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REVIEW

Metabolic Recovery from Exhaustive Exercise in Rainbow Trout

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ABSTRACT. Exercise to exhaustion results in severe metabolic, acid-base, and endocrine disturbances to fish. Recovery metabolism from this type of activity may place limits on performance, since the time requirements for restoration of high energy stores (e.g. glycogen, high energy phosphates) will ultimately determine the frequency of maximal performance. This article reviews the types of metabolic, acid-base, and endocrine disturbances associated with exhaustive exercise in rainbow trout, the mechanism(s) of recovery, and their regulation. In particular, the roles of catecholamines and cortisol in regulating recovery metabolism are explored. COMP BIOCHEM PHYSIOL 113A;1:51–60, 1996.

KEY WORDS. Glycogenesis, lactate, muscle, cortisol, exercise, trout

WHAT IS EXHAUSTIVE EXERCISE?

Exercise is a pervasive force in the lives of all animals. The ability to sprint or sustain high levels of activity may determine whether an animal acquires energy or becomes acquired energy. Given that the capacity for excercise is highly variable both within and between species and is heritable, it is a system which lends itself to study by comparative physiologists to investigate such questions as factors that may be limiting to performance (see 7 for a full discussion).

In fish, exercise which leads to exhaustion involves short bouts of high intensity swimming that is primarily powered by white muscle fibers and supported by anaerobic metabolism. Fish are considered exhausted when they are no longer capable of burst activity though they may be able to sustain swimming at slow speeds. During this type of activity many physiological and biochemical systems approach their limits, which in turn, may limit exercise performance. Recovery metabolism may be an important process in setting limits to performance since the frequency of maximal performance is ultimately set by the time required for recovery, in particular, restoration of energy reserves (e.g., high energy phosphates, glycogen). Exercise to exhaustion therefore, can be a useful model system in which to study regulatory processes, one of which is addressed in this review article: metabolic regulation during recovery from exhaustive exercise.

There are several advantages offered by a fish model to the study of metabolic recovery from exhaustive exercise compared to mammals. For example, the metabolic disturbances associated with exhaustive exercise are greater than in mammals (see Figs. 1 and 2) and require a longer time to be corrected (2). Such long recovery times facilitate the study of the recovery process and its regulation. Furthermore, fish skeletal muscle is highly homogeneous, consisting of mainly white fibers and constitutes the bulk of the fish's body mass (50–60%; 5). In high intensity burst exercise, with recruitment of white muscle, most of the fish's body is involved and therefore, metabolic recovery in white muscle would account for the majority of whole body metabolism post-exercise. In contrast, only a relatively small proportion of the mammalian body mass is recruited during burst exercise. Thus, studying metabolic recovery patterns and regulation in fish white muscle will provide insights into the whole body recovery response.

Several methods have been used to exercise fish to exhaustion. The most commonly used technique is to chase fish around a tank for a fixed period of time (usually 5–10 min.) until they are no longer able to sprint (e.g., 49,30,47,38,15,20,53,54). This method has been frequently criticized because it combined an element of "fright" or "stress" associated with the chasing, which is separate and distinct from the exercise. To eliminate the "fright" associated with chasing, another approach used is to exercise fish to exhaustion by forcing them to swim against a current in a swim tunnel until they are no longer able to maintain station, which usually occurs within 30–45 min. (e.g., 12,43). A third method of exhausting fish is angling them at the end of a hook and line until they are "played out," exhausting the fish within 1–2 min. (e.g., 44,8). This latter method is perhaps the most "stressful" to the fish since it combines exercise, "fright" and some degree of air exposure. However, this type of exercise, and the physiological responses to it, are relevant to the life of many fish, particularly the salmonids which are

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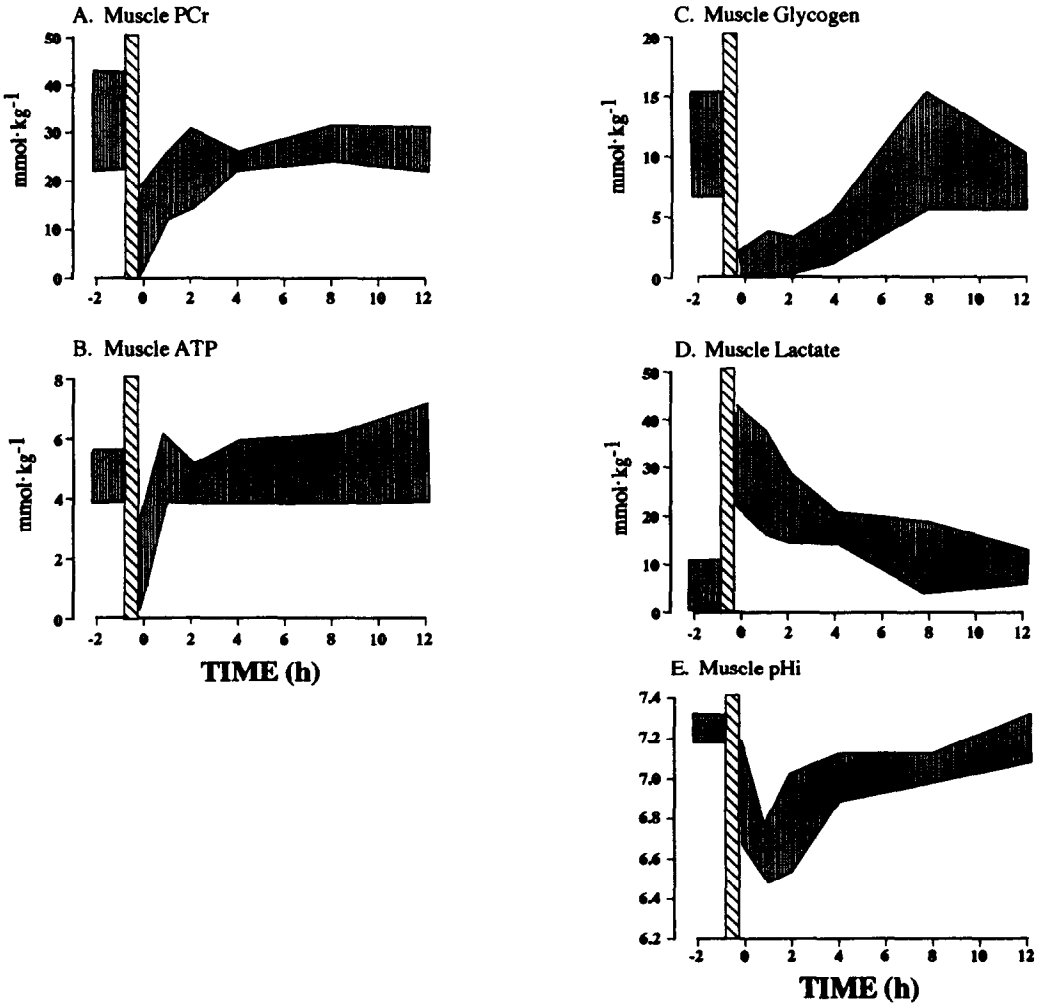

FIG. 1. Typical changes in muscle phosphocreatine (PCr; A), adenosine triphosphate (ATP; B), glycogen (C), lactate (D) and pHi (E) following exhaustive exercise in rainbow trout. Hatched bar represents period of exercise (5–7 min of manual chasing). Shaded area indicates range of values. Data reported are from 29, 46, 15, 20, 53, 54.

an important sports fish. Although seemingly very different, each method of exhaustive exercise results in similar types of metabolic and acid-base disturbances. This review article focuses on the metabolic and acid-base disturbances associated with exhaustive exercise induced by chasing fish around a tank mainly because there are more data available for this form of exercise than any other.

Rainbow trout (*Oncorhynchus mykiss*) are the most frequently studied fish species in terms of exercise physiology, and consequently, there is more information about the exercise response of this species than any other. Rainbow trout, like other salmonids, are, in many respects, a high performance fish. They exhibit some impressive exercise feats: swimming upstream against substantial currents and jumping up waterfalls during their reproductive migration (56). Thus, they make them a good model in which to examine the question of metabolic regulation and the potential limits recovery from exhaustive exercise places upon performance.

METABOLIC, ACID-BASE AND ENDOCRINE CHANGES ASSOCIATED WITH EXHAUSTIVE EXERCISE

The types of metabolic and acid-base disturbances associated with exhaustive exercise have been described in detail by others, most recently by Wang *et al.* (53), and are summarized in Figs. 1 and 2.

In the early stage of exercise the energy for muscular contraction is provided by hydrolysis of phosphocreatine (PCr). Muscle PCr levels tend to decline, though the extent of depletion is somewhat variable (Fig. 1A). Some studies report drops of only 40% (53) and others (29,12,43), as much as 90%. Similarly, muscle ATP levels also decline (Fig. 1B), though again, the

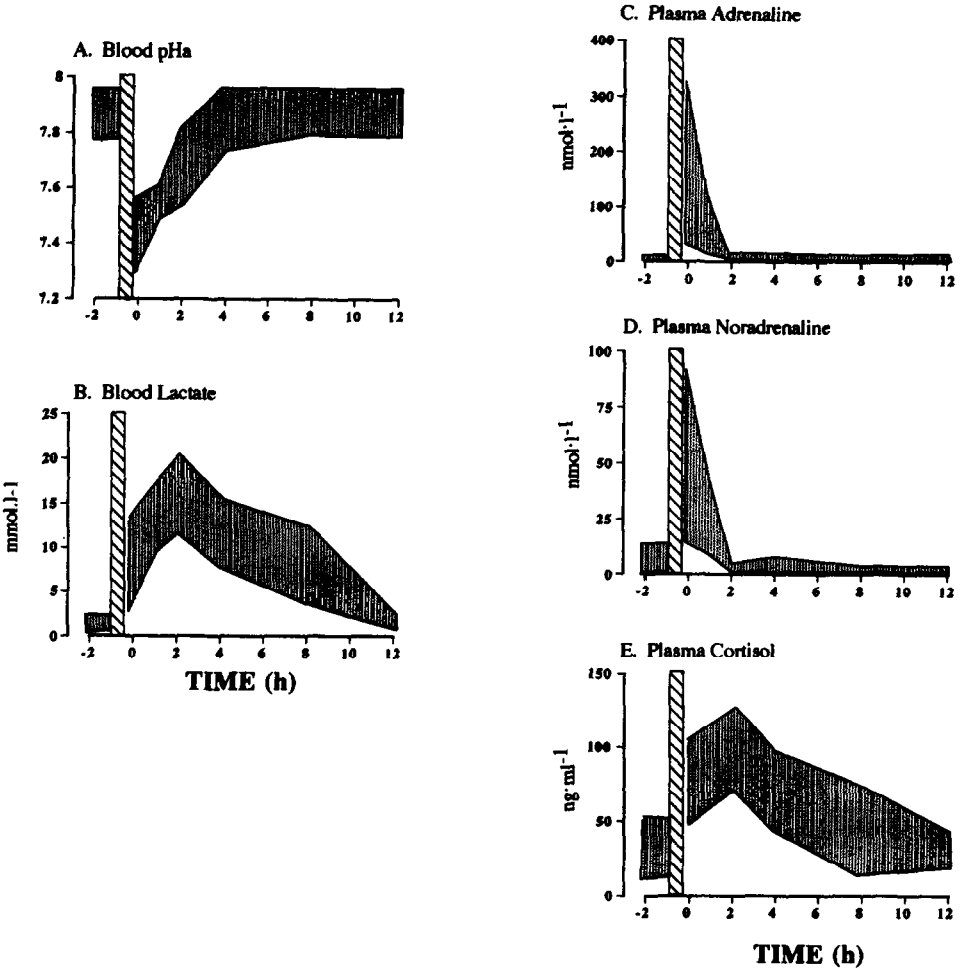

FIG. 2. Typical changes in blood pHa (A), lactate (B) and plasma levels of adrenaline (C), noradrenaline (D) and cortisol (E) following a bout of exhaustive exercise in rainbow trout. Data reported are from 40, 30, 35. Other details as in the legend of Fig. 1.

extent of the decline is variable. Some of this variability may reflect differences in the amount of exercise performed by the fish in various studies and/or differences in sampling protocol. Both PCr and ATP pools are highly labile and difficult to preserve during sampling and analysis; therefore, it is difficult to obtain true estimates of *in vivo* levels (54). In response to the reductions in PCr and ATP levels, glycogenolysis is activated (12,37) and glycogen levels are reduced by as much as 90% (Fig. 1C). Concomitant with the reduction in muscle glycogen is an accumulation of lactate (Fig. 1D) and H^+, the latter reflected by a drop in pHi (Fig. 1E).

Some of the acid and lactate produced in the muscle enters the blood space, resulting in a drop in pHa (Fig. 2A) and increase in blood lactate (Fig. 2B). The nadir in blood pHa is seen immediately after exercise and is of both respiratory and metabolic origin. Blood pHa is restored to rest levels by about 4 hr as acidic equivalents are excreted by the gills (for a review, see 18). Lactate appears in the blood space rather slowly, not reaching peak levels until about 2 hr into recovery and then returns to pre-exercise values by about 8 hr (Fig. 2B).

Few studies have examined the endocrine responses to exhaustive exercise, and even fewer hormones have been considered; glucagon, insulin, T_3, T_4 cortisol and catecholamines. Only the latter two hormones have been studied to any extent probably because they are relatively easy to measure since piscine-specific RIA's are not required. The effects of exercise on plasma catecholamines (Fig. 2C, D) and cortisol (Fig. 2E) have been reasonably well documented (e.g., 40,29,47,35). The general pattern emerging is that the rise in plasma catecholamines is immediate and short-lived, whereas the response of plasma cortisol is delayed and more prolonged. The highest levels of adrenaline and noradrenaline are seen immediately after exercise, with adrenaline levels tending to be 2–3 times those of noradrenaline. Both noradrenaline and adrenaline return to rest levels by about 2 hr. Plasma cortisol levels, on the other hand, do not increase until 1–2 hr post-exercise, and stay elevated through 4–6 hr. These differences in time course probably reflect the fact that the catecholamines are released from storage vesicles, whereas cortisol is not stored, but rather is synthesized *de novo*, then released (39).

Other hormones that are involved in regulation of intermediary metabolism, insulin, glycagon and the thyroid hormones

(T_3 and T_4), have only recently been investigated in terms of their potential roles in post-exercise metabolic recovery. In the single study which have measured them, circulating insulin and glucagon levels did not change in response to exhaustive exercise (35). The authors attribute this lack of responsiveness to the fact that there were no corresponding changes in plasma glucose levels, which are thought to be a factor regulating insulin and glucagon levels (19). A difficulty in assigning a potential regulatory role to the thyroid hormones is that the response of circulating levels to exhaustive exercise is highly variable, both within and between studies. For example, Himick and Eales (18a) report that circulating T_4 levels increase in response to exercise, and suggest the T_4 increase is associated with an increase in plasma glucose levels. Pagnotta *et al.* (35), however, saw no consistent effect of exercise on circulating T_4 levels, which may be correlated to fact that they also saw no consistent response of plasma glucose to exercise. A note of interest is that when the rise in plasma cortisol was blocked with metyrapone treatment prior to exercise, circulating T_4 levels increased after exercise and remained elevated through to 12 hr (35). This observation suggests that there is a link between cortisol and the regulation of circulating T_4 levels and supports the theory that products of the pituitary-interrenal axis inhibit the pituitary-thyroid axis in fish (42,41,22). In mammals, thyroid hormones are known to play a permissive role in the glycogenolytic effects of glucagon and catecholamines and in the glucose uptake effect of insulin (23), but their roles in metabolic regulation in fish are not well understood (22).

GLYCOGEN RESYNTHESIS AND THE FATE OF LACTATE

A question which has been the focus of much research in the field of fish exercise physiology is how and where and how is glycogen resynthesized and what is the metabolic fate of lactate? The early work of Meyerhoff (24,25) on frog skeletal muscle suggested that skeletal muscle can resynthesize glycogen from lactate *in situ*. Subsequent work by others (e.g., 3a,6,23a) using radiolabelled lactate has demonstrated *in situ* glycogen synthesis from lactate in isolated amphibian and mammalian skeletal muscle. While these studies indicate that skeletal muscle is capable of lactate based *in situ* glycogenesis, there is some question as to its physiological significance, particularly in mammals (9). It is clear that vertebrate skeletal muscle is not capable of gluconeogenesis, releases lactate to the blood and at least in mammals, can utilize blood glucose as a precursor for glycogen synthesis. For some time, the latter was thought to be the major route for muscle glycogen synthesis in mammals and was described by the Cori cycle: lactate produced in the muscle is transported to the liver, via the blood, where it is converted into glucose which, in turn, is released to the blood and utilized by the muscle to support glycogen synthesis (33).

The Cori cycle has little, if any, physiological role in glycogen resynthesis in skeletal muscle in fish and other lower vertebrates (16,17,60). Rather, muscle glycogen resynthesis occurs *in situ* using lactate as the primary substrate. There are several lines of evidence from fish which support this notion. Following exhaustive exercise 80–85% of the total lactate produced is retained within the muscle (49,29,36) and its clearance is coincident with glycogen replenishment (see Fig. 1C, D). In addition, *in vivo* lactate turnover rates are not adequate to account for the observed rates of lactate clearance from muscle (11,57,28). The most compelling evidence in support of the idea that Cori cycle activity is low in fish comes from *in vivo* radiotracer studies showing that glucose utilization is extremely low (36,58) and that blood-borne glucose is not readily taken up by skeletal muscle and makes little contribution to muscle glycogen resynthesis (36,59; Table 1). The primary fate of the blood lactate is oxidative, most likely utilized by such aerobic tissues as cardiac and red muscle (4,21,26). However, some blood lactate reappears first in the muscle lactate pool then, in the glycogen pool (27). This most likely represents direct uptake of lactate from the blood space by the muscle. Nothing is known about the mechanism and regulation of lactate transport across the muscle in fish.

While it is now generally accepted that muscle glycogen synthesis occurs via lactate-based *in situ* glycogenesis, the route of glycogen resynthesis from lactate and the energy source(s) are less clear. The great unknown is the pathway

TABLE 1. Contribution of blood glucose and lactate to muscle glycogen resynthesis during recovery from exhaustive exercise in rainbow trout

Hours after exercise	ΔGlycogen ($\mu mol \cdot g^{-1}$)*	% Resynthesized from blood glucose*	% Resynthesized from blood lactate†
0–2	2.23	0.1	6.2
2–4	0.51	0.1	47.8
4–6	1.62	0.4	3.7

*From 39.
†From 30.

by which phosphoenol pyruvate (PEP) is produced from pyruvate. In hepatic gluconeogenesis this step is catalyzed by pyruvate carboxylase and phospho-enol pyruvate carboxykinase (PEPCK), however activity of these enzymes is not detectable in fish muscle (32). There are two other possible pathways for production of PEP from pyruvate: reversal of pyruvate kinase and/or malic enzyme plus PEPCK. Evidence in favour of either pathway is lacking. Given that PEPCK is not readily detectable in fish muscle, with the notable exception of marlin (45), it seems unlikely that this pathway could be of physiological importance. Moyes *et al.* (31) have put forward a very compelling argument that muscle glycogenesis proceeds by reversal of pyruvate kinase. Pyruvate kinase activity in the reverse direction has not been measured directly in fish, but measurements from rabbit muscle (13) suggest pyruvate kinase reversal is a plausible scheme. In most species, PK activity in skeletal muscle are high enough that only a limited reversal (e.g., <2% of the forward rate) would be adequate to explain muscle glycogenesis.

Although fish white muscle is considered a lipid-poor tissue, in terms of capacity for both storage and oxidation, there is recent evidence suggesting that fatty acid oxidation makes a significant contribution to energy production both during exercise and the recovery process. Milligan and Girard (27) report large, though highly variable decreases in total muscle lipid, which persists through 6 hr of recovery. More convincing evidence comes from Wang *et al.* (53) who report increases in white muscle acetyl-CoA, acetyl-carnitine and short-chain acyl-carnitine levels and a decrease in free carnitine levels throughout the 4 hr post-exercise recovery period. These metabolite changes are consistent with an increase in fatty acid oxidation. The suggestion that lipid is used as an energy source both during exhaustive exercise and recovery is not unique to fish, but has been documented in human skeletal muscle as well. The relative contribution of lipids to white muscle metabolism during and after exercise has not been quantified in fish, but in humans lipid oxidation was estimated to contribute as much as 50% to the post-exercise muscle oxygen consumption. Fatty acid oxidation by muscle would inhibit pyruvate dehydrogenase, thus pyruvate and lactate oxidation; the net result would be the channeling of lactate towards glycogen synthesis (31).

Lactate-based *in situ* muscle glycogenesis represents a carbohydrate-sparing strategy, which is not unique to fish but rather appears to be the rule in ectotherms. By retaining the lactate within the muscle and preferentially channeling it towards glycogen, fish are able to "quantitatively" recycle the lactate carbon, which would not be possible with hepatic-based gluconeogenesis (see Fig. 3). The transfer of lactate from muscle to liver via the blood, as required by the Cori cycle, would mean that a significant amount of lactate could be used by other tissues and lost to muscle glycogenesis. Similarly, any glucose produced from muscle lactate could meet fates other than muscle glycogenesis. Thus, limiting the amount of lactate that enters the blood space minimizes the "leakage" of

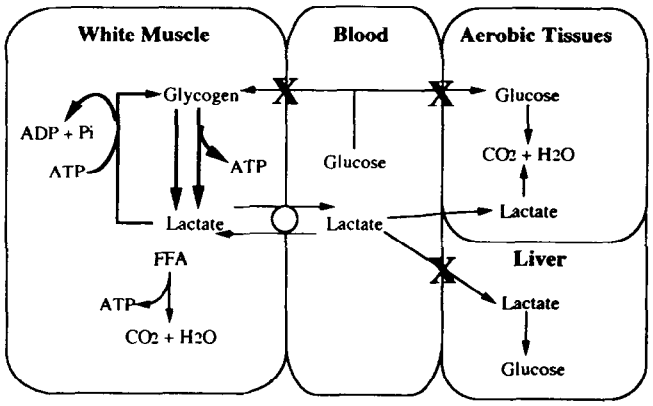

FIG. 3. Schematic diagram describing the quantitative recycling of lactate by muscle following exercise in rainbow trout. The width of the lines indicate the relative magnitude of the various pathways. FFA = free fatty acids. Modified from 9, 17. See text for further details.

lactate carbons and facilitates muscle glycogen resynthesis. The observation that the plugging of one of these "leaks" by hepatectomy results in faster restoration of muscle glycogen (27) lends support to this argument. Furthermore, studies on glucose dynamics in swimming fish (58,59,60) indicate that glucose does not make a significant contribution to white muscle energy metabolism, probably because of the low capacity of fish muscle to take up glucose.

Thus, in fish, the "lactate shuttle," originally proposed by Brooks (9) in which the main fate of muscle lactate is oxidation by aerobic tissue is not important. Furthermore, Gleeson's "ectothermic lactate shuttle," described for reptiles, in which white muscle lactate becomes red muscle glycogen (17) is also not relevant to fish. Rather, the major fate of lactate in fish muscle is *in situ* glycogenesis, supported in part by lipid oxidation. A model summarizing these events is shown in Fig. 3.

ENDOCRINE REGULATION OF THE RECOVERY PROCESS

As noted above, exhaustive exercise results in a mobilization of catecholamines and cortisol into the plasma. The role of these hormones in integrating the metabolic response to and recovery from exhaustive exercise is just beginning to be understood.

Catecholamines

Defining the regulatory role(s) of the catecholamines in the post-exercise recovery process has been problematic. Attempts to block the actions of adrenaline and noradrenaline prior to exercise have been unsuccessful because fish treated with either α- or β-adrenergic antagonists do not swim (C. L. Milligan, unpublished observations). This clearly indicates that

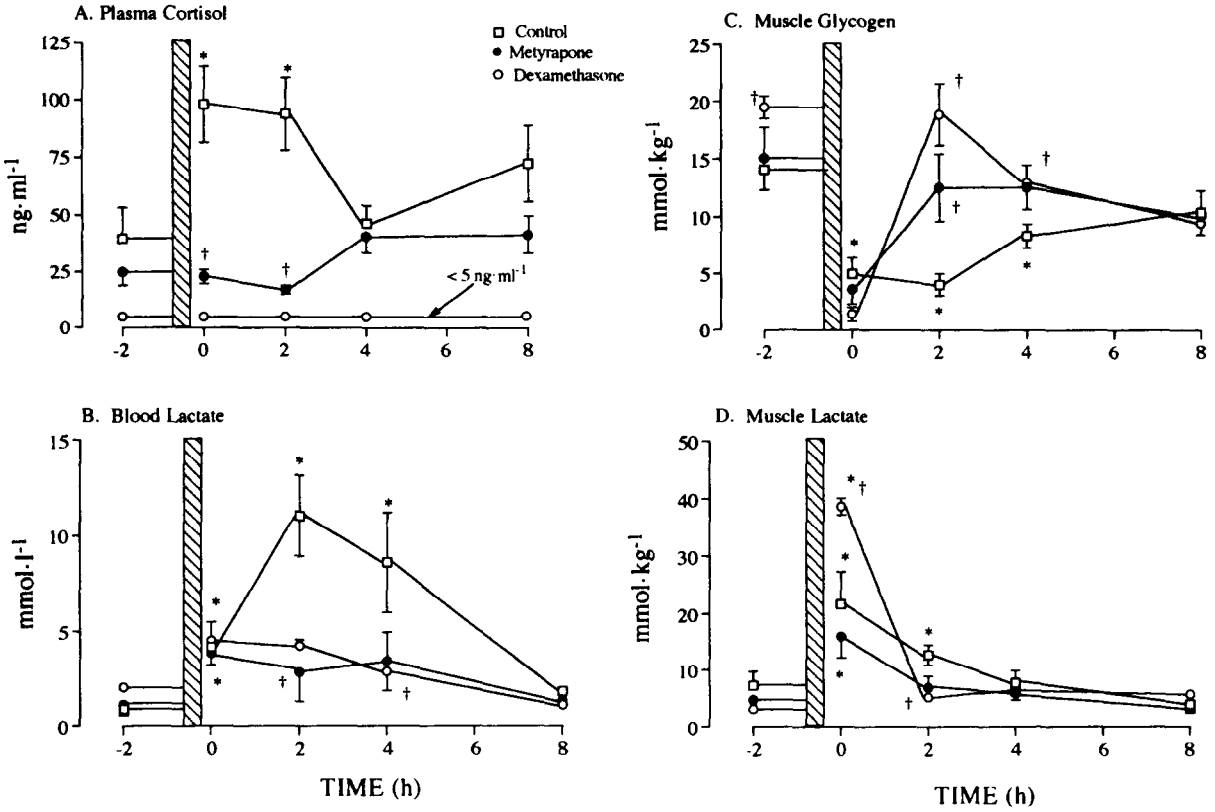

FIG. 4. The effects of dexamethasone treatment 24 hr prior to exercise (o) metyrapone treatment 1 hr prior to exercise (•) or saline treatment (□) on (A) plasma cortisol, (B) blood lactate, (C) muscle glycogen and (D) muscle lactate levels. Note: plasma cortisol levels in dexamethasone-treated fish were not detectable (≤5.0 ng · ml^{-1}). −2 hr indicates sample taken 2 hr prior to exercise, bar indicates 5 min exercise period and 0 indicates immediately after exercise. Mean ± 1 SEM. * Indicates a significant difference ($p < 0.05$) from corresponding −2 hr (i.e., rest) value. † Indicates a significant difference ($p < 0.05$) from corresponding value in saline treated fish. Data from 35.

catecholamines have an important role in coordinating the swimming response.

Adrenergic blockade after a bout of exercise has been a more productive experimental approach. The best documented regulatory role for catecholamines is the regulation of red cell pHi and thus, oxygen transport during periods of extracellular acidosis (34). Another post-exercise regulatory role for catecholamines was suggested by Wardle (55) in a study on plaice in which β-adrenergic blockade after exercise altered blood lactate dynamics; blood lactate levels were higher in fish treated with the β-adrenergic antagonist propranolol, compared to control fish. These results suggested to Wardle that catecholamines were involved in lactate retention in muscle. However, attempts to repeat these experiments by others has met with mixed results. Neither Wood and Milligan (61) nor van Dijk and Wood (51) were able to demonstrate a regulatory role for catecholamines in post-exercise blood lactate or H^+ dynamics in either starry flounder or rainbow trout, respectively. However, Tang *et al.* (48) showed that in rainbow trout, β-adrenergic blockade after exercise slowed recovery of blood pH and reduced H^+ excretion at the gills, but blood lactate dynamics were unaffected. Interestingly, post-exercise β-adrenergic stimulation enhanced recovery of blood pH, without altering branchial H^+ excretion or blood lactate dynamics. The differences between the results of Tang *et al.* (48) and those of van Dijk and Wood (51) may be due to the fact that the latter used a dose of propranolol (a β-adrenergic antagonist) 10 times that used by Tang *et al.* (48).

Based upon the available evidence it would appear that the catecholamines have an important role in coordinating the exercise response and the regulation of post-exercise blood and red cell acid-base balance. It is not clear if catecholamines have any influence on the movement of lactate across the muscle membrane or in regulating post-exercise lactate and glycogen metabolism. The regulation of muscle lactate and glycogen metabolism as well as metabolite transport across the muscle cell membrane are areas ripe for further research.

Cortisol

Cortisol has been implicated as having a regulatory role in teleost intermediary metabolism (50), though its role in post-

TABLE 2. Effects of cortisol replacement post-exercise in trout treated 1 hr prior to exercise with the cortisol synthesis blocker metyrapone

Time (h)	Plasma cortisol ng · ml^{-1}			Blood lactate mmol · l^{-1}		
	S	M	M+C	S	M	M+C
−2	37.6 ± 7.6	28.1 ± 7.4	20.6 ± 6.6	0.7 ± 0.1	1.2 ± 0.2	1.4 ± 0.2
−0.5	23.6 ± 5.4	13.8 ± 2.4	12.3 ± 2.1	0.7 ± 0.2	1.7 ± 0.2	1.7 ± 0.4
0	22.4 ± 4.8	13.8 ± 2.21	20.8 ± 4.7	4.05 ± 0.7*	3.3 ± 0.6*	3.8 ± 0.4*
2	94.4 ± 13.9*	11.75 ± 1.7†*	159.4 ± 45.6*	13.6 ± 2.34*	3.1 ± 0.8*†	10.5 ± 1.5*

Values reported are means ±1 SEM (n = 5 for each group). S = saline treatment followed by saline infused controls; M = metyrapone treatment followed by saline infusion; M+C = metyrapone treatment followed by cortisol infusion. −2 hr is 2 hr prior to exercise and metyrapone treatment, −0.5 is 0.5 hr prior to exercise and 0 represents immediately after exercise. *significantly different from corresponding −2 hr value; †significantly different from corresponding value in saline-treated fish. Data from 14.

exercise metabolism has only recently been investigated. Most studies to date have focused on the metabolic consequences to long term, or chronic elevations in cortisol (e.g., 1) with little attention paid to short-term or acute situations. The period of recovery from exhaustive exercise falls into the latter category. To investigate what role cortisol, if any, is playing after exercise, Pagnotta *et al.* (35) blocked the exercise-induced rise in plasma cortisol by either inhibiting its synthesis with metyrapone or preventing its release with dexamethasone treatment (Fig. 4). Preventing the rise in plasma cortisol altered blood lactate dynamics in trout post-exercise (Fig. 4). In treated fish, blood lactate levels did not increase in the usual fashion; blood lactate was not higher than 4–5 mmol/l and had returned to pre-exercise levels within 2 hr. Also, muscle glycogen was restored and lactate was cleared within 2 hr in treated groups compared to the 8 hr required for controls (Fig. 4). These observations suggest that the increase in cortisol after exercise exerts a negative influence on lactate and glycogen recovery metabolism; recovery in fish without the cortisol proceeds more quickly than in fish with cortisol. These data suggested that cortisol was somehow regulating lactate metabolism. To confirm this hypothesis, Eros (14) conducted a replacement experiment. Fish were treated with metyrapone to inhibit cortisol synthesis, exercised and then one group of fish were infused with cortisol to mimic the exercise-induced rise and the other group received saline. Replacing cortisol in metyrapone-treated fish returned the recovery profile to that of controls (Table 2). Taken together, these observations clearly indicated that increasing plasma cortisol after exercise has negative consequences to the fish, in terms of metabolic recovery. The mechanism(s) by which cortisol is exerting this negative effect is not clear, though recent studies on the regulation of skeletal muscle glycogenolysis in rats may suggest a mechanism. Coderre *et al.* (10) found that in resting rats, epinephrine alone was not adequate to stimulate the activation of glycogen phosphorylase and inhibition of glycogen synthase; hence glycogenolysis, rather glucocorticoids were also required. In exercising muscle, however, exercise itself was enough to stimulate glycogenolysis, presumably via activation of glycogen phosphorylase and inhibition of glycogen synthase by alterations in adenylates and ion levels (e.g., increases in Ca^{2+}, ADP, AMP and decreases in ATP and PCr). These observations suggest that at least in resting muscle, glucocorticoids exert a "permissive" effect on the action of catecholamines. In fish muscle post-exercise, muscle adenylates have returned to rest levels by 2 hr into recovery (Fig. 1A, B) and plasma catecholamines are similarly lowered (Fig. 2C, D), however, plasma cortisol is still elevated (Fig. 2E). This continued elevation of cortisol through to 4–6 hr post-exercise may exert an inhibitory effect on glycogen synthase, thus preventing restoration of muscle glycogen. Previously, it was speculated that correction of pHi is limiting to muscle glycogen restoration (52); that is, muscle pHi must be restored to a level compatible with glycogenesis. Transfer of H^+ from the muscle to the extracellular space then to the water via the gills is mechanism for the initial correction of pHi (18). However, the results of Pagnotta *et al.* (35) and Eros (14) tend to suggest that it is the continued elevation of plasma cortisol which is limiting to restoration of muscle glycogen post-exercise.

There is additional experimental evidence which provides support for the notion that the rise in cortisol is limiting to lactate and glycogen metabolism post-exercise. In a series of experiments designed to examine the consequences of aerobic swimming on recovery from exhaustive exercise, B. Hooke and C. L. Milligan (unpublished observations) found that in fish swimming at 1 bl · s^{-1} post-exercise, there was no rise in plasma cortisol (Fig. 5A), blood lactate levels did not increase to the same extent as seen in fish in still water and had returned to rest levels more quickly (Fig. 5B) as did blood pH (Fig. 5C). Whether the lower blood lactate levels during active recovery are due to enhanced clearance of lactate from the blood by aerobic tissues (e.g., red and cardiac muscle), or enhanced lactate metabolism in the muscle is not clear. A similar phenomenon has been observed in humans where it is suggested that the lower blood lactate during active recovery is due to elevated muscle lactate metabolism.

Whatever the explanation, these results also suggest that the exhaustive exercise per se may not be the causative factor in the mobilization of cortisol but rather what the fish per-

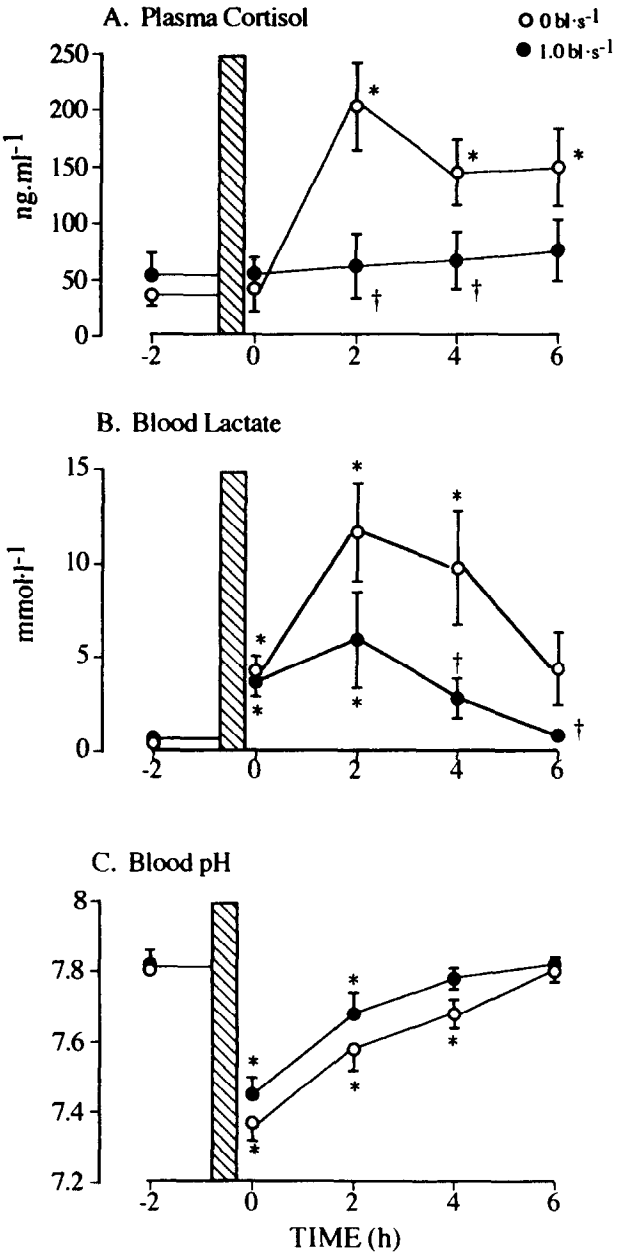

FIG. 5. The influence of aerobic swimming following exhaustive exercise on plasma cortisol (A), blood lactate (B) and blood pHa (C). In this series of experiments, fish were exhausted by chasing them around a circular tank. Fish were then placed in either a Vogel-type swim chamber and allowed to swim at 1 body length per second (●; 1 bl · s^{-1}) during the entire 6 hr recovery period or placed in a chamber of similar size but without current (o; 0 bl · s^{-1}). −2 hr indicates 2 hr prior to exercise and the hatched bar represents the 5 min period of exhaustive exercise. Values reported are means ±1 SEM, $n = 6$ for each group. * Indicates a significant difference ($p < 0.05$) from corresponding −2 hr value; † indicates a significant difference ($p < 0.05$) from corresponding value in 0 bl · s^{-1} group. Data from B. Hooke and C. L. Milligan (unpublished observations).

ceives as stressful is the still water post-exercise. If this in fact proves to be the case, then what has been considered the typical post-exercise recovery profile may not be. Instead, what we and others have been reporting over the years may be confounded by a stress response; the stressful event not being the exercise to exhaustion but rather, the lack of activity following exercise.

SUMMARY AND CONCLUSIONS

The types of metabolic and acid-base changes associated with exhaustive exercise in a high performance fish, such as rainbow trout, are well defined. However, our understanding of the mechanism of recovery and the factors involved in its regulation is still cursory. Ultimately, it is recovery from a bout of exhaustive exercise which may set limits to overall exercise performance: the longer a fish takes to recover, the less frequent are the bouts of high intensity exercise. We know nothing about the role of metabolite transport across membranes in the recovery process, nor how these processes are regulated. Using *in vitro* models (e.g., muscle cell culture and sarcolemmal vesicle preparations) to build upon *in vivo* systems, such as questions could be readily addressed.

The observation that aerobic swimming during recovery from exhaustive exercise alters what has been considered the "standard" recovery pattern suggests that we may have to re-evaluate the work done in this field and alter the definition of a "resting" trout.

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