fig. 2, the salinity challenges had no significant effects upon U_{crit}, which was maintained at the same level as prior to the challenge. As might be expected from the fact that U_{crit} was maintained, the salinity challenges also had no significant effects upon IMR, AMR or aerobic scope (Fig. 3) or upon maximum Q (Fig. 4).

The ability of the seabass to maintain exercise and cardiac performance following the salinity challenges was associated with an extraordinary capacity for homeostatic regulation of plasma osmolality and tissue water balance. That is, the mean plasma osmolality of the seabass when sampled at fatigue from the swim test performed after the various salinity challenges was not significantly different from that of the control seabass fatigued in SW (Table 1). Similarly, the mean water content of cardiac and skeletal muscle from the seabass exposed to the salinity challenges was not significantly different from that of the control seabass in SW (Table 1). Note that water content was not measured in the fish exposed to the SW to FW or FW to SW challenges, but the fact that they regulated plasma osmolality indicates that tissue water balance was also maintained.

Discussion

The ability of the seabass to maintain exercise performance following changes in salinity differs from the responses observed in coho salmon and Adriatic sturgeon, where seawater challenge caused a significant decline in U_{crit} (Brauner et al. 1992, 1994; McKenzie et al. 2001a, 2001b). The impaired responses in these latter species were directly related to significant increases in plasma ion concentrations and osmolality, and significant reductions in tissue water content (Brauner et al. 1992, 1994; McKenzie et al. 2001a, 2001b). Thus, it seems reasonable to assume that the absence of any changes in

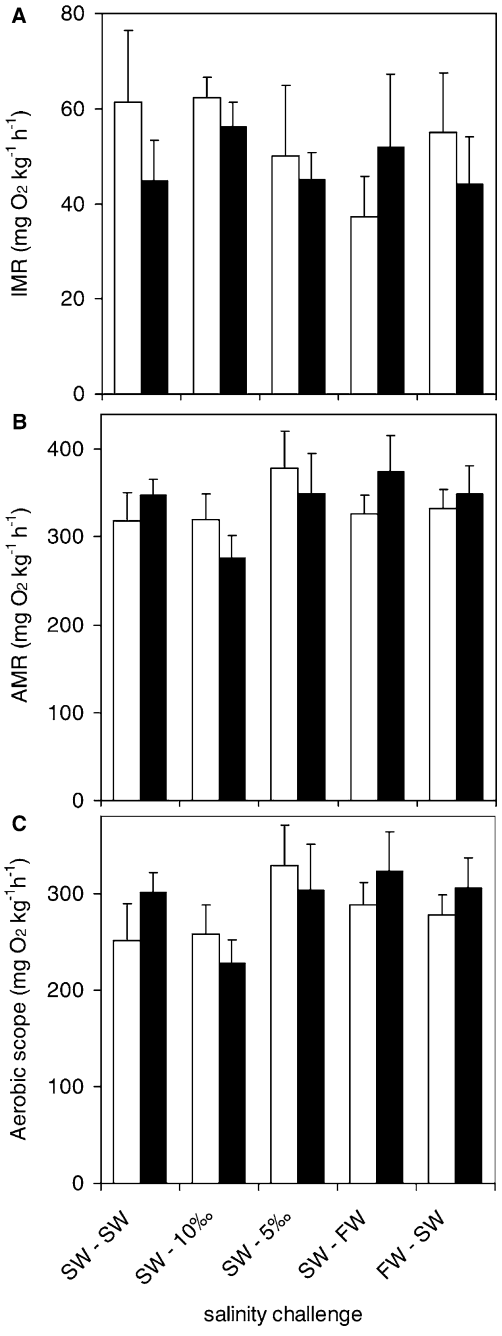

Fig. 3 Effects of acute changes in water salinity on mean (±SEM) immobile metabolic rate (*IMR*) (**a**), active metabolic rate (*AMR*) (**b**) and resultant aerobic scope (**c**). *Unfilled columns* are means of the first swim test in either seawater (*SW*) or fresh water (*FW*). *Filled columns* are means of the second swim test 18 h after the salinity challenges. These comprised an acute change from seawater to either seawater (*SW-SW*, $n=6$); brackish water at a salinity of 10‰ (*SW-10‰*, $n=4$); brackish water at a salinity of 5‰ (*SW-5‰*, $n=6$), or to fresh water (*SW-FW*, $n=3$); or an acute change from fresh water to seawater (*FW-SW*, $n=3$)

the seabass' exercise or cardiac performance following the various salinity challenges was a direct result of their exceptional ability to regulate plasma osmotic homeostasis and tissue water balance.

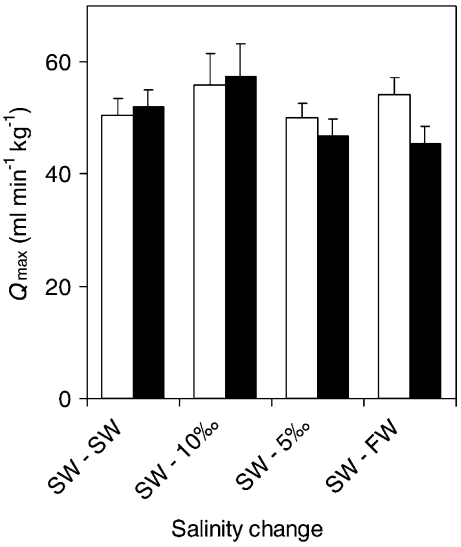

Fig. 4 Effects of changes in water salinity on mean (±SEM) maximum cardiac output (Q_{max}). *Unfilled columns* are means of the first swim test in seawater (*SW*). *Filled columns* are means of the second swim test 18 h after the salinity challenges. These comprised an acute change from seawater to either seawater (*SW-SW*, $n=6$); brackish water at a salinity of 10‰ (*SW-10‰*, $n=4$); brackish water at a salinity of 5‰ (*SW-5‰*, $n=6$), or to fresh water (*SW-FW*, $n=3$)

This extraordinary capacity for homeostatic regulation of osmotic status is consistent with a previous study by Varsamos et al. (2001). Salmonids and sturgeon exhibit significant osmotic imbalances at 24 h following changes in water salinity (Bath and Eddy 1979; Brauner et al. 1992, 1994; Claireaux and Audet 2000; McKenzie et al. 2001a, 2001b), and so do such species as Mozambique tilapia (*Oreochromis mossambicus*) and its hybrids, which are able to adapt to extreme salinities of up to 95‰ (Hwang et al. 1989; Sardella et al. 2004). The European flounder (*Platichthys flesus*) is, however, able to tolerate increases in salinity from 10‰ to 30‰ with only a small increase in plasma osmolality at 24 h exposure (Jensen et al. 2002).

In many euryhaline species, changes in water salinity lead to changes in routine M_{O2} and, although both increases and decreases in M_{O2} have been reported, these have all been attributed to the existence of osmotic stress (e.g. Morgan and Iwama 1991, 1998; Swanson 1998; McKenzie et al. 2001a, 2001b; Sardella et al. 2004). Thus, the absence of any changes whatsoever in IMR of the seabass, whether acclimated to SW or FW, and following the various salinity challenges, may indicate that the maintenance of osmotic homeostasis did not cause any significant stress or metabolic costs. There was one indication that acclimation to FW was, however, significantly stressful to the seabass, which was their inability to tolerate anaesthesia and placement of the ventral aortic flow cuff. It is not clear whether seabass spend extended periods in FW in nature (Pickett and Pawson 1994), but there are anecdotal reports that they are particularly sensitive to husbandry stresses when reared in FW.

Table 1 Mean (±SEM) plasma osmolality and percentage tissue water content in ventricular and skeletal muscle of seabass exposed to various salinity challenges for 18 h and then exercised to U_{crit}. The salinity challenges comprised an acute change from seawater to either seawater (SW-SW); brackish water at a salinity of 10‰ (SW-10‰); brackish water at a salinity of 5‰ (SW-5‰), or to fresh water (SW-FW); or from fresh water to seawater (FW-SW)

Salinity change	Plasma osmolality (mOsm kg^{-1})	Ventricle water content (% by mass)	Muscle water content (% by mass)
SW-SW ($n=6$)	374 ± 11	75.2 ± 0.7	79.6 ± 0.8
SW-10‰ ($n=4$)	384 ± 23	74.7 ± 0.7	79.9 ± 0.5
SW-5‰ ($n=6$)	358 ± 18	76.2 ± 0.9	80.8 ± 1.2
SW-FW ($n=3$)	364 ± 34	NA	NA
FW-SW ($n=3$)	379 ± 11	NA	NA

Varsamos et al. (2002) suggested that the exceptional capacity for osmotic regulation may reflect extremely plastic morphofunctional adaptations of mitochondria-rich (“chloride”) cell populations in the gills, and the osmoregulatory physiology of the seabass clearly represents a fascinating area for future research. Whatever the mechanism by which the seabass can maintain a constant internal milieu when faced by variations in environmental salinity, this ability would allow them to migrate freely throughout estuaries, and between marine, BW and FW habitats (Pickett and Pawson 1994). It is interesting that the European flounder, which is also reported to perform such facultative migrations, also possesses a similar ability to regulate the osmotic status of their internal milieu when exposed to acute changes in water salinity (Jensen et al. 2002).

The performance and energetics of sustained exercise in the seabass at 14°C were comparable to those of farmed and instrumented salmonids of a similar size at temperatures between 10°C and 14°C (e.g. Gallaugher et al. 2001; Shingles et al. 2001; Beaumont et al. 2003). For example, Shingles et al. (2001) exercised cannulated rainbow trout (*Oncorhynchus mykiss*) of a similar size at 14°C in the same respirometer as used in the current study, and reported a U_{crit} of 2.23 BL s^{-1}, compared with 2.25 BL s^{-1} measured in the current study. This required a similar, over fivefold, increase in metabolic rate above IMR (Shingles et al. 2001). A temperature of 14°C is at the lower end of the European seabass's thermal range; they perform better at warmer temperatures around their thermal optimum. Aerobic scope at 26°C is up to double that observed at 14°C (Claireaux and Lagardère 1999) and U_{crit} some 1.5 times higher (G. Claireaux, unpublished observations).

Seabass cardiac scope during exercise, and the maximum Q achieved, were similar to those measured directly by ventral aortic flowprobes in salmonids (Thorarensen et al. 1996b; Gallaugher et al. 2001; Beaumont et al. 2003) and other active (i.e. non-sedentary) teleosts such as the northern squawfish *Ptychocheilus oregonesis* (Kolok and Farrell 1994a, 1994b) of a similar size and at similar temperatures (10–15°C). The fact, however, that seabass exhibit much greater aerobic scope and U_{crit} at temperatures above 20°C (Claireaux and Lagardère 1999; G. Claireaux, unpublished observations) indicates that they may also have much greater cardiac scope and maximum Q at such temperatures. There is preliminary evidence to indicate that this is indeed the case at 20°C (A. Chatelier, unpublished observations).

In many of the active teleost species studied to date, both M_{O2} and Q plateau as the fish approaches U_{crit} (Kiceniuk and Jones 1977; Kolok and Farrell 1994a; Thorarensen et al. 1996b; Gallaugher et al. 2001). This has been proposed as circumstantial evidence that maximum cardiac output is linked to, and may limit, maximum O_2 uptake and aerobic exercise performance in these species (Farrell 2002). This plateau effect occurred in the seabass, and visual observation of the ventral aortic probe trace revealed that both f_H and Q became extremely irregular as the fish approached U_{crit} and started the intermittent “burst and coast” swimming pattern that indicates recruitment of anaerobic white muscle (Day and Butler 1996) and which precedes fatigue. Thus, cardiac performance may limit AMR and U_{crit} in the seabass.

In most teleost species studied to date, increases in V_{SH} are responsible for at least 50% of the increase in Q observed during exercise (reviewed by Farrell and Jones 1992; also Kolok and Farrell 1994a, 1994b; Thorarensen et al. 1996a, 1996b; Gallaugher et al. 2001) whereas, in the seabass, 90% of the increase in Q was attributable to increased f_H. The tunas have often been cited as one of the few teleost groups that accomplish exercise-related increases in Q almost exclusively through increased f_H (Farrell 1996; Brill and Bushnell 2001). It has been suggested that this is because they have extremely high V_{SH} under routine conditions and so have little scope to increase it during exercise (Farrell 1996; Brill and Bushnell 2001). The routine V_{SH} of the seabass was not, however, exceptionally high, being only some 20% higher than the routine values reported for salmonids at similar temperatures (e.g. Taylor et al. 1996; Thorarensen et al. 1996b; Gallaugher et al. 2001). However, routine f_H of the seabass was some 35% lower than resting values for salmonids reported in these previous studies (e.g. Taylor et al. 1996; Thorarensen et al. 1996b; Gallaugher et al. 2001). In tunas, regulation of f_H during exercise appears to occur exclusively as a result of relaxation of inhibitory vagal cholinergic tone, whereas in other teleosts it occurs both by this mechanism and by increased adrenergic stimulation (Farrell and Jones 1992; Brill and Bushnell 2001). Investigating the pharmacology of cardiac control during exercise would be an interesting area for future research in the seabass.

Conclusions

The seabass possesses an exceptional capacity for homeostatic regulation of plasma osmolality and tissue

water balance following changes in water salinity. This adaptation allows them to maintain the performance of their skeletal and cardiac muscle, and so would allow the animals to negotiate facultative migrations through waters of widely differing salinities. At 14°C, seabass exercise performance, and underlying cardiac performance, are similar to those reported for other teleosts of a similar size and at similar temperatures. The seabass increased Q during exercise almost exclusively through increases in f_H, with only a minor change in V_{SH}. This cardiac response to exercise is similar to that of the tunas but different from that of most other fishes, in which the predominant modulation is of V_{SH}.

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