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Changes in blood cortisol, glucose and lactate in carp retained in anglers' keepnets

T. G. POTTINGER

NERC Institute of Freshwater Ecology, Windermere Laboratory, Far Sawrey, Ambleside, Cumbria, LA22 0LP

Tel: 015394 42468 fax: 015394 46914 Email: t.pottinger@ife.ac.uk

Running title: physiological effects of keepnet confinement

Capture of carp from holding tanks by dip-net, or from semi-natural conditions by rod and line, elicits a physiological stress response characterised by elevation of plasma cortisol levels. The transfer of carp to keepnets subsequent to capture does not increase or reduce the magnitude or duration of this response and in both cases plasma cortisol levels have returned to pre-stress levels within 24 hours of the initial disturbance. The post-capture plasma cortisol elevation is accompanied by disturbances in plasma glucose and lactate levels but these are less consistent in severity and duration than the cortisol response. These data suggest that the retention of fish in keepnets following capture, does not represent a source of stress additional to that imposed by capture and has no effect on the rate of recovery of the fish from the initial capture stress.

Keywords: carp, *Cyprinus carpio*, keepnets, angling, stress, physiology

INTRODUCTION

The effects of angling practices on fish which are returned to the water following capture are of significance to the agencies responsible for management of fisheries and the framing of related legislation (Hickley *et al*., 1995; Maitland, 1995) but the impact of angling on fish has not been thoroughly investigated. Critical points of the angling process include capture, hook removal, general handling following capture, and confinement of fish within keepnets subsequent to capture. Physical and physiological factors of concern include the extent of exertion during playing and landing, the physical effect of hooking and handling the fish, exposure of fish within keepnets to harmful deterioration in water quality, and the possible initiation of a physiological stress response consequent to the combined effects of capture, handling, restraint and confinement. Some information is available regarding the welfare of individual fish and the effects of angling pressure on populations of fish (Brana *et al*., 1992; Brobbel *et al*., 1996) and there are limited data available concerning mortality arising from hook damage (Bugley & Shepherd, 1991; Dedual, 1996; Dextrase & Ball, 1991; Muoneke & Childress, 1994; Malchoff & Heins, 1997) and the recovery of rod-caught fish post-capture (Ferguson & Tufts, 1992; Pankhurst & Dedual, 1994; Wilkie *et al*., 1996). However, the species and conditions employed in these studies are not strictly relevant to United Kingdom non-salmonid fisheries, in which catch-and-return angling is prevalent. Furthermore, the use of keepnets is more common in Europe than North America and to date there are few data available which allow any firm conclusions to be reached regarding the effects of their use.

Because of the susceptibility of keepnet use to legislative control interest has recently focused on factors associated with their use. Raat *et al*. (1997) demonstrated that the long-term growth

and mortality rates of cyprinid fish were unaffected by retention for up to 4 h in keepnets. It has also been demonstrated that although water quality may deteriorate significantly within keepnets during the confinement of fish, resulting in reduced levels of dissolved oxygen and increased levels of ammonia and carbon dioxide, the changes do not exceed the limits of tolerance of freshwater fish (Pottinger, 1997) even with above-average loadings of fish and micromesh (2mm mesh diameter) nets.

The physiological effects of keepnet confinement have not been examined. The physiological stress response, although initiated as an adaptive response to destabilising factors, can have damaging effects on fish if prolonged (Barton, 1997; Barton & Iwama, 1991). The aim of the present study was to establish to what extent keepnet confinement may be considered stressful to fish by quantifying several physiological indices of stress in fish exposed to a combination of capture and subsequent confinement within a keepnet, or capture and immediate release. The species utilised for this study, the common carp, *Cyprinus carpio* L. is typical of the cyprinid species widely sought by anglers in the United Kingdom.

MATERIALS AND METHODS

FISH

Common carp (*Cyprinus carpio* L.) were purchased from Humberside Fisheries (Cleaves Farm, Skerne, Driffield, Yorkshire) and maintained in partially covered 1500 l circular glassfibre tanks, each supplied with a constant flow (10 l min^{-1}) of lake water at ambient temperature. The fish were fed 5 times weekly on commercial feed (BP Mainstream) *ad libitum*. All experimental manipulations were carried out in this tank system.

EXPERIMENTAL PROCEDURE

Experiment 1: A range-finding study to determine the time-course of changes in the determinands following capture and confinement of carp

One hundred and twenty common carp (body weight 568 ± 22 g; length 27.6 ± 0.3 cm; n = 30, mean ± SEM) were distributed evenly between three holding tanks (A, B, C) and allowed to acclimate for three weeks. Water temperature during the experiment was 4^{o}C. At time 0 five fish were netted from tank B into a bucket containing anaesthetic (2-phenoxyethanol, 1:2000, Sigma). Blood samples were collected within 2 minutes of capture from the caudal vessels of each fish using heparinized syringes and transferred to polypropylene tubes on ice. The fish were returned to a recovery tank and took no further part in the procedure. The 35 fish remaining in tank B were transferred into a 50 l glassfibre trough containing water and after five minutes were returned to their holding tank ("netted"). The crowding, physical exertion and aerial exposure associated with this procedure were intended to mimic the nature of disturbances associated with capture by rod and line. Five fish were removed from tank C and blood sampled. The remaining 35 fish were transferred to a 50 l trough and after 5 minutes were placed in two keepnets (2.5 m length x 0.4 m diameter, 6mm mesh) suspended in the holding tank, 0.75 m immersed ("keepnet confined"). Five fish were also sampled from tank A but the 35 fish remaining in the holding tank were not disturbed further ("controls"). One hour after the original disturbance, five further fish were removed from each group and blood-sampled. At 4 h after the initial disturbance the fish confined within keepnets were released into their holding tanks. Further blood samples were taken 4, 24, 48 and 168 hours after the start of the experiment. Blood samples were spun down (at 4^{o}C) within 1 h of collection and plasma was aspirated and frozen at -20^{o}C until required for assay.

Experiment 2: To determine the time-course of changes in plasma cortisol, lactate and glucose levels in small carp subjected to simulated capture alone, or capture combined with a period of keepnet confinement.

Six hundred carp were distributed between 15 tanks (40 fish per tank; body weight 44.9 ± 1.9 g; length 11.6 ± 0.2 cm; n = 30, mean ± SEM). Holding conditions were as described for Expt. 1. Water temperature was 15^{o}C. The fifteen tanks were designated as controls (1-5), netted (1-5), or keepnet-confined (1-5). At time 0, six fish were netted from tank 1 within each treatment group, anaesthetized and blood-sampled as for Experiment 1. The forty fish in each of the remaining netted or keepnet-confined tanks were netted and transferred to 50 l troughs. After five minutes, fish from the netted group were returned to their holding tanks, while the fish from the keepnet-confined group were transferred to keepnets (see Expt. 1) suspended within their holding tanks. Fish in the remaining four control tanks were not disturbed. At 4 h after the initial disturbance the fish confined within keepnets were released into their holding tanks. At 1, 4, 24, 48 and 168 h samples of six fish were taken from one control (undisturbed) tank, one netted tank and one keepnet-confined tank. The 168h sample was removed from the same tanks from which the 1h sample had been taken.

Experiment 3: To determine the time-course of changes in plasma cortisol, lactate and glucose levels in large carp subjected to simulated capture alone, or capture combined with a period of keepnet confinement.

A second time-course experiment was carried out using large carp (body weight 505 ± 30 g; length 27.4 ± 0.4 cm; n = 30, mean ± SEM). Sixty fish were distributed evenly between

twelve holding tanks, five fish per tank. The experimental procedure was identical to that for Experiment 2, except that limited numbers of fish dictated that the 24 h and 48 h samples were omitted. Five fish were sampled at each time point. Water temperature during the experiment was 15^{o}C.

Experiment 4: To determine the time-course of changes in plasma cortisol, lactate and glucose levels in carp captured from a semi-natural environment by rod and line and transferred to a keepnet.

This experiment was carried out at a purpose-built earth lagoon, 50m x 30m x 1.5m containing carp bred for re-stocking purposes. Batches of ten common carp (mirror variety; mean weight 120 ± 15 g, n = 40) were captured from a single lagoon by two anglers in quick succession and either immediately anaesthetised and blood-sampled, or placed in keepnets (see Expt. 1) and blood-sampled at 1, 2, 3, 4 or 5 h post-capture. Blood samples were treated as described above and plasma was stored frozen until required for assay. Water temperature during the study was 8^{o}C.

ANALYTICAL PROCEDURES

Plasma cortisol levels were determined by radioimmunoassay following the methodology described in Pickering *et al*. (1987). Plasma glucose and lactate levels were determined spectrophotometrically (Sigma Diagnostics procedures no. 510 and no. 735, respectively). Data were subjected to analysis of variance (ANOVA, Genstat) with treatment (control, netted, keepnet-confined) and time of sample as factors. The data were log-transformed where necessary, to improve homogeneity of variance.

RESULTS

EXPERIMENT 1

Experiment 1 was carried out to provide information on the general time-course and extent of changes to be expected in carp following capture and confinement. Although the experimental design incorporated repeat sampling from the same tanks the data are included here because of the general paucity of information on this species. Levels of plasma cortisol prior to the start of the experiment were slightly, but significantly ($P<0.05$) higher in the keepnet designated group (26 ± 8 ng ml^{-1}; mean ± SEM, n = 5) than in the controls (11 ± 3) or netted (9 ± 3) groups (Fig. 1a). There was a significant increase in plasma cortisol levels in both netted and keepnet-confined groups relative to the controls within 1 hour of the initial disturbance. Levels in both groups were highest 4 hours after netting/confinement, reaching 271 ± 31 in the keepnet-confined fish and 217 ± 31 in the netted fish, both being significantly higher ($P<0.001$) than the controls (76 ± 11) but not different from each other. Within 24 hours of the initial disturbance, levels in both netted (42 ± 8) and keepnet-confined groups (43 ± 8) were statistically indistinguishable from levels in the controls (39 ± 8). Although there were some differences between groups at 48 hours these, although significant, were small compared to the elevations observed at 1 and 4 hours. There were no differences between groups at 168 hours. The moderate rise in plasma cortisol levels within the control group during the first four hours of the study was probably due to repeated disturbance associated with sampling.

There was considerable variation in plasma glucose levels within treatment groups in fish

prior to the start of the experiment (Fig. 1b). At time 0, plasma glucose levels in the keepnet-confined group were significantly (P<0.05) lower than those in the control and netted groups. Overall, there was no significant effect of treatment. However, there was a significant elevation of plasma glucose in the keepnet-confined group (8 ± 0.8 mM) relative to the control (5.1 ± 0.8) and netted groups (5.9 ± 0.6) at 4 and 24 hours after the onset of disturbance. There were no significant differences between the three groups at 48 or 168 hours.

There were no significant differences in blood lactate levels between groups prior to the onset of disturbance (Fig. 1c). A highly significant elevation of plasma lactate levels in both the netted (15.5 ± 2.9 mM) and keepnet-confined groups (13.9 ± 0.4) compared to the control group (3.0 ± 0.2) occurred within 1 hour of the onset of disturbance. Although levels in both handled groups had declined markedly by 4 hours, they were still significantly higher than levels in the control group (P<0.01, P<0.001). There was no significant difference in lactate levels between the handled groups. At 24, 48 and 168 hours only minor differences in lactate levels were apparent.

EXPERIMENT 2

Baseline plasma cortisol levels in undisturbed carp at time 0 in this experiment were within a similar range to those in Experiment 1 (10 - 50 ng ml^{-1}; Fig. 2a). A significant (P<0.001) elevation in plasma cortisol was observed within 1 hour of disturbance in both netted (435 ± 52 ng ml^{-1}; n = 6) and keepnet-confined groups (448 ± 75) compared to the undisturbed controls (74 ± 4). Significantly (P<0.001) elevated levels of plasma cortisol were maintained in both handled groups at 4 hours relative to the control group but by 24 hours after the onset

of disturbance there was only a minor, though significant (P<0.01, 40 ± 6) elevation in the keepnet-confined fish compared to the control fish (16 ± 4). The difference in cortisol levels between the two stressed groups was not significant at any time point. There were no differences between groups at 48 and 168 hours.

Plasma glucose levels were higher in keepnet-confined fish than in control or netted fish at 1 hour and 4 hours (Fig. 2b). This difference was superimposed on a general and significant (P<0.001) increase in glucose levels in all three groups between time 0 and 1 h. The most pronounced difference in glucose levels was observed at 4 h when levels in keepnet-confined fish (7.3 ± 1 mM) were significantly higher than control (3.6 ± 0.2) or netted (4.3 ± 0.3) fish. During the remaining samples at 24, 48 and 168 hours there were no marked differences between groups although keepnet-confined fish at 48 hours had slightly, but significantly, higher glucose levels (3.7 ± 0.2) than control (2.8 ± 0.3) and netted fish (3.1 ± 0.1).

Lactate levels in all three groups of fish were variable for the first four samples (Fig. 2c); levels in keepnet-confined fish were significantly lower at time 0 than levels in control fish (2.6 ± 0.4 cf. 4.1 ± 0.3 mM; P<0.05). Lactate levels in all three groups were higher at 1 hour than at time 0 (P<0.001) and were significantly lower again at 4 hours (P<0.001). Lactate levels in fish in the netted group were significantly lower than levels in the other two groups at 4 hours (P<0.001) and levels in the netted group at 24 hours were significantly higher than the control group, though not the keepnet-confined group (P<0.001). There were no differences between groups at 48 hours and 168 hours.

EXPERIMENT 3

There were significantly higher plasma levels of cortisol in both handled groups at 1 h and 4 h (Fig. 3a). At time 0, cortisol levels in keepnet-confined fish (38 ± 4 ng ml^{-1}; n = 5) were significantly (P<0.001) higher than those in control (17 ± 5) and netted fish (9 ± 1). However, at 1 hour, levels in both netted (207 ± 44) and keepnet-confined groups (352 ± 86) were markedly higher than levels in the control group (7 ± 1; P<0.001) but not significantly different from each other. At 4 hours, levels in the keepnet-confined fish (126 ± 23) were significantly higher (P<0.001) than levels in both netted (38 ± 9) and control groups (10 ± 3). At 168 hours, levels in the netted group (61 ± 15) remained slightly, but significantly (p<0.001) higher than levels in the control (15 ± 3) and keepnet-confined groups (26 ± 6 ng ml^{-1}).

There was a significant elevation of plasma glucose levels in netted and keepnet-confined groups at 1 and 4 h (P<0.001; Fig. 3b). Glucose levels in these groups rose from a baseline of 3.3 - 4.2 mM at time 0 to 6.2 ± 0.4 (netted) and 6.8 ± 0.6 (keepnet-confined) compared to a level in the controls of 3.1 ± 0.4. A slight decline in glucose levels in the handled groups was observed at 4 hours but they remained higher than control levels. No differences between the three groups were apparent at time 0 or 168 hours.

Plasma lactate levels at 1 hour were slightly, but significantly, higher in the netted group (2.7 ± 0.3 mM; P<0.05) and keepnet-confined group (3.7 ± 0.3; P<0.001) than in the control group (2 ± 0.2; Fig. 3c). Levels in the keepnet-confined group at 168 hour were slightly, but significantly, higher than control levels (2.1 ± 0.3 cf. 1.0 ± 0.2; P<0.001).

EXPERIMENT 4

There was a significant increase in cortisol levels immediately following capture. Highest levels were recorded after 1 hour of keepnet confinement ($P<0.01$; Table 1). At the four subsequent hourly samples, cortisol levels were significantly elevated above those at time 0 at 2 hours ($P<0.05$) and 4 hours ($P<0.05$) but were not significantly different from 0 hour values at 3 and 5 hours after capture. Plasma glucose levels were significantly higher 1 hour after capture than at time 0 ($P<0.05$) and continued to rise during the subsequent three hours to peak after four hours confinement (Table 1). Lactate levels rose significantly ($P<0.001$) from time 0 to a maximum within one hour of capture (Table 1). There was little subsequent change in lactate levels during the remaining four hours of confinement, levels remaining significantly higher than those at time 0 throughout ($P<0.01$, $P<0.001$).

DISCUSSION

Effects of capture and confinement on plasma cortisol levels

The results clearly demonstrate that both capture and immediate release, and capture followed by a period of keepnet confinement, evoked a neuroendocrine stress response in carp, exemplified by a rapid elevation of plasma cortisol levels. The mean cortisol levels observed following application of the stressor in Experiments 1 to 3 (150-450 ng ml^{-1}), in which capture was simulated by netting and transfer to a second tank, are similar to or higher than, those measured in rod-caught carp following capture and during keepnet confinement in Experiment 4 (75-200 ng ml^{-1}) suggesting that the experimental procedure constituted a stressor at least as severe as that of rod-and-line capture and was therefore an appropriate approach to simulating

effects of angling.

In none of the experiments was there a substantial difference in the response of the fish to capture alone when compared to capture followed by keepnet confinement. The magnitude and duration of cortisol elevation in both cases was similar. Furthermore, the duration of the cortisol response to handling was relatively short with major perturbations in cortisol levels occurring only during the first four hours following initial disturbance. Although some minor differences in cortisol levels were observed subsequent to this, these were within the range of baseline levels and were also observed prior to the onset of disturbance (time 0, Experiments 1 and 3), suggesting they were unrelated to treatment. Overall, the results suggest that the major determinant of the magnitude of the cortisol stress response in the experimental carp was the initial capture procedure. Only in Experiment 3 was there a significantly higher cortisol level in keepnet-confined fish compared to netted-only fish, at four hours after the start of the experiment (Fig. 3a). At the other time-points and in the other experiments the cortisol response to capture followed by keepnet confinement was indistinguishable from the cortisol response to capture and release.

Mean plasma cortisol levels in Experiment 2 at one and four hours following the onset of stress (300-450 ng ml^{-1}) were higher than those at the equivalent time points in Experiments 1 and 3 (50-300 ng ml^{-1}). The reasons for this are unclear but may be related to the age, size or strain of the carp used in Experiment 2 which were different to those used in Experiments 1 and 3. The stress responsiveness of salmonid fish can vary between strains (Pickering & Pottinger, 1989; Pottinger & Moran, 1993; Pottinger *et al*., 1994) and the same is likely to be

true of non-salmonid fish. There have been no reports that age and size are factors which affect stress responsiveness in fish although that possibility cannot be discounted. Water temperature may also, however, have influenced the dynamics of the cortisol response observed during these studies. In both Experiments 2 and 3, carried out at 15^{o}C, high cortisol levels are achieved within one hour of the onset of disturbance and within a further three hours levels have begun to decline. In Experiment 1, which was carried out at a water temperature of 4^{o}C, cortisol levels continue to rise until at least four hours after the onset of the stress. The salmonid stress response is known to be temperature-dependent (Sumpter *et al*., 1985) and similar temperature-dependent differences in the dynamics of the stress response occur in roach (*Rutilus rutilus* L.; T. G. Pottinger, W. E. Yeomans & T. R. Carrick, unpublished), a species closely related to the carp.

Although most experimental work on the corticosteroid response of fish to stress has been carried out on salmonid fish (Barton & Iwama, 1991), in particular the rainbow trout (*Oncorhynchus mykiss* Walbaum) and Pacific salmon (*Oncorhynchus* sp.), the few data available for carp suggest that the cortisol response to stress observed during the present study is characteristic of that observed in carp under a variety of stressful conditions. Both baseline pre-stress levels and levels of cortisol observed following capture in the present study are similar to those measured in carp before and following forced swimming (Leloup-Hatey, 1960), handling (Ilan & Yaron, 1976) and transport (Davis & Parker, 1976). The magnitude and duration of the response to capture stress in the present study is also very similar to that observed in carp exposed to acid stress (van Dijk *et al*., 1993) in which cortisol levels rose from approximately 35 ng ml^{-1} prior to the onset of stress to peak at approximately 370 ng

ml^{-1} within four hours before returning to baseline within 24 hours.

The absolute levels of cortisol observed in carp in the present study following handling are well in excess of those required to cause serious adverse effects on growth, reproduction, and immunity in salmonid fish if sustained over a prolonged period of time. However, in the current study such levels were reached only briefly (<24 hours) and, although no data specific to these species are available, there is no evidence in the literature to suggest that short exposure to elevated levels of cortisol constitute a threat to the well-being of fish.

Effects of capture and confinement on plasma glucose levels

Plasma glucose levels are elevated during stress in fish primarily as a consequence of elevated blood catecholamine levels, although the involvement of cortisol in glycogenolytic/gluconeogenic processes has not been discounted (Van Der Boon *et al*., 1991). The level of plasma glucose at any time is a function of many factors such as diet, age, time since feeding, and season, and therefore is a more equivocal index of stress than cortisol (Wedemeyer *et al*., 1990). However, quantification of plasma glucose can provide valuable complementary information regarding the severity and duration of the stress response, the time required for recovery from the stimulus, and provides another point of reference with previous work on the same and related species.

In the present study the effects of the experimental procedures on plasma glucose levels were less pronounced and more variable than the effects on plasma cortisol levels. In contrast to the plasma cortisol response, plasma glucose was elevated only in response to combined capture

and confinement in Expts. 1 and 2 whereas both handled groups displayed a significant elevation in Expt. 3. This anomaly is suggestive of a difference in the factors controlling cortisol and glucose elevation during stress and supports the contention that stress-induced hyperglycaemia is not wholly cortisol-dependent. It is conceivable that the data indicate a differential adrenergic stress response to the two procedures. Much less is known of the adrenergic response to stressors in fish than the corticosteroid response, largely because of the difficulties in obtaining estimates of "resting levels" of catecholamines. As yet, no adverse effects can be linked directly with elevated blood catecholamine levels. Glucose levels in unstressed fish in the present investigation (2.5 - 7.0 mM) fall within the range reported for carp in previous work (2.8 - 5.6 mM: Hertz *et al*., 1989; 2.5 - 3.6 mM: Blasco *et al*., 1992; 3.6 - 6.5 mM: Malinovskaya, 1992). Exposure to prolonged hypoxia was reported to cause an increase in blood glucose levels from 5 mM to 10 mM (van Raaij *et al*., 1996) and carp exposed to crowding stress displayed an increase in blood glucose levels from 4.7 mM in unstressed controls to 6.9 mM after prolonged crowding (Yin *et al*., 1995). Both observations are consistent with the levels of glucose recorded following disturbance in the current study (6 mM - 9 mM).

Effects of the experimental procedures on plasma lactate levels

Elevated blood lactate levels occur consequent to respiratory activity under anaerobic conditions in which glycogen stores are depleted and lactate accumulates in the muscle tissue (Milligan & Girard, 1993). Thus elevated blood lactate is not considered to be an indicator of stress *per se* but reflects the imposition of severe exercise in which the tissue requirement for oxygen exceeds supply. Lactate was measured during the present study to establish that the

procedure employed to mimic capture and handling in Expts. 1 to 3 provided comparable physiological disturbance to that observed in rod-caught carp during Experiment 4, and to determine the time required for recovery from the respiratory effects of handling.

The levels of lactate measured in the present study (1.0 - 16 mM) are similar to those reported previously for other active fish species (see references in Milligan & McDonald, 1988) and for carp; both baseline and post-disturbance lactate levels were within the range previously reported for carp before and following stress (3.7 - 5.7 mM, 7.2 - 11.4 mM respectively; Dabrowska *et al*., 1991). While measurement of lactate alone does not provide an exact indicator of the extent of metabolic acidosis, it is an adequate index of the duration of recovery (Pankhurst & Dedual, 1994). The most pronounced effects of the experimental procedures on plasma lactate levels in the present study were observed in Expts. 1 and 4, while only minimal treatment-related variation was apparent in lactate levels in Expt. 3, and none occurred in Expt. 2. This suggests that, for Expt. 1 at least, the handling procedures produced metabolic disturbances similar to those occurring in rod-caught fish. Reasons for the inconsistency in response to handling are not immediately apparent. The physical procedures followed were identical for all three experiments. It is possible that the higher water temperature (15^{o}C) prevailing during Expts. 2 and 3, compared to Expts. 1 (4^{o}C) and 4 (8^{o}C) was a factor. Although environmental temperature does not influence the anaerobic capacity of salmonid fish, the rate of recovery from anaerobiosis is enhanced in warmer water (Brobbel *et al*., 1996; Wilkie *et al*., 1997). Therefore, the sample intervals in Expts. 2 and 3 may have been too great to detect a rapid elevation and recovery in lactate levels. There is no detailed information on lactate dynamics in carp with which to compare these data. Given these

considerations, these results suggest that overall, handling with or without a subsequent period of keepnet confinement elicits similar perturbations in blood lactate levels and recovery is complete within 4 to 24h.

Previous research on angling-related stress in fish

There are few data with which to compare these results and none which specifically examine the effects of post-capture confinement in non-salmonid fish. Hooking and playing fish is consistently reported to elicit increases in one or more of blood catecholamines, cortisol, glucose, and metabolic acidosis/blood lactate in species as diverse as rainbow trout (Pankhurst & Dedual, 1994; Wydoski *et al*., 1976), Atlantic salmon (*Salmo salar* L.; Booth *et al*., 1995: Brobbel *et al*.,1996) blue mao mao (*Scorpis violaceus* Hutton; Lowe & Wells, 1996) and largemouth bass (*Micropterus salmoides* Lacepède; Gustaveson *et al*., 1991). Recovery times, where determined, vary between 4 h to 72 h depending on the parameters measured and factors such as the nutritional status of the fish and water temperature (Booth *et al*., 1995: Brobbel *et al*., 1996; Pankhurst & Dedual, 1994; Wilkie *et al*., 1996; Wydoski *et al*., 1976). No mortality of fish during confinement, or during the weeks following the end of the experiments, was observed during the present study but post capture survival is reported to be significantly affected in rainbow trout which are exercised to exhaustion, and then removed from the water for between 30 and 60 seconds (Ferguson & Tufts, 1992). These authors identify the collapse of respiratory gas exchange when the gill lamellae are removed from water as being a critical factor in subsequent mortality. However, it should be noted that post-capture mortality is not widely reported, for example, Pankhurst and Dedual (1994) played wild rainbow trout to exhaustion but did not observe mortality following capture.

Given that the amount of effort expended by the fish during capture is a function of the size of the fish, environmental conditions (water flow, temperature) and the duration of time the fish is played, which in turn is dictated by whether the angler is using light or heavy tackle, and considering that some species are more tolerant of anoxia than others, it is possible that the results of the present study are not universally applicable. The moderate disturbance of blood lactate levels in the carp in this study suggest that the degree of respiratory stress experienced was not excessive. None of the studies cited above is directly comparable with the present work in terms of species or procedures, but nonetheless the results follow a similar pattern; capture of fish results in both endocrine and metabolic disruption but recovery generally occurs within a period not exceeding 96 h.

In conclusion, capture and retention of carp within keepnets does not result in a more pronounced stress response than capture followed by immediate release; the primary determinant of the physiological response appears to be capture itself. This observation is consistent with a previous report on snapper (*Pagrus auratus*, Bloch and Schneider; Pankhurst & Shaples, 1992) in which capture appeared to be the dominant factor influencing blood cortisol levels, independent of the severity or duration of post-capture handling. On the basis of these results, the use of keepnets appears not to have adverse effects on this species, a conclusion which it may be possible to extend to other cyprinid fish. However, this study was designed specifically to address the physiological aspects of capture and confinement in carp and further work is required to examine the impact of these procedures in other non-salmonid species and the possible effects of physical damage arising from the capture, playing, unhooking and confinement of rod-caught fish.

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References

Barton, B. A. (1997). Stress in finfish: past, present and future - a historical perspective. In *Fish Stress and Health in Aquaculture* (Iwama, G. K., Pickering, A. D., Sumpter, J. P. & Schreck, C. B., eds.), pp. 1-33. Cambridge: Cambridge University Press.

Barton, B. A. and Iwama, G. K. (1991). Physiological changes in fish from stress in aquaculture with emphasis on the response and effects of corticosteroids. *Annual Review of Fish Diseases* **1**, 3-26.

Blasco, J., Fernandez, J. and Gutierrez, J. (1992). Variations in tissue reserves, plasma metabolites and pancreatic hormones during fasting in immature carp (*Cyprinus carpio*). *Comparative Biochemistry and Physiology* **103A**, 357-363.

Booth, R. K., Kieffer, J. D., Davidson, K., Bielak, A. T. & Tufts, B. L. (1995). Effects of late-season catch and release angling on anaerobic metabolism, acid-base status, survival,

and gamete viability in wild Atlantic salmon (*Salmo salar*). *Canadian Journal of Fisheries and Aquatic Sciences* **52**, 283-290.

Brana, F., Nicieza, A. G. &Toledo, M. M. (1992). Effects of angling on populations structure of brown trout *Salmo trutta* L. in mountain streams of northern Spain. *Hydrobiologia* **237**, 61-66.

Brobbel, M. A., Wilkie, M. P., Davidson, K., Kieffer, J. D., Bielak, A. T. & Tufts, B. L. (1996). Physiological effects of catch and release angling in Atlantic salmon (*Salmo salar*) at different stages of freshwater migration. *Canadian Journal of Fisheries and Aquatic Sciences* **53**, 2036-2043.

Bugley, K. & Shepherd, G. (1991). Effect of catch-and-release angling on the survival of black sea bass. *North American Journal of Fisheries Management* **11**, 468-471.

Dabrowska, H., Dabrowski, K., Meyer-Burgdorff, K., Hanke, W. and Gunther, K. D. (1991). The effect of large doses of vitamin C and magnesium on stress responses in common carp, *Cyprinus carpio*. *Comparative Biochemistry and Physiology* **99A**, 681-685.

Davis, K. B. and Parker, N. C. (1986). Plasma corticosteroid stress response of fourteen species of warmwater fish to transportation. *Transactions of the American Fisheries Society* **115**, 495-499.

Dedual, M. (1996). Observed mortality of rainbow trout caught by different angling

techniques in Lake Taupo, New Zealand. *North American Journal of Fisheries Management* **16**, 357-363.

Dextrase, A. J. & Ball, H. E. (1991). Hooking mortality of lake trout angled through the ice. *North American Journal of Fisheries Management* **11**, 477-479.

Ferguson, R. A. and Tufts, B. L. (1992). Physiological effects of brief air exposure in exhaustively exercised rainbow trout (*Oncorhynchus mykiss*): implications for "catch and release" fisheries. *Canadian Journal of Fisheries and Aquatic Sciences* **49**, 1157-1162.

Gustaveson, A. W., Wydoski, R. S. & Wedemeyer, G. A. (1991). Physiological response of largemouth bass to angling stress. *Transactions of the American Fisheries Society* **120**, 629-636.

Hertz, Y., Madar, Z., Hepher, B. and gertler, A. (1989). Glucose metabolism in the common carp (*Cyprinus carpio* L.): the effects of cobalt and chromium. *Aquaculture* **76**, 255-267.

Hickley, P., Marsh, C. & North, R. (1995). Ecological management of angling. In *The Ecological Basis for River Management* (Harper, D. M. & Ferguson, A. J. D., eds.), pp. 415-425. Chichester: John Wiley & Sons.

Ilan, Z. and Yaron, Z. (1976). Stimulation of carp interrenal function by adrenocorticotrophin. *Journal of Endocrinology* **68**, 13-20.

Jeney, Z., Jeney, G., Olah, J. Siwicki, A. and Danko, I. (1986). Propanidid, a new anaesthetic for use in fish propagation. *Aquaculture* **54**, 149-156.

Leloup-Hatey, J. (1960). The influence of enforced exercise on the concentration of plasma corticosteroids in a teleost: the carp (*Cyprinus carpio* L.). *Journal of Physiology, Paris* **52**, 145-146.

Lowe, T. E. & Wells, R. M. G. (1996). Primary and secondary stress responses to line capture in the blue mao mao. *Journal of Fish Biology* **49**, 287-300.

Maitland, P. S. (1995). Ecological impact of angling. In *The Ecological Basis for River Management* (Harper, D. M. & Ferguson, A. J. D., eds.), pp. 443-452. Chichester: John Wiley & Sons

Malchoff, M. H. & Heins, S. W. (1997). Short-term hooking mortality of weakfish caught on single-barb hooks. *North American Journal of Fisheries Management* **17**, 477-481.

Malinovskaya, M. V. (1992). Daily rhythm of free fatty acids and glucose levels in the blood of carp. *Hydrobiological Journal* **28**, 5-12.

Milligan, C. L. and Girard, S. S. (1993). Lactate metabolism in rainbow trout. *Journal of Experimental Biology* **180**, 175-193.

Milligan, C. L. and McDonald, D. G. (1988). *In vivo* lactate kinetics at rest and during recovery from exhaustive exercise in coho salmon (*Oncorhynchus kisutch*) and starry flounder (*Platichthys stellatus*). *Journal of Experimental Biology* **135**, 119-131.

Muoneke, M. I. & Childress, W. M. (1994). Hooking mortality: a review for recreational fisheries. *Reviews in Fisheries Science* **2**, 123-156.

Pankhurst, N. W. and Dedual, M. (1994). Effects of capture and recovery on plasma levels of cortisol, lactate and gonadal steroids in a natural population of rainbow trout. *Journal of Fish Biology* **45**, 1013-1025.

Pankhurst, N. W. & Sharples, D. F. (1992). Effects of capture and confinement on plasma cortisol concentrations in the snapper, *Pagrus auratus*. *Australian Journal of Marine and Freshwater Research* 43, 345-356.

Pickering, A. D. and Pottinger, T. G. (1989). Stress responses and disease resistance in salmonid fish: effects of chronic elevation of plasma cortisol. *Fish Physiology and Biochemistry* **7**, 253-258.

Pickering, A. D., Pottinger, T. G. & Sumpter, J. P. (1987). On the use of dexamethasone to block the pituitary-interrenal axis in the brown trout, *Salmo trutta* L. *General and Comparative Endocrinology* **65**, 346-353.

Pottinger, T. G. (1997). Changes in water quality within anglers keepnets during the confinement of fish. *Fisheries Management and Ecology* **4**, 341-354.

Pottinger, T. G. & Moran, T. A. (1993). Differences in plasma cortisol and cortisone dynamics during stress in two strains of rainbow trout (*Oncorhynchus mykiss*). *Journal of Fish Biology* **43**, 121-130.

Pottinger, T. G., Moran, T. A. & Morgan, J. A. W. (1994). Primary and secondary indices of stress in the progeny of rainbow trout (Oncorhynchus mykiss) selected for high and low responsiveness to stress. *Journal of Fish Biology* **44**, 149-163.

Raat, A. J. P., Klein Breteler, J. G. P. & Jansen, A. W. (1997). Effects on growth and survival of retention of rod-caught cyprinids in large keepnets. *Fisheries Management and Ecology* **4**, 355-368.

Sumpter, J. P., Pickering, A. D. & Pottinger, T. G. (1985). Stress-induced elevation of plasma α-MSH and endorphin in brown trout, *Salmo trutta* L. *General and Comparative Endocrinology* 59, 257-265.

van der Boon, J., van den Thillart, G. E. E. J. M., and Addink, A. D. F. (1991). The effects of cortisol administration on intermediary metabolism in teleost fish. *Comparative Biochemistry and Physiology* **100A**, 47-53.

van Dijk, P. L. M., van den Thillart, G. E. E. J. M., Balm, P. and Wendelaar Bonga, S. (1993). The influence of gradual water acidification on the acid/base status and plasma hormone levels in carp. *Journal of Fish Biology* **42**, 661-671.

Van Raaij, M. T. M., van den Thillart, G. E. E. J., Vianen, G. J., Pit, D. S. S., Balm, P. H. M. & Steffens, A. B. (1996). Substrate mobilization and hormone changes in rainbow trout (*Oncorhynchous mykiss*, L.) And common carp (*Cyprinus carpio*, L.) During deep hypoxia and subsequent recovery. *Journal of Comparative Physiology B* **166**, 443-452.

Wedemeyer, G. A. Barton, B. A. and McLeay, D. J. (1990). Stress and acclimation. In *Methods For Fish Biology,* edited by C. B. Schreck and P. B. Moyle, 451-490, Bethesda, Maryland: American Fisheries Society

Wilkie, M. P., Davidson, K., Brobbel, M. A., Kieffer, J. D., Booth, R. K., Bielak, A. T. & Tufts, B. L. (1996). Physiology and survival of wild Atlantic salmon following angling in warm summer waters. *Transactions of the American Fisheries Society* **125**, 572-580.

Wilkie, M. P., Brobbel, M. A., Davidson, K., Forsyth, L. & Tufts, B. L. (1997). Influence of temperature upon the postexercise physiology of Atlantic salmon (Salmo salar). *Canadian Journal of Fisheries and Aquatic Sciences* **54**, 503-511.

Wydoski, R. S., Wedemeyer, G. A. and Nelson, N. C. (1976). Physiological response to

hooking stress in hatchery and wild rainbow trout (*Salmo gairdneri*). *Transactions of the American Fisheries Society* **105**, 601-606.

Yin, Z., Lam, T. J. & Sin, Y. M. (1995). The effect of crowding stress on the non-specific immune response in fancy carp (*Cyprinus carpio* L.). *Fish & Shellfish Immunology* **5**, 519-528.

Table 1. Experiment 4. Plasma cortisol, plasma glucose and plasma lactate in rod-caught carp immediately following capture (0) and at five hourly intervals following unhooking and transfer to keepnets. Each value is the mean ± SE, n = 10. Significant differences from values at time 0 are denoted by asterisks * $P<0.05$, ** $P<0.01$, *** $P<0.001$.

	Time (h)					
	0	1	2	3	4	5
Plasma cortisol (ng ml-1)	86.4 ± 19.4	178.1 ± 18.1**	146.6 ± 25.1*	121.3 ± 18.7	164.6 ± 32.3*	122.4 ± 19

Plasma glucose (mM)	2.1 ± 0.2	3.0 ± 0.3*	3.2 ± 0.2**	3.5 ± 0.3***	5.0 ± 0.5***	4.0 ± 0.2***
Plasma lactate (mM)	4.7 ± 0.5	11.2 ± 1.9***	9.9 ± 0.6***	9.3 ± 0.9**	9.6 ± 0.8***	10.2 ± 0.7***

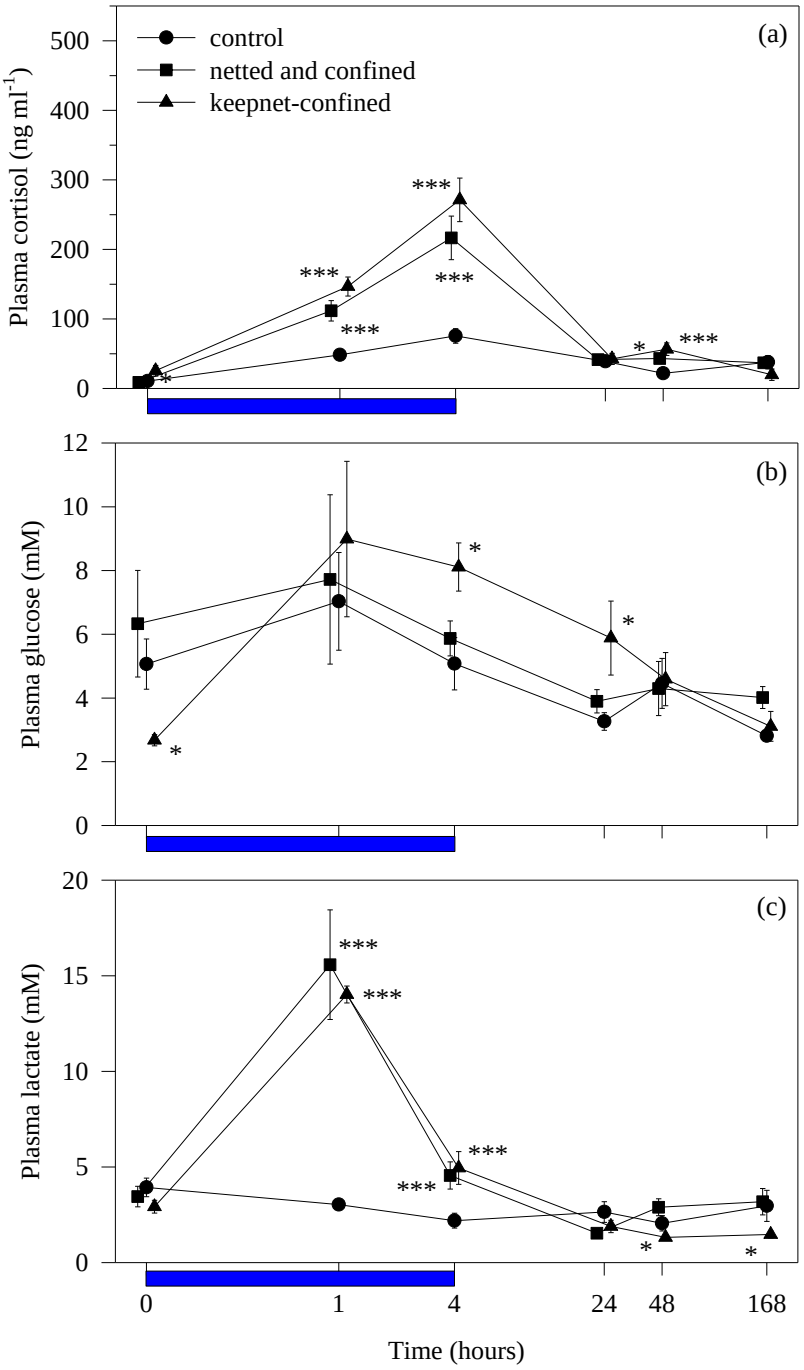

Figure 1. Experiment 1. Plasma cortisol (a), plasma glucose (b) and plasma lactate (c) in undisturbed control fish (●), netted and confined fish (■), and fish keepnet-confined for 4h (▲), prior to and following the onset of disturbance. Each point represents the mean ± SE, n = 5. Significant differences from control values at each time point are denoted by asterisks * P<0.05, *** P<0.001. The solid bar indicates the period of keepnet confinement.

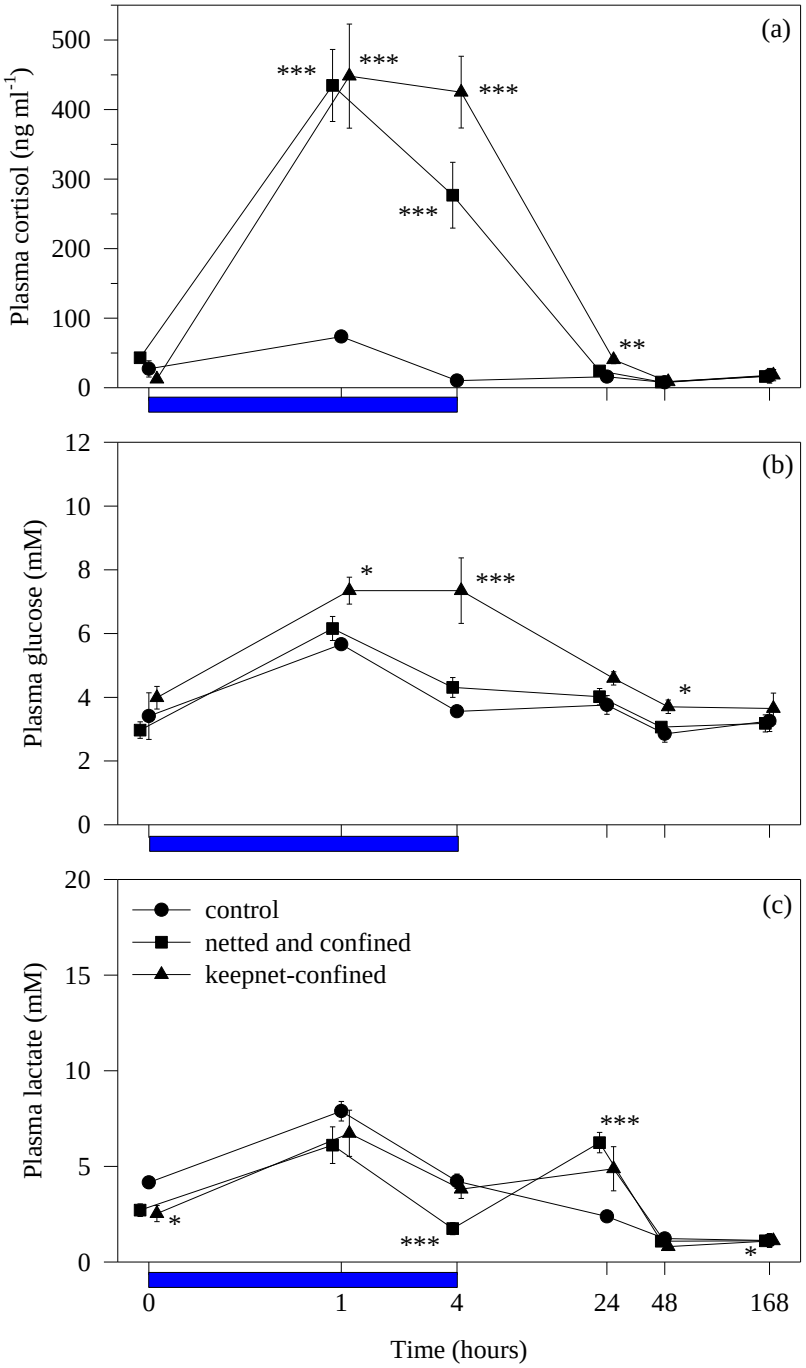

Figure 2. Experiment 2. Plasma cortisol (a), plasma glucose (b) and plasma lactate (c) in undisturbed control fish (●), netted and confined fish (■), and fish keepnet-confined for 4h (▲), at intervals prior to and following the onset of disturbance. Each point represents the mean ± SE, n = 6. Significant differences from control values at each time point are denoted by asterisks * P<0.05, ** P<0.01, *** P<0.001. The solid bar indicates the period of keepnet confinement.

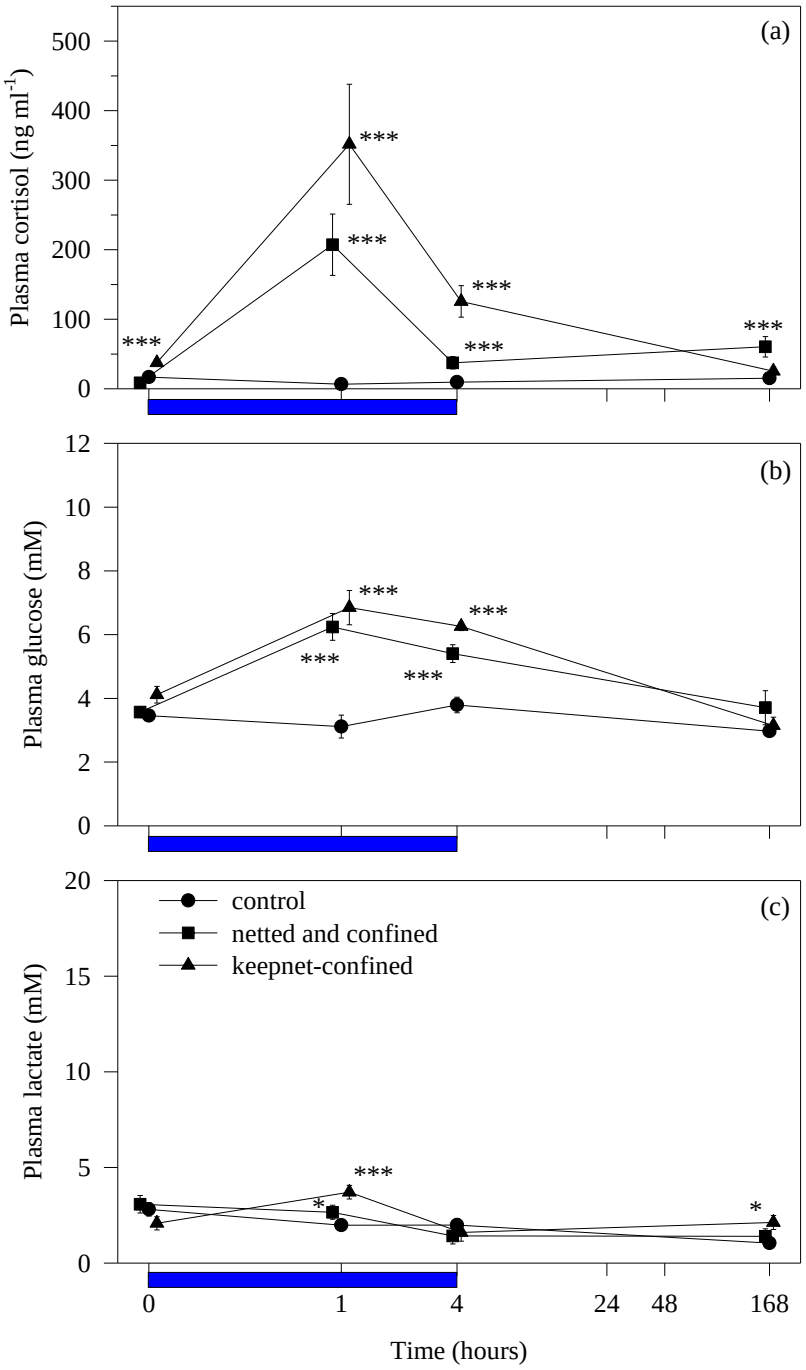

Figure 3. Experiment 3. Plasma cortisol (a), plasma glucose (b) and plasma lactate (c) in undisturbed control fish (●), netted and confined fish (■), and fish keepnet-confined for 4h (▲), at intervals prior to and following the onset of disturbance. Each point represents the mean ± SE, n = 5. Significant differences from control values at each time point are denoted by asterisks * P<0.05, *** P<0.001. The solid bar indicates the period of keepnet confinement.