

# Physiological Effects of Brief Air Exposure in Exhaustively Exercised Rainbow Trout (*Oncorhynchus mykiss*): Implications for "Catch and Release" Fisheries

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Rainbow trout (*Oncorhynchus mykiss*) which were air exposed for 60 s after exhaustive exercise initially had a much larger extracellular acidosis than trout which were only exercised. In both groups, however, plasma pH returned to normal by 4 h. Blood lactate concentrations were also greater in the air-exposed fish and continued to increase throughout the experiment. During air exposure, there was retention of carbon dioxide in the blood, and oxygen tension (PO<sub>2</sub>) and hemoglobin:oxygen carriage (Hb:O<sub>2</sub>) both fell by over 80%. After 30 min of recovery, however, blood gases resembled those in fish which were only exercised. Finally, survival after 12 h was 10% in control fish and 88% in the exercised fish but fell to 62 and 28% in fish which were air exposed for 30 and 60 s, respectively, after exercise. These results indicate that the brief period of air exposure which occurs in many "catch and release" fisheries is a significant additional stress which may ultimately influence whether a released fish survives.

Des truites arc-en-ciel (*Oncorhynchus mykiss*) exposées à l'air pendant 60 s après une activité physique épuisante ont présenté, tout d'abord, une acidose extracellulaire beaucoup plus élevée que celles qui n'avaient pas été exposées à l'air. Dans les deux groupes, toutefois, le pH plasmatique est revenu à la normale dans les quatre heures suivantes. La concentration de lactate sanguin était également plus élevée chez les poissons exposés à l'air, et elle a continué à augmenter tout au long de l'expérience. Pendant l'exposition à l'air, on a enregistré une rétention de dioxyde de carbone dans le sang, la tension en oxygène (PO<sub>2</sub>) et le transport de la molécule d'hémoglobine oxygénée (Hb:O<sub>2</sub>) diminuant tous deux de plus de 80%. Cependant, après 30 min de repos, les gaz sanguins se situaient à des niveaux comparables à ceux des poissons n'ayant pas été exposés à l'air. Finalement, le taux de survie après 12 h était de 100% chez les témoins et de 88% chez les poissons ayant été soumis à des exercices épuisants, mais il a chuté à 62 et à 28% chez ceux qui ont été exposés à l'air pendant 30 et 60 s respectivement après ces exercices. Ces résultats indiquent que, dans de nombreux cas de pêche avec remise à l'eau des prises, la courte période pendant laquelle les truites sont exposées à l'air représente un stress supplémentaire important, qui peut en fin de compte, avoir une incidence sur la survie des poissons relâchés.

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An integral component of the management strategy in commercial and recreational fisheries is the release of a significant portion of the catch. In commercial fisheries, this may include species caught out of season or individuals which do not meet size restrictions. In recreational fisheries, "catch and release" policies have also been implemented to offset the impact of increased angling pressure on limited fish stocks. For example, recreational fishermen on Canada's east coast must now release all multi-sea-winter salmon (i.e. over 63 cm in length) and all smaller salmon over and above the daily or seasonal limit. Similar legislative restrictions apply to a diversity of species throughout North America (Barnhart 1989). Furthermore, in a number of sport fisheries, individuals as well as tournament organizers are promoting the live release of fish even in the absence of legislation.

Fish caught either by commercial or recreational methods often struggle to the point of complete exhaustion. Black (1957a, 1957b, 1957c, 1958) has shown that a significant percentage of these fish may die from the ordeal. Death does not

occur immediately, but often well into the recovery period. While other investigators have documented similar mortalities in exhaustively exercised fish (Bouck and Ball 1966; Beggs et al. 1980; Graham et al. 1982; Wood et al. 1983), some studies indicate that exhaustive exercise is not associated with significant mortality (Wydoski et al. 1976; Tufts et al. 1991). It is clear nonetheless that the period of exhaustive exercise associated with angling or struggling in commercial fishing gear results in a significant physiological disturbance in a fish (Wood and Perry 1985). Furthermore, complete recovery is not guaranteed simply because the exhausted fish is eventually released.

In both commercial and recreational fisheries, exhaustive exercise is often followed by a brief period of air exposure prior to release. During this time, the gill's delicate lamellae will collapse and gas exchange may be largely inhibited. The importance of this additional stress on the disturbance associated with exhaustive exercise and on the process of recovery has not previously been investigated. The purpose of the present study was therefore to examine the additive effect of brief air exposure on the physiological disturbance associated with exhaustive exercise in the rainbow trout (*Oncorhynchus mykiss*). In view

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of the rapidly growing importance of catch and release policies in the management of fisheries, it is hoped that this study will provide some insight into an additional stress which has often been overlooked, but which may ultimately influence the survival of released fish.

## Methods

### Animals

Freshwater-adapted rainbow trout (300- to 500-g males and females) were obtained from a local supplier and were maintained for at least 1 mo prior to experiments in dechlorinated Kingston tap water (8–10°C). The animals were fed to satiation every other day with commercially prepared trout pellets. At least 2 d prior to surgery, feeding was halted and the animals were transferred to an acclimation tank at the experimental temperature (15°C).

### Surgery

The fish were anaesthetized in a 3-aminobenzoic acid ethyl ester (MS-222, Sigma) – NaHCO<sub>3</sub> – dechlorinated tap water mixture (1 : 2 : 10 000, w/w) prior to surgery. During surgery, the gills were continuously irrigated with a MS-222–NaHCO<sub>3</sub> mixture (10<sup>-1</sup> the original concentration). The dorsal aorta was cannulated with PE 50 tubing (Clay Adams) filled with heparinized (20 U·mL<sup>-1</sup>, Sigma) freshwater teleost saline (Hoar and Hickman 1983). Following the 5- to 10-min surgical procedure, animals were revived and placed in blackened perspex chambers with flowing dechlorinated tap water (15°C) where they remained for 24–48 h of recovery. This procedure allows the collection of dorsal aortic blood from unrestrained animals (Smith and Bell 1964).

### Experimental Protocol

Following recovery, an 800-μL blood sample was removed from the resting fish with a Hamilton gas-tight syringe. The fish was then moved to a cylindrical tank where it was exhaustively exercised by manual chasing. After about 10 min, the fish would no longer respond to chasing and the exercise period was terminated. At this point, another blood sample was removed and the fish was returned to the blackened perspex box. Additional blood samples were taken 30, 60, and 240 min after exercise. A second group of fish was subjected to a similar protocol. However, these fish were moved to a damp cloth for 60 s immediately following the exercise period and the second blood sample was removed at the end of this brief period of air exposure. Finally a third group of control fish was subjected to a similar blood sampling procedure but was not exercised or air exposed.

Survival of fish was recorded after 12 h. For this data set, an additional group of fish was exercised and air exposed for 30 s but no blood samples were taken.

### Analyses

True plasma pH (extracellular pH, pH<sub>e</sub>) was determined with a PHM 73 pH meter and associated micro-pH unit (Radiometer, Copenhagen, Denmark) thermostatted to 15°C. Blood plasma was separated from the corpuscular component by 2 min of centrifugation in an Eppendorf centrifuge. Oxygen partial pressure in whole blood (P<sub>O<sub>2</sub></sub>) was measured with an E5046 oxygen electrode (Radiometer) thermostatted to 15°C and an associated oxygen meter (Cameron Instrument Co., Texas,

USA). A similar oxygen electrode was used to determine the total oxygen content (TO<sub>2</sub>) of blood by the method of Tucker (1967). Total carbon dioxide contents (TCO<sub>2</sub>) of whole blood and plasma were measured with a Corning model 965 CO<sub>2</sub> analyzer (CIBA Corning Canada Inc.). Hemoglobin (Hb) content of blood samples was measured by Drabkin's method using Sigma reagents and procedures (Sigma Bulletin No. 525). Whole-blood lactate concentrations were determined on neutralized perchloric acid extracts by the method of Lowry and Passonneau (1972). Measured values of true plasma total carbon dioxide and pH<sub>e</sub> were used to calculate PCO<sub>2</sub> and true plasma bicarbonate concentration ([HCO<sub>3</sub><sup>-</sup>]<sub>tpl</sub>) via a rearrangement of the Henderson–Hasselbach equation with the values for pK' and αCO<sub>2</sub> determined according to Boutilier et al. (1984). The concentration of metabolic protons added to the plasma (Δ[H<sup>+</sup>]<sub>m</sub>) over any given time period (e.g. time 1 to 2) was calculated according to McDonald et al. (1989) using the following equation:

$$\Delta[\text{H}^+]_m = [\text{HCO}_3^-]_{\text{tpl},1} - [\text{HCO}_3^-]_{\text{tpl},2} - \beta(\text{pH}_{e,1} - \text{pH}_{e,2})$$

where β is the nonbicarbonate buffer value of true plasma.

### Statistics

Two sample Student *t*-tests (unpaired) were employed to determine the significance (*p* < 0.05) of differences observed between treatment groups. A one-way ANOVA was followed, where appropriate, by Dunett's multiple comparisons test to determine significance (*p* < 0.05) between resting and recovery values within groups. All values are presented as the mean ± 1 SE.

## Results

One minute of air exposure following exhaustive exercise promotes more severe acid–base disturbances than does exercise alone. In trout which were exercised, the pH<sub>e</sub> fell by 0.239 pH unit whereas in trout which were also air exposed, the

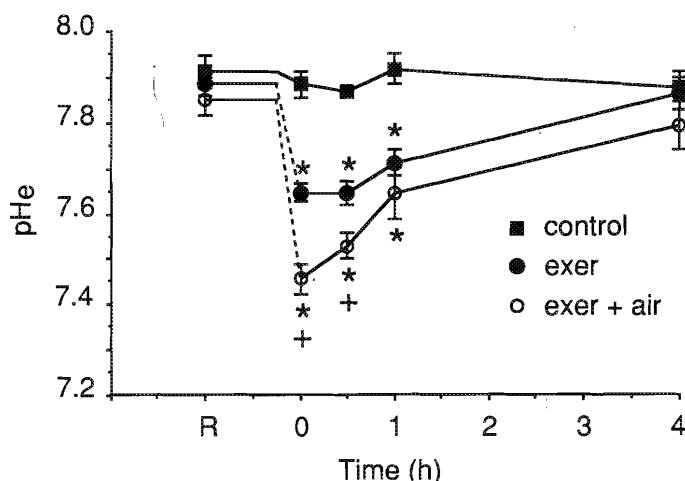


FIG. 1. Extracellular pH (pH<sub>e</sub>) in rainbow trout at rest (R) and after 0, 0.5, 1, and 4 h under control conditions (■), following exhaustive exercise (●), or following exhaustive exercise plus 60 s of air exposure (○). Values are means ± SE (control, N = 6; exercise, N = 8; exercise + air, N = 7). An asterisk denotes a significant difference from the resting value. A plus sign denotes a significant difference between exercise and exercise + 60 s of air exposure.

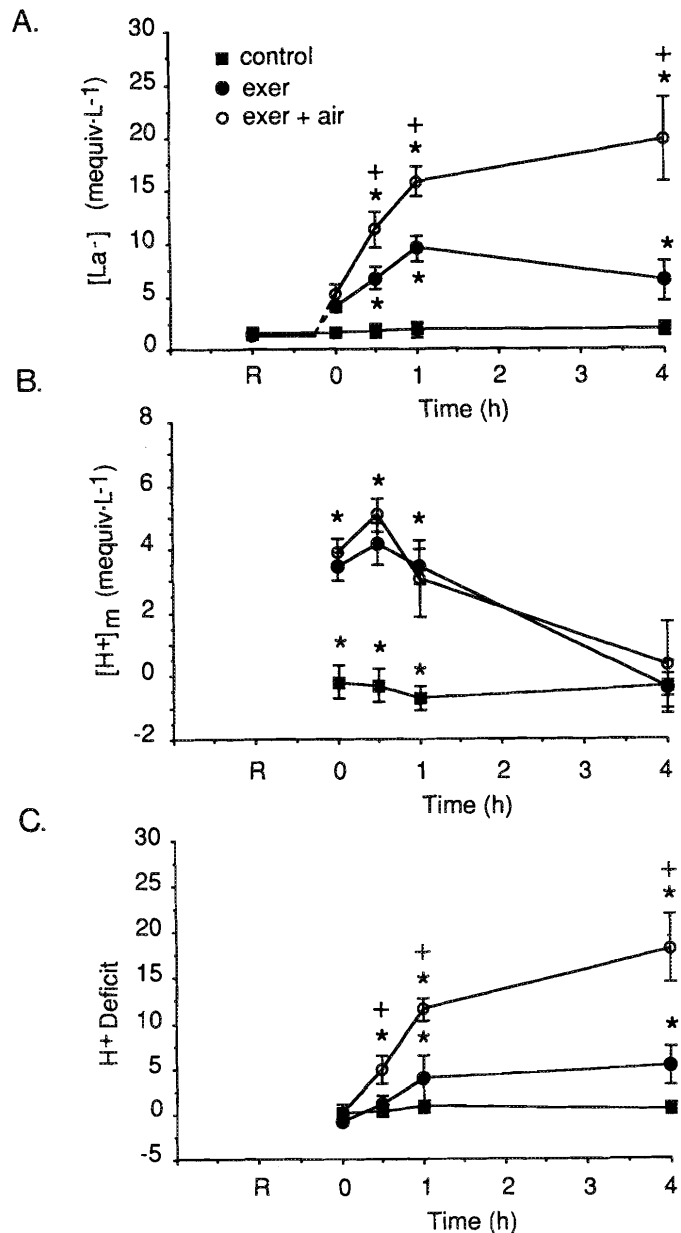


FIG. 2. (A) Blood lactate concentration ( $[La^-]$ ), (B) metabolic proton load ( $[H^+]_m$ ), and (C) proton ( $H^+$ ) deficit in rainbow trout at rest (R) and after 0, 0.5, 1, and 4 h under control conditions (■), following exhaustive exercise (●), or following exhaustive exercise plus 60 s of air exposure (○). Values are means  $\pm$  SE (control,  $N = 6$ ; exercise,  $N = 8$ ; exercise + air,  $N = 7$ ). An asterisk denotes a significant difference from the resting value. A plus sign denotes a significant difference between exercise and exercise + 60 s of air exposure.

decrease in  $pH_e$  was 0.396 pH unit (Fig. 1). The  $pH_e$  remained significantly lower in the air-exposed group until the 1-h sample. By 4 h, the  $pH_e$  of both groups had returned to values which were no longer significantly different from those at rest.

A large increase in blood lactate was observed following exhaustive exercise as well as exhaustive exercise followed by brief air exposure (Fig. 2A). In the latter, the amount of lactate accumulated was significantly greater than that seen following exercise alone. The peak blood lactate in the exercise group was  $9.6 \pm 1.3$  mequiv·L<sup>-1</sup> and occurred after 1 h of recovery. In contrast, the blood lactate in the air-exposed fish continued to increase throughout the experiment and the maximal concentration of  $19.8 \pm 3.9$  mequiv·L<sup>-1</sup> was observed at 4 h.

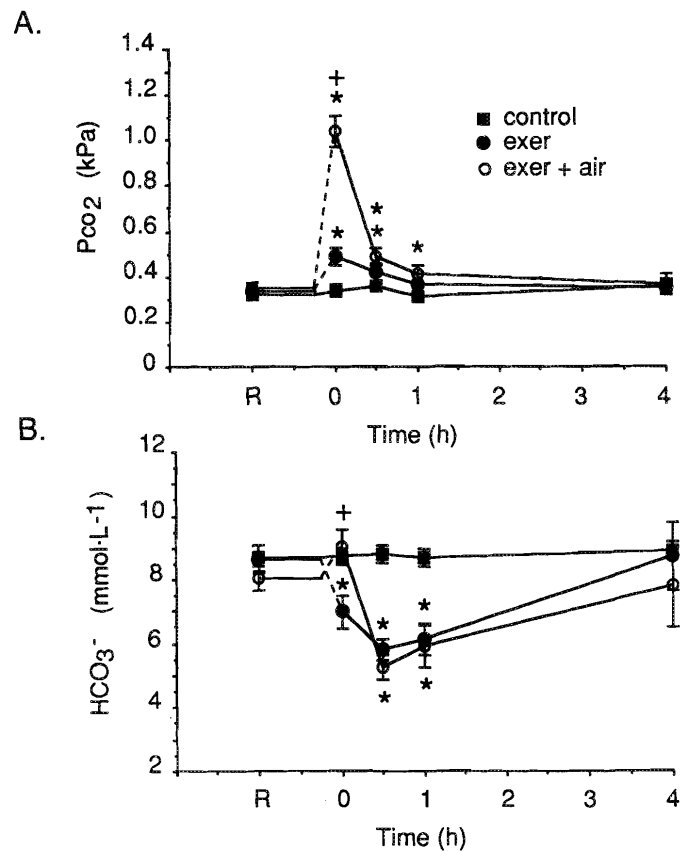


FIG. 3. (A)  $CO_2$  tension ( $PCO_2$ ) and (B) bicarbonate concentration ( $[HCO_3^-]$ ) in rainbow trout at rest (R) and after 0, 0.5, 1, and 4 h under control conditions (■), following exhaustive exercise (●), or following exhaustive exercise plus 60 s of air exposure (○). Values are means  $\pm$  SE (control,  $N = 6$ ; exercise,  $N = 8$ ; exercise + air,  $N = 7$ ). An asterisk denotes a significant difference from the resting value. A plus sign denotes a significant difference between exercise and exercise + 60 s of air exposure.

There were no significant differences in the blood metabolic proton loads of the two groups throughout the recovery period (Fig. 2B). Thus, due to the greater blood lactate concentrations, the air-exposed fish had a much greater proton deficit during the recovery period (Fig. 2C).

The acidosis following exercise also contains a respiratory component (Fig. 3). Although there is a significant reduction in plasma  $TCO_2$  immediately after exercise, there is a 44% increase in  $PCO_2$  (Fig. 3A). After air exposure, however, there is a significant increase in  $TCO_2$  and a much greater increase (200%) in  $PCO_2$ . Air exposure after exercise also causes a significant increase in plasma  $[HCO_3^-]$  rather than the observed decrease in fish which were only exercised (Fig. 3B). Following 30 min of recovery, there were no longer any significant differences between these groups of fish, and by 4 h, these variables had returned to resting values.

Blood  $PO_2$  was significantly reduced by 28% after exhaustive exercise, but there were no significant changes in hemoglobin:oxygen carriage ( $Hb:O_2$ ) (Fig. 4).  $PO_2$  returned to resting values after 1 h. In contrast, these variables were reduced by 82 and 87%, respectively, when the fish were also exposed to air for 1 min. Again, after 30 min of recovery in water, there were no significant differences between the two groups.

Exercise and brief exposure to air after exercise both had an impact on survival of the fish during the next 12 h (Fig. 5). Survival after 12 h ranged from 100% in the control fish to 28%

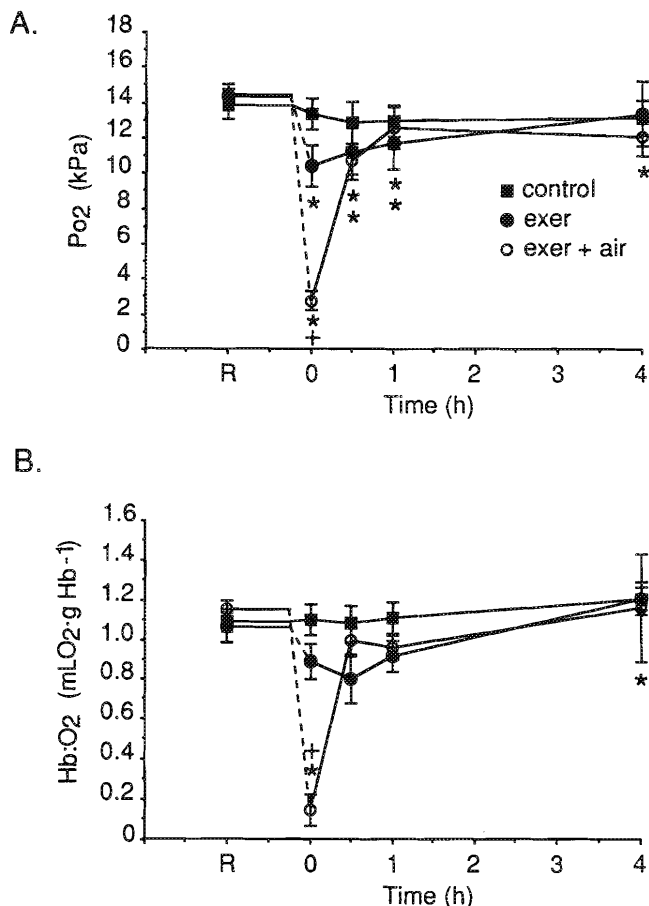


FIG. 4. (A) Blood oxygen tension ( $P_{O_2}$ ) and (B) hemoglobin:oxygen carriage ( $Hb:O_2$ ) in rainbow trout at rest (R) and after 0, 0.5, 1, and 4 h under control conditions (■), following exhaustive exercise (●), or following exhaustive exercise plus 60 s of air exposure (○). Values are means  $\pm$  SE (control,  $N = 6$ ; exercise,  $N = 8$ ; exercise + air,  $N = 7$ ). An asterisk denotes a significant difference from the resting value. A plus sign denotes a significant difference between exercise and exercise + 60 s of air exposure.

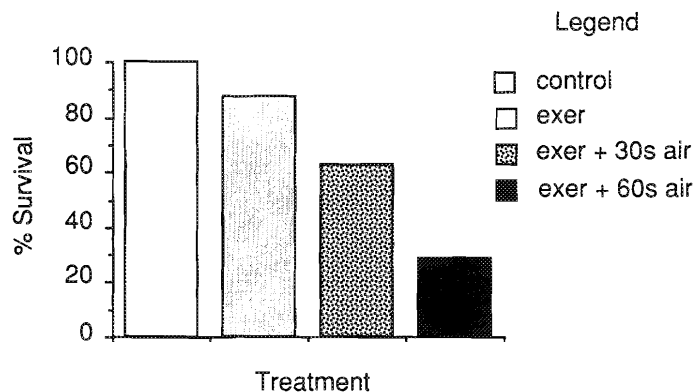


FIG. 5. Survival of rainbow trout 12 h following control conditions, exhaustive exercise, exhaustive exercise plus 30 s of air exposure, and exhaustive exercise plus 60 s of air exposure.

in the fish which were exposed to air for 60 s after exercise. When the period of air exposure was reduced to 30 s, survival increased to 62%, and in fish which were only exercised, survival was 88%.

## Discussion

Physiological responses to exhaustive exercise have been well described in a number of fish species (e.g. reviewed in Wood and Perry 1985; Heisler 1986). The magnitude of the disturbance we observed in exhaustively exercised rainbow trout and the dynamics of the recovery process were very similar to that documented by previous investigators (Holeton et al. 1983; Turner et al. 1983; Milligan and Wood 1986; Primmitt et al. 1986; McDonald et al. 1989; Tang et al. 1989). However, the impact of brief air exposure on exhausted fish has not been examined previously; thus, the following discussion will focus primarily on this aspect.

In many commercial fisheries, fish which have struggled to exhaustion during capture are routinely exposed to air for sorting and identification prior to release. Similarly, in recreational fisheries, exhausted fish may be exposed to air for photos, measurements, or weighing before release. Indeed, this is common practice during angling contests and tournaments which have become very popular in recent years.

As demonstrated in previous studies, exhaustive or "burst" exercise in rainbow trout is associated with a considerable extracellular acidosis (Fig. 1). In fish which were also air exposed, the fall in plasma pH was much greater. After exhaustive exercise, the extracellular acidosis is normally composed of both a metabolic and a respiratory component (Wood and Perry 1985; Heisler 1986). The metabolic acidosis is caused by anaerobic production of lactate in the poorly perfused white muscle and subsequent release of the associated protons into the extracellular fluid. Our results indicate that the production of lactic acid is probably greater in fish which are briefly air exposed after exhaustive exercise (Fig. 2A). In the air-exposed fish, the blood lactate concentration was higher and it continued to increase throughout the experiment. Thus, even 60 s of air exposure following exhaustive exercise appears to cause a much greater degree of anaerobiosis within the muscle. Despite the higher plasma lactate concentrations, however, the metabolic proton load in the plasma of the air-exposed fish was not significantly different from that of the exercised group (Fig. 2B). Consequently, the proton deficit was larger in the air-exposed group. (Fig. 2C). This suggests either that there was a greater excretion rate of protons in the air-exposed fish after they were returned to water and/or that a significant portion of the metabolic proton load remained within the muscle. These two possibilities cannot be differentiated in the present experiments. However, according to Wood et al. (1983), mortality after exhaustive exercise is probably caused by the extent of the intracellular acidosis within the muscle. Thus, the increased mortality in the air-exposed fish may be evidence that a significant portion of these metabolic protons were retained within the muscle (Fig. 5). Clearly, further study into the muscle acid-base status of air-exposed fish is warranted and may explain the observed differences in survival.

In teleost fish, the majority of gas and ion transfer takes place across the delicate secondary lamellae which are aligned on the gill filaments. These lamellae are largely supported by the water flowing between them and, with few exceptions, the lamellae of most species will collapse if exposed to air (Boutillier 1990). Our results indicate that this reduction in respiratory surface area upon air exposure causes an almost complete inhibition of gas transfer across the gills in the rainbow trout (Fig. 3 and 4). In exhaustively exercised fish which remain in water, there is a significant reduction in the  $TCO_2$  of plasma wherein bicarbonate is titrated by metabolic protons to form carbon dioxide

which is excreted (Fig. 3). This results in a transient increase in  $\text{PCO}_2$  and a reduction in plasma bicarbonate after exhaustive exercise (Turner et al. 1983; Wood and Perry 1985; Heisler 1986; Milligan and Wood 1986; McDonald et al. 1989) (Fig. 3). However, in fish which are also air exposed following exhaustive exercise,  $\text{TCO}_2$  significantly increases, indicating that gas transfer is largely inhibited while the animals are in air. The marked rise in blood  $\text{PCO}_2$  during air exposure also explains the significantly greater reduction in blood pH (Fig. 1). Upon return to water, normal gill function is restored and the dynamics of carbon dioxide excretion are not significantly different between the two groups of fish (Fig. 3).

The large reduction in the oxygen content of blood during air exposure is possibly the most critical effect of exposing exhausted fish to air. This reduction was not attributable to any differences in hematocrit or total blood hemoglobin concentration but can be explained by the 81% reduction in  $\text{PO}_2$  and associated 87% reduction in the amount of oxygen bound to hemoglobin after 60 s of air exposure (Fig. 4). The normal physiological responses of an exhaustively exercised fish combine to enhance oxygen transport to the tissues to compensate for the increased oxygen requirements immediately following exercise (Nikinmaa et al. 1984; Wood and Perry 1985; Primmatt et al. 1986; Milligan and Wood 1987). Our results indicate, however, that if the fish is even briefly exposed to air immediately after exhaustive exercise, the tissues will be temporarily deprived of oxygen during this critical period. Indeed, the large difference in plasma lactate concentration created by the air exposure is probably evidence of the detrimental effects of brief air exposure on the tissue metabolism of exhausted fish (Fig. 2A).

The issue of mortality in exhaustively exercised fish is an important management concern in catch and release fisheries. Black (1957a, 1957b, 1957c, 1958) originally demonstrated that significant mortality may occur in exhaustively exercised fish. Similar results have been obtained by a number of other investigators (Bouck and Ball 1966; Beggs et al. 1980; Graham et al. 1982; Wood et al. 1983). In contrast, there is also considerable evidence that mortality from exhaustive exercise is possibly very minimal in many catch and release fisheries (Wydoski et al. 1976; Barnhart 1989; Tufts et al. 1991). Differences in observed mortality may be due to a large number of variables which will undoubtedly be the focus of a great deal of study as the importance of both commercial and recreational catch and release fisheries continues to increase. In this regard, our study clearly demonstrates differences in mortality between exhaustively exercised fish and those which were also briefly exposed to air (Fig. 5). In fact, only 28% of those fish which were exposed to air for 60 s after exercise survived the next 12 h as compared with 88% of those fish which were only exercised. In each case, the extracellular acid-base status of the fish initially appeared to be returning toward normal, but the animals died between 4 and 12 h later. This "delayed mortality" has been observed by other investigators and, in the wild, could give the false impression that released fish always survive (Black 1958; Wood et al. 1983).

The purpose of the present experiments was not to predict actual percentages of mortality in the wild when exhausted fish are briefly exposed to air. The use of hatchery fish and repetitive blood sampling may have influenced our results in this regard. On the other hand, our results clearly indicate that the brief period of air exposure which commonly occurs in many catch and release fisheries is an important additional stress in an exhausted fish and may ultimately have a significant impact

on the number of released fish which survive. Finally, as the importance of catch and release fisheries continues to increase, fisheries managers may wish to place greater emphasis on the proper handling of exhausted fish.

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