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Influence of Retention and Air Exposure on Physiology and Behaviour of Common Carp (*Cyprinus carpio* L.): A Field Study

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Table of Contents

List of Tables

List of Figures

Abstract

Specialised angling for large size common carp (*Cyprinus carpio* L.) is often conducted as voluntary catch-and-release angling involving various handling measures. Among the most prominent handling measures is retention in carp sacks for the purpose of photographing the fish prior to release. When carp are captured during unfavourable light conditions for photography, they are retained until the light conditions have improved. Photography is also associated with extended air exposure. So far, little is known about potential sub-lethal and lethal impacts associated with carp sack retention and air exposure on large size common carp. To address these issues, a field study was conducted from September to December 2007 at Dow's Lake in Ottawa, Ontario, Canada. Carp were captured by angling and allocated to a control group and five treatment groups (N = 10 each). Three treatments involved retention in carp sacks for periods up to 9 h. One treatment received air exposure for 10 min following capture, and a final treatment involved air exposure for 10 min following 9 h of retention. All fish were blood sampled to assess physiological changes, and radio transmitters were attached to monitor short-term behaviour and long-term fate of carp post-release.

Retention in carp sacks induced a prolonged physiological stress response in carp indicated by elevated blood plasma cortisol concentrations, and hyperglycaemia in long-term retained fish. Blood plasma lactate levels decreased during retention, suggesting recovery from playing. Observed blood plasma ion changes were minor and likely a result of playing. Long-term retention was also found to be associated with tissue damage. Physiological changes were reflected by impaired post-release behaviour in long-term retained fish indicated by reduced minimum displacement. However, recovery was fast and no mortalities occurred. Air exposure following capture did not result in physiological changes relative to fish captured only (i.e. control), whereas exposing carp to air following retention increased blood plasma lactate levels relative to retained fish. Air exposed carp required more time to leave the release site, and minimum displacement between 31 min and 60 min post-release was reduced in fish air exposed following capture compared to control fish. However, behaviour normalised quickly, and air exposure did not result in mortality. From a practical perspective, the results indicate that despite being sub-lethally affected by retention and air exposure, carp are able to cope with these stressors. From a fish welfare perspective, however, it is recommended to avoid retention in carp sacks and minimise exposure to air.

1. Introduction

Recreational fishing has become a popular leisure activity throughout the world (Cowx 2002, Cooke & Cowx 2004, Cooke & Cowx 2006, Arlinghaus & Cooke 2009), and has largely replaced commercial fisheries in many temperate freshwater ecosystems (Arlinghaus *et al.* 2002). Estimates suggest a scale of more than 700 million anglers worldwide (Cooke & Cowx 2004, Arlinghaus & Cooke 2009) catching about 47.1 billion fish annually (Cooke & Cowx 2004). A portion of this catch is harvested by the anglers, but many fish are released alive (Cooke & Suski 2005, Arlinghaus *et al.* 2007). The implementation of catch-and-release can either occur mandatorily on the basis of fishing regulations (e.g. fish captured during closed seasons or fish protected by size limits must be released; Quinn 1996, Policansky 2002), or voluntarily where the angler decides to release a fish that could be retained (Quinn 1996, Policansky 2002), or both. Voluntarily catch-and-release can be practised for various reasons (Quinn 1996, Aas *et al.* 2002), and for more specialised anglers, total catch-and-release is a strict policy practised to conserve fish stocks (Aas *et al.* 2002). Voluntary release rates near 100 % have been reported for some specialised fisheries such as Western European coarse fishing including specialised common carp (*Cyprinus carpio* L.) angling (North 2002, Arlinghaus & Mehner 2003) and fishing for elitist resources such as bonefish (*Albula* spp.) (Policansky 2002) and Atlantic white marlin (*Tetrapturus albidus*) (Cramer 2004). Overall, it is estimated that global release rates could account for up to 2/3 of all captured fish (Cooke & Cowx 2004).

Any form of catch-and-release can benefit recreational fisheries by reducing fishing mortality (Arlinghaus *et al.* 2007). However, the efficiency of catch-and-release depends on adequate survival rates (Wydoski 1977, Muoneke & Childress 1994, Bartholomew & Bohnsack 2005, Arlinghaus *et al.* 2007), and minimisation of sub-lethal impacts, such as injuries, physiological disturbances, behavioural alterations and fitness impairments (Cooke *et al.* 2002*a*, Arlinghaus *et al.* 2007). Previous research has often focused on direct mortality following catch-and-release angling (Wydoski 1977, Muoneke & Childress 1994, Batholomew & Bohnsack 2005, Arlinghaus *et al.* 2007). Less attention has been paid to sub-lethal consequences as they arise from various stressors during multiple portions in a recreational fishing event (Cooke *et al.* 2002*a*). Hence, there are only a few species for which a reasonable understanding of the sub-lethal catch-and-release angling effects exist at present time (Cooke & Suski 2005).

These species include largemouth bass (*Micropterus salmoides*), walleye (*Sander vitreus*), rainbow trout (*Oncorhynchus mykiss*), striped bass (*Morone saxatilis*) and Atlantic salmon (*Salmo salar*). All these species are endemic to North America, aside from Atlantic salmon which is also endemic to Europe (Cooke & Suski 2005). Information for other species is comparatively scarce. One of these poorly studied, but rather important species in recreational fisheries is common carp (Cooke & Suski 2005) which is the study species of the present work.

Common carp originated in Eastern Europe and central Asia (Kohlmann *et al.* 2003), but have been introduced to all continents for aquaculture purposes and to support inland fisheries (Lever 1996, Cambray 2003). In Europe carp has developed into a popular target species in recreational fisheries (Czech Republic: Vacha 1998; France: Boisneau & Mennesson-Boisneau 2001; Germany: Wedekind *et al.* 2001, Arlinghaus 2007, Arlinghaus 2008; Poland: Wolos 1996; Slovakia: Hensel 1996; United Kingdom: Hickley & Chare 2004) and is increasingly sought on other continents such as Africa (Økland *et al.* 2003) and North America (Spitler 1987, Faaroqi 2006). However, the consumptive orientation (i.e. the attitude towards catch and harvest aspects of the fishing experience) of carp anglers differs between countries as do release rates. For example, in many Eastern European countries, carp are often captured for personal consumption (Vacha 1998), while in central Europe and the United Kingdom, highly specialised carp angling for trophy size fish is often conducted as total catch-and-release fisheries (Arlinghaus & Mehner 2003), involving voluntary catch-and-release (Arlinghaus 2007). Important catch-related motivations of specialised carp anglers are catching trophy size fish and photographing them prior to release to serve as memories (Arlinghaus & Mehner 2003). In the context of photography, carp are subjected to various handling measures potentially lasting for extended periods. Two of the most common handling practises in specialised carp angling are retention of fish post-capture and the photography itself which is associated with extended exposure to air.

Trophy and large size carp are commonly retained when they are captured during unfavourable light conditions under the belief that a better photograph of a memorable fish can be produced when the light conditions have improved. Retention periods can last several hours to the entire night if a fish is captured soon after dusk. In specialised carp angling, so-called “carp sacks” are widely used to retain the fish during this period, which are collapsible, dark knotless mesh bags made of synthetic fibre cloth. Typically, a zip cord is incorporated to close the sack and prevent the escape of

fish during retention. After capture, carp are individually confined in carp sacks and retained in shallow water, with the zip cord of the sack fixed to a stick on the bank or some hard structure at the shoreline.

During photography, fish are exposed to air for extended time periods as carp anglers typically aim at photographing both sides of the fish to be able to later recognise individual carp by comparing pictures (Arlinghaus 2007). In addition, air exposure also occurs directly following angling during unhooking. It is currently unknown how retention in carp sacks and air exposure affects the physiology, behaviour, and survival of carp subjected to catch-and-release angling. Shedding light on this question is the objective of the present study.

2. Literature Review

2.1 The Stress Response in Fish

Stressors are factors that threaten or disturb the internal homeostasis of an organism and evoke a set of physiological and behavioural responses that, under normal conditions, are adaptive and enable the organism to deal with and overcome the threat and return to a prestress physiological state or to an altered resting state with no long- term consequences for health and fitness (Barton & Iwama 1991, Wendelaar Bonga 1997, Chrousos 1998). If the stressor is excessive or long-lasting, compensation may not be possible and the stress response can become maladaptive and detrimental to the fish's health (Barton & Iwama 1991, Barton 2002, Barton *et al.* 2002). This view of an organism's ability or inability to cope with a stressor is consistent with the general adaptation syndrome (G.A.S.) paradigm of Selye (1950). According to the G.A.S. paradigm, the organism passes three distinct stages in response to stress, which include: (1) the alarm reaction as the initial response comprising the perception of the stimulus and its recognition as a thread to homeostasis, (2) the stage of resistance describing the mobilisation of resources to adjust to the thread and maintain or regain homeostasis, and (3) the stage of exhaustion describing the incapability of the organism to cope with the disturbance finally resulting in pathological condition or death (Selye 1950). However, the concept of G.A.S. is controversial (Levine 1985, Moberg 1985, Munck *et al.* 1984) and might not apply to all situations, partly because of questions about its supposed non-specificity (Mason 1971, Schreck 1981, Schreck 1982). Schreck (1981) concluded that a G.A.S.-type stress response is evoked in teleosts by situations which cause fright, discomfort or pain. During the recreational fishing event (e.g. playing, retention and air exposure) fish experience clear disturbances in terms of fright and, possibly, in terms of discomfort or pain (Schreck 1981), wherefore the G.A.S. paradigm provides a useful approach for discussing stress in a recreational fishing context. The first two stages of the G.A.S. are usually manifested by measurable physiological changes at different levels of organisation, whereas the final stage is maladaptive and associated with pathological state, altering health and condition and potentially resulting in mortalities (Barton *et al.* 2002).

According to the sequence of an organism response to a stimulus, a broad distinction between primary, secondary, and tertiary stress response has been introduced (Wedemeyer & McLeay 1981). The primary stress response involves the initial

response on the endocrine level and includes the release of catecholamines from chromaffin cells via the hypothalamic-sympathetic-chromaffin cell axis, which stimulate the hypothalamic-pituitary-interrenal axis to release corticosteroids (mainly cortisol) from interrenal cells (Mazeaud & Mazeaud 1981, Wendelaar Bonga 1997, Barton *et al.* 2002). Coping with stress is an energy-demanding process, wherefore catecholamines and corticosteroids are released into general circulation under conditions that require mobilisation of energy substrate to cope with increased energy demands and enhanced oxygen uptake (Wendelaar Bonga 1997, Barton *et al.* 2002).

The actions and effects of the stress hormones at the blood and tissue levels are described as the secondary stress response (Wedemeyer & McLeay 1981, Wendelaar Bonga 1997), and appear within a few minutes to an hour after the onset of a stressor (Barton *et al.* 2002, Portz *et al*. 2006). As noted above the secondary stress response includes metabolic changes such as the mobilisation of energy substrate (e.g. the release of glucose from the liver; Barton & Iwama 1991, Wendelaar Bonga 1997, Portz *et al.* 2006) and the formation of lactate through anaerobic consumption of the three endogenous fuels adenosine triphosphate (ATP), phosphocreatine (PCr) and glycogen during increased muscular activity and anaerobiosis (Driedzic & Hochachka 1978, Kieffer 2000). Furthermore, stress hormones evoke haematological alterations (e.g. release of erythrocytes from the spleen; Barton & Iwama 1991, Barton 1997, Barton 2002, Barton *et al.* 2002) and increase cardiovascular and respiratory functions to enhance the oxygen uptake and maintain adequate oxygen supply to tissues (Mazeaud & Mazeaud 1981, Randall & Perry 1992, Wendelaar Bonga 1997). However, the enhanced gas exchange also increases gill permeability to water and ions which can result in an influx of water and an efflux of ions from the blood causing a disturbance of the hydromineral balance (McDonald & Milligan 1997, Wendelaar Bonga 1997, Barton *et al.* 2002). In addition, ionic/osmotic disruptions occur at the kidney including increased filtration, reabsorption, and excretion (Wood 1991) and in response to haematological alterations, for example through changes of membrane permeability to water and ions in adrenergically stimulated erythrocytes (Wood 1991, Borgese *et al.* 1987). But non-hormonally disturbances of the ionic/osmotic balance are also associated with stress and disruptions can occur via other processes. For example, during strenuous exercise, the CO_2 partial pressure increases in the blood of fish, resulting in a respiratory acidosis (Wood *et al.* 1983, Wood 1991, McDonald & Milligan 1997). Furthermore, the accumulation and dissociation of lactate causes a pH

decrease in the white muscle and, due to proton leakage into the plasma, also causes decrease in the blood pH (i.e. metabolic acidosis) (Wood *et al.* 1983, Wood 1991, McDonald & Milligan 1997). These acid-base disturbances are a direct consequence of the secondary stress response (i.e. lactate accumulation), and can be associated by fluid and electrolyte shifts between extracellular and intracellular spaces and can also result in ion exchange via the gills to restore acid-base equilibrium (Wood *et al.* 1983, Holeton *et al.* 1983, Wood 1991, McDonald & Milligan 1997). These mechanisms affect the ionic/osmotic balance of fish unrelated to any hormonal mechanism (Wood *et al.* 1983, Holeton *et al.* 1983, Turner *et al.* 1983, Wood 1991, McDonald & Milligan 1997).

Physiological demands that exceed a threshold level can, potentially, not be overcome by coping mechanisms and lead to debilitating effects. These consequences, impairing the whole-animal performance, are described as tertiary stress-response (Wedemeyer & McLeay 1981, Barton & Iwama 1991, Wendelaar Bonga 1997, Barton 2002, Barton *et al.* 2002, Portz *et al.* 2006), and can be affected directly or indirectly by catecholamines and corticosteroids (Wendelaar Bonga 1997, Barton *et al.* 2002). The tertiary stress response includes behavioural impairments and reduced metabolic scope for activity as well as growth depression, reduced immune response, reduced reproductive capacity, and may alter the capacity of the organism to tolerate subsequent stressors, ultimately affecting the survivability (Wedemeyer & McLeay 1981, Barton & Iwama 1991, Wendelaar Bonga 1997, Barton *et al.* 2002, Portz *et al.* 2006). Thus, the effects of the tertiary stress response impact not only the individual, but may also have negative consequences at the population level.

The stress response of fish can further be influenced by non-stress factors regarding influencing the magnitude of the stress response and the ability of the individual to recover (Barton *et al.* 2002). These factors comprise species-specific differences, genetic variability within species, developmental stage, nutritional status and almost all environmental conditions (Barton 1997, Barton 2002, Barton *et al.* 2002).

2.2 Catch-and-Release Angling-Induced Stress in Fish

Angling is considered as one of the most physically demanding forms of exercise for fish (Wood 1991, Booth *et al.* 1995), and causes a suite of physiological alterations (Cooke & Suski 2005, Arlinghaus *et al.* 2007). Previous research on a variety of species has shown that stress hormones are released during the playing of the fish often

measured on basis of elevated cortisol levels (Lowe & Wells 1996, Pottinger 1998, Meka & McCormick 2005) and less frequently by an increase in adrenalin and noradrenalin (Lowe & Wells 1996). As a direct consequence of elevated circulation of these hormones hyperglycaemia was frequently reported (Wydoski *et al.* 1976, Gustaveson *et al.* 1991, Pottinger 1998, Suski *et al.* 2007*a*, Wedemeyer & Wydoski 2008). The heightened physical activity during playing is accompanied by oxygen debt in white muscle tissues indicated by an increase in cardiac output and its components (i.e. stroke volume and heart rate; Schreer *et al.* 2001, Suski *et al.* 2004, Killen *et al.* 2006), and the anaerobic consumption of energy stores (Suski *et al.* 2004, Suski *et al.* 2006, Killen *et al.* 2006, Arlinghaus *et al.* 2009), resulting in an accumulation of lactate (Gustaveson *et al.* 1991, Pottinger 1998, Suski *et al.* 2004, Meka & McCormick 2005, Killen *et al.* 2006). In addition, haematological disruptions were frequently observed following playing, such as changes in haematocrit (Beggs *et al.* 1980, Lowe & Wells 1996, Suski *et al.* 2007*a*, White *et al.* 2008) and haemoglobin (Beggs *et al.* 1980, Gustaveson *et al.* 1991, Lowe & Wells 1996, Suski *et al.* 2007*a*, Wedemeyer & Wydoski 2008), as well as acid-base disturbances (Beggs *et al.* 1980, Booth *et al.* 1995, Kieffer *et al.* 1995, Skomal & Chase 2002, Thompson *et al.* 2002). Finally the primary and secondary stress response evoked by angling causes ionic/osmotic disturbances manifested as alterations of osmolality and electrolyte concentrations (Wydoski *et al.* 1976, Gustaveson *et al.* 1991, Suski *et al.* 2004, Killen *et al.* 2006, Wedemeyer & Wydoski 2008).

It has generally been accepted that species differ in their sensitivity to catch-and-release angling stressors (Cooke & Suski 2005). But also differences within species are apparent arising from angling operation, environmental conditions and intrinsic factors. It was frequently observed that the magnitude of physiological disruptions increase with playing duration (Gustaveson *et al.* 1991, Kieffer *et al.* 1995, Lowe & Wells 1996, Thompson *et al.* 2002, Meka & McCormick 2005), which is in turn dependant on other variables, including gear type (Arlinghaus *et al.* 2007) and size of captured fish (Thorstad *et al.* 2003, Meka 2004, Meka & McCormick 2005). In addition, physiological disturbances tend to increase when fish are captured in greater water depth and subjected to barotrauma (Morrissey *et al.* 2005, Gravel & Cooke 2008). Regarding environmental conditions, previous research demonstrated that water parameters including water hardness (Kieffer *et al.* 2002), and water temperature (Cooke & Suski 2005, Arlinghaus *et al.* 2007) can affect the magnitude of the

physiological stress response following angling exercise. In particular, high water temperatures were noted to increase physiological disruptions (Gustaveson *et al.* 1991, Anderson *et al.* 1998, Thompson *et al.* 2002, Meka & McCormick 2005) and impact survival, especially when above the optimum range of the species (Wilkie *et al.* 1997, Anderson *et al.* 1998, Wilde 1998, Wilde *et al.* 2000, Thorstad *et al.* 2003), but this may also be applicable to low water temperatures in warm water adapted fish species (Arlinghaus *et al.* 2007). Intrinsic factors can potentially influence susceptibility to catch-and-release angling stressors including differences among strains or populations (Hanson *et al.* 2008, Redpath 2008), gender (Arlinghaus *et al.* 2007), life history stage (Brobbel *et al.* 1996), condition (Meka & McCormick 2005) and fish size (Wydoski *et al.* 1976, Ferguson *et al.* 1993, Kieffer *et al.* 1996).

Physiological disturbances caused by angling-induced exercise are typically not lethal during appropriate environmental conditions assuming the fish are in good physiological condition when released (Wydoski *et al.* 1976, Wydoski 1977, Gustaveson *et al.* 1991, Pankhurst & Dedual 1994, Wedemeyer & Wydoski 2008) and recovery occurs rapidly (Arlinghaus *et al.* 2007). Recovery dynamics are influenced by the magnitude of disturbance (Cooke *et al.* 2002*b*), environmental conditions such as water temperature and dissolved oxygen (Wilkie *et al.* 1997, Schreer *et al.* 2001, Suski *et al.* 2006). In addition, it was demonstrated that swimming at low velocities can reduce the time required to recover from exhaustive exercise (Meyer & Cook 1996, McDonald & Milligan 1997, Milligan *et al.* 2000), though this does not apply to all species (Suski *et al.* 2007*b*).

Previous research has indicated that angling-induced exercise potentially results in consequences beyond physiological disruptions. It was shown that capture of fish can cause leakage of cytoplasmatic enzymes into the blood stream (Morrissey *et al.* 2005). The function of these enzymes is normally restricted to the intracellular space, and they are only released by cell defects or death, wherefore they are a useful indicator of tissue damage (Henry 1996). Catch-and-release angling for trophy size carp is frequently associated with extended handling, often involving retention and prolonged air exposure periods. Previous catch-and-release angling research revealed that stressors can be cumulative and interactive which might aggravate physiological disruptions and its consequences (e.g. during fishing tournaments; Suski *et al.* 2003*a*, Killen *et al.* 2003) and delay or prohibit recovery (Cooke *et al.* 2002*b*).

Retention of fish after capture represents one of the most pronounced stressors in recreational fishing on the top of intrinsic stressors associated with playing (Arlinghaus *et al.* 2007). Match or coarse anglers commonly use net retention gear when angling for European cyprinids (North 2002), in particular keepnets (Pottinger 1997, Pottinger 1998, Raat *et al.* 1997, Meinelt *et al.* 2008). Keepnets are also often used in coarse fish tournaments when their use is not restricted (Meinelt *et al.* 2008). In specialised carp angling, retention of fish is accomplished in carp sacks as previously described. Various impacts of keepnet retention on fish have been investigated, but less attention has been paid to retention in carp sacks. Pottinger (1998) examined short-term physiological consequences of keepnet retention following angling in carp, including release of cortisol and metabolic changes, and concluded his research that post capture retention in keepnets does not result in a more pronounced stress response than capture alone. Furthermore, no adverse long-term effects of keepnet retention, such as growth depression or elevated mortality rates, were observed in multiple cyprinid species including carp (Raat *et al.* 1997). However, Cooke & Hogle (2000) found that keepnet retention is injurious to smallmouth bass (*Micropterus dolomieu*) and results in mortalities especially during periods of high water temperature. So far, only one laboratory study investigated the impact of carp sack confinement on fish (Hallermann unpublished data). In contrast to keepnet retention, the results of this study suggest that retention in carp sacks induces a prolonged stress response in small carp indicated by elevated cortisol and glucose levels. Similar results were observed in a number of other studies examining the influence of net retention in a non-angling context (Davis & Parker 1986, Ruane *et al*. 2001, Ruane *et al.* 2002). Apart from hormone-induced stress response occurring directly from the retention, previous studies assessing effects of net retention on physiology in a recreational fishing context consistently showed that fish recovered from playing during confinement indicated by decreasing lactate levels and a restoration of acid-base and ionic/osmotic equilibrium (Pottinger 1998, Hallermann unpublished data). This was also observed during live-well confinement in largemouth bass and walleye if fish were supplied with adequate water conditions (Suski *et al.* 2004, Killen *et al.* 2006). Consequently, Lewin *et al.* (2006) concluded that post-capture retention can facilitate recovery from recreational angling-induced stressors when the conditions are appropriate.

In addition to retention, a second pronounced stressor in carp angling is extended air exposure, which represents one of the most important stressors influencing the

physiology (Cooke & Suski 2005, Arlinghaus *et al.* 2007). Air exposure causes a collapse of the gill lamellae, an adhesion of the gill filaments, and the resultant reduction of the gill surface area causes an inhibition of gas exchange (Boutilier 1990, Ferguson & Tufts 1992). The physiological disruptions occurring from impaired oxygen uptake are similar to those induced by playing (Ferguson & Tufts 1992). Previous research demonstrated that air exposure following angling aggravates the physiological disturbances as indicated by a further raise in stress hormones (Haukenes & Buck 2006) and blood glucose levels (Haukenes & Buck 2006, Thompson *et al.* 2008, White *et al.* 2008, Arlinghaus *et al.* 2009) and induces more pronounced cardiovascular changes indicated by a longer return time to resting levels (Cooke *et al.* 2001, Cooke *et al.* 2002*b*). In addition, anoxia following playing results in an increase in the blood and muscle lactate load (Ferguson & Tufts 1992, Killen *et al.* 2006, Suski *et al*. 2007*a*, Hanson *et al*. 2008), resulting in more pronounced acid-base disturbances (Ferguson & Tufts 1992) and aggravating ionic/osmotic disruptions (Haukenes & Buck 2006), though many studies did not observe ionic/osmotic disturbance following air exposure beyond those resulting from playing (Suski *et al.* 2007*a*, Thompson *et al.* 2008, Arlinghaus *et al.* 2009, Hallermann unpublished data). Since carp held in keepnets and carp sacks have been reported to recover from the physiological disturbances caused by exercise (Pottinger 1998, Hallermann unpublished data), exposing carp to air after an extended retention period, as is typical in specialised carp angling for large size fish, probably results in a second bout of hypoxia accompanied by a depletion of energy stores, an accumulation of lactate and disruptions of the acid-base equilibrium (Suski *et al.* 2004, Killen *et al.* 2006, Hallermann unpublished data).

Similar to playing, the degree of physiological disturbances caused by air exposure correlates positively with its duration (Cooke *et al.* 2002*b*, White *et al.* 2008) as does the time required for the restoration of the physiological status (Cooke *et al.* 2001, Cooke *et al.* 2002*b*, Suski *et al.* 2004, White *et al.* 2008). In addition, environmental conditions (Davis & Schreck 2005, Thompson *et al.* 2008) and intrinsic factors, such as age of fish (Davis & Schreck 2005), can influence the magnitude of physiological disturbances following air exposure. In certain circumstances air exposure can contribute to mortalities (Ferguson & Tufts 1992, Arlinghaus & Hallermann 2007, Gingerich *et al.* 2007), though many studies have reported no impact on survival even following extended air exposure periods (Thompson *et al.* 2008, White *et al.* 2008, Arlinghaus *et al.* 2009, Hallermann unpublished data). Currently it is unclear how

stressors in carp angling associated with exercise, retention in carp sacks, and air exposure affect the physiological state, tissue damage and physiological recovery dynamics of large size common carp that are targeted by specialised carp anglers.

The primary and secondary stress responses of fish to catch-and-release angling stressors potentially leads to tertiary effects such as reduced fitness of fish, for example through inhibition of reproductive hormones (Pankhurst & Dedual 1994) or reduced reproductive output (Ostrand *et al.* 2004), and may cause a suite of behavioural alterations resulting in further ecologically relevant consequences. In fact, investigation of fish behaviour has proved to be a sensitive indicator of the complex biochemical and physiological changes resulting from stressors (Schreck *et al.* 1997, Little 2002). Behavioural alterations may be adaptive and increase the probability of survival or may reflect deleterious changes in how an animal senses and responds to its environment (Schreck *et al.* 1997, Little 2002). Several individual and inter-individual behavioural changes have been reported as indicators of catch-and-release angling impacts on fish. Siepker *et al.* (2006) observed temporarily altered feeding behaviour potentially affecting growth rate or condition factor (Clapp & Clark 1989, Diodati & Richards 1996, Siepker *et al.* 2006, Klefoth & Arlinghaus 2008), and Beukema (1970) and Raat (1985) found changes in vulnerability to angling post-release. Furthermore temporarily altered habitat choice (Klefoth *et al.* 2008) and altered homing behaviour following tournament displacement have been reported (Wilde 2003). Catch-and-release angling might also impact reproductive behaviour of fish and potentially alter lifetime reproductive success. It was demonstrated that common snook (*Centropomus undecimalis*) changed their spawning behaviour (Lowerre-Barbieri *et al.* 2003) and Atlantic salmon altered upstream spawning migration patterns post-release (Mäkinen *et al.* 2000, Thorstad *et al.* 2003, Thorstad *et al.* 2007). In addition, several studies revealed that removal of nest guarding black bass (*Micropterus* spp.) from their nests by catch-and-release angling resulted in higher rates of nesting abandonment, especially when brood predation occurred during absence of the fish (Kieffer *et al.* 1995, Philipp *et al.* 1997, Suski *et al.* 2003*b*, Hanson *et al.* 2007). Among the most frequently measured behavioural parameters for the assessment of catch-and-release angling impacts is movement activity and related parameters (e.g. time rested). These behavioural variables offer clear advantages, such as the high sensitivity to stressors (Little 2002) and the feasibility of data sampling in the field using biotelemetry (Cooke *et al.* 2002*a*, Little 2002, Donaldson *et al.* 2008). Post-release movement activity can

increase and show signs of hyperactivity (Cooke *et al.* 2000, Thorstad *et al.* 2004) or can decrease (Klefoth *et al.* 2008, Arlinghaus *et al.* 2008). Both behavioural alterations potentially result in ecological relevant consequences. Increased activity causes an unnecessary expenditure of energy reserves, thereby reducing capacity for other ecologically relevant processes (Black *et al.* 1958). Decreased activity and impaired locomotion might interfere with foraging behaviour (Klefoth & Arlinghaus 2008), impair parental care ability of nest guarding species (Cooke *et al.* 2000), or increase susceptibility to aquatic or terrestrial predators (Cooke & Philipp 2004, Thorstad *et al.* 2004, Danylchuk *et al.* 2007).

Despite the growing body of literature on behavioural effects of catch-and-release angling (Donaldson *et al.* 2008) little is known about the influence of retention on post-release behaviour. Previous research assessed behavioural impairments following live-release fishing tournaments that include retention in live-wells (Young & Isely 2006, Hanson *et al.* 2007), but it is currently unknown how the application of net retention gear, as it is used in shore angling for cyprinids, affects fish behaviour (Arlinghaus *et al.* 2007). Evidence suggests that net retention might cause behavioural impairments, as previously demonstrated by decreasing swimming performance in dip net retained striped bass in a non-angling context (Strange & Chech 1992).

Prolonged air exposure periods during handling were identified to aggravate impacts of catch-and-release angling on post-release behaviour. Observed effects following air exposure include alterations of movement activity, such as hyperactivity (Cooke & Philipp 2004), decreased swimming performance (Schreer *et al.* 2005), increased time to resume swimming post-release (Arlinghaus *et al.* 2009), and longer post-release rest periods relative to non-air exposed fish (Cooke & Philipp 2004, Arlinghaus *et al.* 2009). In addition, it was demonstrated that extended air exposure periods increase the likelihood of equilibrium loss relative to non- or gently air exposed fish (Cooke & Philipp 2004, Danylchuk *et al.* 2007, Gingerich *et al.* 2007, Thompson *et al.* 2008), possibly representing a generalised breakdown of systemic homeostatic mechanisms that normally function to allow fish to avoid life-threatening situations (Beitinger *et al.* 2000). The consequences of air exposure on behaviour potentially result in more pronounced ecologically relevant behavioural consequences compared to angled fish that experienced no air exposure, such as higher post-release predation rates (Danylchuk *et al.* 2007) and longer return time of nest guarding species to their nest increasing the likelihood of nesting abandonment (Philipp *et al.* 1997).

Several studies demonstrated that the magnitude of behavioural impairments following catch-and-release angling typically correlates with length of stress exposure (i.e. playing and air exposure duration; Philipp *et al.* 1997, Cooke & Philipp 2004, Schreer *et al.* 2005, Danylchuck *et al.* 2007, Gingerich *et al.* 2007, Thompson *et al.* 2008, Arlinghaus *et al.* 2009) and can be influenced by environmental conditions such as water temperature (Gingerich *et al.* 2007, Thompson *et al.* 2008).

Recovery periods of behavioural alterations following catch-and-release angling are highly variable depending on the investigated parameter. While behavioural consequences arising from physical impairments are often reversed within hours to few weeks (Cooke *et al.* 2000, Skomal & Chase 2002, Arlinghaus *et al.* 2008, Klefoth *et al.* 2008, Arlinghaus *et al.* 2009), other behavioural changes can in fact last for extended time periods as demonstrated by reduced angling vulnerability in carp which can be maintained for one year or even longer (Beukema 1970, Raat 1985). Generally, the recovery period from behavioural alterations arising from physical impairments correlates with the longevity of stress exposure (Gingerich *et al.* 2007, Thompson *et al.* 2008, White *et al.* 2008) but can be influenced by environmental conditions such as water temperature (Gingerich *et al.* 2007). In addition, the recovery can be delayed during reproductive season. Cooke *et al.* (2000) demonstrated that locomotory activity in nest guarding largemouth bass required more than 24 h to recover from catch-and-release angling while non-nesting fish appeared to recover as early as 2 h post-release.

Ultimately catch-and-release angling and its various stressors can result in mortalities. Mortality rates are reported for a number of species and range between 0 and 95 % influenced by various factors associated with angling operation, environmental conditions and intrinsic differences (Wydoski 1977, Muoneke & Childress 1994, Bartholomew & Bohnsack 2005, Arlinghaus *et al.* 2007). Specifically for carp, reported catch-and-release mortality rates are low and vary between 0 % and 2 % (Beukema 1970, Raat 1985). According to the time of occurrence, mortalities are categorised as immediate, short-term, and long-term mortalities (Pollock & Pine 2007). Immediate mortality is measured when a fish is dead upon landing or dies prior to release (e.g. through acute injuries or predation resulting from the fish being hooked, Pollock & Pine 2007). Short-term mortality occurs within 24 h to 72 h post-release often as a result of hooking and handling injury or indirect effects such as predation (Pollock & Pine 2007), and long-term mortality is measured when a fish dies more than 72 h post-release, for example through injuries that are not directly lethal, but impair

foraging ability and cause mortality at a later time (Pollock & Pine 2007). Currently, the consequences of a carp angling event and the additional handling stressors carp sack retention and air exposure on short-term behaviour and long-term fate of large carp in their natural environment are unknown.

2.3 Study Objectives and Hypotheses

Laboratory studies are the most common approaches to quantify sub-lethal impacts of catch-and-release angling and provide substantial insight into physiological consequences of catch-and-release angling (Cooke *et al.* 2002*a*, Cooke *et al.* 2004). In addition to laboratory settings, pond studies are often used to assess effects of catch-and-release angling on growth and mortality (Raat *et al.* 1997, Arlinghaus & Hallermann 2007). However both kinds of experimental set-ups are subjected to clear limitations (Cooke *et al.* 2002*a*, Donaldson *et al.* 2008) and concerns exist regarding the applicability of those data to natural environments (Cooke *et al.* 2004). Limitations can arise regarding experimental animals as hatchery-reared fish or wild fish held in captivity typically respond differently to stressors compared to free-swimming wild fish (Cooke *et al.* 2002*a*). In addition, the size of fish used in laboratory and pond settings for evaluation of catch-and-release angling impacts do often not correlate with the size of fish targeted by anglers (Raat *et al.* 1997, Pottinger 1998, Hallermann unpublished data), which could potentially affect the stress response (Wydoski *et al.* 1976, Ferguson *et al.* 1993, Kieffer *et al.* 1996). Further limitations arise from exclusion of stressors in the natural environment of fish such as predator-prey interactions, rapidly changing environmental conditions, and changes in water quality. In turn laboratory and pond settings provide a variety of other stressors fish do not face in their natural environment including crowding and additional physiological demands arising from artificial holding environments (Cooke *et al.* 2004, Portz *et al.* 2006). Limitations can also arise from the sampling procedure itself especially through repeated chasing during sequential fish removal from the holding facility (Redgate 1974, Pickering *et al.* 1982). These limitations might bias results of laboratory and pond studies and restrict the transferability to a regular angling scenario (Cooke *et al.* 2004). Therefore Cooke *et al.* (2002*a*) called for *in situ* data sampling to add realism to catch-and-release angling studies. An approach that may help to advance catch-and-release angling science in field settings includes coupling physiological assessments with behavioural observation and survivorship using biotelemetry (Cooke *et al.* 2005, Skomal 2007). Biotelemetry

allows fine-scale movement measurements as well as examination of short-term and long-term survival and other endpoints associated with catch-and-release angling in the natural environment of fish (Donaldson *et al.* 2008). However, despite its obvious advantages, biotelemetry is currently still underused in catch-and-release angling science (Donaldson *et al.* 2008).

To heed the call for more field studies in catch-and-release angling science, the objective of the present study was to examine the effects of carp sack retention and air exposure on physiology, short-term behaviour, and long-term fate of large size common carp in their natural environment. Physiological changes were measured by means of several blood plasma parameters including cortisol as indicator of the primary stress response and glucose as an indicator of metabolic changes stimulated by stress hormones. Blood plasma lactate served as an indicator of physical exhaustion, anaerobiosis, and metabolic recovery. Osmolality, sodium, chloride, and potassium concentrations in the blood plasma were used to assess ionic and osmotic disturbances. Presence of the enzymes lactate dehydrogenase (LDH) and aspartate transaminase (AST) in the blood plasma served as indicators of cell damages. The tertiary stress response was assessed by examination of behavioural impairments with biotelemetry using movement activity and its components (i.e. distance moved, time required to leave the release site, time rested, and minimum displacement) as indicators. Four hypotheses were tested in the present work.

Hypothesis 1

Retention in carp sacks results in physiological stress response in carp indicated by an increase in cortisol and glucose values and a decrease in osmolality and ion concentration in the blood plasma. At the same time, retention facilitates recovery from angling exercise indicated by a decrease in blood plasma lactate concentrations.

Hypothesis 2

Retention negatively affects short-term movement activity and its components, but does not impact survival of carp.

Hypothesis 3

Air exposure aggravates the physiological disturbances of carp relative to appropriate controls as indicated by increased concentrations of cortisol, glucose and lactate in the blood plasma, but does not result in a heightened disturbance of osmolality and ion equilibrium in the blood plasma relative to appropriate controls.

Hypothesis 4

Air exposure negatively affects short-term movement activity and its components relative to appropriate controls, but does not impact survival of carp.

3. Materials & Methods

3.1 Study Area

The present study was conducted at Dow's Lake, an embayment of the Rideau Canal, which is located in the centre of Ottawa, Ontario, Canada, between Hartwell's Lockstation and Ottawa Lockstation (N 45°23'39.86", W 75°42'05.56"). Its surface area is about 18 ha (Jaakson 1984) and the maximum depth is about 6 m (Fisheries and Oceans Canada 2005). Submerged macrophytes are highly abundant during the summer month comprising numerous species with fluctuating proportions (Cooke *et al.* 2007; Table 1).

Table 1: Macrophyte species community in Dow's Lake observed in 2006 (according to Cooke *et al.* 2007).

Common name	Species name
Bushy pondweed	*Najas flexilis*
Canada waterweed	*Elodea Canadensis*
Coontail	*Ceratophyllum demersum*
Creeping spearwort	*Ranunculus flammula*
Eurasian water milfoil	*Myriophyllum spicatum*
Needle spikerush	*Eleocharis acicularis*
Clasping-leaf pondweed	*Potamogeton richardsonii*
Sago pondweed	*Potamogeton pectinatus*
Tape grass	*Vallisneria americana*
Water bulrush	*Scirpus subterminalis*
Water stargrass	*Heteranthera dubia*

A recent species survey that included seine netting identified 17 fish species in the study area (Cooke *et al.* 2007; Table 2). The fish community is dominated by centrarchids in particular *Lepomis* spp. (Cooke *et al.* 2007; Table 2). Top predators are muskellunge and northern pike (Cooke *et al.* 2007; Table 2). Common carp were infrequently captured in the course of the species survey (Cooke *et al.* 2007; Table 2), but might be underrepresented in this study due to sampling methodology (Barthelmes & Doering 1996). Natural reproduction in Dow's Lake and the Rideau Canal was observed for

various fish species including bluegill, pumpkinseed, largemouth bass, smallmouth bass, yellow perch, brown bullhead and common carp, and there is evidence that also golden shiner, log perch and rock bass attempt to spawn in the study area (Cooke *et al.* 2007). Dow's Lake and the Rideau Canal are important local recreation areas and utilized for many leisure activities (Jaakson 1984) including recreational angling which is predominantly conducted as a catch-and-release fishery (Cooke *et al.* 2007). Along with several native species, common carp are frequently targeted by local anglers (Cooke *et al.* 2007).

Table 2: Fish species community in Dow's Lake observed in 2006. * denote species that were not encountered in the species survey by seine netting, but infrequently observed in surveys by snorkeling or in angler catches (according to Cooke *et al.* 2007).

Common name	Species name	Relative abundances (%)
Black crappie	*Pomoxis nigromaculatus*	0.44
Bluegill	*Lepomis macrochirus*	57.0
Bluntnose minnow	*Pimephales notatus*	0.10
Brook silverside	*Labidesthes sicculus*	0.29
Brown bullhead	*Ameiurus nebulosus*	0.39
Common carp	*Cyprinus carpio*	0.15
Golden shiner	*Notemigonus crysoleucas*	6.7
Largemouth bass	*Micropterus salmoides*	1.6
Log perch	*Percina caprodes*	0.63
Muskellunge	*Esox masquinongy*	*
Northern pike	*Esox lucius*	0.05
Pumpkinseed	*Lepomis gibbosus*	26.4
Rock bass	*Ambloplites rupestris*	2.0
Smallmouth bass	*Micropterus dolomieu*	0.24
Walleye	*Sander vitreus*	*
White sucker	*Catostomus commersonii*	0.29
Yellow perch	*Perca flavescens*	4.2

3.2 Study Design

To assess the impact of retention and air exposure on physiology, post-release behaviour, and survival, individual carp were randomly allocated to a control group and five different treatment groups. Each group was comprised of N = 10 individuals (Figures 1 and 2). The focus of this study was on the additional stressors of retention and air exposure, wherefore all fish, including the control fish, were captured by rod and reel. Control fish experienced no further treatment, while other fish were subjected to additional treatments as shown in Figures 1 and 2. Investigation of carp sack retention effects on carp involved the control group and three treatment groups of retention periods of either 3 h, 6 h or 9 h (Figure 1). These retention periods correspond to short-, medium- and long-term retention in a typical specialised carp angling event. Investigation of air exposure effects on physiology and behaviour of carp included the control group and two air exposure treatment groups, which consisted of a 10 min air exposure period either applied directly after capture or following retention for 9 h (Figure 2). The combined treatment of retention and air exposure served to mimic a photography event after retention as is typical in specialised carp angling. In addition to the control fish and the air exposure treatments, the 9 h retention treatment group was included in this investigation and provided a further control to determine whether the impact of the combined treatment is based upon interactive effects of retention and air exposure or simply resulting from retention alone. The air exposure duration of 10 minutes was chosen on basis of a pre-test. It comprised the time needed for tranquillisation of body movements of carp and time required for the photography event. Indeed, to photograph a trophy size carp, it is essential that the fish is calm while holding it in front of the camera. This is typically achieved by extended air exposure resulting in hypoxia and exhaustion. Thus, the treatment of air exposure mimicked the behaviour of carp anglers in field settings. Following the applications of the treatments, all fish were blood sampled (Figures 1 Figure 2, see below for description) using caudal venipuncture. Various haematological parameters indicative of the primary and secondary stress response as well as tissue damage were examined as described in detail below. Additionally, external radio transmitters were attached to the carp for investigation of behaviours indicative of a tertiary stress response (Figures 1 and 2, see below for description) as has been successfully applied in a number of catch-and-release angling studies (Thompson *et al.* 2008, Gravel & Cooke 2008, Arlinghaus *et al.* 2008, Arlinghaus *et al.* 2009).

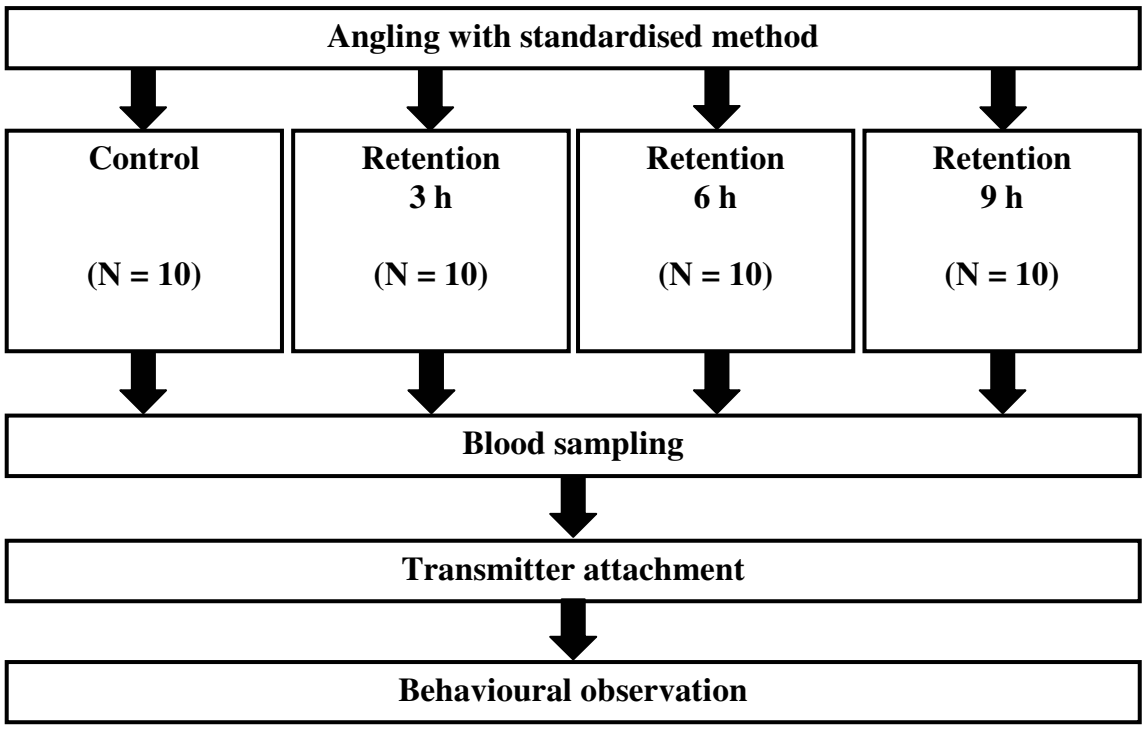

Figure 1: Study design for investigation of retention effects on physiology and behaviour in carp.

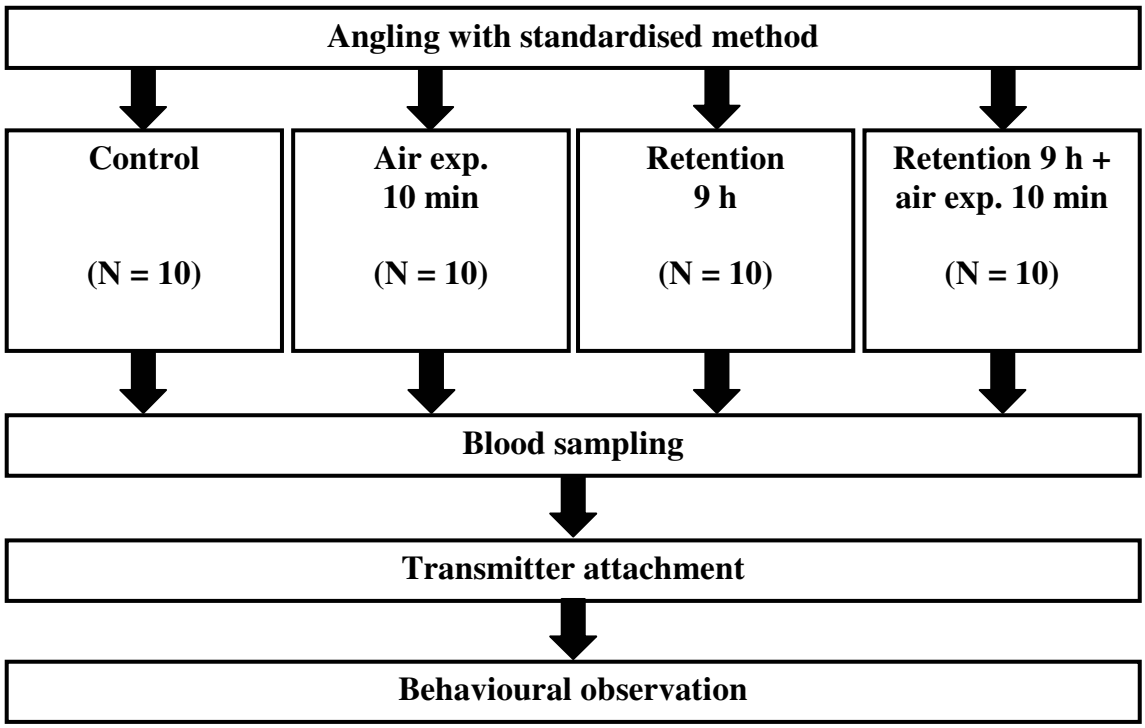

Figure 2: Study design for investigation of air exposure effects on physiology and behaviour in carp.

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3.3 Carp Angling

Angling was conducted across 21 individual fishing days between September 9 and October 3, 2007. Fish were captured by the same experienced angler during the morning hours from sunrise until noon. All angling was conducted from shore and took place at the same fishing site. Water temperature and dissolved oxygen were measured daily at 8.00 am and ranged from 18.3 °C to 23.1 °C (mean ± SD: 20.2 ± 1.30 °C) and 8.43 mg/L to 12.79 mg/L (mean ± SD: 10.5 ± 1.44 mg/L), respectively. The angling method conformed to a standard bottom fishing technique in specialised carp angling with a fixed lead sinker and a short leader (Figure 3a). The bait was not directly attached to the hook, but to a short piece of line, which constituted an extension of the leader. The so-called "hair" had a length of 2 cm (measured from the bent of the hook) and the leader had a total length of 15 cm (11.3 kg test). This method is known among carp anglers as the hair rig and is assumed to facilitate quick hooking after the bait is ingested as the hook remains uncovered by bait (Figure 3b). For bait, 3 kernels of maize (corn) threaded onto the hair using a crochet hook and fixed on the hair with a small piece of plastic, called boilie stopper (Figure 3b). The leader was directly tied to a swivel (Figure 3a). A plastic clip was fixed on the swivel, by pushing the swivel into the clip (Figure 3a). A lead sinker (weight of 84 g) was attached to the plastic clip and fixed with a flexible cone-shaped rubber tube (Figure 3a). The other parts of the terminal rig consisted of a 50 cm long braided line with a lead core to avoid loosing large carp that became snagged in dead woody debris, macrophytes or other structure during the fight (Figure 3a). This set-up contributes to a shallow hooking depth, avoids hooking in critical (i.e. deep) locations (Rapp *et al.* 2008) and facilitates quick unhooking to avoid the influence of additional handling potentially influencing physiology and behaviour post-release. The terminal rig was connected to an 18.1 kg test braided mainline via a second swivel (Figure 3a). All carp were captured with carp rods (length: 3.60 m, test curve: 1.24 kg) near shore within a macrophyte-free patch. Playing time was standardised to 3 min. If the fish was exhausted within a shorter time period, it was played near the shore until the 3 min time limit was reached. All fish were landed with a knotless landing net that minimises injury to the fish (Barthel *et al.* 2003). Following landing the fish were immediately transferred into a water-filled trough to avoid air exposure during unhooking and allow total fish length to be recorded to the nearest 1 cm. Thereafter, with the exception of control fish that were only captured, the fish experienced their respective treatments as shown in Figures 1 and 2.

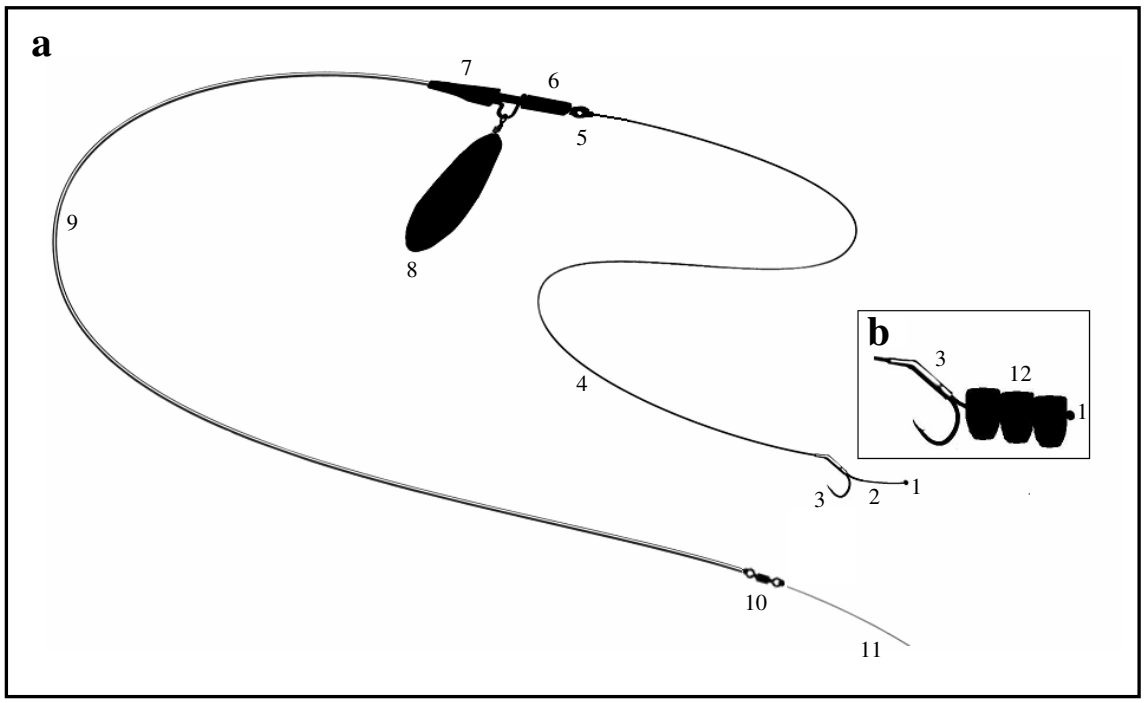

Figure 3: Illustrations of the angling rig used in this study (plot a) and the hair baited with corn (plot b): 1: boilie stopper, 2: hair, 3: hook, 4: leader, 5: swivel, 6: plastic clip, 7: rubber tube, 8: lead sinker, 9: braided line with a lead core, 10: swivel, 11: braided main line, 12: corn (modified from Rapp *et al.* 2008).

3.4 Treatments

After capture, carp were randomly allocated to the different treatment groups. For the retention treatments, captured fish were transferred into carp sacks of the dimension 140 × 120 cm using a commercially available model (Daiwa-Cormoran Sportartikelvertriebs GmbH, Groebenzell, Germany, model: Cormoran Karpfensack "De Luxe"). This carp sack model is made of black coloured polyester with knotless mesh of the size 3 × 2 mm. The horizontal distance between the meshes is 5 mm and the vertical distance is 1 mm. Vertical mesh rows are staggered in arrangement. At the open end of the carp sack, a zip cord is incorporated to close the sack. Every fish was confined individually as this approach is usually used by specialised carp anglers. The carp sacks were transferred into the lake in a distance of about 5 m from the angling site and fixed to the shoreline with a metal stick to avoid drifting. Carp were held in a distance of about 1 m from shore, which is about the length of the zip cord after tying the fish to the metal stick. During retention the fish were not further disturbed by handling. For air exposure treatments, fish were placed on a commercially available unhooking mat of the size of 90 × 50 cm (Daiwa-Cormoran Sportartikelvertriebs GmbH, Groebenzell, Germany;

model: Cormoran Standard Abhakmatte). This unhooking mat model is filled with soft foam and the outer material is made of polyester and coated with polyvinyl chloride. The unhooking mat served to avoid dermal injuries (Arlinghaus 2007) that might have occurred through contact with hard structures such as stones and roots. Carp were held in place on the unhooking mat to prevent jumping or sliding off. The air exposure period was measured with a stop watch.

3.5 Blood Sampling

Following the treatment, fish were placed back into the water-filled trough to avoid air exposure during blood sampling. An assistant held the fish while the blood sample was taken by using vacutainer syringes (Becton-Dickinson, Inc., Franklin Lakes, New Jersey, U.S.A.; model: vacutainer: 3 mL, lithium heparin anticoagulant; needle: 21 gauge, 38.1 mm). Blood was collected by caudal venipuncture and the caudal vein was approached laterally by inserting the needle just below the lateral line (Dyer & Cervasio 2008). Activation of the vacutainer mostly resulted in an immediate blood flow, but if blood did not enter the vacutainer immediately, the syringe was slightly readjusted. Blood collection duration was limited to 1 min, and any fish that required more than a minute for sampling was excluded from the study. One vacutainer of blood was collected per fish. The blood samples were stored in an ice slurry and then transferred to a centrifuge and spun at 10,000 × gravity for 5 min to separate the blood plasma from the blood constituents. The blood plasma was extracted with a pipette, transferred into vials, and stored in a dewar with liquid nitrogen. The vials remained in this dewar until the field sampling period was over and were afterwards stored at -80 °C for future biochemical analyses.

3.6 Biochemical Blood Analyses

For preparation of blood plasma cortisol analysis, 100 µL of blood plasma were extracted in 3 mL diethylether. Upon phase separation, samples were incubated at -80 °C to separate the frozen aqueous phase. The liquid phase was transferred to a glass vial, followed by a second extraction in 3 mL diethylether. Following complete evaporation of the solvent, the samples were dissolved in 1 mL PBS buffer and blood plasma cortisol was determined with an enzyme-linked immunoabsorbent assay (ELISA) kit (IBL International GmbH, Hamburg, Germany, test: RE 52611) according to the manufacturer protocol. The ELISA test is based upon the principle of competitive

binding. The microtiter wells are coated with a monoclonal antibody directed towards a unique site on the cortisol molecule. Endogenous cortisol in the blood plasma sample competes with a cortisol horseradish peroxidase conjugate for binding to the coated antibody. After incubation the unbound conjugate is washed off. The amount of bound peroxidase conjugate is reverse proportional to the cortisol concentration in the blood plasma sample. Optical density was measured at 450 nm using a plate reader (Tecan Group Ltd., Maennedorf, Switzerland, model: Spectrafluor Plus).

Blood plasma glucose analysis was based on Trinder's glucose oxidase method and was conducted with an autoanalyser (F. Hoffmann - La Roche, Basal, Switzerland, model: Roche/Hitachi 917) and appropriate reagents according to the manufacturer protocol. This photometric method relies on the oxidation of glucose to gluconic acid by the enzyme glucose oxidase with the formation of hydrogen peroxide. In presence of the enzyme peroxidase, phenol and 4-aminoantipyrine are oxidised by hydrogen peroxide to form quinonimine, a colorimetric indicator, which is proportional to the amount of glucose in the blood plasma sample. Quinonimine was measured at 500 nm.

Determination of blood plasma lactate was conducted with a lactate assay kit based on an enzymatic method according to the manufacturer protocol (Trinity Biotech plc, Bray, Ireland, test: 735-10). This approach is based on conversion of lactic acid into pyruvate and hydrogen peroxide by lactate oxidase. Due to chromogen formation, catalysed by peroxidase in the presence of hydrogen peroxide, absorbance was measured at 540 nm using a plate reader (Tecan Group Ltd., Maennedorf, Switzerland, model: GENios, software: Magellan V5.0).

Blood plasma osmolality was determined using a freezing point osmometer (Gonotec GmbH, Berlin, Germany, model: Osmomat 030). The principle of this method relies on comparative measurements of freezing points of distilled water and the test solution, by what osmolality of the sample was determined.

Methodologies of blood plasma sodium, blood plasma chloride and blood plasma potassium analysis relied on the ion selective electrode principle and were conducted with an autoanalyser according to the manufacturer protocol (F. Hoffmann - La Roche, Basal, Switzerland, model: Roche/Hitachi 917). Ion selective electrodes have a selective membrane in contact with the test solution at the outer side and an internal filling solution containing the test solution in a fixed concentration. The test ions associate with the membrane on each side and an electrical potential develops. The

electromotive force is determined by the difference in concentration of the test solution and the internal filling solution according to the Nernst equation.

LDH concentration in the blood plasma was determined using an autoanalyser (F. Hoffmann - La Roche, Basal, Switzerland, model: Roche/Hitachi 917) and appropriate reagents according to the manufacturer protocol. The method was based on the catalysis of l-lactate to pyruvate whereby NAD^+ is reduced to NADH. The velocity of NADH formation is proportional to the catalytic LDH activity and absorbance was determined at 340 nm.

Analysis of AST in the blood plasma sample was conducted with an autoanalyser (F. Hoffmann - La Roche, Basal, Switzerland, model: Roche/Hitachi 917) and appropriate reagents according to the manufacturer protocol. Blood plasma AST analysis relied on catalysis of α-ketoglutarate and l-aspartate to l-glutamate and oxalacetate. The increase of oxalacetate is determined by an indicator reaction catalysed by malate-dehydrogenase, in which NADH is oxidised to NAD^+. The velocity of NADH decrease is proportional to the velocity of formation of oxalactetate and consequently the AST activity. NADH decrease was measured photometrically at 340 nm.

3.7 External Transmitter Attachment

Transmitter attachment was conducted after blood sampling. The fish was placed in the water-filled trough and an assistant held the fish in place during the radio transmitter attachment. No anaesthetic was used as this might have altered post-release behaviour of the fish (Cooke *et al.* 2005). The transmitters (Holohil Systems Inc., Carp, Ontario, Canada; model: PD-2 transmitters; weight in air: 3.8 g, battery life: 6 month, dimensions L × W × H (mm): 23 × 12 × 7) were attached externally below the dorsal fin as has been previously successfully applied in a study with common carp (Økland *et al.* 2003). To attach the transmitters, two interconnected syringes with 22 gauge hypodermic needles were used. On these needles a plastic backing of about 1 × 2.5 cm was attached. The needles were pushed through the dorsal musculature beneath the caudal end of the dorsal fin. From the opposite side, stainless steel wires that were threaded through the transmitters were inserted into the lumen of needles. The wires were pulled out on the opposite side of the fish, and when the needles were removed, the plastic backing was left in place to protect the body of the fish. The wires were twisted carefully and trimmed prior to release (Cooke 2003). Due to ongoing carp

fishing by other anglers in the study lake, an additional anchor tag (Floy Manufacturing Inc., Seattle, Washington, U.S.A.) with an individual identity number and a phone number was inserted into the dorsal musculature. During the study period, no carp was reported as recaptured either by local anglers or the research team.

3.8 Tracking

Following transmitter attachment the fish were released directly at the angling site. Tracking was conducted manually from shore using a handheld radio receiver (Lotek Wireless, Newmarket, Ontario, Canada, model: SRX 400) and a three element Yagi antenna. Movement activity of all fish was observed constantly for the first 30 min post-release using successive gain reductions (i.e. zero-point tracking; Gravel & Cooke 2008). Tracking calibrations were previously conducted at the Rideau Canal and Dow's Lake. For this purpose a line was attached to a transmitter, which was subsequently positioned in different water depth and distances to the tracker to evaluate reception range of the receiver with different gain settings. To estimate the fish position during data sampling, detailed notes were recorded on a map including shoreline positions and estimated distances of the fish from shore. Shoreline positions were quantified by descriptions of the shoreline and GPS data points (Garmin International Inc., Olathe, Kansas, U.S.A., model: eTrex Summit). Accuracy of the GPS ranged from 5 to 10 m depending on satellite reception and weather conditions. Distance from shore was estimated by adjusting gain settings of the receiver and the angle of the antenna. All fish swam in close distance to the shoreline when released where water depth was relatively shallow and did not vary decisively (based on assessments with an echosounder and the information given in a detailed depth map; Figure 4). Therefore, it was assumed that the influence of water-depth on signal reception was negligible and the reception ranges established during tracking calibrations were valid. The coordinates of each fish position were determined with an electronic GIS map (Google Inc., Mountain View, California, U.S.A., model: Google earth) based on the shoreline GPS position where the fish was tracked and shoreline descriptions coupled with the estimated distance of the fish from shore. The accuracy of the this GIS map in the study area was previously tested by comparing GPS data points taken with the handheld GPS receiver with those assessed with the GIS map at the same landmark and differences were negligible which validates the applied approach. With these data, distance moved within the first 30 minutes, time required to leave the release site, and time rested within 30 min post-

release were calculated (Rogers & White 2007). Further tracking points were taken from each fish at 1 h, 12 h, 24 h, 36 h, 48 h, 60 h, and 72 h post-release. Tracking was conducted using triangulation from at least two points (Mech 1983). Triangulation was not possible when the fish was in the main basin of the lake close to one shoreline or in deep water due to insufficient transmitter signal strength resulting in imprecise accuracy of this position estimation. In these cases, the position was estimated from the nearest shoreline using successive gain reductions (i.e. zero-point tracking; Gravel & Cooke 2008). In addition to the previously described approach, the obtained data on distance to shore were matched with a detailed depth map (Figure 4) to increase accuracy of the position estimation. This was necessary due to varying water depths on the different sides of the lake (Figure 4) and the decrease of signal strength with increasing water depth. The final coordinates of the fish position were determined as described above and these coordinates were used to generate minimum displacement between successive locations for all time periods mentioned above (Rogers & White 2007). Minimum displacement between successive time intervals was determined as the straight line between successive locations and/or the nearest water distance between tracking points if a fish swam around a headland (Arlinghaus *et al.* 2008). The accuracy of the tracking methods was tested on two days by comparing results received from shoreline tracking and tracking from boat which was expected to provide accurate position measures. The difference between both methods was within ± 17 m and most similar near the shore with decreasing congruence at great distance to shore and deep water. Due to similar findings between boat- and land-based tracking for transmitters located a short distance to shore, it was assumed that the accuracy during the constant observation period post-release was also appropriate, since fish moved along the shoreline when released. Less regular tracking was conducted for a period of two months post-release to estimate long-term mortality. Moving fish between tracking dates were assumed alive since it is impossible that the large carp were predated by any predatory fish in the system.

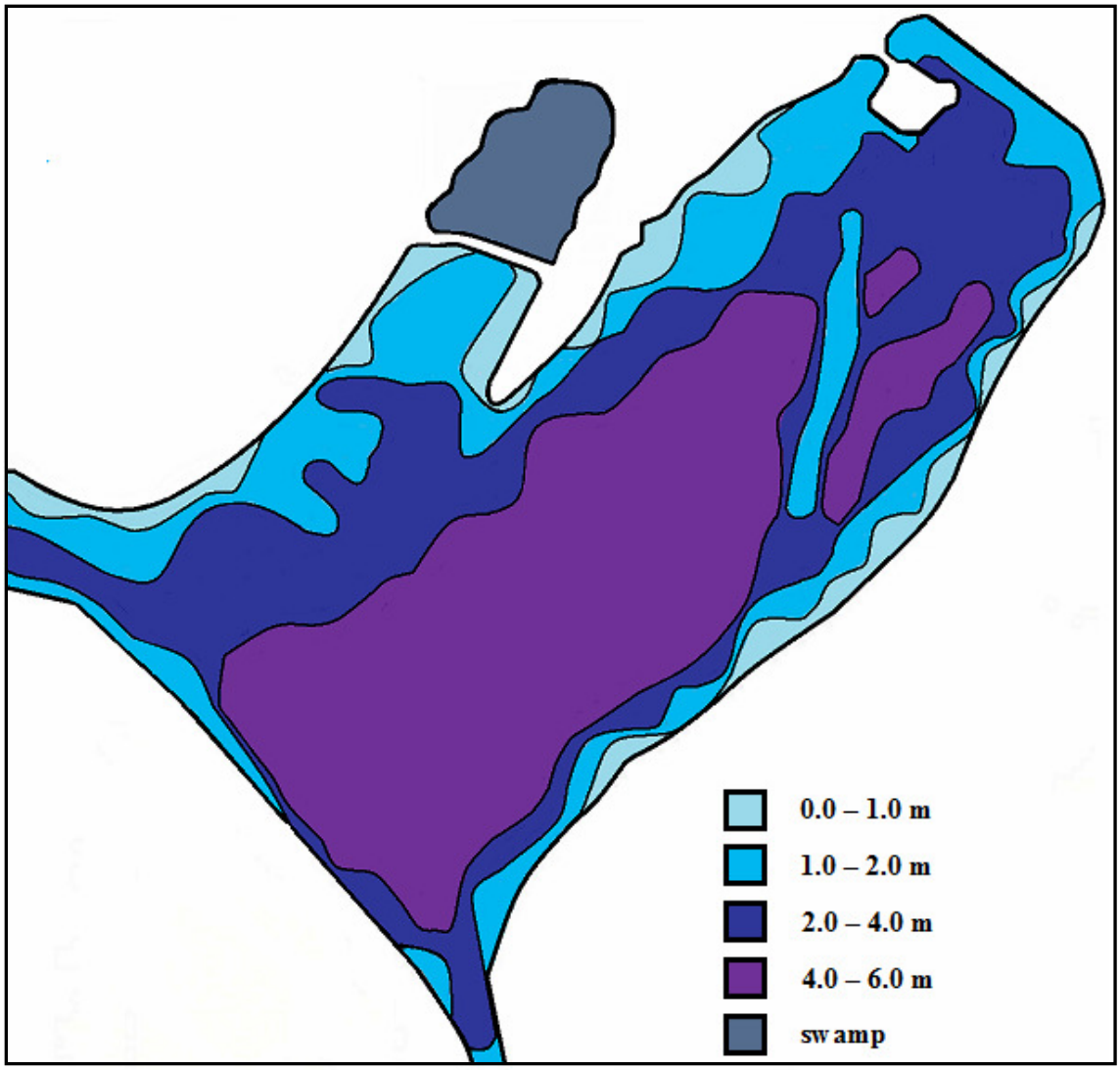

Figure 4: Depth map of Dow's Lake (modified from Fisheries and Oceans Canada 2005).

3.9 Statistical Analyses

Blood plasma samples of 8 fish showed signs of either haemolysis, icterus or lipemia which can interfere with analysis of blood plasma constituents and cause errors in test results (Bellamy & Olexson 2000). Consequently these fish were excluded from statistical analysis of haematological variables and sample sizes for physiological assessments deviate from the initially reported sample sizes (Table 3). Analysis of behavioural parameters included all fish (i.e. N = 10 fish per treatment group).

Table 3: Sample sizes of treatment groups included in the analyses of haematological parameters in response to retention or air exposure after exclusion of blood plasma samples that showed signs of haemolysis, icterus or lipemia.

Retention		Air exposure	
Treatment group	**Sample size**	**Treatment group**	**Sample size**
Control	9	**Control**	9
Retention 3 h	9	**Air exposure**	9
Retention 6 h	8	**Retention 9 h**	8
Retention 9 h	8	**Retention 9 h + air exposure**	9

Comparisons between groups were made using one-way analyses of variance (ANOVA) and Tukey *post-hoc* tests after verifying that data meet the assumptions for parametric tests. Normality was tested by using Kolmogorov-Smirnov tests and homogeneity of variances was tested with Levene's tests. In case of deviations from underlying assumptions a logarithmic transformation [ln (x + 1)] was applied. If the logarithmic transformation did not result in normal distribution the non-parametric Kruskal-Wallis-H test was used to compare treatment groups. In these cases, to identify which treatment groups differed from each other in significant models, multiple comparisons were conducted by non-parametric Nemenyi *post-hoc* tests (i.e. sample size independent Nemenyi *post-hoc* tests for comparisons of haematological data and sample size dependent Nemenyi *post-hoc* tests for comparisons of behavioural data) as described in Sachs & Hedderich (2006). If the logarithmic transformation did not result in variance homogeneity, differences between treatments groups were tested by one-way ANOVAs and an appropriate *post-hoc* test for inhomogeneous variances (Dunnett-T3) was applied.

Environmental variables (i.e. water temperature and dissolved oxygen) that could not be controlled in the field but might influence haematological variables, behaviour or survival of carp in response to retention and air exposure were compared separately for physiology and behaviour due to the differences in sample sizes occurring from exclusion of fish. Comparisons were made using one-way ANOVAs or Kruskal-Wallis-H tests as described before. Environmental parameters did not differ significantly between treatment groups (Table 4), indicating that carp in each treatment group experienced similar environmental conditions. Therefore, water temperature and

dissolved oxygen were not included in the final analyses of retention and air exposure effects on physiology and behaviour of carp.

Table 4: Test statistics of mean water temperature and dissolved oxygen at capture among treatment groups, each for physiological and behavioural assessments (ANOVA, $P < 0.05$ for all tests; Kruskal-Wallis-H, $P < 0.05$ for all tests).

Treatment	Investigation	Water parameter	F-value	Chi^2-value	df	P-value
Retention	Physiology	Temperature	0.672		3	0.567
		Dissolved oxygen		0.949	3	0.814
	Behaviour	Temperature	0.424		3	0.737
		Dissolved oxygen		1.919	3	0.589
Air exp.	Physiology	Temperature	0.055		3	0.983
		Dissolved oxygen		1.139	3	0.786
	Behaviour	Temperature		0.093	3	0.993
		Dissolved oxygen		1.252	3	0.741

To exclude an influence of fish length on haematological and behavioural parameters, treatment groups were compared using either one-way ANOVAs or Kruskal-Wallis-H tests as described before. Also fish length was analysed separately for haematological and behavioural parameters due to exclusion of fish. Significant differences were identified between retention treatment groups (physiology: ANOVA, $F = 3.563$, $df = 3$, $P = 0.026$, Tukey *post-hoc* test; behaviour: ANOVA, $F = 3.952$, $df = 3$, $P = 0.016$, Tukey *post-hoc* test; Table 5), while no differences in fish length between air exposure treatments were observed (physiology: Kruskal-Wallis-H, $Chi^2 = 3.304$, $df = 3$, $P = 0.347$; behaviour: ANOVA, $F = 1.278$, $df = 3$, $P = 0.296$; Table 5). Subsequent analyses of covariance (ANCOVA) for parametric data or generalised linear models (GLM) for non-parametric data revealed no significant influence of fish length as a covariate on haematological variables and the behavioural parameters of distance moved, time rested, and minimum displacement (Table 6). Cox regression models revealed no significant influence of fish length as a covariate on time required to leave the release site (Table 6). Since fish length did not influence stress response in carp following retention and mean fish length did not differ significantly between air exposure treatments, it was not included in the final analyses of retention and air exposure effects on physiology and behaviour of carp.

Haematological variables, and the behavioural parameters distance moved, time rested and minimum displacement were compared between treatment groups by either using ANOVAs or Kruskal-Wallis-H tests as described before. Time required to leave the release site served as an indicator to assess the point in time when fish resume swimming post-release. The release site was defined as a semi-circle with a radius of 10 m around the specific point where the fish were released and was compared among treatment groups overall by using Kaplan-Meier analyses with Breslow statistics. All analyses were conducted using the software package SPSS (SPSS Inc., Chicago, Illinois, U.S.A., Version: 15.0) and significance was judged at $P < 0.05$. Results are presented as mean ± standard error (SE). All presented results are non-transformed values to improve interpretation.

Table 5: Comparison of mean fish length (mm) among treatment groups in response to retention and air exposure for physiological and behavioural assessments. Different letters indicate significant differences (ANOVA, $P < 0.05$ for all tests; Kruskal-Wallis-H, $P < 0.05$ for all tests).

Treatment	Investigation	Treatment group	Mean length ± SD (mm)
Retention	Physiology	Control	643 ± 73^{ab}
		3 h	736 ± 88^{b}
		6 h	631 ± 91^{a}
		9 h	704 ± 55^{ab}
	Behaviour	Control	644 ± 69^{a}
		3 h	736 ± 83^{b}
		6 h	645 ± 86^{a}
		9 h	709 ± 53^{ab}
Air exposure	Physiology	Control	643 ± 73
		Air exposure	697 ± 111
		Retention 9 h	704 ± 55
		Retention 9 h + air exposure	700 ± 83
	Behaviour	Control	644 ± 69
		Air exposure	690 ± 107
		Retention 9 h	709 ± 53
		Retention 9 h + air exposure	697 ± 79

Table 6: Influence of fish length as a covariate in ANCOVAs or GLMs on haematological variables and the behavioural parameters distance moved, time rested and minimum displacement in response to retention. Influence of fish length as a covariate in the Cox regression model on the behavioural parameter time required to leave the release site in response to retention. The P-value refers to the significance of fish length. The influence of fish length was not significant (ANCOVA, $P < 0.05$ for all tests; GLM, $P < 0.05$ for all tests; Cox regression model, $P < 0.05$).

Haematological parameter	**P-value**
Blood plasma cortisol (ng/mL)	0.911
Blood plasma glucose (mmol/L)	0.855
Blood plasma lactate (mmol/L)	0.844
Blood plasma osmolality (mOsmol/kg)	0.797
Blood plasma sodium (mmol/L)	0.843
Blood plasma chloride (mmol/L)	0.474
Blood plasma potassium (mmol/L)	0.398
Blood plasma LDH (U/L)	0.847
Blood plasma AST (U/L)	0.902
Behavioural parameter	**P-value**
Distance moved 0 - 30 min (m)	0.626
Time required to leave the release site (min)	0.890
Time rested 0 - 30 min post-release (min)	0.993
Minimum displacement 31 - 60 min (m)	0.403
Minimum displacement 1 - 12 h post-release (m)	0.885
Minimum displacement 12 - 24 h post-release (m)	0.252
Minimum displacement 24 - 36 h post-release (m)	0.866
Minimum displacement 36 - 48 h post-release (m)	0.508
Minimum displacement 48 - 60 h post-release (m)	0.130
Minimum displacement 60 - 72 h post-release (m)	0.747

4. Results

4.1 Influence of Retention on Physiological Status of Carp

All carp retained in carp sacks exhibited significantly higher blood plasma cortisol concentrations relative to control fish (ANOVA, F = 19.782, df = 3, P < 0.001, Dunnett-T3 *post-hoc* test; Figure 5). Retention duration did not significantly influence the magnitude of cortisol increase, but there was a clear trend of mean blood plasma cortisol concentrations rising with retention duration.

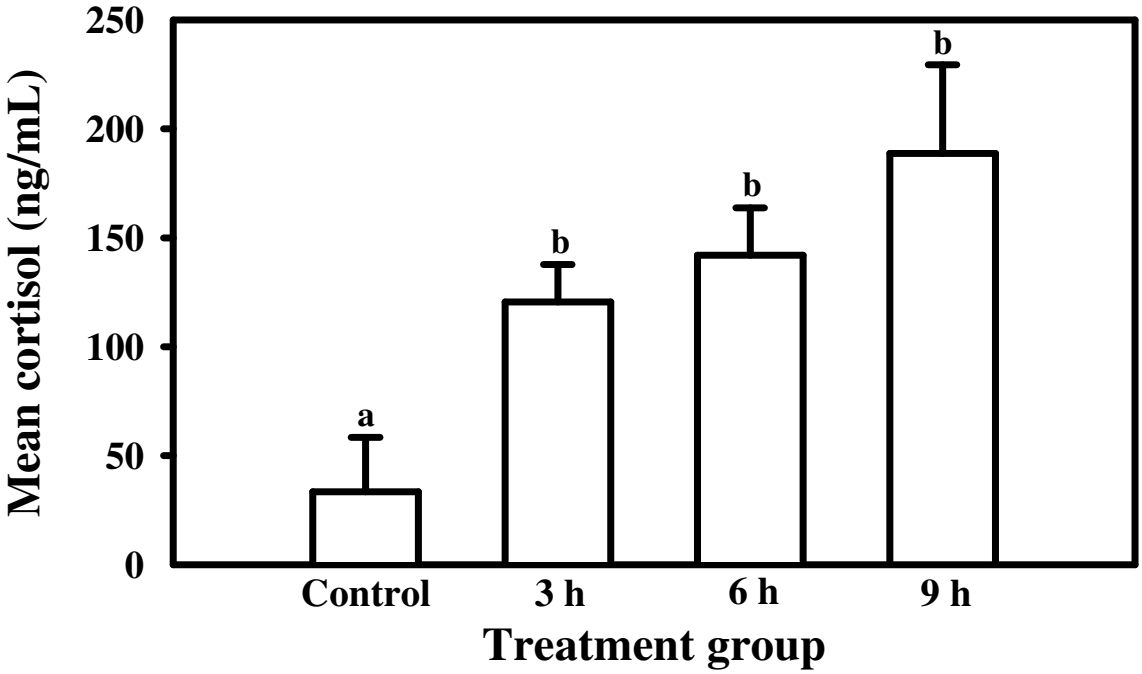

Figure 5: Mean blood plasma cortisol concentrations ± SE (ng/mL) in control fish and carp retained in carp sacks for up to 9 h. Different letters indicate significant differences between treatment groups (ANOVA, F = 19.782, df = 3, P < 0.001, Dunnett-T3 *post-hoc* test).

In terms of indicators of metabolic changes, mean concentrations of blood plasma glucose tended to increase with retention, and they significantly differed between carp retained for 9 h and control fish (ANOVA, F = 3.554, df = 3, P = 0.026, Tukey *post-hoc* test; Figure 6). Mean blood plasma lactate concentrations were significantly lower in fish retained for 9 h relative to control fish and carp retained for 3 h (ANOVA, F = 10.468, df = 3, P < 0.001, Dunnett-T3 *post-hoc* test; Figure 7). In addition, mean blood plasma lactate was significantly lower in fish retained for 6 h relative to carp retained for 3 h (Figure 7).

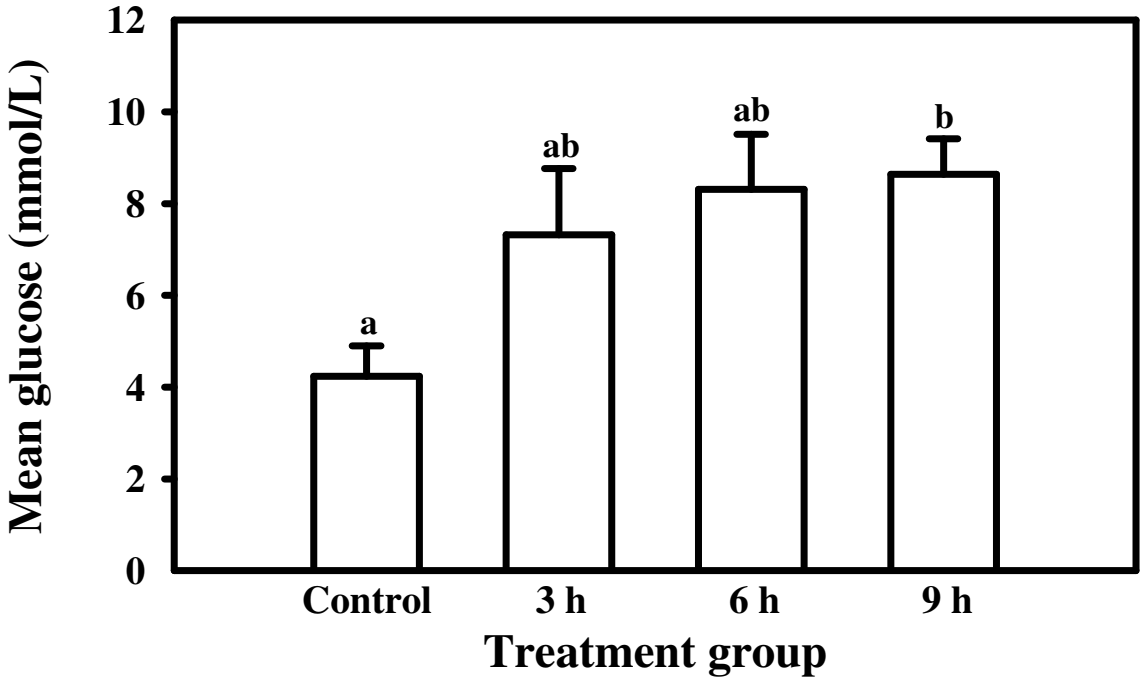

Figure 6: Mean blood plasma glucose concentrations ± SE (mmol/L) in control fish and carp retained in carp sacks for up to 9 h. Different letters indicate significant differences between treatment groups (ANOVA, F = 3.554, df = 3, P = 0.026, Tukey *post-hoc* test).

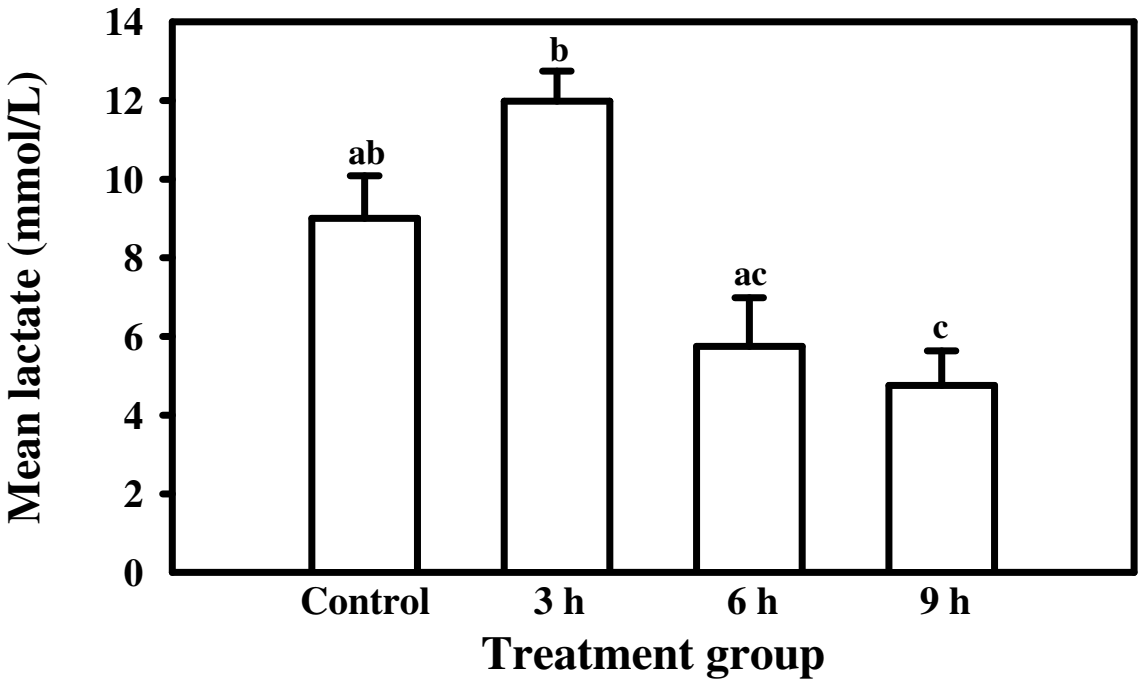

Figure 7: Mean blood plasma lactate concentrations ± SE (mmol/L) in control fish and carp retained in carp sacks for up to 9 h. Different letters indicate significant differences between treatment groups (ANOVA, F = 10.468, df = 3, P < 0.001, Dunnett-T3 *post-hoc* test).

Retention in carp sacks did not result in a significant alteration of mean blood plasma osmolality (Kruskal-Wallis-H, $Chi^2 = 0.291$, df = 3, P = 0.962; Figure 8), but analysis of mean blood plasma electrolyte concentrations revealed significant differences between treatment groups. Carp retained for 6 h and 9 h in carp sacks exhibited a significant decrease in mean blood plasma sodium concentrations relative to control fish (ANOVA, F = 7.284, df = 3, P = 0.001, Dunnett-T3 *post-hoc* test; Figure 9). Average blood plasma chloride concentrations dropped significantly in carp retained for 3 h relative to control (Kruskal-Wallis-H, $Chi^2 = 8.735$, df = 3, P = 0.033, Nemenyi *post-hoc* test; Figure 10), and mean blood plasma potassium concentration was significantly higher in fish retained for 6 h relative to control fish (ANOVA, F = 3.271, df = 3, P = 0.035, Tukey *post-hoc* test; Figure 11).

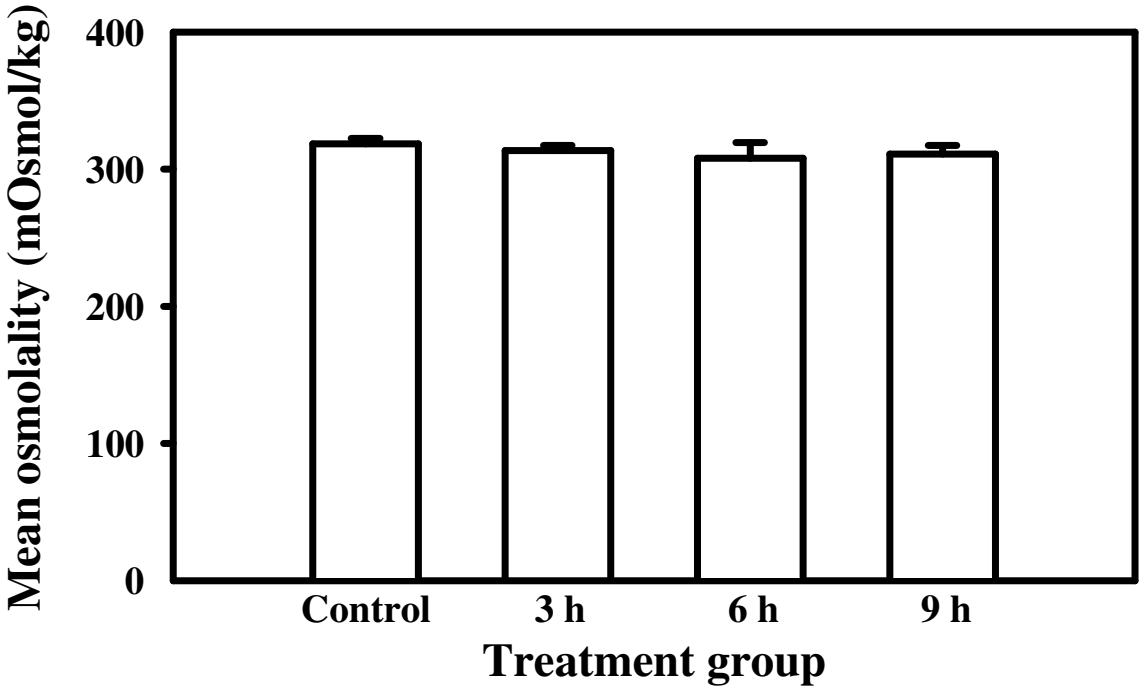

Figure 8: Mean blood plasma osmolality concentrations ± SE (mOsmol/kg) in control fish and carp retained in carp sacks for up to 9 h. Differences were not significant (Kruskal-Wallis-H, $Chi^2 = 0.291$, df = 3, P = 0.962).

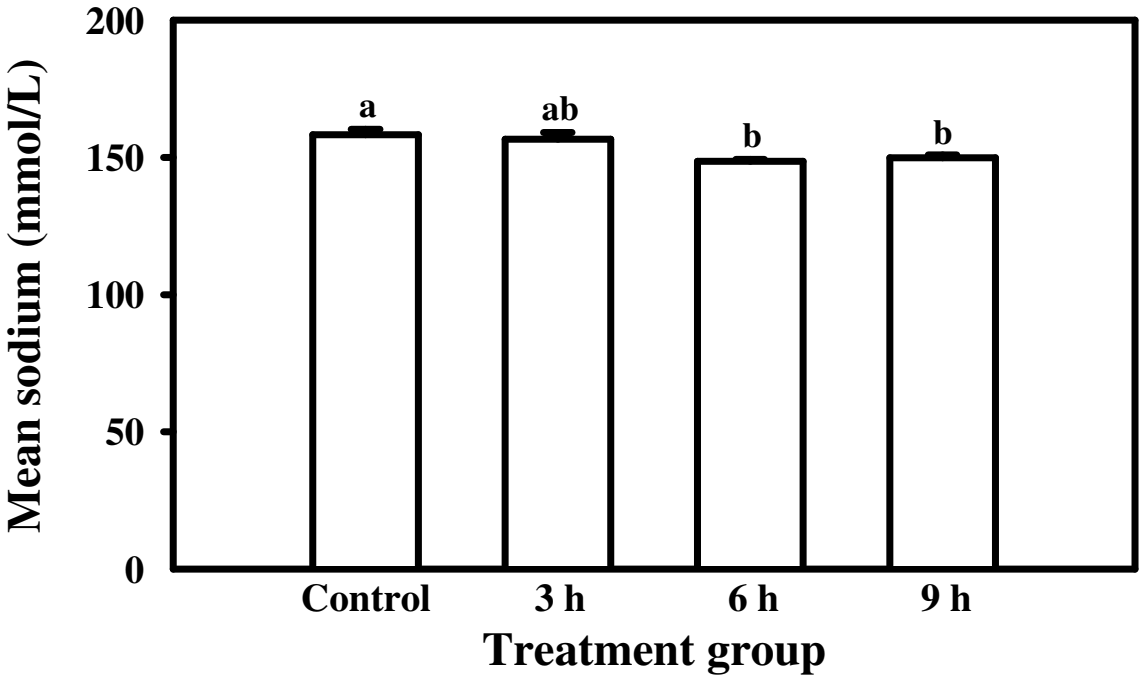

Figure 9: Mean blood plasma sodium concentrations ± SE (mmol/L) in control fish and carp retained in carp sacks for up to 9 h. Different letters indicate significant differences between treatment groups (ANOVA, F = 7.284, df = 3, P = 0.001, Dunnett-T3 *post-hoc* test).

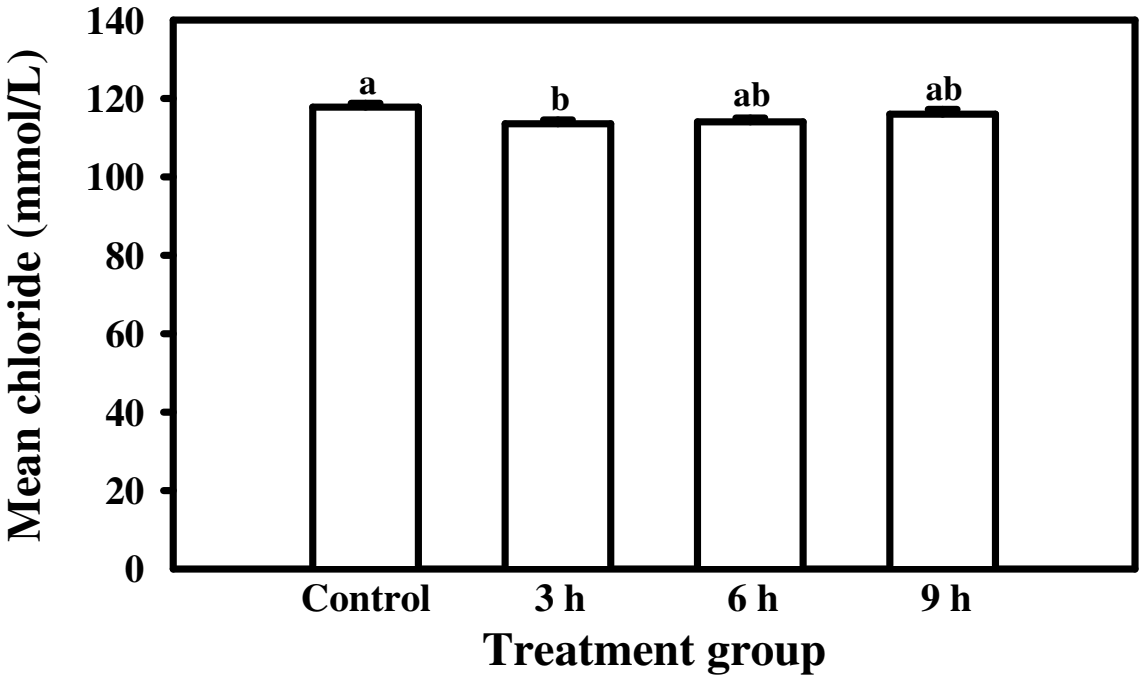

Figure 10: Mean blood plasma chloride concentrations ± SE (mmol/L) in control fish and carp retained in carp sacks for up to 9 h. Different letters indicate significant differences between treatment groups (Kruskal-Wallis-H, Chi^2 = 8.735, df = 3, P = 0.033, Nemenyi *post-hoc* test).

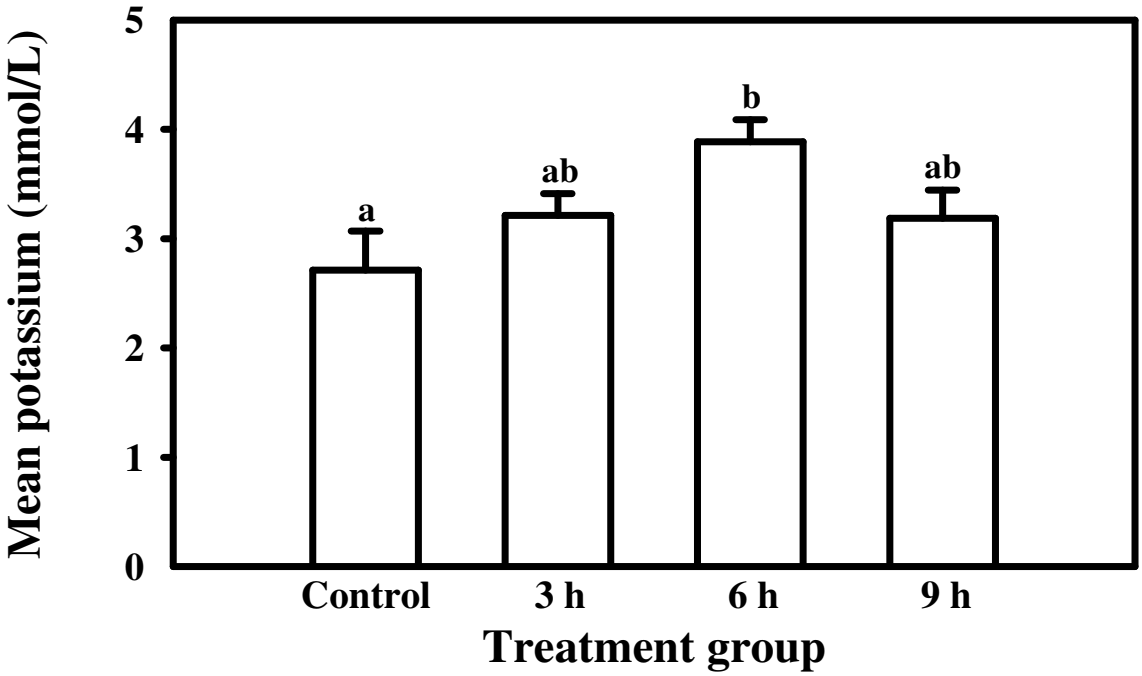

Figure 11: Mean blood plasma potassium concentrations ± SE (mmol/L) in control fish and carp retained in carp sacks for up to 9 h. Different letters indicate significant differences between treatment groups (ANOVA, F = 3.271, df = 3, P = 0.035, Tukey *post-hoc* test).

In terms of indicators of tissue damage, mean blood plasma LDH concentrations were significantly higher for fish retained for 9 h relative to control (ANOVA, F = 3.495, df = 3, P = 0.028, Tukey *post-hoc* test; Figure 12). Shorter retention durations did not significantly affect blood plasma LDH values. Mean blood plasma AST concentrations tended to increase with retention duration and carp retained for 9 h showed significantly higher mean AST concentrations in the blood plasma relative to the control (ANOVA, F = 4.834, df = 3, P = 0.007, Tukey post-hoc test; Figure 13).

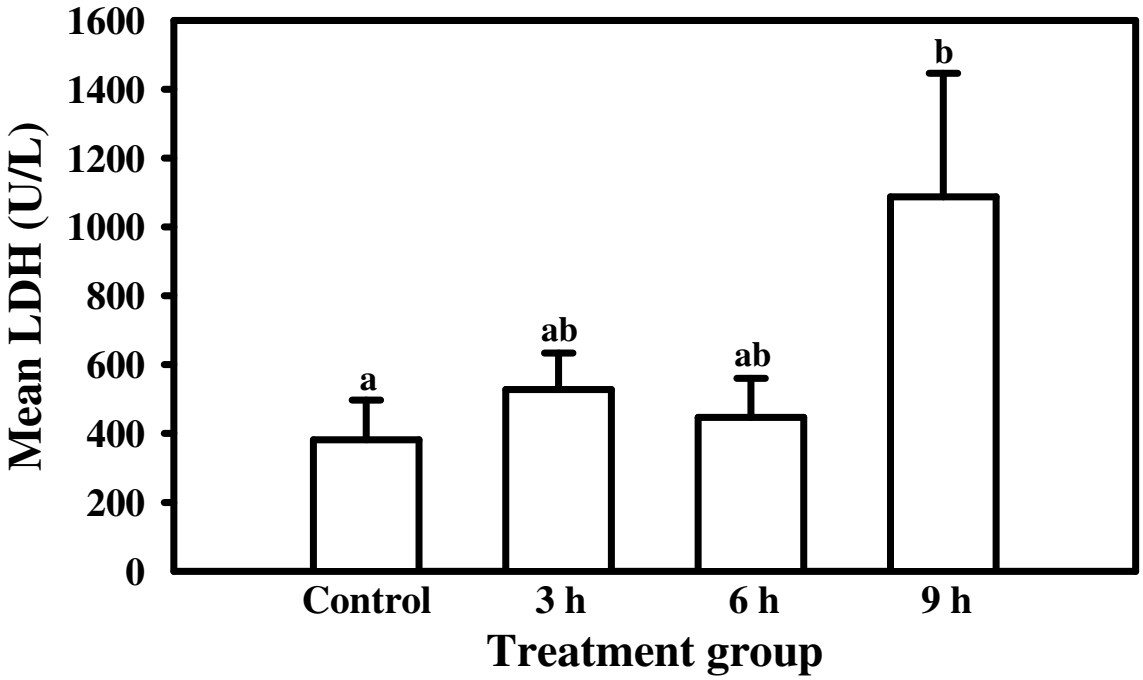

Figure 12: Mean blood plasma LDH concentrations ± SE (U/L) in control fish and carp retained in carp sacks for up to 9 h. Different letters indicate significant differences between treatment groups (ANOVA, F = 3.495, df = 3, P = 0.028, Tukey *post-hoc* test).

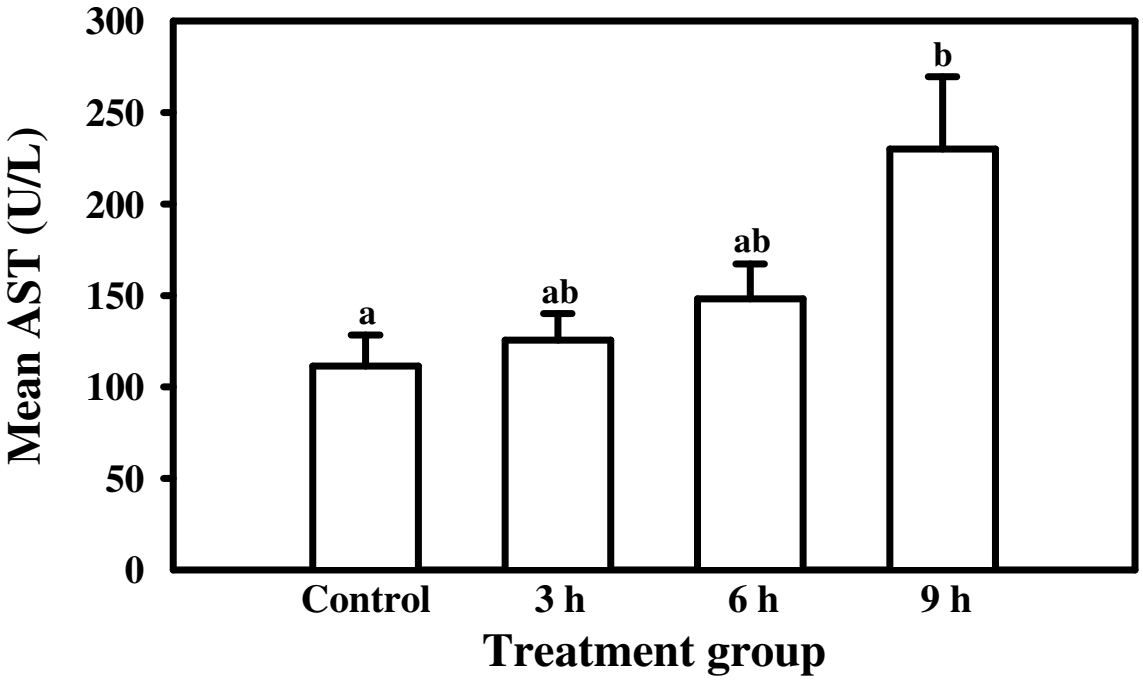

Figure 13: Mean blood plasma AST concentrations ± SE (U/L) in control fish and carp retained in carp sacks for up to 9 h. Different letters indicate significant differences between treatment groups (ANOVA, F = 4.834, df = 3, P = 0.007, Tukey *post-hoc* test).

4.2 Influence of Retention on Behaviour and Survival of Carp

Mean distance moved by carp did not vary significantly among treatment groups within 30 min post-release (ANOVA, F = 1.750, df = 3, P = 0.174; Figure 14), and no significant differences in patterns leaving the release site were observed between treatment groups (Kaplan-Meier, Breslow, Chi^2 = 2.14, df = 3, P = 0.54; Figure 15). However, there was a trend that carp retained for 6 and 9 h moved less compared to control and carp retained for only 3 h (Figure 14), and fish retained for 3 h tended to also spend less time at the release site relative to all other treatments (Figure 15). Finally, fish retained for long periods of 9 h were found to spend significantly more time resting relative to fish retained for short periods of only 3 h (Kruskal-Wallis-H, Chi^2 = 8.225, df = 3, P = 0.042, Nemenyi *post-hoc* test; Figure 16).

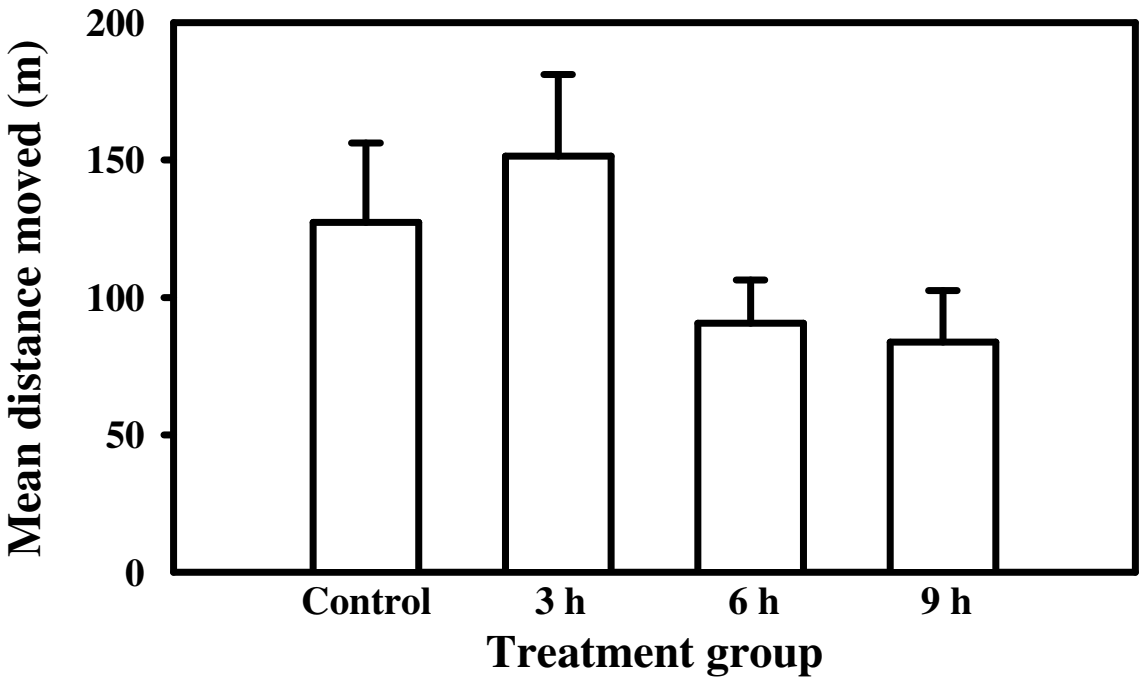

Figure 14: Mean distance moved ± SE (m) from 0 to 30 min post-release in control fish and carp retained in carp sacks for up to 9 h. Differences were not significant (ANOVA, F = 1.750, df = 3, P = 0.174).

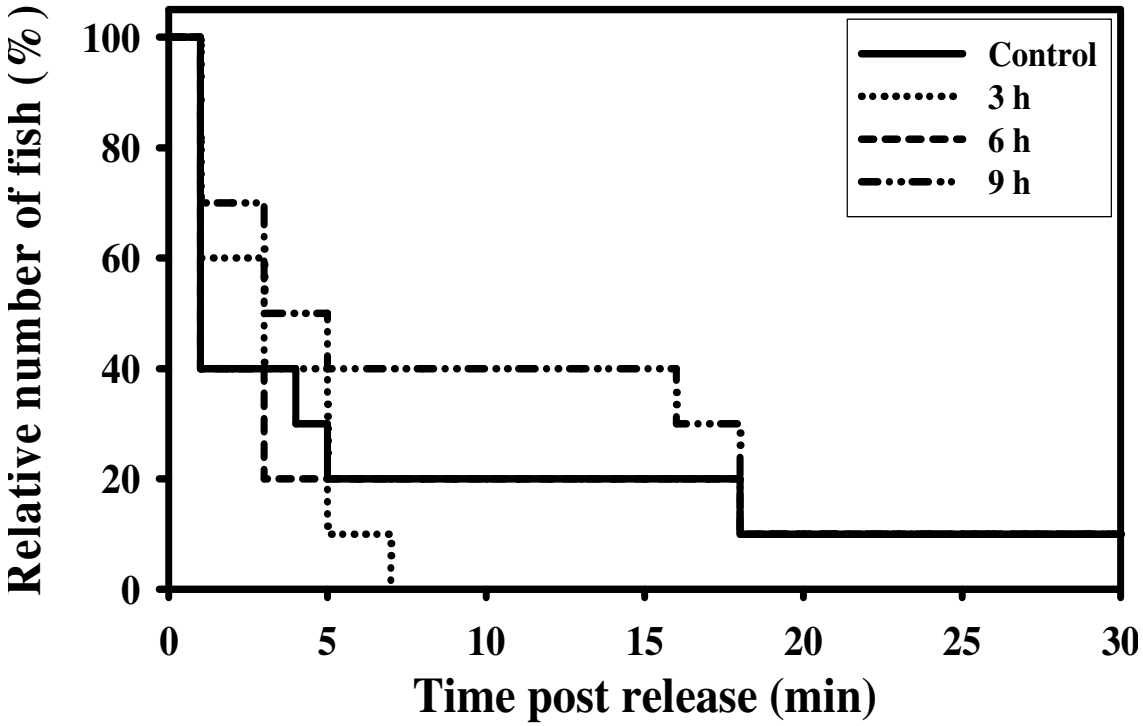

Figure 15: Relative number of fish at the release site (%) plotted against the time post-release (min) for control fish and carp retained in carp sack for up to 9 h. Differences were not significant (Kaplan-Meier, Breslow, $Chi^2 = 2.14$, df = 3, P = 0.543).

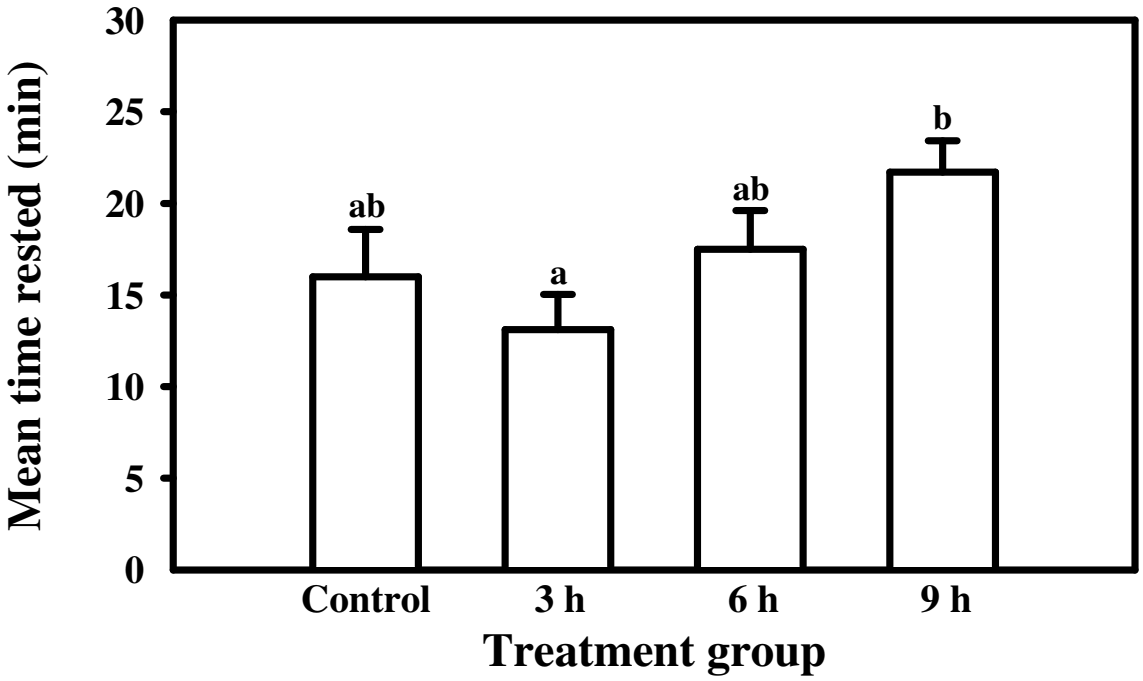

Figure 16: Mean time rested ± SE (min) from 0 to 30 min post-release in control fish and carp retained in carp sacks for up to 9 h. Different letters indicate significant differences between treatment groups (Kruskal-Wallis-H, $Chi^2 = 8.225$, df = 3, P = 0.042, Nemenyi *post-hoc* test).

From 31 min to 60 min post-release, fish retained for 9 h moved significantly less relative to control fish (ANOVA, 3.438, df = 3, P = 0.027, Tukey *post-hoc* test; Figure 17). Short- and medium-term retention periods did not result in significantly reduced mean minimum displacement. However, there was a trend that carp retained for these periods also exhibited reduced movement activity relative to control fish (Figure 17). Mean minimum displacement of carp during subsequent tracking intervals after the first hour post-release did not differ significantly between treatment groups indicating reversed behavioural patterns (Table 7). In a 2 month observation period, no initial and delayed mortality was observed and hooking and retention mortality was 0 %.

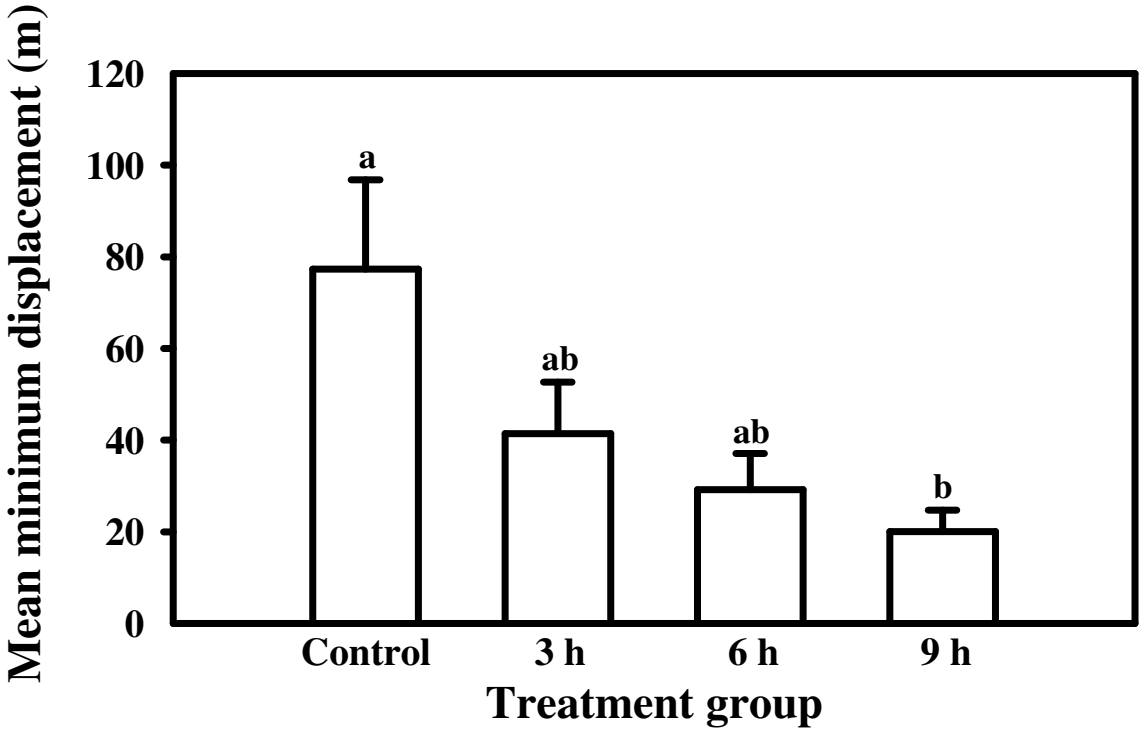

Figure 17: Mean minimum displacement ± SE (m) from 31 to 60 min post-release in control fish and carp retained in carp sacks for up to 9 h. Different letters indicate significant differences between treatment groups (ANOVA, 3.438, df = 3, P = 0.027, Tukey *post-hoc* test).

Table 7: Mean minimum displacement (MDP) (m) in control fish and carp retained in carp sacks for up to 9 h from 12 h to 72 h post-release. Differences were not significant (ANOVA, $P < 0.05$ for all tests; Kruskal-Wallis-H, $P < 0.05$ for all tests).

Time post-release (h)	**Mean MDP (m) Control (± SE)**	**Mean MDP (m) 3 h (± SE)**	**Mean MDP (m) 6 h (± SE)**	**Mean MDP (m) 9 h (± SE)**	**F-value/ Chi^2-value; df; P-value**
1 – 12	346.57 (± 60.28)	301.49 (± 41.61)	197.95 (± 61.56)	278. 62 (± 53.80)	$Chi^2 = 3.120$; df = 3; P = 0.373
12 – 24	287.94 (± 91.60)	349.85 (± 95.39)	237.16 (± 53.99)	271.78 (± 48.26)	F = 0.138; df = 3; P = 0.936
24 – 36	220.61 (± 55.62)	172.59 (± 47.60)	182.97 (± 37.56)	305.36 (± 62.15)	F = 1.386; df = 3; P = 0.267
36 – 48	158.65 (± 27.03)	228.08 (± 65.07)	155.32 (± 41.23)	248.36 (± 42.81)	F = 1.090; df = 3; P = 0.369
48 – 60	306.09 (± 65.44)	244.20 (± 52.11)	253.38 (± 70.69)	201.90 (± 65.62)	F = 0.458; df = 3; P = 0.714
60 – 72	265.14 (± 59.26)	177.77 (± 43.67)	190.14 (± 57.72)	201.76 (± 77.46)	F = 0.410; df = 3; P = 0.747

4.3 Influence of Air Exposure on Physiological Status of Carp

Analysis of blood plasma cortisol revealed no differences between air exposure treatments relative to directly comparable treatment groups without air exposure (Kruskal-Wallis-H, Chi^2 = 15.281, df = 3, P = 0.002, Nemenyi *post-hoc* test; Figure 18). Statistical differences are a result of underlying retention in carp sacks as indicated by lack of statistical differences between directly comparable treatment groups (i.e. air exposure of 10 minute relative to control, and air exposure of 10 minutes following 9 h of retention relative to 9 h retention only; Figure 18).

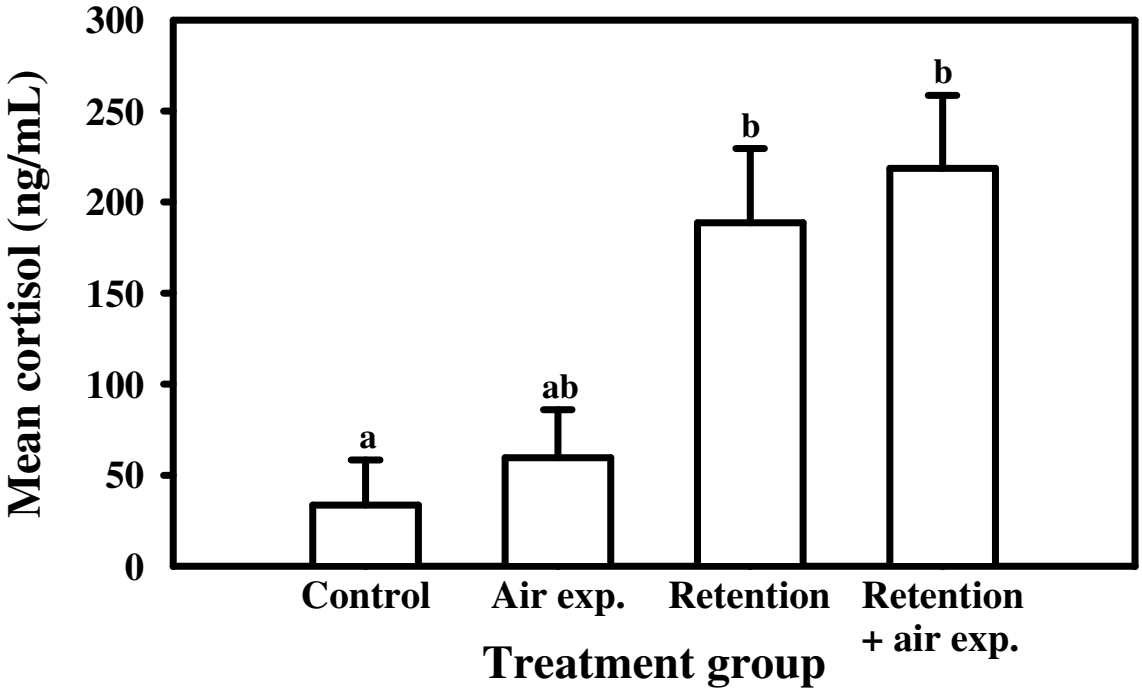

Figure 18: Mean blood plasma cortisol concentrations ± SE (ng/mL) in carp without and with air exposure of 10 min and either not retained in carp sacks or retained for 9 h. Different letters indicate significant differences between treatment groups (Kruskal-Wallis-H, Chi^2 = 15.281, df = 3, P = 0.002, Nemenyi *post-hoc* test).

Similarly, mean blood plasma glucose concentrations were not affected by air exposure (ANOVA, F = 15.971, df = 3, P < 0.001, Tukey *post-hoc* test; Figure 19). The lack of statistical differences among directly comparable treatment groups indicate that statistical significance is due to retention and not caused by air exposure. Air exposure following capture did not significantly alter mean blood plasma lactate concentrations relative to control, but air exposure following 9 h of retention caused a significant increase in blood plasma lactate compared to fish retained only (ANOVA, F = 10.348, df = 3, P < 0.001, Dunnett-T3 *post-hoc* test; Figure 20).

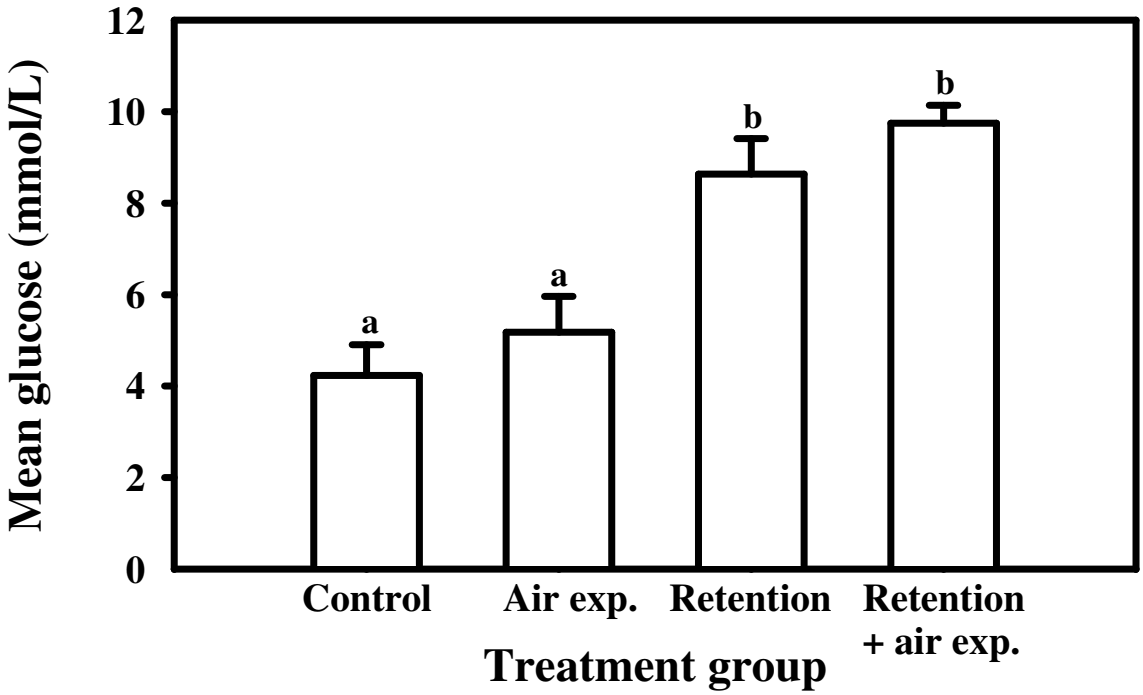

Figure 19: Mean blood plasma glucose concentrations ± SE (mmol/L) in carp without and with air exposure of 10 min and either not retained in carp sacks or retained for 9 h. Different letters indicate significant differences between treatment groups (ANOVA, $F = 15.971$, $df = 3$, $P < 0.001$, Tukey *post-hoc* test).

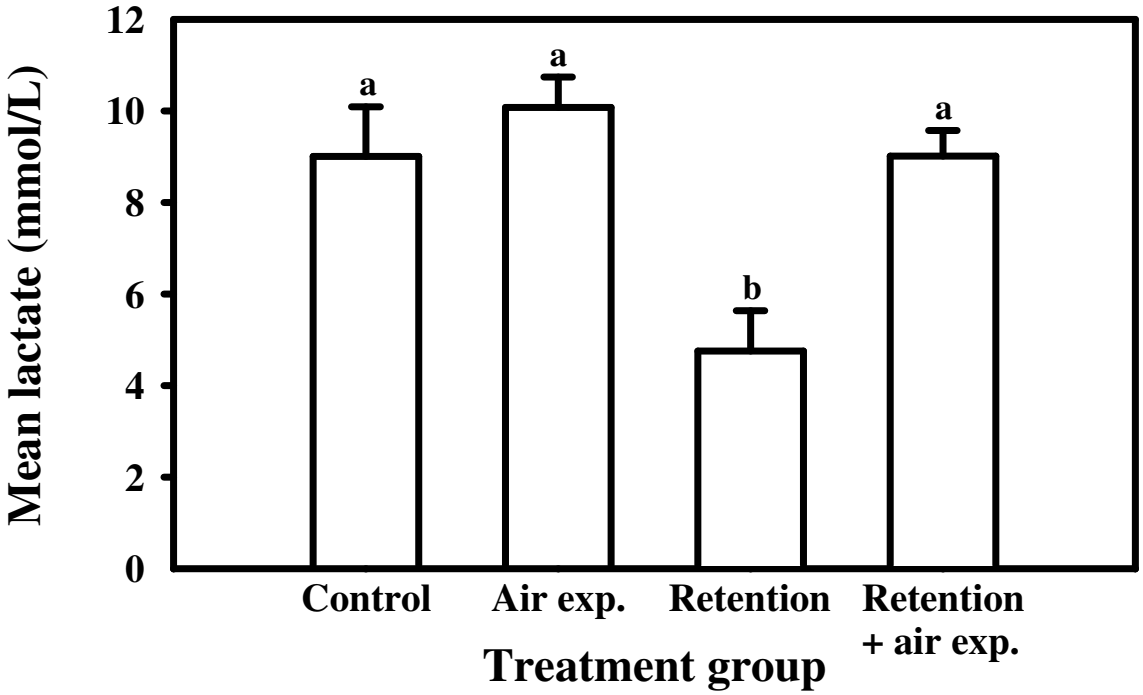

Figure 20: Mean blood plasma lactate concentrations ± SE (mmol/L) in carp without and with air exposure of 10 min and either not retained in carp sacks or retained for 9 h. Different letters indicate significant differences between treatment groups (ANOVA, $F = 10.348$, $df = 3$, $P < 0.001$, Dunnett-T3 *post-hoc* test).

Air exposure following capture or 9 h of retention did neither significantly affect mean blood plasma osmolality concentrations (Kruskal-Wallis-H, Chi^2 = 0.602, df = 3, P = 0.896; Figure 21), nor mean blood plasma sodium (ANOVA, F = 10.072, df = 3, P < 0.001, Tukey *post-hoc* test; Figure 22), mean blood plasma chloride (Kruskal-Wallis-H, Chi^2 = 14.337, df = 3, P = 0.002, Nemenyi *post-hoc* test; Figure 23) and mean blood plasma potassium concentrations (Kruskal-Wallis-H, F = 5.043, df = 3, P = 0.169; Figure 24) of carp relative to appropriate controls. The pattern in decreases in blood plasma sodium and blood plasma chloride between retained and non-retained carp indicate that significant differences are due to retention and not caused by air exposure.

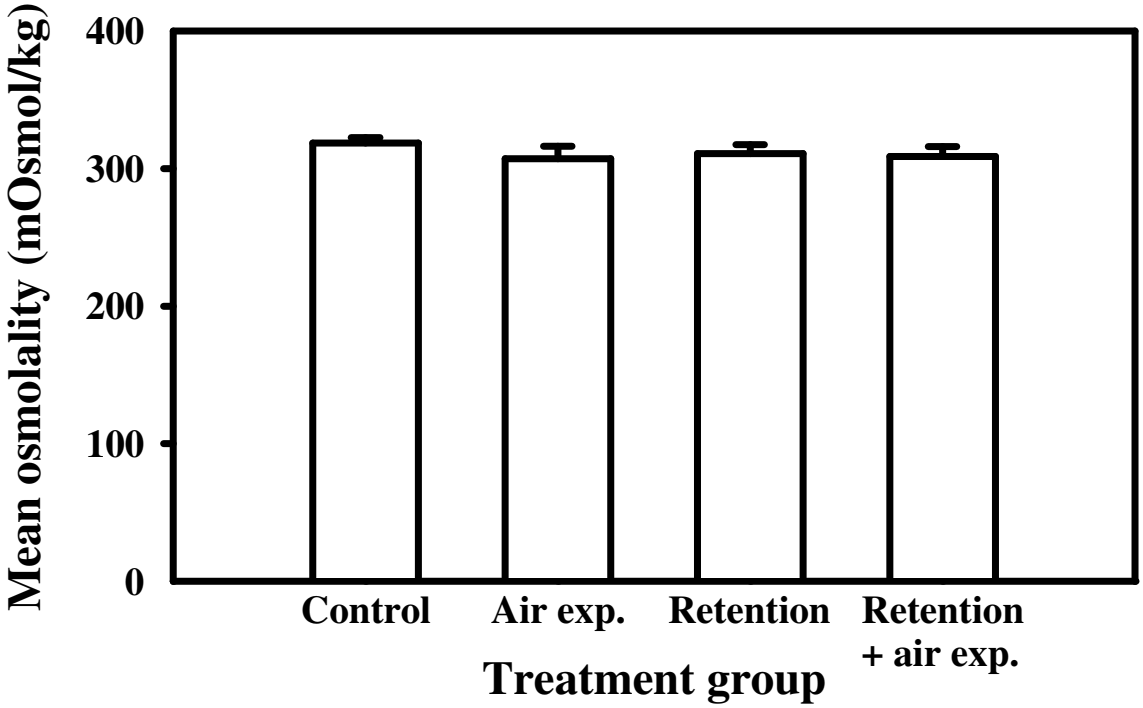

Figure 21: Mean blood plasma osmolality concentrations ± SE (mOsmol/kg) in carp without and with air exposure of 10 min and either not retained in carp sacks or retained for 9 h. Differences were not significant (Kruskal-Wallis-H, Chi^2 = 0.602, df = 3, P = 0.896).

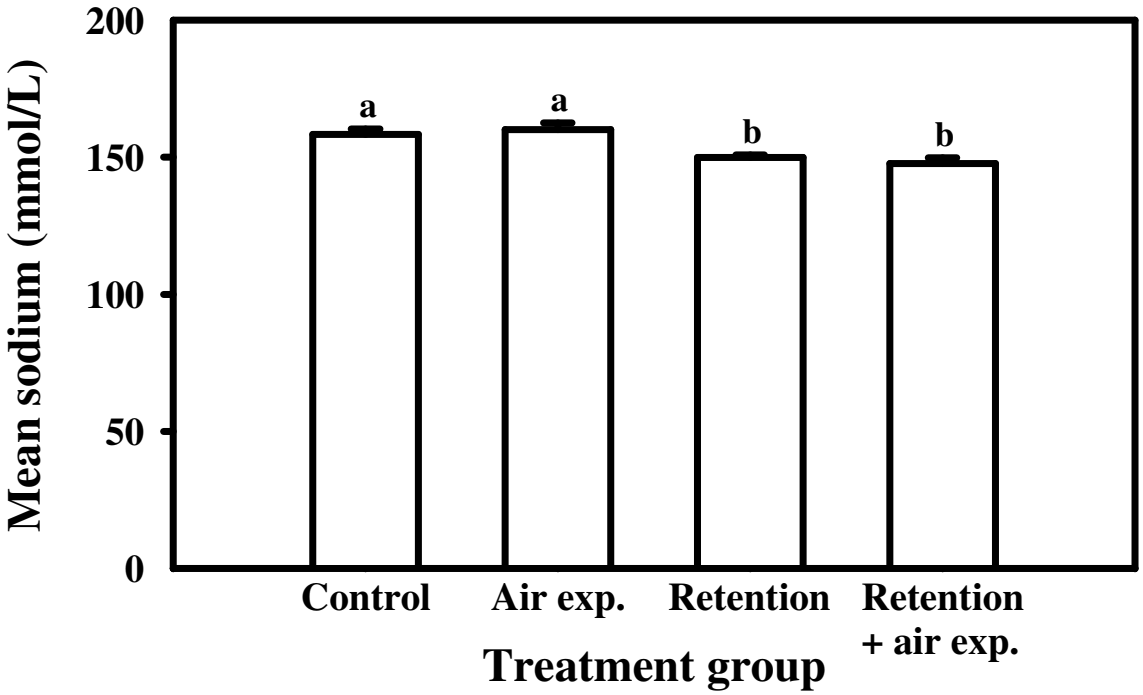

Figure 22: Mean blood plasma sodium concentrations ± SE (mmol/L) in carp without and with air exposure of 10 min and either not retained in carp sacks or retained for 9 h. Different letters indicate significant differences between treatment groups (ANOVA, $F = 10.072$, $df = 3$, $P < 0.001$, Tukey *post-hoc* test).

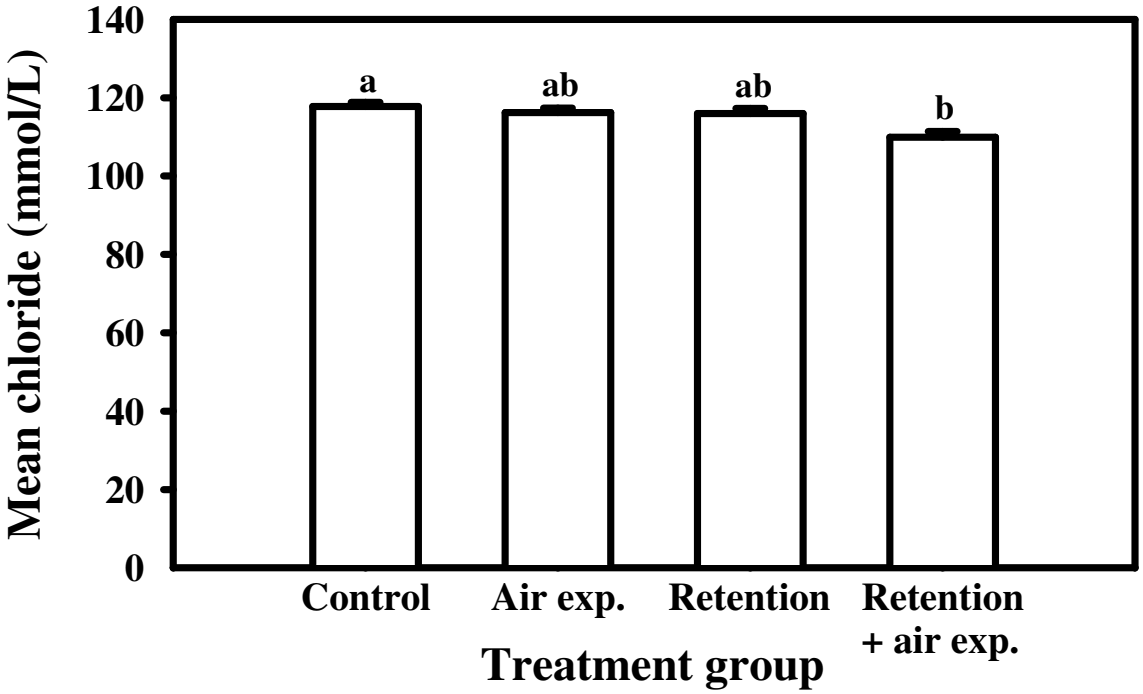

Figure 23: Mean blood plasma chloride concentrations ± SE (mmol/L) in carp without and with air exposure of 10 min and either not retained in carp sacks or retained for 9 h. Different letters indicate significant differences between treatment groups (Kruskal-Wallis-H, $Chi^2 = 14.337$, $df = 3$, $P = 0.002$, Nemenyi *post-hoc* test).

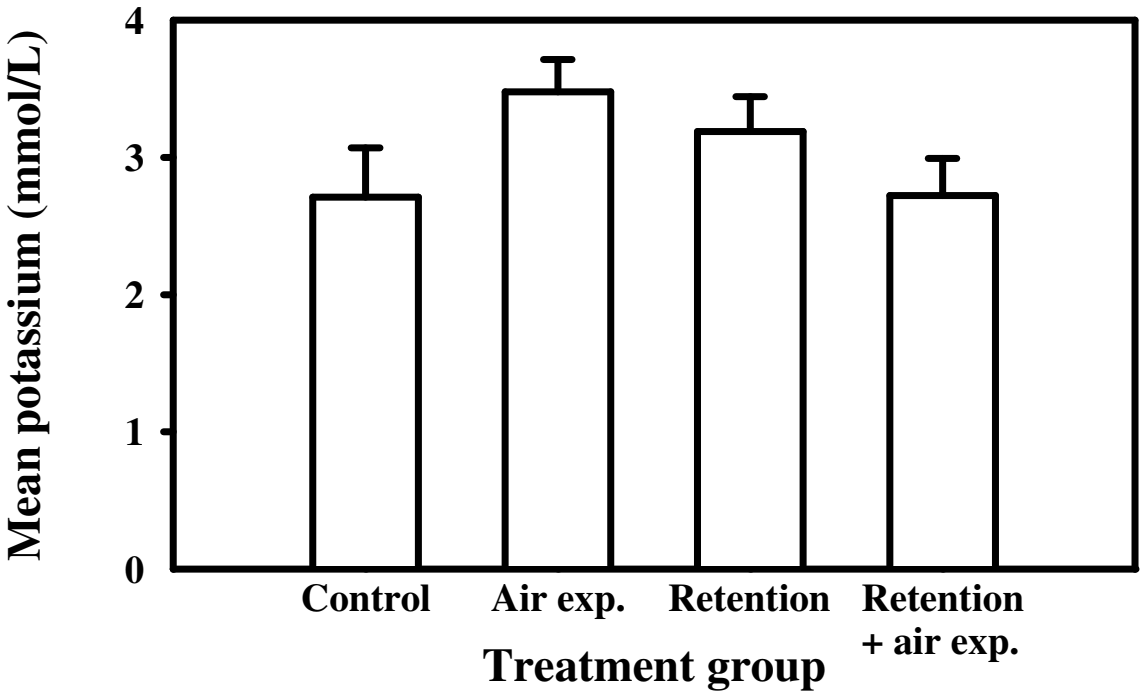

Figure 24: Mean blood plasma potassium concentrations ± SE (mmol/L) in carp without and with air exposure of 10 min and either not retained in carp sacks or retained for 9 h. Differences were not significant (Kruskal-Wallis-H, F = 5.043, df = 3, P = 0.169).

ANOVAs revealed no significant differences in mean blood plasma LDH concentrations (ANOVA, F = 2.533, df = 3, P = 0.075; Figure 25) and mean blood plasma AST concentrations (ANOVA, F = 3.393, df = 3, P = 0.030, Tukey *post-hoc* test; Figure 26). Significant differences of blood plasma AST are a result underlying retention in carp sacks and do not result from differences between air exposed fish and appropriate controls.

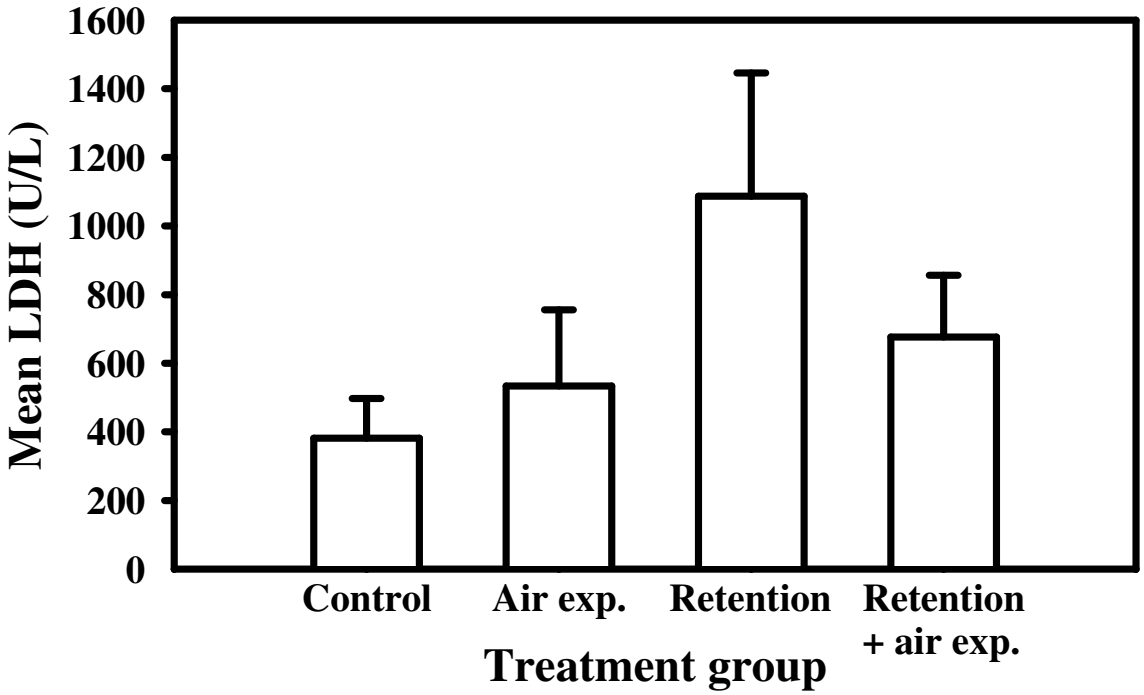

Figure 25: Mean blood plasma LDH concentrations ± SE (U/L) in carp without and with air exposure of 10 min and either not retained in carp sacks or retained for 9 h. Differences were not significant (ANOVA, F = 2.533, df = 3, P = 0.075).

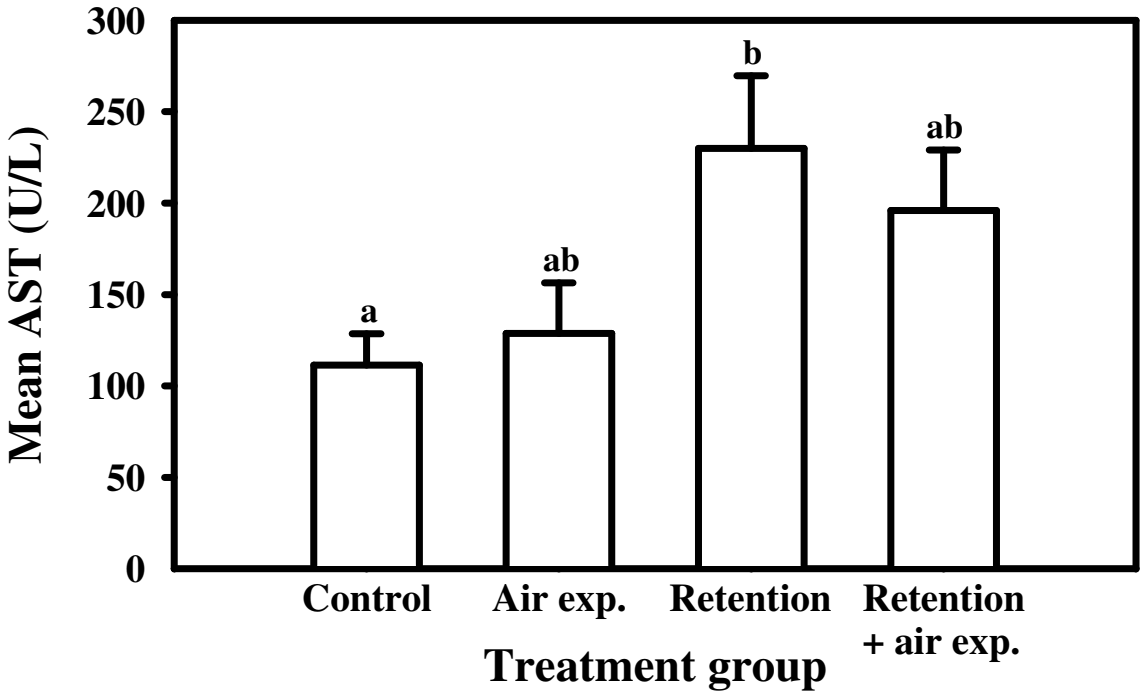

Figure 26: Mean blood plasma AST concentrations ± SE (U/L) in carp without and with air exposure of 10 min and either not retained in carp sacks or retained for 9 h. Different letters indicate significant differences between treatment groups (ANOVA, F = 3.393, df = 3, P = 0.030, Tukey *post-hoc* test).

4.4 Influence of Air Exposure on Behaviour and Survival of Carp

Although not significant, there was a trend that air exposure of 10 min decreased movement activity of carp within the first 30 min post-release relative to directly comparable treatment groups (ANOVA, F = 2.601, df = 3, P = 0.067; Figure 27), and treatment groups showed differences in patterns leaving the release site (Kaplan-Meier, Breslow, Chi^2 = 9.46, df = 3, P = 0.024; Figure 28). Thirty percent of fish exposed to air after capture and 50 % of fish air exposed following retention did not leave the release site compared to 10 % of fish in directly comparable treatment groups (Figure 28). However, overall mean time rested within the first 30 min post-release did not differ significantly between air exposure treatments and appropriate controls (ANOVA, 3.813, df = 3, P = 0.018, Dunnett-T3 *post-hoc* test, significant differences were not between directly comparable treatment groups; Figure 29).

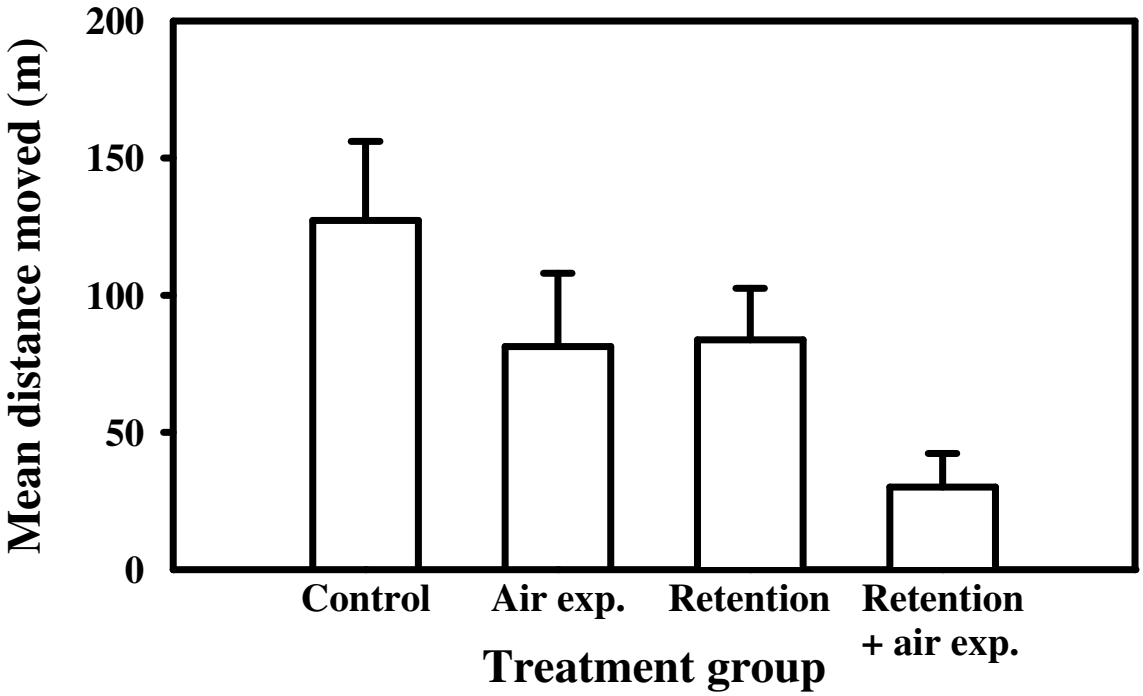

Figure 27: Mean distance moved ± SE (m) from 0 to 30 min post-release in carp without and with air exposure of 10 min and either not retained in carp sacks or retained for 9 h. Differences were not significant (ANOVA, F = 2.601, df = 3, P = 0.067).

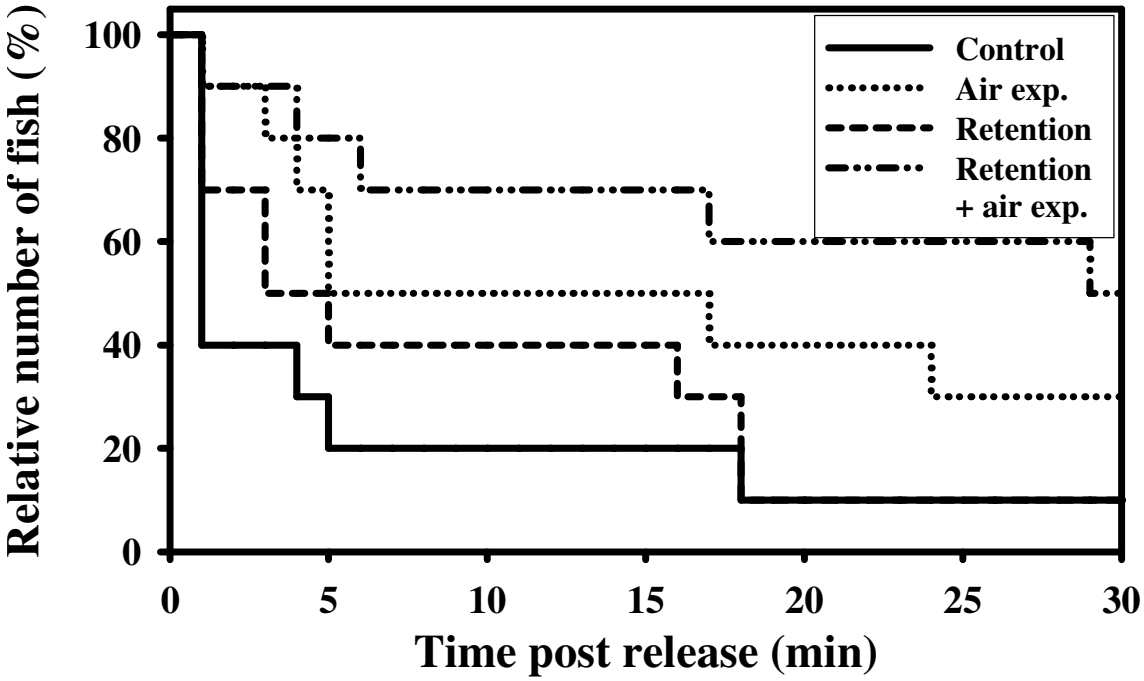

Figure 28: Relative number of fish at the release site (%) plotted against the time post-release (min) in carp without and with air exposure of 10 min and either not retained in carp sacks or retained for 9 h. Differences between treatment groups were significant (Kaplan-Meier, Breslow, $Chi^2 = 9.46$, df = 3, P = 0.024).

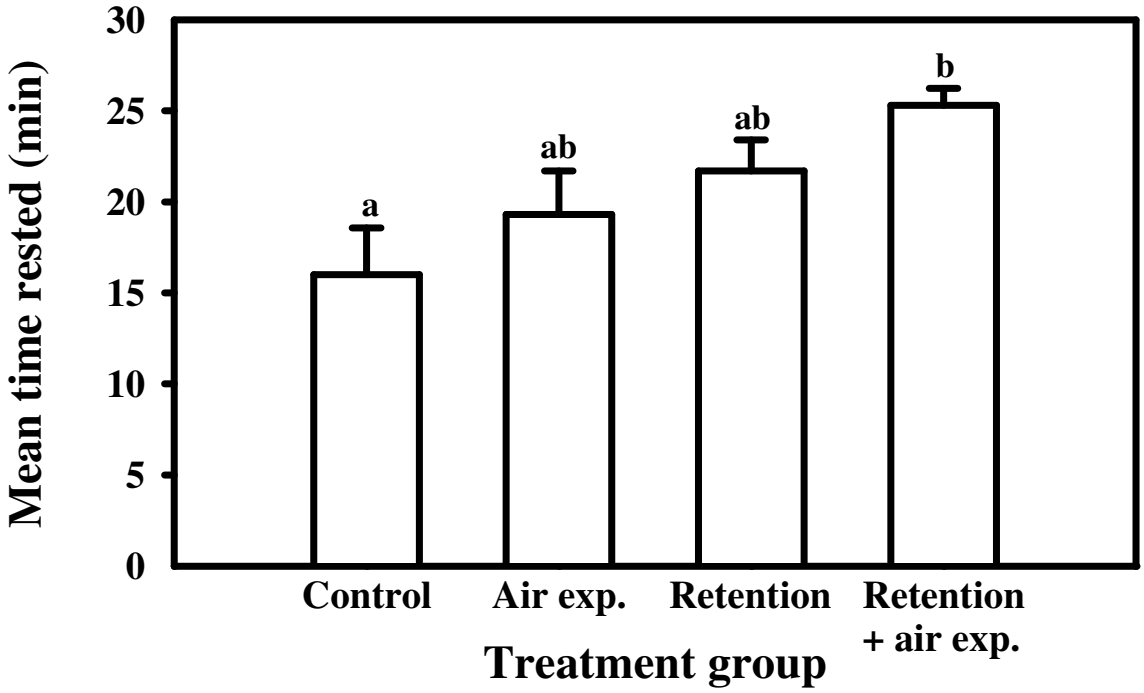

Figure 29: Mean time rested ± SE (min) from 0 to 30 min post-release in carp without and with air exposure of 10 min and either not retained in carp sacks or retained for 9 h. Different letters indicate significant differences (ANOVA, 3.813, df = 3, P = 0.018, Dunnett-T3 *post-hoc* test).

From 31 to 60 min post-release, mean minimum displacement was significantly reduced in fish air exposed following capture relative to control fish, but no difference between fish air exposed following retention and fish retained only was observed (ANOVA, F = 5.244, df = 3, P = 0.004, Tukey *post-hoc* test, significant differences were not between directly comparable air exposure treatment groups; Figure 30). Mean minimum displacement during subsequent tracking intervals was not statistically different among treatment groups (Table 8). Air exposure of 10 minutes following playing or playing and a 9 h retention period in a carp sack did not result in mortalities during an observation period of 2 month.

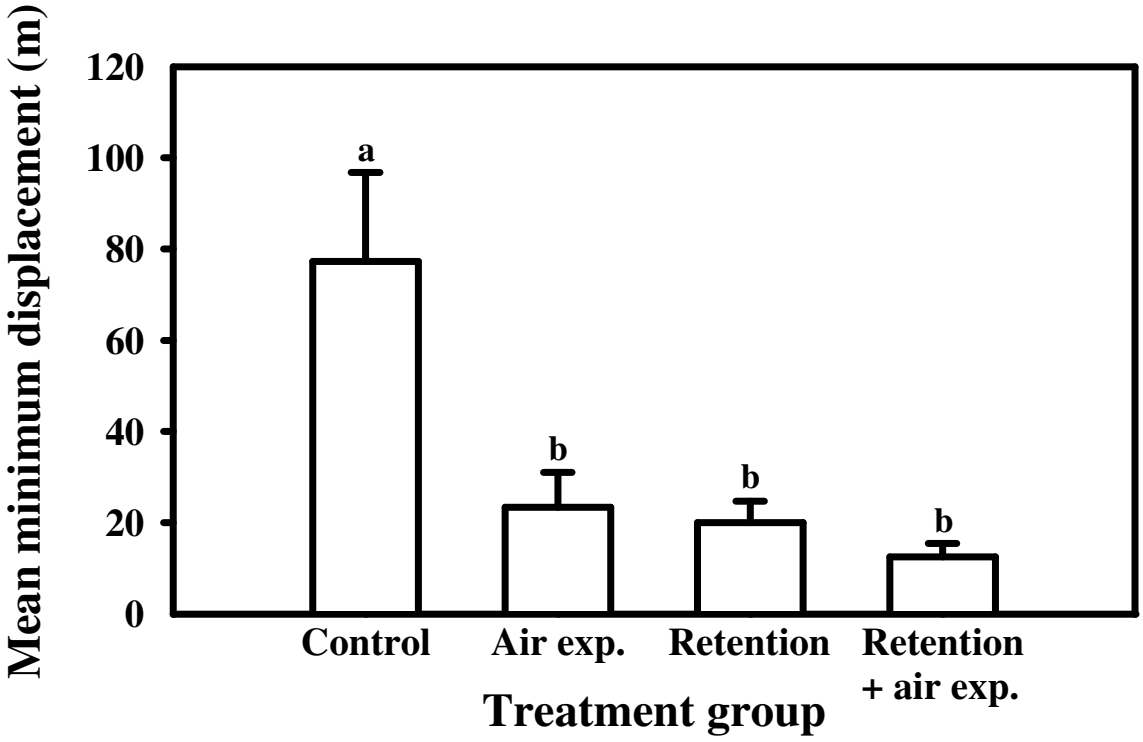

Figure 30: Mean minimum displacement ± SE (m) from 31 to 60 min post-release in carp without and with air exposure of 10 min and either not retained in carp sacks or retained for 9 h. Different letters indicate significant differences (ANOVA, F = 5.244, df = 3, P = 0.004, Tukey *post-hoc* test).

Table 8: Mean minimum displacement (MDP) (m) in carp without and with air exposure of 10 min and either not retained in carp sacks or retained for 9 h from 12 h to 72 h post-release. Differences were not significant (ANOVA, $P < 0.05$ for all tests).

Time post-release (h)	Mean MDP (m) Control (± SE)	Mean MDP (m) Air exp. (± SE)	Mean MDP (m) Retention (± SE)	Mean MDP (m) Retention + air exp. (± SE)	F-value; df; P-value
1 – 12	346.57 (± 60.28)	224.75 (± 52.70)	278.62 (± 53.80)	354.74 (± 33.94)	F = 1.489; d.f = 3; P = 0.238
12 – 24	287.94 (± 91.60)	371.04 (± 86.29)	271.78 (± 48.26)	184.01 (± 65.29)	F = 1.055; df = 3; P = 0.383
24 – 36	220.61 (± 55.62)	227.65 (± 49.65)	305.36 (± 62.15)	221.26 (± 92.61)	F = 1.403; df = 3; P = 0.263
36 – 48	158.65 (± 27.03)	191.25 (± 47.43)	248.36 (± 42.81)	192.40 (± 55.30)	F = 0.690; df = 3; P = 0.565
48 – 60	306.09 (± 65.44)	131.08 (± 41.89)	201.90 (± 65.62)	212.32 (± 52.53)	F = 1.565; df = 3; P = 0.218
60 – 72	265.14 (± 59.26)	176.24 (± 57.38)	201.76 (± 77.46)	170.36 (± 40.59)	F = 0.521; df = 3; P = 0.671

5. Discussion

Carp is a popular target species in recreational fisheries and despite its importance little is known about the sub-lethal consequences of handling-related stressors on the fish (Cooke & Suski 2005). The present study examined physiological disturbances, short-term behavioural alterations, and survival of carp following carp sack retention and air exposure, two stressors associated with handling practises common to catch-and-release angling for this species. It is the first study of its kind to assess the influence of fish retention in an open water body and complements laboratory and pond studies looking at short-term and long-term effects of retention in carp (Raat *et al.* 1997, Pottinger 1998, Hallermann unpublished data). In addition, this study provides insight into the consequences of air exposure on carp in a catch-and-release fishing event, which has not been investigated under field conditions before. Thus, the results of the present study are of importance to fisheries managers and decision makers to inform management decisions, but they are also of relevance for specialised carp anglers that are particularly interested in minimising damage on released fish (Arlinghaus 2007).

5.1 Methodological Issues

The present study was conducted in the wild and simulated the various stressors that occur in specialised carp angling. The examination of catch-and-release fishing impacts in the natural environment of fish is a useful approach that adds realism to catch-and-release angling science (Cooke *et al.* 2002*a*, Donaldson *et al.* 2008) and, therefore, may be more applicable to and accepted by anglers. Advantages of field studies include the exclusion of additional physiological demands related to laboratory settings including various confinement stressors that can influence physiology, behaviour and survivorship of fish (Cooke *et al.* 2004, Portz *et al.* 2006). In addition, field techniques exclude potential stressors during sequential fish sampling from the confinement facility (e.g. repeated chasing of fish with dip nets; Redgate 1974, Pickering *et al.* 1982). These factors potentially influence the stress response of fish and contribute to differences in results between laboratory and field settings (Cooke *et al.* 2004). In addition, the field approach includes factors in the natural environment that have the potential to influence behavioural response and survival including interactions with other organisms such as predators (Cooke & Philipp 2004, Thorstad *et al.* 2004, Danylchuk *et al.* 2007). It is assumed that the applied approach in the present study offers a more realistic estimate

of physiological disturbance, behavioural alteration, and survival after catch-and-release angling compared to laboratory studies (Cooke *et al.* 2002*a*, Cooke *et al.* 2004, Pollock & Pine 2007, Donaldson *et al.* 2008). However, any field study has the drawback whereby some of the most important environmental factors affecting the stress response of fish cannot be standardised (e.g. water temperature and dissolved oxygen). These factors may increase variance in measurements and reduce statistical validity. Thus, to compare results between treatment groups, individuals must experience similar environmental conditions and must have similar baseline levels of blood constituents at the onset of the study.

During the sampling period, water temperature was monitored as this is a prominent factor manipulating the physiological (Gustaveson *et al.* 1991, Anderson *et al.* 1998, Thompson *et al.* 2002, Meka & McCormick 2005) and behavioural responses of fish to angling-induced stressors (Gingerich *et al.* 2007, Thompson *et al.* 2008), as well as survival after release (Wilkie *et al.* 1997, Anderson *et al.* 1998, Wilde 1998, Wilde *et al.* 2000, Thorstad *et al.* 2003). In the present study, there were no differences in mean water temperature experienced by fish from each treatment group, effectively controlling for any physiological and behavioural impacts of water temperature. In addition, a current study assessing the impacts of carp sack confinement in a controlled laboratory setting revealed no influence of temperature on stress response of retained carp (Hallermann unpublished data), corroborating the validity of the present study. Another crucial factor that cannot be controlled in field experiments is dissolved oxygen. In the present study, dissolved oxygen was recorded to exclude hypoxia as a factor influencing the study results. Mean dissolved oxygen was not significantly different between the treatment groups, and dissolved oxygen levels remained within the optimum range reported for carp during all sampling days (Panek 1987, Bohl 1999, Schreckenbach 2002), effectively controlling for the impact of dissolved oxygen on the physiological and behavioural stress response of test animals.

Regarding the test animals, it was necessary that fish had similar baseline levels of blood constituents after the angling experience and prior to exposure to the treatments. The stress response of fish is correlated with longevity of exposure to stressors (Gustaveson *et al.* 1991, Kieffer *et al.* 1995, Thompson *et al.* 2002, Thorstad *et al.* 2003, Meka & McCormick 2005, White *et al.* 2008) and aggravates by an accumulation of stressors (Suski *et al.* 2003*a*, Killen *et al.* 2003). Landing time and additional stressors beyond the treatments were standardised so that the physiological

measures reflected variation from focal treatments alone. For example air exposure during handling and prior to execution of the experimental procedures was minimised in the experimental approach by holding fish underwater during unhooking, blood sampling and external transmitter attachment. The magnitude of physiological disturbances (Wydoski *et al.* 1976, Kieffer *et al.* 1996, Ferguson *et al.* 1993) and behavioural alterations (Little 2002) in response to stressors can also be influenced by fish size. The body sizes of carp captured during the study were within the size range targeted by specialised carp anglers. Therefore, it is assumed that the results of the present study reflect the physiological and behavioural stress response of trophy size fish in a "real-world" carp angling event. However, minor differences in mean fish length between treatment groups for retention effects were observed for random reasons. These differences in fish length did not have a statistically significant influence on the magnitude of physiological and behavioural stress response, so an impact of fish length was not apparent in the present study.

The major disadvantage of experimental approaches in the field is the lack of an unstressed control group since all capture methods cause stress to fish (Cooke *et al.* 2002*a*, Cooke & Schramm 2007; Donaldson *et al.* 2008). For this reason, the influence of the angling event (i.e. playing) itself could not be examined because unstressed control fish were lacking. A comparison between fish held in a laboratory with fish from the field is not valid as wild fish, such as those used in the present study, respond differently to stressors when held in captivity compared to when free-swimming in the wild (Cooke *et al.* 2002*a*). Such knowledge gaps can be addressed with complementary laboratory studies using acclimated experimental animals (i.e. hatchery-reared fish) to provide managers and decision-makers with the most reliable information. Indeed, a recent laboratory study examining the physiological stress response of carp to simulated capture revealed a neuroendocrine stress response and mobilisation of glucose as well as an accumulation of lactate and acid-base and ionic disturbances (Hallermann unpublished data). However, this does not affect the validity of the present study since the relative difference in physiological and behavioural response of the different treatments was the focal point of the study rather than comparisons to an unstressed control group.

5.2 Influence of Retention on Physiological Status of Carp

It was initially predicted that retention results in a physiological stress response. Consistent with this hypothesis, all retention durations caused a neuroendocrine stress response relative to fish sampled immediately after angling, indicated by an increase in blood plasma cortisol concentrations. Concentrations rose about 3.6 fold during retention for 3 h compared to control values and remained elevated throughout the entire retention period with a further, however not significant increase during medium- and long-term confinement. Observed blood plasma cortisol concentrations in the control group after angling were lower than reported values after exercise in previous studies involving carp (Leloup-Hatey 1960, Pottinger 1998, Hallermann unpublished data). Since cortisol increases in the blood significantly 0.5 h to 1 h after angling (McDonald & Milligan 1997), it cannot be excluded that values continued to rise after blood sampling, and control values directly after exercise do not fully reflect the stress response to the angling event. Nonetheless, the cortisol response in the present study was maintained throughout the entire retention period and tended to be positively correlated with retention duration. Similar results were observed in studies examining net confinement stress in a non-angling context (Davis & Parker 1986, Ruane *et al.* 2001, Ruane *et al.* 2002). However, Pottinger (1998) and Hallermann (unpublished data) observed decreasing cortisol concentrations in the blood during longer lasting net retention periods in a recreational fishing context. During long duration net confinement, cortisol levels were lower than the level after angling, but still above resting levels (Pottinger 1998, Hallermann unpublished data). These conflicting findings might be related to the origin of the experimental animals. Fish used in the present study were wild fish, whereas fish used by Pottinger (1998) and Hallermann (unpublished data) originated from hatcheries. Fish reared in aquaculture settings are often selected for stress resistance as this trait confers a benefit to growth potential and disease vulnerability, and it might be possible that cultured fish do not react in the same manner on the examined stressors as wild fish (Pottinger 2000). In addition, repeated brief stressors during hatchery operation (i.e. netting and transfer of fish) can influence the stress response in fish. Auperin & Geslin (2008) demonstrated that the exposure of rainbow trout a brief stressor during early life-history reduces the cortisol response in later life and Rush & Umminger (1978) and Barton *et al.* (1987) observed a training effect and lower stress response in goldfish (*Carassius auratus*) and rainbow trout following repeated exposure to mild stressors. This might explain the relatively higher

cortisol stress response to confinement stress in the present study compared to the studies by Pottinger (1998) and Hallermann (unpublished data). Similarly, Woodward & Strange (1987) noted a higher cortisol response in wild rainbow trout subjected to net confinement compared to similarly treated hatchery-reared fish. In turn, hatchery-reared fish are rarely required to swim at high speeds, whereas it is the case for wild fish in their natural environment (Booth *et al.* 1995). The unfamiliar exercise for hatchery-reared fish might explain the higher cortisol response following angling and angling simulation in the studies conducted by Pottinger (1998) and Hallermann (unpublished data) compared to the present study. Similarly, Wydoski *et al.* (1976) observed more severe physiological changes in hatchery-reared fish in response to playing compared to wild fish. It is unlikely that the stress response to retention in the present study was caused by a deterioration of water quality. The mesh material of net retention gear used in shore angling for cyprinids, such as carp sacks and keepnets, support water exchange (Pottinger 1997, Hallermann unpublished data). In fact, reported differences between water parameters inside the retention gear and the surrounding water are not of biological significance during environmental conditions as they were observed in the present study (Pottinger 1997, Hallermann unpublished data).

Blood plasma glucose concentrations rose during retention and observed values in fish confined for 9 h were double as high as those of control fish. Hyperglycaemia is a frequently observed response to an acute stressor. The rapid rise of glucose in the blood is mainly caused by the effects of catecholamines on glycogenolysis (Wendelaar Bonga 1997) and longer lasting indirect actions by the effect of cortisol on gluconeogenesis (Suarez & Mommsen 1987, Vijayan *et al.* 1994, Mommsen *et al.* 1999). Through that link between the primary stress response and the mobilisation of glucose, it was revealed that blood glucose is an appropriate indicator of the secondary stress response (Barton & Iwama 1991, Barton 1997, Barton *et al.* 2002). The results of the present study are in accordance with previous studies reporting increasing blood glucose concentrations during net confinement (Ruane *et al.* 2001, Ruane *et al.* 2002, Hallermann unpublished data). The observed blood plasma glucose concentrations after 9 h are within or above the range of post-stress levels previously observed in net confined carp (Pottinger 1998, Ruane *et al.* 2001, Ruane *et al.* 2002, Hallermann unpublished data) and support the notion that carp sack confinement is in fact a serious stressor in recreational fisheries.

It was initially hypothesised that retention in carp sacks facilitates physical recovery from angling exercise. Consistent with this hypothesis, blood plasma lactate levels were significantly lower in long-term retained fish relative to control and short-term retained fish. The high concentrations of blood plasma lactate in the control group are a result of angling exercise (Gustaveson *et al.* 1991, Pottinger 1998, Meka & McCormick 2005, Suski *et al.* 2004, Killen *et al.* 2006). The observed mean blood plasma lactate concentration of 9 mmol/L in the control group is in the upper range previously reported for carp after exercise (Pottinger 1998, Hallermann unpublished data), suggesting that the relatively short angling period of 3 min in the present study was sufficient to exhaust the fish. During short-term confinement blood plasma lactate levels increased slightly which is likely still a result of the anaerobic exercise during angling as lactate stored within the muscle moves slowly into the blood and reaches its peak in the bloodstream within 2 h to 4 h after exercise (Holeton *et al.* 1983, Turner *et al.* 1983, Wood *et al.* 1983, Wang *et al.* 1994, Booth *et al.* 1995, McDonald & Milligan 1997). Medium and long-term retention resulted in a decrease of blood plasma lactate levels, indicating that fish recovered consistent with the findings of other studies that investigated retention following capture in various fish (Pottinger 1998, Suski *et al.* 2004, Killen *et al.* 2006, Hallermann unpublished data). However, blood plasma lactate concentrations after 9 h confinement in carp sack were somewhat higher than initial values reported for carp (Ruane *et al.* 2001, Ruane *et al.* 2002, Hallermann unpublished data), which suggests that clearance of blood plasma lactate was not entirely completed.

Blood plasma osmolality was not affected by the examined treatments, though blood plasma electrolyte concentrations differed between treatment groups. Despite the fact that inorganic ions are considered to account for most of the osmolality in fishes (Wedemeyer 1996), osmotic pressure is further dependant on other osmotically active particles in the blood, such as glucose, protein, lactate and HCO_3^- (Wood 1991, Suski *et al.* 2003*a*). The unchanged blood plasma osmolality during retention despite changes in blood plasma electrolyte concentrations might be attributable to these other variables. Ionic disturbances during confinement are considered to result from ion loss via the gills, diuretic ion loss, and haemodilution due to net influx of water through the gills (McDonald & Milligan 1997, Wendelaar Bonga 1997, Barton *et al.* 2002). In the present study, a decrease in blood plasma ion concentrations was evident for sodium and chloride. However, blood plasma potassium concentrations increased during retention, which is contrary to reported ion changes during confinement stress

(McDonald & Milligan 1997, Wendelaar Bonga 1997, Barton *et al.* 2002), but consistent with a number of studies examining stress involving physical exercise (Holeton *et al.* 1983, Turner *et al.* 1983, Wood *et al.* 1983, Wang *et al.* 1994, Arlinghaus *et al.* 2009). Since all fish in the present study were captured by rod and reel prior to carp sack retention, it is possible that the observed ion changes in the blood plasma are a result of angling rather than of retention and, thereby, are similar to the results of studies examining exercise stress. The osmotic disturbances following exhaustive exercise are more complex, for in addition to increased branchial permeability to ions and water and diuretic ion loss, mediated by adrenalin (McDonald & Milligan 1997). A number of studies indicated that elevated muscle lactate concentrations during exercise increase muscle intracellular osmotic pressure leading to a net fluid shift into white muscle (Holeton *et al.* 1983, Turner *et al.* 1983, Wood *et al.* 1983, Wood 1991), though other studies did not observe fluid shifts into white muscle instead speculating that fluid shifts into erythrocytes, tissue other than white muscle, and ion movements across the gills account for changes in blood plasma electrolyte concentrations due to exercise (Wang *et al.* 1994, Arlinghaus *et al.* 2009). Irrespective of the exact mechanism, both assumptions result in initial haemoconcentration to a more or lesser extend (Holeton *et al.* 1983, Turner *et al.* 1983, Wood *et al.* 1983, Wood 1991, Wang *et al.* 1994, Arlinghaus *et al.* 2009). Although such a likely haemoconcentration could not be demonstrated in the present study due to the lack of an unstressed control group, the observed subsequent blood plasma ion patterns are highly comparable to those observed in these studies. Blood plasma sodium values were highest in the control group and decreased during retention. The higher initial concentrations might be a result of haemoconcentration during playing and an uptake of sodium from the environment in exchange for either ammonia (Holeton *et al.* 1983) which is produced in carp muscle fibre during anaerobic exercise (Driedzic & Hochachka 1976) or for protons to buffer acid-base disturbances (Holeton *et al.* 1983, Wood 1991). After 6 h of carp sack retention, blood plasma sodium concentrations reached a level which was also evident after 9 h. This likely indicates a return to resting levels, similar to the findings of previous studies that observed an initial peak of blood plasma sodium following exercise and a subsequent decline to resting levels within a similar time frame (Turner *et al.* 1983, Wood *et al.* 1983). In terms of blood plasma chloride, concentrations dropped within 3 h of retention with an increase to an intermediate level thereafter. This is in accordance with other studies that demonstrated an initial increase due to

haemoconcentration and a subsequent sharp decline likely caused by transbranchial net excretion of chloride in exchange for bicarbonate to buffer acid-base disturbances (Holeton *et al.* 1983). Slightly increasing blood plasma chloride concentrations following 6 h and 9 h of retention in the present study can again be interpreted as a return to resting levels during recovery as previously observed by other authors within a similar time frame (Holeton *et al.* 1983, Turner *et al.* 1983, Wood *et al.* 1983 Arlinghaus *et al.* 2009). The decreases of sodium and chloride in the blood plasma were in a magnitude of up to 6.1 % and 3.6 %. According to Barton *et al.* (2002), blood plasma ion concentrations change during stress in fish by a magnitude of about 10 %. The observed changes in blood plasma sodium and blood plasma chloride in carp are well below this threshold. Thus it is concluded that the fish were probably well able to cope with the observed disturbances. The rise in blood plasma potassium during retention for 6 h potentially reflects extrusion of potassium from muscle cells in response to intracellular acidosis (Turner *et al.* 1983, Wood *et al.* 1983, Wood 1991) or it might originate from other sources such as gill tissue (Wood & LeMoigne 1991) and adrenergically stimulated and swollen red blood cells (Borgese *et al.* 1987). Following retention for 9 h blood plasma potassium was not significantly elevated anymore, indicating quick recovery similar to the results of previous studies (Holeton *et al.* 1983, Turner *et al.* 1983, Wood *et al.* 1983, Arlinghaus *et al.* 2009). Blood plasma potassium increased in a magnitude of 30.3 %, which is clearly above the usual ion changes during stress noted by Barton *et al.* (2002). However the absolute mean blood plasma potassium concentration after 6 h of retention, which reflects the highest mean concentration, was 3.9 mmol/L and thus much lower than the concentrations of about 10 mmol/L that causes heart failure in mammals (Guyton 1981), wherefore it is unlikely that the observed changes in blood plasma potassium are of significance for the fish's viability (Wood *et al.* 1983). Taken together, it is very likely that ionic disturbances in the present study were caused by angling rather than by retention as observed blood plasma ion patterns are similar to those previously reported for exercised fish. However the ionic disturbances were only of low magnitude and ionic status seemed to recover after 6 h to 9 h.

Blood plasma concentrations of the enzymes LDH and AST increased during retention reaching the highest levels after 9 h. Both enzymes are considered to be indicators of tissue damage with no known function in the blood and are released from the intracellular space during cell damage or death (Henry 1996). However, enzyme

activity differs between species, and even between tissues within the same species. For example, LDH activity between white and red muscle varies due to the different requirements of the tissues (Johnston 1977). Therefore, higher values in the blood might occur from a severe damage of tissue with lower enzymatic activity or from moderate damage of tissue with higher enzymatic activity. Hence, it is difficult to specify the degree of damage and the affected tissue especially since LDH is a non-specific indicator and present in a number of tissues beyond muscle (Morrissey *et al.* 2005). In the present study, differences in indicators of tissue damage might be due muscle tissue damage occurring from struggling (Moyes *et al.* 2006) or it might be a result of stress causing a leakage of cytoplasmatic enzymes into the blood via tissue damage or increased cell membrane permeability (Meltzer 1971, Arakawa *et al.* 1997, Sánchez *et al.* 2002). One can only speculate about the reason for the observed elevated blood plasma enzyme concentrations. Since beside LDH also AST increased in the blood plasma during carp sack confinement, which is predominantly located in heart and liver (Morrissey *et al.* 2005), a stress induced enzyme leakage, for example due to heart tissue damage as observed in mice (Sánchez *et al.* 2002), seems to be more plausible than simple white muscle damage by exercise or struggling. Due to exclusion of blood plasma samples that showed signs of haemolysis from statistical analysis it is unlikely that differences between treatment groups were caused by the sampling procedure unrelated to the treatment. Taken together, fish retained in carp sack experience a certain degree of cell damage though it remains unclear which tissues are damaged.

5.3 Influence of Retention on Behaviour and Survival of Carp

It was initially hypothesised that retention would decrease movement activity and, in turn, increase post-release rest periods. Consistent with this assumption minimum displacement between 31 min and 60 min post-release was reduced in long-term retained fish relative to control fish. In addition long-term retained fish rested significantly longer than short-term retained fish within 30 min post-release. Both, control fish and short-term retained fish experienced physiological disruptions emerging from exercise as described above, but showed lesser stress response than long-term retained fish that seemed to recover from the angling event. Thus, the results of the present study suggest that the stress experience and its consequences had a larger impact on movement activity in carp than physiological disturbances emerging from angling exercise. Therefore the conclusion that post-exercise retention facilitates recovery from

angling (Lewin *et al.* 2006), which suggest beneficial effects of retention, should be modified as retention can be disadvantageous in terms of post-release behaviour despite physiological recovery. Impaired swimming performance following net retention was previously demonstrated for striped bass (Strange & Chech 1992). One explanation for decreased movement activity might be that behavioural impairments following confinement reflects increased metabolic demands arising from confinement stress reducing metabolic capacity for activity (Strange & Chech 1992). An alternative explanation for the observed movement patterns is based on different coping styles during short- and long-term stress. Haller *et al.* (1998) stated that during short-term stress it is advantageous for an organism to actively cope with the situation (i.e. fight or flight response). However, an affected energetic background during longer-lasting stress situations hampers the ability of the organism to perform demanding behaviours, and thus the individual would benefit from adopting an energy-saving wait-and-see mode (i.e. conservation-withdrawal response) to minimise losses of energy (Haller *et al.* 1998). Such a transition from behavioural activation to inhibition is typically seen with increasing duration or severity of a stressor (Haller *et al.* 1998, Øverli *et al.* 2002). In the context of the present study, it seems possible that, in control fish and short-term retained fish, the release of stress hormones was stimulating and enabled the fish to actively cope with the situation (i.e. fight or flight response) indicated by high movement activity despite physical exhaustion and physiological disruption. This short-term response might also be expected from an evolutionary perspective since the typical response of an animal is to escape and avoid the stressor (Peake *et al.* 1997, Haller *et al.* 1998). But a behavioural flight or fight response is not appropriate in all situations such as when the behavioural options of an animal are limited as is the case during confinement (Ladewig 2000). In such situations a behavioural down-regulation (i.e. conservation-withdrawal response) through inhibitory effects of stress hormones may indeed have adaptive value at minimising energy losses (Haller *et al.* 1998). However, despite the general acceptance that glucocorticoids play a major role in this time-dependant behavioural pattern, the exact physiological mechanisms and the involved processes are not well understood (Haller *et al.* 1998).

Irrespective of the exact mechanism, the present study demonstrated that behavioural alterations resulting from retention in carp sacks are largely reversed 12 h post-release as indicated by similar minimum displacement of all treatment groups after this time-period which is consistent with a number of other studies (Cooke *et al.* 2000,

Skomal & Chase 2002, Arlinghaus *et al.* 2008, Klefoth *et al.* 2008, Arlinghaus *et al.* 2009) and indicates that the deleterious effects of catch-and-release angling practises are reversible within the short-term. However, this study did not include a non-angled control, and potentially longer lasting effects on behaviour compared to a pre-angled state remain unknown.

As initially hypothesised no mortalities were recorded within a post-release observation period of 2 months. This is in accordance with previous studies reporting only low catch-and-release mortalities in carp, varying from 0 % to 2 % (Beukema 1970, Raat 1985) and no additional lethal consequences of net retention in various cyprinid species (Raat *et al.* 1997).

5.4 Influence of Air Exposure on Physiological Status of Carp

Contrary to the prediction that air exposure aggravates the physiological stress response, no differences in blood plasma cortisol concentrations between air exposure treatments and directly comparable treatments without air exposure were observed. As previously mentioned, a significant increase of cortisol in the blood often happens with a time delay of 0.5 h to 1 h (McDonald & Milligan 1997). As such, it might be possible that the impact of air exposure on blood plasma cortisol response was not fully reflected by the results of the present study. Indeed, Reynolds *et al.* (2009) and Hallermann (unpublished data) used a similar approach by taking blood samples following the treatment as it was employed in the present study and also did not observe an additional impact of air exposure, whereas Haukenes & Buck (2006) reported significantly elevated cortisol levels in the blood in fish sampled 0.5 h after air exposure compared to those sampled directly following exposure to air. Similarly, Dabrowska *et al.* (1991) observed elevated blood plasma cortisol levels in carp that were exposed to air for 30 s in a non-angling context by taking the blood sample 1 h after handling. Therefore, one can hypothesise that a peak of blood plasma cortisol concentrations happened in the aftermath of the blood sampling, and could not be revealed due to the sampling time.

Similar to cortisol, air exposure had no additional effect on blood plasma glucose concentrations relative to appropriate controls. Consistent with the present study, several authors did not observe an increase in glucose concentrations in the blood when sampling was conducted directly following air exposure (Hanson *et al.* 2008, Arlinghaus *et al.* 2009, Reynolds *et al.* 2009, Hallermann unpublished data). However studies using another approach by taking blood samples temporally delayed revealed

that air exposure results in a substantial blood glucose increase (Haukenes & Buck 2006, Thompson *et al.* 2008, White *et al.* 2008, Arlinghaus *et al.* 2009). The difference in glucose patterns between the present studies and other studies that observed a temporally delayed increase in blood glucose might partially be attributable to a delayed cortisol increase following stress exposure (McDonald & Milligan 1997) as cortisol stimulates glucose release indirectly via gluconeogenesis (Suarez & Mommsen 1987, Vijayan *et al.* 1994, Mommsen *et al.* 1999). However, as previously mentioned, glucose mobilisation is not only cortisol dependant and is also mediated by the effects of catecholamines on glycogenolysis. Haukenes & Buck (2006) assumed that delayed glucose appearance in the blood stream is likely due to lag-time between stimulation of glucose release through glycogenolytic and gluconeogenic pathways and its subsequent diffusion into the circulatory system. Both possible explanations allow the assumption that blood plasma glucose concentrations might have increased in the aftermath of blood sampling in response to air exposure.

Exposing fish to air after angling did not result in blood plasma lactate increase relative to controls which is consistent with a recent study examining effects of carp angling practises in the laboratory setting (Hallermann unpublished data), but conflicts with a number of other studies that demonstrated that impaired oxygen uptake during air exposure results in anaerobic consumption of endogenous fuels and an accumulation of lactate as the end product of anaerobic metabolism (Ferguson & Tufts 1992, Suski *et al.* 2007*a*, Hanson *et al.* 2008). The reason for the similar blood plasma lactate concentrations in the angled control group and the air exposure treatment group might be due to species-specific characteristics. Carp accumulate only low levels of lactate up to the point of fatigue (Driedzic & Hochachka 1975) compared to, for example, salmonids (Black *et al.* 1962, Stevens & Black 1966, Hammond & Hickman 1966). Blood plasma lactate concentrations following angling were in the upper range of previously reported values for carp (Pottinger 1998, Hallermann unpublished data) and therefore one can speculate that blood plasma lactate had already accumulated to a maximal threshold after angling. During retention in carp sacks, lactate was removed and blood plasma lactate levels returned to similar levels as unstressed fish (Ruane *et al.* 2001, Ruane *et al.* 2002, Hallermann unpublished data). Subsequent air exposure resulted in a rise in blood plasma lactate to a level observed directly after playing, indicating that air exposure after confinement poses a second exhaustive event for the fish with similar consequences on anaerobic metabolism as the original angling

exercise. Similar findings were observed in a study with carp that were air exposed following carp sack retention (Hallermann unpublished data) as well as largemouth bass and walleye exposed to air during weigh-in simulation after live-well confinement (Suski *et al.* 2004, Killen *et al.* 2006).

As predicted, blood plasma osmolality and blood plasma ion concentrations were not altered by air exposure following angling and retention. These findings are generally in agreement with a number of catch-and-release angling studies that mostly did not observe an additional influence of air exposure on these variables following either angling (Suski *et al.* 2007*a*, Thompson *et al.* 2008, Arlinghaus *et al.* 2009, Hallermann unpublished data) or retention (Killen *et al.* 2006, Hallermann unpublished data).

Concentrations of tissue damage indicators LDH and AST did not significantly change in the blood plasma during the air exposure period among treatments and appropriate controls, similar to the results of a study with largemouth bass (Thompson *et al.* 2008). This suggests that air exposure might not be associated with tissue damage. An alternative explanation for this finding might be related to sampling time. Sánchez *et al.* (2002) reported that, for mice, a moderate increase of LDH and AST in the blood plasma occurred directly post-stress, and a further substantial increase occurred during a 160 min recovery period. In the present study blood samples were taken directly following the treatments and in the study conducted by Thompson *et al.* (2008) the control group was sampled following angling, and the air exposure groups were sampled 20 min post-air exposure. In the context of the study conducted by Sánchez *et al.* (2002), it might also be possible that changes in LDH and AST concentrations in the blood plasma could not be revealed by the applied experimental approach, and blood plasma concentrations of these enzymes increased further subsequent to blood sampling.

5.5 Influence of Air Exposure on Behaviour and Survival of Carp

In accordance with the hypothesis that air exposure impairs movement activity post-release, differences in patterns leaving the release site were observed. Thirty percent of fish exposed to air following capture and 50 % of fish exposed to air following retention did not leave the release site within 30 min post-release compared to 10 % of fish in comparable treatment groups without air exposure. Despite differences in time to resume swimming, distance moved did not differ significantly between comparable

treatments, likely as a result of high inter-individual variation. Differences in patterns of movement while leaving the release site were caused in some cases by equilibrium loss in both treatment groups involving air exposure which has been previously observed in other species following extended air exposure periods (Cooke & Philipp 2004, Danylchuk *et al.* 2007, Gingerich *et al.* 2007, Thompson *et al.* 2008). A loss of equilibrium indicates a generalised breakdown of systemic homeostatic mechanisms due to multiple biochemical and physiological disruptions (Beitinger *et al.* 2000). In the present study visual observation of equilibrium loss was only possible for fish that rested directly at the release point. However, equilibrium loss can also be time delayed (Danylchuk *et al.* 2007) or last only for short time periods (i.e. seconds, Thompson *et al.* 2008), and quantitative assessment of these types was not feasible in the context of present study. Minimum displacement from 31 min to 60 min post-release was reduced in air exposed fish following capture relative to control fish, though no differences in measured physiological parameters between these treatment groups were observed. This might partly be attributed to a temporally delayed response of some blood plasma parameters, though other authors have noted a lack of concordance between physiological responses to stress and various organismal endpoints (Davis & Schreck 2005, Thompson *et al.* 2008, Arlinghaus *et al.* 2009). In fact, behaviour culminates a multitude of complex biochemical and physiological processes and is a sensitive indicator of organismal changes in response to stress (Schreck *et al.* 1997, Little 2002). It is very likely that decreased post-release movement activity following air exposure reflects cumulative disruptions of various biochemical processes involving parameters not measured in the present study. Behavioural alterations following prolonged air exposure periods have been observed in other species and the degree of impairment often correlated with air exposure duration (Cooke & Philipp 2004, Schreer *et al.* 2005, Danylchuk *et al.* 2007, Gingerich *et al.* 2007, Arlinghaus *et al.* 2009). In the present study, fish were exposed to air for 10 min, assuming that this is a realistic time period mimicking the average air exposure duration during a regular specialised carp fishing event involving weighing and photographing a large fish. It is unknown how different air exposure periods influence behaviour of carp, and which durations can be regarded as critical thresholds as they were observed in other species (Schreer *et al.* 2005; Arlinghaus *et al.* 2009). Several authors demonstrated that post-release behavioural impairments in conjunction with equilibrium loss increase the susceptibility of angled fish to predation (Cooke & Philipp 2004, Thorstad *et al.* 2004, Danylchuk et *al.* 2007).

Large size carp reached refuge size most likely for all predatory freshwater fish species, but they do contribute to the diets of otters (Britton *et al.* 2005). Despite the fact that no mortalities were observed in the present study, it cannot be assured that angling would not increase mortality in areas with established piscivorous mammal populations as post-release predation rates are highly dependent on predator occurrence (Cooke & Philipp 2004).

Consistent with other studies reporting quick recovery of behavioural changes resulting from angling (Cooke *et al.* 2000, Skomal & Chase 2002, Arlinghaus *et al.* 2008, Klefoth *et al.* 2008, Arlinghaus *et al.* 2009), behavioural alterations in the present study seemed to be reversed by 12 h post-release, and subsequent tracking intervals revealed no differences in minimum displacement between treatment groups. But again, this study did not include an unstressed control group, therefore potential longer-lasting alterations compared to a pre-angled state remain unknown.

During the 2 month post-release observation period no mortalities occurred, whereas several authors have noted mortalities in fish subjected to air exposure treatments for shorter time periods (Ferguson & Tufts 1992, Arlinghaus & Hallermann 2007, Gingerich *et al.* 2007). These patterns might be due to species-specific differences. Carp are capable of coping with severe hypoxia, or even anoxic conditions, for up to 2 h (Van den Thillart & Van Waarde 1991), thereby enabling them to survive periods of low dissolved oxygen common in their natural habitats, potentially contributing to their tolerance against air exposure. Taken together, an air exposure period of 10 min results in behavioural alterations post-release, but there is no indication that it affects behaviour and survival of carp in the long-term.

6. Conclusions

The examination of the use of carp sacks to retain large carp following angling in the present study revealed additional stress effects above and beyond those associated with capture alone. The present study showed that carp sack confinement results in a prolonged physiological stress response, tissue damage, and altered post-release behaviour, specifically decreased movement activity. Retention of carp in carp sacks must be considered as a severe stressor for fish, though physiological disorders were not within the range to cause mortalities and behaviour normalised within 12 h post-release.

The present study revealed no additional effect of air exposure following angling on the physiological stress response of carp. However, air exposure subsequent to retention resulted in the recruitment of anaerobic respiration that shows similarities to a second capture event. Overall, the observed physiological disturbances associated with air exposure were minor, but might be influenced by the experimental set-up and do not reflect the entire set of possible adverse effects. Analysis of post-release movement activity revealed that air exposure disrupts the homeostasis and organismal performance of carp as differences in movement patterns revealed. Air exposed fish experienced equilibrium loss and resumed movement temporally delayed relative to non-air exposed fish. Furthermore air exposed fish showed reduced minimum post-release displacement. However, the stress effects of air exposure are below a lethal threshold as air exposure following angling or following retention did not result in any mortality and behaviour of treated animals approximated that of controls in a relatively short time (i.e. 12 h post-release).

Two largely divergent conclusions can be drawn depending on the personal values attached to the issue. From a fish population perspective which is interested in survival endpoints, the short-term impacts of retention in carp sacks and air exposure associated with handling and photographing are of minor concern as carp showed high resiliency to these stressors and suffered no mortality and limited behavioural alterations. However, if one takes an individual-based fish welfare perspective (Arlinghaus *et al.* 2007), the physiological stress responses and behavioural impairments associated with retention and air exposure suggest that the use of carp sacks is to be discouraged. Even without formal management regulations, anglers with a strong conservation ethic could voluntarily abandon the use of carp sacks for the purpose of facilitating the recovery of angled fish and should engage an immediate,

quick photographing session followed by release of the fish without further handling. Recommendations such as this should be easily put in place as after the angling event the fish is exhausted and is thus easily photographed. The present study shows that this approach is the least deleterious to the fish.

This is the first study that investigated the impacts of retention in carp sacks in an open water body and it provided insight into the physiological and behavioural impairments resulting from retention and air exposure under “real-world” angling conditions. Of particular interest, based upon qualitative assessments during this study, is the investigation of physical injuries to the epidermis of the fish. Those injuries occurred during retention in carp sacks and could potentially result in long-term infection from water borne pathogens and might also reduce the aesthetic value for specialised carp anglers.

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Hiermit erkläre ich an Eides statt, die vorliegende Masterarbeit selbstständig verfasst und keine anderen als die angegebenen Quellen und Hilfsmittel benutzt zu haben.

Berlin, den 28.05.2009

Tobias Rapp