FISH WELFARE LITERATURE REVIEW

Prepared for the Angling Governing Bodies Liaison Group
and the British Field Sports Society

by

T. G. Pottinger BSc PhD

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The Institute of Freshwater Ecology
The Windermere Laboratory
The Ferry House, Far Sawrey
Ambleside, Cumbria LA22 0LP

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i. SUMMARY

This study sought to identify, summarise, and interpret published research which has addressed three areas of concern to the angling community:

(a) Physiological stress. Prolonged activation of the physiological stress response can have harmful effects in animals. Do the procedures associated with the capture of fish by angling; hooking, playing, landing, unhooking, confinement within keepnets, weighing, cause a stress response? If so, is the response severe enough to cause concern regarding the welfare of the fish?

(b) Physical damage. Do the processes associated with the capture of fish cause damage that might affect the subsequent welfare and survival of the fish?

(c) Pain. It has been suggested that hooked fish experience pain during capture and unhooking. Is there evidence that fish can experience "pain and suffering" in a manner analogous to higher vertebrates?

The main points highlighted by the literature review are summarised below.

1. The physiological stress associated with capture

1.1 - Capture by angling is always accompanied by physiological disturbances typical of activation of the hypothalamic-pituitary-interrenal axis. Capture by angling may therefore be considered to cause physiological stress in fish.

1.2 - Physiological recovery is complete within 24 - 72 h of capture. The stress associated with capture may be considered acute, rather than chronic, and unlikely to have long-term impact on the well-being of the fish.

1.3 - Mortality of fish within 72 h of capture is rare.

1.4 - Water temperature may be a significant factor in determining the severity of the stress

response to capture.

1.5 - The stress of capture may be more severe for larger fish.

1.6 - There is an overwhelming requirement for well-designed studies to examine the physiological effect of capture, and time-course of recovery following capture, in non-salmonid European fish. Such studies should encompass an examination of the effect of water temperature, fish size and delayed release.

2. The respiratory and metabolic effects of capture by angling

2.1 - Even when exposed to an exercise regime arguably more severe than that imposed by rod and line capture, the available data suggest that most species of fish recover baseline respiratory and metabolic levels within 8 - 24 h.

2.2 - Fish size and water temperature both affect the severity and duration of the metabolic and respiratory effects of severe exercise.

2.3 - Severe exercise can, under certain circumstances, cause mortality in fish. Whether this arises under experimental conditions because of the imposition of unrealistically extreme levels of activity, or because of the use of "unfit" fish is not clear.

3. The impact of capture stress on post-release behaviour

3.1 - Capture by angling is likely to result in some short-term modification of behaviour. In prey fish, this may result in an increased susceptibility to predation.

3.2 - The duration of behavioural modification following stress is shorter than the period required for physiological recovery from stress.

3.3 - There is no research in this area that has examined species native to the UK.

4. Physical damage associated with capture

4.1 - There is a measurable level of mortality associated with hooking of fish. Most studies have not monitored survival beyond 72 h following capture.

4.2 - Mortality is low to negligible in fish that are hooked in the jaw, but can be extremely high in fish hooked in the throat, gills and deep-hooked in the gut. The majority of fish caught under experimental conditions were hooked in the jaw.

4.3 - There may be differences in mortality rate associated with natural and artificial baits and with hook size and type. The species and size of fish may also be factors. There are too few data available at present to draw firm conclusions.

4.4 - Almost all the data available on hooking mortality originate from North America. The species studied are almost exclusively predators, captured with either lures or bait. Although this information may be applicable to some species of fish native to the UK (perch, pike, zander, salmonids), there are no data in the literature derived from studies on cyprinid fish that constitute the majority of species sought by freshwater anglers in the UK.

5. Pain perception in fish

5.1 - It is advantageous for an animal to be aware of damage to its body and to be able to avoid potentially damaging situations. A system that alerts the animal to damage has survival value.

5.2 - Several of the anatomical and biochemical components involved in pain perception in mammals are present in fish. However, some key elements (e.g. unmyelinated nerve fibres) are absent from certain species and other elements (e.g. the forebrain/cerebral cortex) are considerably less well developed in fish than in mammals.

5.3 - The neurophysiological mechanisms underlying pain perception in man and other mammals are complex and not fully understood. Many components of the nociceptive/pain perception system have other, unrelated, functions. Identification of such components in fish cannot be

considered proof that fish experience "pain".

5.4 - It is the opinion of experts in the field that animals may possess mechanisms allowing them to avoid damage and facilitate recuperation without conscious perception of pain, in human terms.

5.5 - Our understanding of pain perception in fish is hampered by the lack of research on this subject, and by the difficulties inherent in interpreting the behavioural and physiological responses of an animal taxonomically far removed from mammals.

5.6 - There is no information available in the literature at present which provides firm evidence that fish perceive pain as mammals apparently do or, conversely, that they cannot perceive pain as mammals do. On balance, it seems unlikely that fish experience pain as understood by humans. However, the problem of assessing exactly what a fish perceives when exposed to stimuli considered to be noxious or unpleasant in human terms may prove to be intractable.

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iii. AIMS

The aim of this review is to assess the current state of knowledge regarding two areas of key importance in fish welfare: physiological stress and pain perception, with particular reference to the relationship between angling practices and fish welfare.

iv. METHODS

The following resources were utilised for the literature search:

(a) The library of the Freshwater Biological Association.

The library of the FBA houses one of the world's finest collections of information on freshwater science, built up over a period of more than 60 years. The collection is made up of 7,500 books, l,500 journal titles and 70,000 reprints and reports, covering all aspects of freshwater ecology, as well as pollution, algology, microbiology, invertebrate taxonomy and ecology, sediment and water chemistry, aquaculture and fisheries management and hydrology.

(b) Aquatic Sciences and Fisheries Abstracts on CD-ROM (1978-1994)

Aquatic Sciences & Fisheries Abstracts (ASFA) covers all aspects of marine, brackish, and freshwater environments. Subjects include biology; ecology; fisheries; aquaculture; oceanography; limnology; resources and commerce; pollution; biotechnology; marine technology and engineering; marine meteorology and climatology; and non-living resources (oil, gas, minerals, chemicals).

Citations are drawn from a variety of sources, including journal articles, conference papers, books, monographs, theses, technical reports, and non-conventional literature. Over 40 languages are included, although the database is produced exclusively in English. Original, non-English titles are given wherever possible.

Information is supplied by the Aquatic Sciences and Fisheries Information System and maintained by research centres throughout the world, including:

FAO: The Food and Agriculture Organization of the United Nations
IOC: The Intergovernmental Oceanographic Commission of Unesco
UNEP: The United Nations Environment Programme
UNDOALOS: The United Nations Division for Ocean Affairs and the Law of the Sea

(c) Water Resources Abstracts on CD-ROM (1967-1994)

The Water Resources Abstracts database is produced by the Water Resources Scientific Information Centre, U.S. Geological Survey, U.S. Department of the Interior.

The database includes abstracts of current and earlier journal articles, monographs, reports and other publication formats. Records in the database cover the development, management and research of water resources.
(d) BIOSIS Previews

BIOSIS Previews is the computer-readable version of the citations found in Biological Abstracts from 1969 to the present. At the end of 1993, the database contained almost nine million records. Nearly seven thousand serial publications are monitored for inclusion, encompassing the entire field of life sciences.

(e) The Science Citation Index Online (Bath Information and Data Services)

The SCI represents the Institute of Scientific Information's online database that contains virtually every scientific article published since 1982. It is updated weekly and can be searched by author or keyword. In addition, the identity of articles that cite specific papers can be accessed, making this a very powerful search engine.

Keyword in title (and where possible abstract) searches were carried out using search terms designed to locate relevant information on stress in fish, pain perception in fish, and the impact of angling practices on fish. In addition, references already available were utilised in citation searching to identify papers that have referred to specific key work.

1. INTRODUCTION

1.1 Purpose of the review

Animal welfare is a subject of concern to many people. Pressure groups have for many years targeted the research community, and commercial organisations that employ animals for product testing, such as the cosmetics and pharmaceutical industries. However, in recent years, agricultural practices have also come under scrutiny, in particular the conditions prevalent in intensive rearing of animals. In addition to these areas of concern, those activities broadly termed "blood sports" have traditionally been the object of protest. In the past, such protest was largely directed at activities involving the hunting of mammals and birds. However, in recent years, the sport of angling has been subjected to increasing levels of criticism by a vocal minority.

Anglers, for the most part and by nature, are conservationists; they want to see the development of well balanced, healthy and thriving fish populations. They are, therefore, just as concerned as others to ensure that fish are treated in the best possible way, commensurate with the pursuance of the sport. Anglers were, however, never wholly convinced by the conclusions of the Medway Report (Medway, 1980), hence their reaction to it at the time. This report was commissioned by the Angling Governing Bodies (the National Federation of Anglers, the Salmon and Trout Association and the National Federation of Sea Anglers) and the British Field Sports Society in order to determine whether, in the intervening fifteen years, any progress has been made in determining the scientific position.

Most concern associated with the subject of animal welfare is focused on the likelihood of suffering arising through exposure of the animal to an environment, procedures, or stimuli perceived to invoke pain and/or stress. While methods for assessing the well-being of mammals, and to a lesser extent birds, receive serious discussion (Curtis, 1985), there is less understanding of the factors that underlie the well-being of lower vertebrates, including fish.

This situation has arisen because, inevitably, most research on animal welfare, and particularly on pain and stress in animals, is either clinically or agriculturally orientated (Moberg, 1985). However, during recent years, and to a large extent as a result of the rapidly expanding

worldwide aquaculture industry, there has been a substantial amount of research carried out on aspects of the physiology of fish. Much of this research is relevant to discussion of welfare concerns. In addition, there has been a limited amount of research that is directly concerned with the impact of angling on fish and fish populations. Some of this material is also relevant to a discussion of the welfare of fish in relation to angling practices. Nonetheless, because of the comparatively limited amount of information on the factors affecting the welfare of fish, and perhaps as a result of the inaccessibility of these data to the general populace, there has been little informed debate on the subject.

The intention of this report is to identify and summarise the currently available knowledge on aspects of fish welfare relevant to angling practices. This will permit the effects of such practices on fish to be considered in the context of informed scientific evidence.

1.2 Possible adverse effects associated with angling practices

There are three main areas of concern related to the treatment of fish by anglers that will be addressed within this review:

1. Physiological stress. Prolonged activation of the physiological stress response can have harmful effects in animals. Do the procedures associated with the capture of fish by angling; hooking, playing, landing, unhooking, confinement within keepnets, weighing, cause a stress response? If so, is the response severe enough to cause concern regarding the welfare of the fish?

2. Physical damage. Do the processes associated with the capture of fish cause damage that might affect the subsequent welfare and survival of the fish?

3. Pain. It has been suggested that hooked fish experience pain during capture and unhooking. Is there evidence that fish can experience "pain and suffering" in a manner analogous to higher vertebrates?

Both stress and pain are emotive words that tend to conjure up subjective images and perceptions in the reader. However, both phenomena comprise well-documented physiological mechanisms.

Although physiological stress can be accurately detected and quantified, the measurement and detection of pain responses in animals is fraught with difficulties (Kitchell and Johnson, 1985; Bateson, 1991). It is generally accepted that adverse effects on animal well-being, or the cause of suffering in animals, predominantly relate to these factors.

Although stress and pain are often linked semantically, and can be linked causally, they are on the whole, completely independent physiological processes. This report will therefore consider each in turn.

2. PHYSIOLOGICAL STRESS

2.1 Terminology and concepts

The concept of *stress* as it relates to animals, can be traced to the work of Cannon (1929), who identified mechanisms underlying the "fight or flight" response of animals to challenge, and to the seminal work of Selye (1936) who conceived a "General Adaptation Syndrome" which sought to explain the adverse effects of prolonged activation of the animals adaptive response to stress. Almost all reviews of the field of stress physiology in mammals draw attention to the possible confusion that can arise through the use of inappropriate terminology, and the fact that the term *stress* is used in many different contexts. Common to all definitions of stress is the concept of "threatened homeostasis" (Chrousos, 1992; Johnson *et al*., 1992; Dorn and Chrousos, 1993; Sutanto and de Kloet, 1994; Tsigos and Chrousos, 1994). *Homeostasis* describes the maintenance by an animal of a stable internal environment, essential for normal function. Threats to homeostasis are countered by an adaptive response, the *stress response*, comprising "neuroanatomical and functional structures that function to produce the behavioural, physiological and biochemical changes directed towards maintaining homeostasis" (Johnson *et al*., 1992). A *stressor* or *stress* is considered to be any perturbation that disrupts homeostasis, or is perceived as a threat to homeostasis.

In response to a stressor, the animal alters its behaviour and physiology to maintain homeostasis. The adaptive measures employed by the animal to overcome the stressor have been summarised by Chrousos and Gold (1992) and are reproduced in Table 1. .

The adaptive response to stress can be summarised as the redirection of both behaviour and energy (Johnson *et al*., 1992). The net effect is to enhance those behavioural processes likely to enhance survival, while suppressing those of no survival value. Similarly, only those physiological processes involved in increasing the likelihood of survival are enhanced, while as far as possible, non-essential physiological processes are shut down or reduced in activity. There is a general switch from an anabolic state to a catabolic state.

It is worth emphasising at this point that stress is a normal component of an animal's life, and it

Table 1. Behavioural and physical adaptation during stress

Behavioural adaptation:	Adaptive redirection of behaviour Acute facilitation of adaptive, and inhibition of nonadaptive, neural pathways Increased arousal, alertness Increased cognition, vigilance and focused attention Suppression of feeding behaviour Suppression of reproductive behaviour Containment of the stress response
Physical adaptation:	Adaptive redirection of energy Oxygen and nutrients directed to the central nervous system and stressed body site(s) Altered cardiovascular tone, increased blood pressure and heart rate Increased respiratory rate Increased gluconeogenesis and lipolysis Detoxification from toxic products Inhibition of growth and reproductive systems Containment of the stress response Containment of the inflammatory/immune response

(From Chrousos and Gold, 1992)

has even been argued, on behalf of domestic animals, that some degree of stress is a *necessary* part of life to ensure well-being (Curtis, 1985).

2.2 The response of fish to stress

There are two major differences between the study of the stress response in mammals and in fish; the importance placed on the emotional or psychological content of stressors in mammals, and the better understanding of behavioural responses to stress in mammals. These factors are rarely considered in the context of research on fish, although some behavioural work is now appearing (see Schreck, 1990). Nevertheless, there has been a considerable amount of work on the physiology of the stress response in fish and the available data suggests that the response of fish to stress is directly comparable to that of higher vertebrates. That these physiological mechanisms have been strongly conserved is unsurprising, given the adaptive value of the stress response.

The stress response in fish has, for convenience, been classified into three stages (Wedemeyer *et*

al., 1990). These can be summarised as:

1. Primary response. This is the neuroendocrine component of the response. Perception of the stressor by the fish initiates a rapid release of catecholamines from the chromaffin tissue (the adrenomedullary homologue in fish), predominantly adrenaline (epinephrine) and noradrenaline (norepinephrine) (Gingerich & Drottar, 1989). At the same time as the catecholamine response is initiated, the hypothalamic-pituitary-interrenal axis is activated. This hormonal cascade is initiated by the release of corticotropin-releasing hormone (CRH) from the hypothalamus (Fryer & Lederis, 1986; Okawara *et al*., 1992; Weld *et al*., 1987), which, in turn, stimulates the release of adrenocorticotropic hormone (corticotropin, ACTH) from the pituitary (Sumpter *et al*., 1986; Pickering *et al*., 1987). ACTH acts on the interrenal to promote the synthesis and release of the steroid hormone cortisol (Balm & Pottinger, 1993).

2. Secondary responses. These comprise changes in physiology arising directly or indirectly from the primary response. The catecholamines act on the cardio-respiratory system, increasing the heart rate and stroke volume (Satchell, 1991) and causing a brief vasoconstriction followed by a prolonged vasodilation of the gill vasculature (Wood, 1975). The overall effect being an increase in the functional surface area of the gill available for gas exchange (Booth, 1979). These catecholamine-mediated branchial and cardiac adjustments lead to a disruption of the fish's water and solute balance (Gonzalez and McDonald, 1992), such that there is a net loss of ions in freshwater, and a net gain in seawater (Avella *et al*., 1991). Contraction of the spleen also occurs, with a resultant increase in the number of circulating erythrocytes (Hadj-Kacem *et al*., 1987; Pottinger *et al*., 1994b). The catecholamines also affect aspects of intermediary metabolism. Following the initiation of the stress response there is mobilisation of energy by glycogenolysis and gluconeogenesis, both of which processes result in elevated blood glucose levels (Haux *et al*., 1985; Laidley and Leatherland, 1988; Morales *et al*., 1990) and lipolysis, leading to elevated plasma free fatty acid levels (Sheridan, 1986; Waring *et al*., 1992). These processes are believed to be under the control of the catecholamines (Ottolenghi *et al*., 1986; Hayashi and Ooshiro, 1977) and cortisol, although a consensus on the exact role of these hormones has yet to be established (Suarez and Mommsen, 1987; van der Boon *et al*., 1991). Elevated blood lactate levels are also often reported following exposure to certain forms of stress (Pickering *et al*., 1982; Schwalme and Mackay, 1985) although this is less a direct effect of activation of the

hypothalamic-pituitary-axis than a reflection of the metabolic consequences of exhaustive exercise. The physiological effects of exhaustive exercise in fish are reviewed in detail by Wood (1991).

In addition to these adjustments, the net result of which is to provide the fish with oxygen and energy to fuel activity in the short term, there are effects on other physiological processes. These include a suppression of growth hormone secretion (Pickering *et al*., 1991; Farbridge and Leatherland, 1992), a generalized reduction of activity within the reproductive system (Pickering *et al*., 1987; Donaldson, 1990; Carragher and Pankhurst, 1991; Campbell *et al*., 1992; Pankhurst and Dedual, 1994) and suppression of components of the immune system (Anderson, 1990; Barton and Iwama, 1991). In addition, behavioural modification has been reported to be a consequence of stress, manifested as alterations in feeding behaviour and aggression for up to 24 h post-stress (Mesa and Schreck, 1989).

These responses can all be seen to have adaptive value to the fish in the context of a stressor which is short in duration, or which the fish can avoid or overcome. However, the stress response ceases to be of adaptive value, and actually becomes maladaptive, under conditions in which the duration of the stress or stress response is prolonged, or repeated incidences of stress occur. It is under conditions of chronic stress that the deleterious *tertiary* effects of stress become apparent.

3. Tertiary responses. Prolonged activation of the stress response results in reduction in growth (Pickering, 1993), reproductive success (Campbell *et al*., 1992; 1994), and survival (Angelidis *et al*., 1987; Peters *et al*., 1988; Pottinger and Pickering, 1992). It is assumed that these effects at the level of the individual fish can lead to adverse effects on population and community structure.

2.3 Chronic stress and maladaptation: the role of cortisol

2.3.1 Factors influencing cortisol levels during stress. It has become apparent that most of the adverse effects of chronic stress in fish arise due to the action of cortisol. As noted above, this steroid hormone is released from the interrenal tissue into the blood, within minutes of the onset of stress.

Salmonid fish first show a corticosteroid stress response within two to five weeks of hatching, depending on the water temperature (Pottinger and Mosuwe, 1994; Barry *et al*., 1995). Normal "resting levels" of this hormone in the plasma of unstressed fish are < 5 ng/ml, rising by up to a hundred-fold within 30 mins of the onset of stress (Barton and Iwama, 1991).

The magnitude and duration of the elevation in plasma cortisol levels following the onset of stress tends to be proportional to the severity and duration of the stress, but species (Davis and Parker, 1986; Waring *et al*., 1992), strain (Woodward and Strange, 1987: Pottinger and Moran, 1993), and water temperature (Sumpter *et al*., 1985; Barton and Schreck, 1987) are all factors which modify the response to stress.

If the stress is short in duration (acute) cortisol levels return to pre-stress levels within hours (Pickering and Pottinger, 1989; Waring *et al*., 1992) whereas if the stress is continuous (chronic) cortisol levels may remain elevated throughout the period of stress, although evidence of acclimation may be observed (Pottinger and Moran, 1993; Pottinger *et al*., 1994a).

There is also evidence that under some circumstances, fish will acclimate to a repeated stressor and cease to show a stress response despite initially responding with elevated cortisol levels (Pickering and Pottinger, 1985) while in other circumstances, repeated acute stressors may result in an additive stress response (Barton *et al*., 1986).

The level of corticosteroid responsiveness to stress of a proportion of fish within a population is consistent with time (Pottinger *et al*., 1992) and corticosteroid stress responsiveness appears to have a genetic basis (Fevolden *et al*., 1993; Pottinger *et al*., 1994b).

2.3.2 Effects of chronic elevation of cortisol levels. Prolonged elevation of plasma cortisol levels in otherwise unstressed fish, achieved by implanting fish with cortisol-releasing pellets or by administering cortisol via food, has been shown to reduce growth and condition (Barton *et al*., 1987; Davis *et al*., 1985; Pickering *et al*., 1989). In addition, the administration of cortisol to fish has a major impact on the reproductive system. Elevated cortisol levels cause a reduction in the concentration of circulating gonadal steroids, reduce gonad size, reduce the circulating levels of

vitellogenin and reduce the levels of gonadotropic hormone in the pituitary and the number of hepatic estradiol receptors (Carragher *et al*., 1989; Pottinger and Pickering, 1990; Foo and Lam, 1993a, b). The impact of chronic or repeated acute stress on reproductive success is severe, resulting in delayed ovulation, reduced egg size, lower sperm counts and a reduction in survival of the progeny (Campbell *et al*., 1992; 1994).

The most profoundly damaging effects of prolonged elevation of plasma cortisol levels relate to the impact of cortisol on the immune system. The link between stress, elevated cortisol levels and immunosuppression in fish has been reviewed by Barton and Iwama (1991) and Schreck *et al*. (1993). Administration of cortisol to otherwise unstressed fish significantly increases mortality due to bacterial and fungal pathogens (Pickering *et al*., 1989; Pickering and Pottinger, 1989) and increases the susceptibility of fish to parasitic infections (Woo *et al*., 1987). The effects of cortisol on specific components of the immune system have been highlighted in numerous reports. Cortisol will cause marked reductions in the number of circulating lymphocytes (Pickering, 1984; Ellsaesser and Clem, 1987), suppress the production of specific antibody-producing cells (Maule *et al.,* 1987), reduce the mitogenic response of lymphocytes (Ellsaesser and Clem, 1987) and reduce immunoglobulin M levels (Nagae *et al*., 1994).

2.4 Species limitations of stress research in fish

Most research on the impact of stress on fish, including much of that cited above, has been carried out on salmonid fish, in particular the rainbow trout (*Oncorhynchus mykiss*), brown trout (*Salmo trutta*) and Pacific salmon (*O. nerka, O. kisutch, O. keta* and *O. tshawytscha*) thus the response to, and effects of, stress are best understood in these few species. This situation has arisen largely because of the economic importance of the salmonids worldwide in both natural fisheries and aquaculture, and the widespread use of the rainbow trout as a basic experimental model.

For reasons related both to diversification of aquaculture, and interest in comparative physiology, there is an increasing body of work which has examined the response to stress of other species of fish, including salmonids such as the Atlantic salmon (*Salmo salar,* Waring *et al*. 1992), brook trout (*Salvelinus fontinalis*, Biron and Benfey 1994) and lake trout (*Salvelinus namaycush*, Barry

et al. 1993), and non-salmonids such as flounder (*Platichthys flesus*, Waring *et al*. 1992), golden perch (*Macquaria ambigua*, Braley and Anderson 1992, Carragher and Rees 1994), yellow perch (*Perca flavescens*; Schwalme and Mackay, 1991), channel catfish (*Ictalurus punctatus*, Limuswam *et al*. 1983, Mazik *et al*. 1994), red drum (*Sciaenops ocellatus*, Thomas and Robertson 1991), and striped bass (*Morone saxatilis*, Young and Cech 1993a, b).

However, there has been no detailed work carried out on the physiological response to stress of those species of non-salmonid fish native to the United Kingdom and which represent the principle quarry of freshwater "coarse" anglers. Limited data are available for only four non-salmonid fish found in British waters, the common carp (*Cyprinus carpio*, Canals *et al*. 1989, Dabrowska *et al*. 1991, Jeney *et al*. 1992, Kakuta and Murachi 1992, van Dijk *et al*. 1993), the perch (*Perca fluviatilis*, Haux and Sjöbeck 1985; Vinogradov and Klerman, 1987), the roach (*Rutilus rutilus*; Vinogradov and Klerman, 1987), and the pike (*Esox lucius*, Schwalme and Mackay 1985a, b). The effects of stress on seafish have been little studied, although some examples are provided in Section 2.5 below. What is apparent from the data available on those species that have been studied and are listed above is that, with minor exceptions, the basic qualitative response to stress remains the same regardless of species studied. This is unsurprising considering the adaptive importance of the stress response.

2.5 Effects of stress in freshwater and seawater

Many of the salmonid fish on which the bulk of research into stress has been carried out are anadramous and thus euryhaline, adapting readily to full-strength seawater. In seawater-adapted salmonids there is no evidence that the component parts of the stress response vary when compared to the response of the same species to stress under freshwater conditions. The only difference, which is immediately apparent, is that post-stress ionoregulatory disturbances lead to an efflux of ions in freshwater and an influx of ions in seawater (Avella *et al*., 1991).

With regard to species that are permanent seawater or brackish water residents, there are few data, but those that are available suggest no radical differences from the established "salmonid model". The physiological response of flounder to a brief period of net confinement differed from that of Atlantic salmon only in degree; in qualitative terms the response of both species was

similar (Waring *et al*., 1992). Similarly, the red drum (Robertson *et al*., 1987) and European sea bass (*Dicentrarchus labrax*; Hadj-Kacem *et al*., 1986, 1987) show a standard "salmonid" type response to physical disturbance. Atlantic cod, *Gadus morhua*, also display a typical "salmonid" pattern response to handling and transport stress (Hemre *et al*., 1991).

There may, however, be differences in the response to stress when a component of the stressor results in the imposition of exhaustive exercise on the fish. In particular, the degree of metabolic acidosis experienced by the fish may vary according to environment. Seawater-adapted rainbow trout recover more rapidly from the acidosis induced by exercise than freshwater-adapted trout (Tang and Boutilier, 1991) due to their ability to excrete more H^+ equivalents to the environment and more rapid removal of H^+ as a result of lactate metabolism in white muscle. It has also been suggested that recovery from exhaustive exercise may be a function of the lifestyle of the fish. Sluggish, sedentary fish such as flatfish may employ a different strategy for post-exercise recovery than active pelagic fish (Turner *et al*., 1983) that results in lower blood lactate levels, but is coupled with a slower return to normality.

2.6 Angling practices and stress

2.6.1 Physiological stress associated with capture. It is reasonable to infer from the data cited above, and from the nature of stimuli which evoke a stress response in fish (novelty, physical disturbance, noxious or toxic stimuli), that the process of hooking, playing, landing, unhooking and confining fish within keepnets is likely to elicit a physiological stress response. This also applies to the subsequent handling of fish associated with weighing and release. For game fish or sea fish destined to be humanely despatched immediately following capture, such a response may be considered irrelevant. For fish destined for return to the water the severity and duration of the response evoked by capture and related procedures is of great significance. If the process of capture elicits a short-term response, lasting hours, the experimental data cited above suggest that no lasting adverse effects on the well-being of the fish will occur. However, if the severity of the response to capture is such that the physiological stress response is prolonged, there is the possibility of adverse effects on growth, reproductive processes and the immune system.

Unfortunately, there has been very little work directed at quantifying the degree of stress

imposed on fish by angling procedures. In the most recent study on this topic, Pankhurst and Dedual (1994) examined indices of stress in rainbow trout captured from a natural population in a New Zealand river. The fish were caught by rod and line and separated into two groups on the basis of time required to land the fish; those fish which were rapidly captured (time from hooking to landing < 5 mins) and those with an extended capture time (15 mins). Fish were transferred within 3 mins of capture to recovery tanks. The authors found that plasma cortisol levels immediately following capture in the rapidly captured group were slightly higher than baseline levels in comparable unstressed fish. Within 1 h of capture, plasma cortisol levels were significantly elevated in all groups but in some cases were lower again within 24 h of capture. The authors indicate that stress associated with the holding conditions may have contributed to high cortisol levels at 24 h post-capture in some fish and suggest that given appropriate holding conditions, angled trout will recover from capture as quickly as laboratory-maintained fish. These authors also found that plasma lactate levels, elevated within 1 h of capture, had returned to baseline levels within 24 h. Finally, the authors also examined plasma levels of reproductive steroid hormones in the fish. Within 24 h of capture they observed a significant suppression of plasma testosterone and 17β-estradiol levels in females, though no change in 17α, 20β-dihydroxy-4-pregnen-3-one levels was observed. No mortality occurred as a consequence of capture. The authors concluded that catch and release angling results in negligible mortality but may have an inhibitory effect on some reproductive processes.

The only other study of which the author is aware, which examined angling-related stress and included plasma cortisol among the determinands, was commissioned jointly by the National Federation of Anglers (NFA) and the National Rivers Authority (NRA) to establish the impact of keepnet confinement on the physiology of a freshwater fish. The plasma cortisol, lactate and glucose response of carp was investigated after simulated capture and either immediate release, or transfer to keepnets for 4 h (Pottinger, 1995). In the course of three separate experiments, capture was found to elicit a significant elevation of plasma cortisol relative to levels in undisturbed control fish. The duration of the corticosteroid response to capture, with or without subsequent confinement was limited to between 4 and 24 h after the onset of disturbance. There was no evidence of chronic elevation of cortisol levels. The post-capture elevation of plasma corticosteroid levels was accompanied in some cases by disturbances in plasma glucose and lactate levels but, where such changes occurred, they were limited to between 4 h and 48 h after

the onset of the procedure. No mortality was observed following the experimental procedures. Similar perturbations in plasma cortisol, glucose and lactate were observed in carp caught by angling from a large earth pond. These results are interpreted by the author to indicate that the major factor responsible for stress-induced physiological perturbations in angled fish is capture; post-capture confinement does not appear to contribute substantially to the degree of physiological stress experienced by the fish.

A similar comparison between captured and released, and captured and restrained fish formed the basis of a North American study, in which a number of indices of stress were determined in walleye (*Stizostedion vitreum vitreum*) subjected to hooking, playing and restraint on "stringers" (Sobchuk and Dawson, 1988). The study was carried out to establish the likely impact on walleye released after capture and a period during which the fish are retained on stringers. Although walleye are normally killed after capture, anglers may retain a number of fish on a stringer during the session, smaller fish being released from the stringer as larger fish are captured and replace them. The procedure is therefore broadly analogous to the practice of retaining fish in keepnets before release, although keepnet confinement does not involve the physical restraint associated with attaching the fish to a stringer; fish in keepnets remain free-swimming. The authors of this study employed mean blood clotting time as an index of the physiological stress response, citing earlier work on fish which described effects of capture on this parameter (Bouck and Ball, 1966; Casillas and Smith, 1977). Stress appears to enhance the rapidity of clotting, an advantage under situations in which physical trauma may occur, but a disadvantage in that the occurrence of intravascular clots may result in mortality (Smith, 1980). The formation of intravascular clots is opposed by the stress-induced enhancement of the fibrinolytic system (van Vliet *et al.*, 1985). The authors found that mean blood clotting time decreased significantly with 1 min of playing; increasing the length of playing the fish did not further reduce clotting time. A marked hyperglycemic response was also observed with 1 min of playing and there was a significant increase in haematocrit. These changes were maintained for up to 4 h during retention on a stringer. In fish subject to capture only, clotting time and haematocrit had returned to pre-stress levels within 72 h of capture. Plasma glucose levels required between 48 and 72 h post-stress to return to normality. For those fish retained on a stringer for 4 h following capture, clotting time and haematocrit again required up to 72 h to approach pre-stress levels. Plasma glucose levels also returned to pre-stress levels within 72 h of

capture.

Although the authors suggest that the lack of difference in the duration of the response to hooking and playing alone, and hooking and holding, may reflect an inadequacy in the severity of the procedures, compared to angler-inflicted stress, or an ability of the walleye "to compensate for a greater level of stress", this presupposes that capture and retention are more stressful than capture and immediate release. The results of Pottinger (1995), cited above, suggest this is not necessarily the case.

In addition, the authors speculate that the lack of post-stress mortality in these experiments may be accounted for by the relatively restrained nature of their handling and hooking procedures. They suggest that "rougher" treatment by anglers may result in a greater degree of physical injury and more severe physiological responses. These assertions are not supported by evidence. However, the authors make the valid point that low water temperatures and favourable dissolved oxygen levels during the experiment may have reduced the overall level of stress experienced by the fish.

The effects of water temperature on the severity of capture-related stress was among the factors considered by Wydoski *et al*. (1976). In this study hatchery-reared rainbow trout were blood-sampled after being hooked and played for 0, 1, 2, 3, 4, or 5 mins, ten fish per time period. In addition, a second group of forty fish were played for 5 mins and transferred to a separate recovery tank where they were sampled at intervals following transfer. Wild rainbow trout were captured by angling from a lake and either blood-sampled after being played for specific time periods, or transferred to a recovery tank after 5 mins playing, and blood-sampled at intervals following transfer. Significant elevation of plasma glucose and reduction in plasma chloride levels was observed in all captured fish after 5 mins playing. The magnitude of these changes was more pronounced in hatchery than wild trout. Estimation of time to recovery was complicated by the possible additional stress of confinement following capture. The onset of hypochloremia and hyperglycemia was delayed in wild trout caught at 4^{o}C when compared to trout caught at 12^{o}C, and the magnitude of changes seen in hatchery fish at 20^{o}C was slightly greater than that observed at 10^{o}C. Larger hatchery-reared fish (43-48 cm) showed a more severe response to capture than smaller fish (20-25 cm). The authors reported no mortality associated

with the experimental procedures and suggested that, overall, recovery from hooking and capture required up to 72 h.

In a study closely patterned on that of Wydoski *et al*., (1976) the physiological response of largemouth bass (*Micropterus salmoides*) to angling stress was examined (Gustaveson *et al*., 1991). This study was more comprehensive in design than many of those cited in this review. The authors captured bass by rod and line, from one site (Lake Powell) at three different water temperatures (11-13, 16-20, 28-30^{o}C) and at a second site (Mantua Reservoir) at one water temperature only (23-26^{o}C). The fish were either immediately landed (within 15 secs, controls) or played for 1, 2, 3, 4, or 5 mins. To establish recovery times, groups of captured bass were released into net pens and sampled at intervals.

Overall, a similar response to capture, in qualitative terms, was seen in all groups. There was a significant elevation of blood glucose (except at the coolest water temperature) and blood lactate levels, within 1 - 5 mins of hooking. Curiously, a marked hyperchloremia was apparent in all groups together with a progressive increase in plasma osmolality during capture. Post-stress osmoregulatory dysfunction is usually characterised by a hypochloremia in freshwater (Avella *et al*., 1991). These effects were more pronounced at warmer temperatures, although no mortality was observed. The authors interpreted changes in blood parameters observed during the recovery period, in which fish were confined in net pens, to indicate continued stress, associated with the confinement rather than the capture event. They observed that physiological disturbances were minimal in fish played for less than 1 min, and within limits of tolerance even in fish played for 5 mins. They concluded that hooking and playing stress alone could not be responsible for acute or delayed mortality following capture.

The influence of capture method on selected blood parameters, and on survival, in rainbow trout was studied by Bouck and Ball (1966). Included in the comparisons was a group of fish caught by lure from their holding tank. Each fish captured was played to exhaustion, and transferred to a holding tank. The authors measured a number of haematological parameters (haemoglobin concentration, total protein, erythrocyte numbers and erythrocyte length) and examined the influence of capture on various plasma protein fractions. Unfortunately, there are several areas in which this study is open to criticism. The experimental fish were provided with only 6 days

acclimation to holding conditions before the onset of the experiment. All the fish captured by angling were removed sequentially from the same holding tank, inevitably leading to additional stress as the fish in the tank were disturbed by each successive capture event (sixteen in all). For the estimation of post capture mortality, the fish were maintained within a tank which was drained to within 3" of empty every three days, again a procedure likely to result in additional stress. Finally, the effect of capture procedures was assessed, not against control, unstressed fish, but by comparison with groups of fish caught either by electronarcosis or seine net. Significant reductions in total protein concentration, and in the abundance of various protein fractions, were observed in all groups of fish. The only result clearly related to capture by angling was an 87% mortality within 10 days of capture. However, given the inadequacies of the experimental design, it is impossible to attribute this to capture by angling alone. The study contributes little to our understanding of effects of capture.

One of the authors cited above was also involved in a subsequent study on the effects of capture stress on plasma enzyme activities in rainbow trout (Bouck *et al*., 1978). The aim of the study was to establish whether certain enzymes, of possible diagnostic value, could be measured in fish without changes due to capture method obscuring the underlying baseline value. In this study, baseline controls were included. Although the methodology is not clearly described, it is assumed that hooked fish were captured by lure from their holding tanks. They were allowed to struggle for approximately one minute before being sampled. The authors found that within one minute of capture, no changes in the selected enzyme levels could be detected in fish caught by angling, compared to the control fish, caught with a minimum of disturbance. However, haematocrits were significantly elevated within this time period.

The levels of plasma lactate, glucose, and cyclic adenosine monophosphate (cAMP) were measured in rainbow trout at intervals following simulated capture by angling (Perrier *et al*., 1978). This procedure involved the manual insertion of a hook in the upper jaw of hatchery-maintained rainbow trout, which were then "played" on rod and line for 90 secs. The authors observed a significant increase, in all three parameters measured, within minutes of capture. There was a complete recovery of cAMP and glucose levels within 16-64 h of capture and of lactate within 4-16 h of capture. The authors are not clear regarding the physiological significance of plasma cAMP, suggesting it reflects general metabolic activity. Lactate

production undoubtedly arises as a consequence of the enforced exercise and consequent anaerobic metabolism in white muscle, while the rise in plasma glucose levels can be presumed to indicate the occurrence of a classic stress response.

Despite the limited amount of information available in the literature, several generalisations can be made arising from the studies described above:

1. Capture by angling is always accompanied by physiological disturbances typical of activation of the hypothalamic-pituitary-interrenal axis. Capture by angling may therefore be considered to cause physiological stress in fish.
2. Physiological recovery is complete within 24 - 72 h of capture. The stress associated with capture may be considered acute, rather than chronic, and unlikely to have long-term impact on the well-being of the fish.
2. Mortality of fish within 72 h of capture is rare.
3. Water temperature may be a significant factor in determining the severity of the stress response to capture.
4. The stress of capture may be more severe for larger fish.
5. There is an overwhelming requirement for well-designed studies to examine the physiological effect of capture, and time-course of recovery following capture, in non-salmonid European fish. Such studies should encompass an examination of the effect of water temperature, fish size and delayed release.

2.6.2 The respiratory and metabolic effects of capture. In addition to the activation of the pituitary-interrenal axis by the process of capture and handling, which is evident from the studies cited above, an additional important factor to be considered is the effect of what may, in some cases, amount to exhaustive exercise as a consequence of struggling during "playing" and during handling. Therefore this section will examine the literature pertaining to the metabolic/respiratory effects of the exercise associated with capture.

The effects of capture by angling on the metabolic and respiratory physiology of a sedentary fish, the muskellunge (*Esox masquinongy*), were examined by Beggs *et al*. (1980). Unfortunately, the

experimental procedure employed by these authors is open to criticism. The fish were not blood-sampled until approximately 22 mins following capture. During this period the fish were captured, anaesthetised, transported, unhooked and subjected to cannulation, an invasive surgical procedure. A series of blood samples was then collected at intervals up to 72 h following capture. The authors noted a reduction in blood pH, elevated lactic acid levels and a drop in total carbon dioxide and bicarbonate concentrations. Recovery from this acidotic state required 12 - 18 h. Thirty percent of the experimental fish died during the procedure. Although the authors claim that the changes in blood variables observed following capture arise primarily from angling stress and not anaesthetisation and cannulation, this is open to some debate. The anaesthetization and cannulation procedures were "controlled" for by monitoring the effects of these procedures on fish already cannulated and confined, not on fish that had *not* been subjected to capture stress. Therefore it is not really established what effect the surgical procedures would have on otherwise unstressed fish, nor is it established what resting levels of the parameters examined are in uncannulated unstressed fish. In addition, only parameters associated with respiratory stress were examined, despite apparent recovery of these variables within 18 h, it is conceivable that the pituitary-interrenal axis remains activated for longer. In a subsequent study by Schwalme and Mackay (1985b), in which pike (*E. lucius*) were caught by angling and blood sampled without cannulation, mortality level was less than 3% more than 48 h after capture. These authors suggest high mortalities in other studies may arise because of inter-species differences, the use of more severe exercise regimes, or effects of cannulation. In this study indices of respiratory disturbance had returned to baseline within 6 - 48 h, although plasma glucose levels remained elevated up to 96 h after capture.

A more recent, and better-designed, study has examined in detail the respiratory effects of exhaustive exercise and brief air exposure on rainbow trout (Ferguson and Tufts, 1992). In this study, rainbow trout were exercised to exhaustion and exposed to air for 60 secs. This group of fish were compared to a group that were exercised but not exposed to air. The recovery of both groups was monitored for 4 h. The authors examined the severity of disturbances to acid-base balance in the fish. Plasma (or extracellular) pH was more markedly reduced, and blood lactate levels were more than twice as high, in air-exposed fish than in exercised-only fish. In fish exposed to air, gas transfer across the gills was impeded, because of the collapse of the delicate lamellar structure of the gill surface, and the blood CO_2 concentration rose while blood O_2 levels declined. The authors highlight the reduction in blood O_2 as the most critical effect of exposing

exhausted fish to air. There was significant mortality among the exercised and air-exposed fish, compared to the exercised only fish. Of those fish that were exposed to air for 60 secs, 28% survived the 12 h following exercise compared to 88% survival among the exercise-only group. Even in those fish that subsequently died, the extracellular acid-base balance of the fish appeared to be returning to normal although death occurred between 4 and 12 h later. The authors did not report the time required for complete recovery of the surviving fish. The possible mechanisms underlying fish death after exercise are discussed by Wood (1983) who suggests that it is intracellular acidosis which is the crucial factor leading to mortality.

The authors acknowledge that their experimental regime; in particular their use of hatchery-reared fish and repetitive blood sampling, may have influenced their results but conclude that the brief air exposure which occurs in catch and release fisheries is an important additional stress in an exhausted fish and may have a significant impact on the number of released fish which survive. One factor that undermines the immediate applicability of these results to fish captured by angling, and is common to almost all the exercise studies described here, is the severity of the exercise protocol employed by the authors. Fish were exercised by manual chasing until they failed to respond to further chasing. This required about 10 minutes. Arguably, most fish landed by anglers are unlikely to experience such prolonged and severe exercise, being brought to the net within a shorter period of time and probably before complete exhaustion has occurred. However, the authors regime may be appropriate when considering fish caught on inappropriately light tackle, where the angler is unable to exert sufficient pressure to land the fish rapidly.

In addition, the authors use of hatchery-reared fish may have been a factor of more significance than they acknowledge. In a previous study by one of the authors (Tufts *et al*., 1991), "wild" Atlantic salmon were exhaustively exercised by exactly the same method as employed in the study described above. However, despite a physiological response almost identical to that observed in the hatchery-reared rainbow trout employed in the later study, *no mortalities* resulted, full recovery of the parameters occurring within 24 h. The authors conclude that wild salmonids may be better adapted for exhaustive exercise though what the nature of such an adaptation would be is not clear. Young and Cech (1993b) have demonstrated that the rate of recovery from handling stress in striped bass is increased in fish which have been "exercise conditioned", a phenomenon the authors attribute to higher capillary density in the muscle of

exercise-conditioned fish enhancing the clearance of lactate and cortisol. Conversely, Woodward and Strange (1987) reported that stress-induced plasma cortisol elevation was more extreme in wild rainbow trout than in hatchery-reared fish, a feature that may arise from generations of selection leading to "domesticated" strains of trout. These observations suggest that it may be inadvisable to assume that results obtained with fish reared under aquacultural conditions for many generations are wholly applicable to wild fish.

The effect of water temperature on the metabolic response to exhaustive exercise was recently examined (Kieffer *et al*., 1994). These authors employed manual chasing of trout for 5 mins at one of two temperatures (5^{o}C or 18^{o}C) to induce a state of exhaustion in the fish. Interestingly, although there was a more severe acidosis in the fish exercised at 18^{o}C than at 5^{o}C , and blood lactate levels post-exercise were two-fold greater in the fish at the warmer temperature, the time required for recovery to pre-exercise levels was similar at the two temperatures, about 8 h. This result is perhaps contradictory to the intuitive assumption that higher temperatures result in more severe physiological disruption following stress and suggests that the effect of water temperature on the metabolic response to capture may not a major consideration.

Further to the observation by Wydoski *et al*. (1976) that larger fish may experience a more severe stress response following capture, Ferguson *et al*. (1993) examined the effects of size on the metabolic status of rainbow trout exercised to exhaustion. By examining the relationship between fish length and the response of a range of metabolic indices to exhaustive exercise, they demonstrated that in rainbow trout, size has an important influence on the storage and utilization of the metabolic fuels required to sustain anaerobic exercise and on the acid-base status of white muscle following severe exercise. Exercise to exhaustion resulted in higher lactate concentrations in large fish than in small and in higher metabolic H^{+} production in large fish. However, because larger fish did not possess a greater buffering capacity than smaller fish, intracellular pH dropped to a greater extent than in small fish. Time to recovery was not reported. The authors make the point that these observations may be of significance to consideration of catch and release angling in that larger individuals may experience a greater physiological disturbance than smaller fish following similarly exhaustive exercise. The likely reasons for constraints on anaerobic capacity increasing with size are discussed by Goolish (1991).

In addition to the substantial body of work on the exercise physiology of salmonids there has been limited work on the response of certain non-salmonid fish to exhaustive exercise that is of relevance to this review. The effect of acclimation temperature on the metabolism of roach (*Rutilus rutilus*) has been examined (Dalla Via *et al*., 1989). These authors measured the whole-body concentrations of certain key metabolites in roach prior to, during and following subjection to exhaustive exercise (chasing). Total tissue lactate levels rose rapidly following exercise, the increase being two-fold greater at 20^{o}C than at 4^{o}C. Recovery to pre-exercise values after exhaustive exercise required between 2 and 8 h. The authors also noted that peak lactate levels were reached within minutes of the cessation of exercise, in contrast to trout in which peak levels may not be observed until several hours following exercise. The authors do not discuss whether this is of adaptive significance to the fish. The authors do not report any mortality following the experimental procedure. In a similar study on the effects of forced exercise on roach longer recovery times of up to 24 h were reported (Wieser *et al*., 1986). The reasons for this disparity are not apparent.

The impact of exercise training on the metabolic recovery from handling stress has also been examined in chub (*Leuciscus cephalus*; Lackner *et al*., 1988). Although the authors describe the stress procedure as handling, effectively the fish were exercised to exhaustion by mechanical chasing and sampled at intervals before, during and after exercise. Overall, the authors found the metabolic response of untrained chub to exercise to be similar to that of untrained roach (Dalla Via *et al*., 1989). The recovery time for return to physiological normality following exercise was found to be significantly reduced in the exercise-trained chub, compared to the untrained chub although in both cases recovery was complete within 2 h of exercise. The authors speculate that the aerobic capacity of the swimming muscle has been improved via hypertrophy and hyperplasia of red muscle fibres rather than an increase in the activity of oxidative enzymes.

These data, and the data cited above for the effects of training on recovery from exercise in salmonids and bass, raise an interesting point regarding the possible importance of the environment from which a fish is caught. Is tolerance of the physical activity imposed by rod and line capture in a given species likely to be a function of whether the fish inhabits, at one extreme, a still water or, at the other extreme, a rapidly flowing river?

Overall, a number of generalisations may be made regarding the effect of severe exercise on fish:

1. Even when exposed to an exercise regime arguably more severe than that imposed by rod and line capture, the available data suggest that most species of fish recover baseline respiratory and metabolic levels within 8 - 24 h.
2. Fish size and water temperature both affect the severity and duration of the metabolic and respiratory effects of severe exercise.
3. Severe exercise can cause mortality in fish. Whether this arises under experimental conditions because of the imposition of unrealistically extreme levels of activity, or because of the use of "unfit" fish is not clear.

2.6.3 The impact of capture stress on post-release behaviour. Although none of the studies discussed in this section refer directly to the imposition of stress by angling, it is nonetheless appropriate to consider their results as indicative of the likely effects of capture by angling.

In a study designed to determine the effect of electrofishing, a common fisheries management tool, on the behaviour and physiology of cutthroat trout (*Oncorhynchus clarki*) Mesa and Schreck (1989) observed marked behavioural alterations in fish following release. Fish captured from a stream by electroshock and marked before being released were observed to display lethargy and cover-seeking behaviour. These wild fish required at least 24 h to recover normal behaviour and activity patterns. The authors noted that aggressive behaviour declined, and feeding, although observed within 4 h of release, did not reach precapture intensity until 24 h after capture. The authors found there was a significant inverse correlation between plasma cortisol levels and behaviour in fish post-capture; feeding and aggressive interactions increased as plasma cortisol levels declined.

In a study in which the stressor employed was a 1 min period of air exposure, juvenile coho salmon were observed to attain control levels of predator avoidance within 90 mins of release (Olla and Davis, 1989). However, plasma cortisol levels remained significantly elevated for at least 240 mins after release suggesting that basic behaviours may not be strictly dependent on, or correlated with, physiological alterations arising from stress (Olla *et al*., 1992).

A more recent study (Mesa, 1994) also failed to demonstrate a significant correlation between the recovery of physiological indices from stress and the return of behaviour patterns to normality. Juvenile chinook salmon were exposed to multiple handling (brief netting and air exposure) or a more severe "agitation" stress in which fish were poured from one bucket to another for a period of 5 mins. The stressed fish, and a group of control unstressed fish, were then transferred to a tank containing a group of northern squawfish (*Ptychocheilus oregonensis*). Juvenile salmon subjected to multiple handling were lethargic and were more vulnerable to predation in the first hour after release than unstressed controls. salmon that experienced multiple agitation stress were also more vulnerable to predation. In longer term tests, differences in predation levels between controls and stressed fish were not apparent, probably, the author suggests, because the salmon congregated within the tank to form a tight school. The author concludes that juvenile salmon are capable of avoiding predators soon after experiencing a stressful event.

In a study designed to assess the effects of physical damage incurred during hatchery release and dam passage on vulnerability to predation, Gadomski *et al*. (1994) exposed control and descaled (10-20% of body area) juvenile chinook salmon to a predator. Despite the severe physical damage and associated stress response in the descaled fish, there was no difference in the predation risk experienced by either control or descaled salmon.

Overall, it can be concluded that:

1. Capture by angling is likely to result in some short-term modification of behaviour. In prey fish, this may result in an increased susceptibility to predation.
2. The duration of behavioural modification following stress is shorter than the period required for physiological recovery from stress.
3. There has been no research on species native to the UK.

2.6.4 Physical damage. There is little or no information on the general physical consequences of the handling associated with capture of fish by angling. However, there is a considerable body of work that specifically addresses the effect of hooking on survival of fish, almost all of which

originates from North America. In general, most of the work has been carried out to establish the impact on undersized fish caught and then returned to the water by anglers, although the increasing prevalence of catch-and-return and no-kill fisheries in North America has also led to research in this area. An excellent review of work in this field was recently published (Muoneke and Childress, 1994) but it is nonetheless appropriate to examine a range of studies in detail for the purposes of the present review, including many that have been published since Muoneke and Childress's review was completed. It is striking that no work of European origin on this subject could be located. The following summary is not intended to be exhaustive, but focuses on the more recent studies.

In a 4-year study on hooking mortality in land-locked Atlantic salmon, 18% of hooked fish died compared to 4% of control (trapped) fish during the spring season during the 5-day post-capture period. In autumn the figures were 8% and 2% respectively (Warner, 1978). No differences in mortality were observed between single and treble hooks, or between fly and spinner. Unsurprisingly, mortality was higher among fish hooked in the gill area (63%) than those hooked in the mouth, and fish which bled following hooking (35%) were more likely to die than those that did not (10%). As will be apparent from the work cited below, these observations are typical of the results obtained by many authors on a variety of fish species.

A higher mortality among Atlantic salmon caught on worm (35%) than fly (4%) was attributed to the fact that nearly 37% of worm-caught fish were hooked in the oesophagus whereas most fly-caught fish were hooked in the mouth or jaws (Warner and Johnson, 1978). The authors relate this difference to feeding behaviour; if not hooked immediately, fish will reject a fly, whereas fish accepting the worm bait ingest it even if not hooked rapidly. As in the study described above, the mortality of fish that bled after hooking (86%) was significantly greater than mortality among fish that did not bleed (15%). Size appeared to have no effect on hooking mortality.

In a study of the effect of hooking on brown trout beneath the legal size limit, the effect of hook size was examined (Hulbert and Engstrom-Heg, 1980). Hatchery-reared brown trout were captured by angling with worm-baited hooks from a large artificial pond. After capture, the fish were transferred to large aquaria and monitored for 14 days. Post capture mortality was 13.5%, and of this 77% occurred within 24h of capture although losses continued throughout the 14-day holding period. Approximately 67% of the fish captured were lightly hooked and mortality

among these fish showed no significant difference from that of control fish. The overall mortality of deeply hooked fish was 38.7% but this was markedly improved by clipping the leader (mortality 17.5%) rather than attempting to remove the hook (mortality 59%). The authors emphasise that hooking and rapidly landing a fish is a minor source of mortality and that lethal injuries most often occurred when hooks were removed from the upper digestive tract and gills. They suggest that if leaders had been clipped on all the deeply hooked fish in this study, overall short-term mortality would have been approximately 7%.

These authors also found that fish caught with the larger hook size employed (no. 4) had a greater rate of mortality than those caught on other hook sizes (6, 8, 10). This they attributed to a greater number of gill-hooked or deep-hooked fish caught with this hook size. Larger hooks were also less efficient than small hooks; the time taken to catch 50 fish with worm-baited large hooks was greater than the time required to catch the same number of fish with smaller hooks. A greater occurrence of eye hooking was also noted with the large hooks. Hook size as a factor in post-capture mortality was also considered by Titus and Vanicek (1988) who found that at least under high temperature conditions, lower mortalities were observed with barbless treble hooks, compared to barbed treble hooks and barbless single hooks. This was not ascribable to the anatomical site of hooking, as almost all fish caught with each hook type were jaw-hooked. These data are in accordance with the conclusions of Klein (1965) who suggested that treble hooks are less often swallowed as deeply as single hooks.

In a detailed study, Dotson (1982) determined mortality among cutthroat trout following capture with fly or spinning tackle employing three hook types (no. 10 barbed, no. 10 barbless, no. 12 treble). Fish were captured from hatchery raceways, reeled in as quickly as possible, and after capture, released into adjacent raceways where recovery was monitored for 30 days. The proportion of fish caught on single hooks and clean-hooked in the upper jaw was approximately 75%. Of those fish caught on treble hooks 79% had only one point penetrating the flesh. Only one mortality was observed among the caught fish, although the author may have biased the result by including in the study only those fish hooked in the jaw. Mortality rate for the 25% of fish not hooked in the jaw is not provided. The data from other studies cited here suggest it would be high.

In a second part of this study, rainbow trout were caught on fly (no. 10 barbed hook) and played to exhaustion. In six experiments (388 fish) an overall mortality of 6.7% was observed within 30 days of capture, most deaths occurring within the first few days. Higher mortality was noted in those groups caught at higher water temperatures but mortality did not appear to be size-related.

The survival of wild-caught lake trout (*Salvelinus namaycush*) after hooking was examined by Loftus *et al*. (1988). Fish were caught by anglers, and post-capture mortality was established by returning hooked fish to the lake after tethering them to a line-buoy system. This system involved clipping a line to the lower jaw of the fish and attaching the fish to a horizontal tether of 45-70m in length. Attaching the tether to a weighted line descending from a buoy via a two-way swivel permitted vertical movement. The fish were inspected after 48h had elapsed. During the two-year period in which the experiment was conducted overall hooking mortality was 14.9%. The time required to land fish during the study ranged from 53 secs to 5 mins 3 secs. No effect of time to landing was observed on mortality. Three types of lure were employed by anglers, plugs with treble hooks, spoons with treble hooks and spoons with single hooks; no statistically significant relationship between the type of lure and post capture mortality was observed. Approximately 72% of the fish in the study were hooked in the upper or lower jaw and these fish experienced a mortality of 6.9% while fish that were hooked internally displayed a mortality rate of 71.4%. In contrast to some of the studies reported here, these authors observed that smaller fish experienced a higher mortality than larger fish.

In a study in which the post-capture mortality of walleye (*Stizostedion vitreum*) was monitored for 12 days after capture, mortality was determined to be 1.1% (Fletcher, 1987). The fish were caught on baited lures and transferred to large net pens to recover after unhooking. The author notes that mortality may have been greater if water temperatures had been higher, citing a number of studies in which a relationship between post-capture mortality and water temperature was recorded (e.g. Pelzman, 1978). The author also highlights the possibility that type of tackle may influence mortality level, suggesting that for example trolled spinners are ingested more deeply than the jigged lures employed in his study.

Several factors affecting the mortality of largemouth (*Micropterus salmoides*) and smallmouth bass (*M. dolomieui*) during angling matches were examined by Bennet *et al*. (1989). These

authors monitored 15 tournaments over a four year period in which fish were transferred to holding cages and mortality for up to 36 h after capture was recorded. Overall, mean mortalities of 4.6% were recorded. Although initial mortalities (within 1 h) were higher for largemouth bass than smallmouth bass the authors suggest this may be related to the fact that smallmouth bass tournaments tend to be held earlier in the year when water temperatures are lower. There was a strong overall correlation between water temperature and initial mortality and also a positive relationship between the number of bass caught in a tournament and initial mortality. This latter result may be related to the crowding of fish within live wells before transfer to observation pens. Hartley and Moring (1993) demonstrated that oxygen is rapidly depleted from live wells when large numbers of fish were confined, or aeration systems were not run continuously. The authors also noted additional mortality of between 0.8% and 9% for largemouth bass held for up to 36 h. They conclude that fish mortalities associated with tournaments do not represent a significant impact on populations in large waters, although this may not be the case in smaller waters, and suggest that restricting tournaments during periods of warmer water may be advisable. Significant effects of temperature on hooking mortality were also reported by Titus and Vanicek (1988) who observed mortality among lure-caught cutthroat trout to be less than 1.5% at water temperatures below 15.5^{o}C but nearly 50% as water temperature rose to 21^{o}C.

A more recent study which considered the mortality of walleye during live-release angling tournaments found higher levels of mortality (mean 21.7%) than the study above, during two matches (Fielder and Johnson, 1994). Mortality estimates were subdivided into fish at the weigh-in and fish monitored in cages for 3 days after capture. Mean mortality at the weigh-in stage was 15.8% while that occurring during the 3-day observation period was 7%. These authors also summarise the results of previous studies that suggest that weather-related variables and water temperatures influence weigh-in and delayed mortality for walleye. They suggest that survival of fish caught by "pleasure" anglers may be greater because these are released immediately and not retained in live wells on board boats and propose that tournaments be restricted to periods of water temperature lower than 21^{o}C.

The role played by different types of lure/bait in causing post-capture mortality was studied by Clapp and Clark (1989). These authors examined hooking mortality in smallmouth bass caught on either live minnows or spinners. The study was carried out under controlled conditions in

artificial streams. The authors employed an experimental design in which the streams were fished for several periods (duration unspecified) and captured fish were returned to the channel after unhooking and noting the tag number on the fish. Forty-eight hours after the end of the test period the channels were drained and dead fish were counted. No control population was maintained; instead the authors compared the mortality of hooked fish to that of fish not caught from within the same channel. The authors found that the mortality of bass hooked on live minnows (11%) was significantly greater than that of bass hooked on spinners (0%) or those not hooked at all (4%). There was no statistically significant difference between mortality among spinner-caught and control fish. Interestingly, the authors noted marked differences in the "catchability" of individual fish, 35% of the fish were never caught, while 18% of the fish were caught three or more times including one individual caught on nine occasions. The authors acknowledge that implicit in their experimental design is the assumption that mortality within a test period occurred as a result of the last hooking event, and that cumulative effects of hooking did not influence the mortality figures. They also draw attention to the possibility of long-term sublethal effects of catch and release, a factor rarely addressed by other studies that consider the effects of hooking.

The contrast in mortality rates following capture on either artificial or live baits was also noted by Payer *et al*. (1989) who examined the post-capture mortality rates of walleye captured on either leeches or artificial lures. The mortality rate of fish caught on leeches (10%) was significantly greater than that of fish caught on lures (0%) and appeared to be primarily related to the tendency for leech-caught fish to be hooked more deeply in throat and gut. Bluegills caught on natural bait (worms) also display a higher post-capture mortality rate (88%) than fish caught with flies (32%) or lures (28%) and this again appeared to be related to deeply ingested hooks (Siewert and Cave, 1990). However, less severe mortality rates for bluegill caught on natural baits were reported by Muoneke (1992) who recorded 1% (winter) and 25% (summer) mortality within 72 h of capture on live hook bait. A distinct difference in the mortality of superficially hooked and deep-hooked fish was also observed in lake trout caught on live minnows (Dextrase and Ball, 1991). There was no mortality among fish hooked superficially in the mouth whereas 18.5% of fish hooked in the gills, deep in the buccal cavity, or in the stomach, died within 48 h of capture. Persons and Hech (1994) reported that lake trout caught by jigging showed much lower mortality rates than trout captured on static dead baits (9% cf. 32%) and that mortality was

closely related to the position of hooking. Fish hooked in the gills or gut showed a 36% mortality rate compared to 0% mortality among fish that were lip-hooked.

The likelihood that mortality related to lure type may also be species dependent is highlighted by the very low mortality (4.7%) observed during the 48 h post-capture period in black sea bass (*Centropristis striata*) caught on natural baits (Bugley and Shepherd, 1991). All these mortalities were, however, deep-hooked fish. The authors suggested that hook ingestion might increase mortality by increasing the degree of damage incurred during hook removal and by increasing the handling time necessary for hook removal. These authors also raise the question of depth of capture as a contributory factor to post-capture mortality, noting that displacement of the swim bladder through cloaca or mouth may result in severe trauma. Matlock *et al*. (1993) also failed to observe pronounced differences in the mortality of red drum (*Sciaenops ocellatus*) and spotted seatrout (*Cynoscion nebulosus*) caught with either single or treble hooks or on natural baits or lures. Mortality within 3 days of capture was 4.1% and 7.3% for the two species respectively. Several factors examined by other studies were included in an experiment by Nuhfer and Alexander (1992) who monitored mortality for 48 h post-capture in angled brook trout. All the fish were captured using artificial lures. Overall the mortality rate was 4.3% but there were nonetheless significant differences in mortality associated with lure type, ranging from 0% to 10.9%. The authors attribute these differences to the frequency with which the gill arch and oesophagus were damaged. The effect of elevated temperature on mortality was most pronounced in fish that were bleeding when unhooked. These authors also make the interesting point that the lack of correlation between fish size and hooking mortality reported by others may arise because of the limited size range of fish employed in some studies. They suggest that larger fish can more easily engulf multi-hooked lures deeply because of their larger gape. This fact may also account for some studies finding single hooks more damaging than treble hooks (Klein, 1965; Warner, 1976).

The few studies to examine the hooking-related mortality of a non-salmonid fish native to the UK focus on the capture of pike (*Esox lucius*). DuBois *et al*. (1994) describe the short-term (48 h) hooking mortality of pike captured by live fish on treble hook mounts or dead bait on large pike hooks (Swedish hooks). The authors found that mortality associated with capture on live bait was low (<1%) whereas capture on the Swedish hook rig resulted in 33% mortalities. In

accordance with many of the studies cited here, mortality was particularly pronounced for deeply hooked fish. Perhaps surprisingly, 84% of fish noted to be bleeding at the time of hook removal survived. Nor was the length of time the fish were allowed to run with the bait related to mortality or hooking location. Lower levels of mortality (<10%) than those observed for capture on Swedish hooks were reported for pike and muskellunge (*E. masquinongy*) caught on artificial lures (Falk and Gillman, 1975; Weithman and Anderson, 1978).

Muoneke and Childress (1994) have reviewed the information available on hooking mortality up to 1993, confirming that most studies have dealt with salmonids, centrarchids (particularly black basses) and percids (in particular walleye). They report that differences in mortality have been linked to bait type (artificial versus natural), hook type (number of hooks, hook size and barbs) season/temperature, depth at which fish were hooked, anatomical location of the hook wound, and fish size, and emphasise the variability in results obtained by various studies.
In recent years there have been several studies utilising more sophisticated means of addressing the problem of hook-related mortality in angler-caught fish. Bendock and Alexandersdottir (1993) employed radio telemetry to track the movements and fate of chinook salmon captured by angling for 5 days after release into the river at the point of capture. By these means they were able to overcome the problem common to almost all other studies on this subject, the fact that fish must be confined following capture for observation. They observed a mean post-capture mortality of 7.6% over a three-year period and found that mortality was highest among small males (<75 cm) compared to larger males and all females. Location of the hook wound and whether or not bleeding occurred were identified as the main factors associated with mortality. Most deaths (80%) occurred within 48 h of release.

Gjernes *et al*. (1993) employed a complex recursive causal model to analyse their data on hooking mortality in chinook and coho salmon during their first year of ocean life. They were able to demonstrate that hooking mortality is a two-stage process; injury location is a function of hook type and barb type (first stage) and mortality is a function of injury location and species (second stage). They estimated that overall mortality following release by anglers was approximately 30% for chinook and 14% for coho salmon.

A meta-analysis of hooking mortality in nonanadromous trout was carried out by Taylor and

White (1992) in which the data from 18 published studies was integrated and factors of significance in determining mortality were identified. The authors found that trout caught on bait had higher mortality rates than those caught on flies or lures, that barbed hooks caused greater mortality than barbless hooks, that brown trout had lower mortality rates than other non-migratory salmonids, and that wild trout showed greater mortality than hatchery-reared fish. Other variables (size of hooks, number of hooks and water temperature) did not show a statistically significant relationship to hooking mortality. The average mortality rate in the 18 studies was <12%, and if only barbless flies or lures are considered, was <3%. The authors suggest that because most studies required the holding of fish for monitoring and this may have exacerbated the mortality rate, actual figures may in practice be lower.

In summary, the effect of hooking on the subsequent survival of rod-caught fish represents the most-studied aspect of angling in relation to fish welfare. However, the data available deal almost exclusively with salmonid fish or non-salmonid fish native to North America and even here the range of species is limited. No data on hook-related mortality in British freshwater fish, other than the pike, could be located during the preparation of this review. To some extent it can be assumed that the general conclusions reached may be applied to other species. However, there are several aspects of the studies that make this less than satisfactory. First, in most studies involving wild-caught fish, fish were held for period following capture to allow the determination of mortality rates. No account was taken by most authors of the additional stress this may have caused. Mortality is ascribed throughout to "hooking damage". The possibility that mortality may occur due to other factors such as those discussed in preceding sections is not considered. Most, though not all, of the studies did not monitor the released fish much beyond a few days, therefore delayed mortality, or sublethal effects of capture and release were not discernible.

The main points that are apparent from consideration of the studies above are:

1. There is a measurable level of mortality associated with hooking of fish. Most studies have not monitored survival beyond 72 h following capture.
2. Mortality is low to negligible in fish that are hooked in the jaw, but can be extremely high in fish hooked in the throat, gills and deep-hooked in the gut. The majority of fish caught are

hooked in the jaw.

3. There may be differences in mortality rate associated with natural and artificial baits and with hook size and type. The species and size of fish may also be factors. There are too few data available at present to draw firm conclusions.

4. Almost all the data available on hooking mortality originate from North America. The species studied are almost exclusively predators, captured with either lures or bait. Although this information may be applicable to some species of fish native to the UK (perch, pike, zander, salmonids), there are no data in the literature derived from studies on cyprinid fish that constitute the majority of species sought by freshwater anglers in the UK.

3. PAIN PERCEPTION

3.1 Background

While it is possible to quantify stress in fish by measuring changes in physiological indices known to be involved in the response to stress, and to directly assess the physical damage caused by hook penetration or handling, and even to assess behavioural modification arising from stressful procedures, determining whether fish perceive pain as a result of procedures associated with capture by angling is far less straightforward.

The perception of pain by animals is a much-studied subject, largely because of the clinical importance of pain relief. Short and Van Poznak (1992) emphasise the extensive research effort expended on the control and prevention of pain by the medical, basic science and veterinary research communities. These authors consider pain to be a "natural" response, and that the existence of pain in animals other than man need not be debated, because of the evident benefits of possessing a pain response (see below). However, all workers in this field do not share this undiscerning and simplistic view. Bateson (1991, 1992) in particular, considers that the question of whether or not animals other than humans feel pain is far from being answered.

It is in laboratory animals, such as the rat and mouse, in which the best mechanistic understanding of pain has been formulated. Analogous anatomical and physiological features are inferred in humans but may be erroneous (Cross, 1994). In a useful overview of pain in animals (The Report of a Working Party of the Institute of Medical Ethics; Smith and Boyd, 1991) it is emphasised that discussion of pain in humans deals with a subjective state, that the neural mechanisms responsible for the production of pain sensation are complex, and that pain perception, even in humans, has yet to be fully understood.

3.2 The role of pain

Clearly, pain has a role in promoting survival of the organism, by alerting the organism that damage to its body has occurred or is likely to occur, and to encourage the avoidance of similar situations in the future. Bateson (1991) lists a number of attributes that would provide significant

survival advantages to the animal in possession of them. They are reproduced in full below:

1. Distinguish at the peripheral level between potentially harmless stimulation and that which is intense but, nevertheless, is harmless and carried further information that might be useful.
2. Learn to avoid conditions previously associated with potentially harmful stimulation.
3. Give top priority to escape from or removal of potentially dangerous stimulation and to avoidance in conditions previously associated with such stimulation. This feature would involve inhibiting other competing activities.
4. Inhibit activities that might delay recovery from disease or injury.
5. Inhibit the inhibition of all other activities under special conditions. Despite the need for limiting further damage, inhibition of all activity while some parts of the body signal injury might increase the risk of an even quicker death through exposure or predation. So peripheral mechanisms that shut out powerful input to the central nervous system and central control of the damage avoidance system would be advantageous.

However, Bateson points out that identifying the functional advantage of possessing a mechanism to detect damage does not reveal why a subjective sense of pain has evolved in humans. Can the system not work successfully without the self-consciousness or awareness of the animal? Bateson argues that it should be possible for an individual without an evolved consciousness to avoid damage and recuperate efficiently without feeling pain, in human terms. *He suggests that caution should be employed in assuming on evolutionary and functional grounds that conscious perception of pain in all animals is a foregone conclusion.*

3.3 The physiology of pain perception in mammals

Two components to the pain response can be identified (Smith and Boyd, 1991):

1. Nociception: the *physiological* perception of and response to painful stimuli that can be an automatic, reflex process not involving the higher centres of the brain.
2. Pain proper: the conscious *emotional* experience of pain, which involves nerve pathways in the highest part of the brain, the cerebrum.

Several review articles provide a good introduction to the physiology of pain perception and the description that follows was taken from Edmeads (1983), Greer and Hoyt (1990), Smith and Boyd (1991), and Cross (1994).

3.3.1 Receptors and the ascending nociceptive pathways. The pain pathway comprises of a chain of three neurons transmitting pain signals from the periphery to the cerebral cortex. The first-order neuron has its cell body in the dorsal root ganglion, and two axons, one projecting distally to the tissue, the other extending centrally to the dorsal horn of the spinal cord. This axon synapses with the second-order neuron, the axon of which ascends in the spinothalamic tract to the thalamus. Here it synapses with the third order neuron, which terminates in the cerebral cortex.

The first event in the generation of pain is the stimulation of *nociceptors.* These are free nerve endings that are sensitive to specific stimuli; mechanoreceptors respond to strong mechanical stimuli and thermoreceptors respond to heat. In addition, there is a third group of receptor cells which are polymodal and respond to a number of stimuli including strong heat, strong mechanical stimuli and chemical factors. When the stimulus exceeds a certain threshold level, electrical activity in the nociceptor evokes nerve impulses that are transmitted along the nerve fibre to the spinal cord. Two types of nerve fibre are associated with nociceptors; A-delta axons which are myelinated and characteristic of high-threshold mechanoreceptors, and C fibres, which are unmyelinated, and characteristic of polymodal nociceptors. The presence or absence of the myelin sheath radically alters the speed with which nerve impulses are transmitted. The A-delta axons transmit impulses at 15 metres per second, in contrast to the rate of transfer along C fibres that is approximately 1 metre per second (Edmeads, 1983).

Following a stimulus, the nociceptor initiates a train of impulses which ascend the C and A-delta fibres to the dorsal horn of the spinal cord and thence to the lower parts of the brain, the brain stem and thalamus. The frequency of the impulses encodes the severity of the pain perceived by the individual and a sufficiently high frequency of nerve impulses is necessary to pass a threshold and be recognised as pain (Edmeads, 1983).

High-threshold mechanoreceptors transmit "first pain", a well-localised sensation (in human

terms, sharp, stinging, pricking) that lasts only as long as the acute painful stimulus. This system permits the individual to analyse the nature of the stimulus, and the location, intensity and duration of stimulation. Polymodal receptors are recruited as a result of strong nociceptive stimulus and carry the sensation of "second pain" which is a more diffuse and persistent burning sensation that lasts beyond the termination of an acute painful stimulus, giving rise to the unpleasant character of painful sensation (Cross, 1994).

3.3.2 Gate control theory. The perception of pain takes place in the brain, but this is governed by a neural mechanism known as the action system (Melzack and Wall, 1965; Greer and Hoyt, 1990). Within the dorsal horn of the spinal cord there are controlling neurons that are activated by large myelinated nerve fibres, carrying non-noxious input, and are inhibited by C fibres, carrying noxious input. Impulses from the large fibres keep the "gate" partially closed and diminish the sensation of pain by causing the controlling neurons to inhibit the transmission of stimuli from both the large and small fibres to the pain transmitting neurons in the posterior horn of the spinal cord. Descending impulses from the brain exert an overall control over this mechanism. Thus the sensation of pain is modulated by a descending efferent system that enables the control of nociceptive thresholds.

3.3.3 Neurotransmitters associated with pain perception. Certain endogenous substances can facilitate or inhibit the firing of neurons involved in pain transmission (Edmeads, 1983). The undecapeptide, substance P, appears to be the neurotransmitter of first order pain afferent neurons. Administration of the compound will induce the firing of pain transmitting neurons, and compounds that reduce the amount of substance P in tissue also reduce the reaction to painful stimuli. Substance P is widely distributed within the central and peripheral nervous system but is particularly localised in the dorsal horn of the spinal cord that receives terminals from primary sensory neurons (Abrams and Recht, 1982). It should be noted that substance P is also known to have a diverse range of functions outside the immediate sphere of nociceptive activity; being involved with blood pressure regulation and associated phenomena, peristalsis of the gut, salivation, and immune regulation (Bost and Pascual, 1992; Walsh *et al*., 1993).

The neurotransmitter, 5-hydroxytryptamine, is believed to be involved in the modulation of nociception. The precise effects are dependent on the receptor sub-type involved, but, on the

whole, 5-HT appears to reduce nociceptive sensitivity by reducing the concentration of substance P (Eide and Hole, 1993).

A number of other endogenous compounds possess opiate-like characteristics, binding to opiate receptors and displaying analgesic qualities similar to morphine. These compounds (including enkephalin, β-endorphin and dynorphin) act as part of an internal antinociceptive system and receptors for the compounds are found in areas of the brain known to be involved with the modification of pain perception (Smith and Boyd, 1991; Cross, 1994). They constitute a negative feedback system that is activated by nociceptive stimulation and produces inhibition of nociceptive signals. This system is also believed to be involved in stress-induced analgesia. In some cases pain may be insensitive to modulation by opioids, for example where damage, disruption or disorder of peripheral nerves are concerned (Dickenson, 1991). β-Endorphin has a two-fold action, functioning as both a peripheral hormone and as a neuroregulator via interference with the production of other neurotransmitters (Dalayeun *et al*., 1993).

3.3.4 Pain associated with tissue damage. Tissue injury results in the rapid accumulation of a number of chemical factors that can cause nociceptors to fire, or, in lower concentrations, lower the threshold of nociceptors to other stimuli. These factors include bradykinin, prostaglandins, serotonin, histamine and hydrogen and potassium ions (Edmeads, 1983; Dickenson, 1991).

3.4 Pain perception in non-mammals and fish

3.4.1 The comparative approach. The question addressed by this section is "what evidence exists to support the contention that fish feel pain?". The most obvious approach, to consult the literature and identify previous research carried out in this area, is not enlightening. The Institute of Scientific Information Science Citation Database identifies 18411 scientific publications as having been published between 1982 and 1995 containing the word "pain" in the title. Of these, 2 publications contain the terms "pain" and "fish" (Stoskopf, 1994; Peters, 1988), one of which is wholly in German.

An alternative approach to answering this question is to identify the anatomical and biochemical correlates of pain perception in higher vertebrates and determine which of these are present in

fish. However, there are difficulties inherent in this approach.

It might be assumed that the mechanisms underlying pain perception, and the assessment of pain, are fully understood in mammals and that it is therefore a question of applying this knowledge to lower vertebrates. Inspection of the literature reveals this to be a fallacy. For example, the understanding and study of pain in man is aided immeasurably by the ability of the subject to convey sensation verbally. Wall (1992) highlights the difficulties inherent in assessing pain without access to the subject's perceptions by reference to the newborn human infant. If a blood sample is removed from a prematurely born infant by heel lancing, without a local anaesthetic, the infant shows a grossly exaggerated flexion reflex for days afterwards. If a local anaesthetic is employed, the flexion reflex does not display prolonged exaggeration. But Wall suggests that this tells nothing regarding the baby's ability to feel pain, only that it has a mechanism in its spinal cord, associated with tenderness in adults. He concludes that the assessment of pain in babies and animals is so difficult that wrong conclusions are often drawn. He also suggests that these difficulties extend beyond the lack of verbal communication for four reasons:

1. There is no reason to associate pain with the possession of any one neural component.
2. Reflex responses are parts of overall behavioural responses and may be uncoupled in particular situations.
3. Overall behavioural responses require, for their observation, an intimate familiarity with each species, which is rarely achieved and might be unachievable.
4. Animal responses to analgesic therapy designed for humans can be an excellent criterion when the animal responds, but the failures that mark the intractable pains experienced by humans could be more common in animals.

The problem of assessing pain in animals is considered at length in a persuasively argued paper by Bateson (1991). One of his central themes is the necessity or otherwise of a reflective, self-aware, consciousness for perception of pain. In the absence of self-awareness, can a damage-avoidance system operate successfully without a subjective feeling of pain? Bateson concludes that this should be possible. However, he also notes that the choice is not a simple one between the animal as an automaton and the fully self-aware animal; different species may possess differing levels of self-awareness and, if it can be established that an animal possesses some

degree of self-awareness, then the grounds for supposing that that animal can "suffer" are strengthened.

Bateson considers the possibility that pain perception may be assessed in animals by comparison with what is known of the human condition, in terms of behaviour and physiology. In mammals, behavioural indices of suffering can be misleading. The lack of a reaction in a severely injured animal can represent high levels of neural inhibitory gating. In addition, quite complex behavioural patterns can be the result of simple neural mechanisms that do not require conscious self-awareness on the part of the animal. Although Bateson acknowledges that the similarities between mammals are more remarkable than their differences he points out that fish are particularly refractory to understanding "even though there are strong grounds for supposing that these complex, long-lived animals possess highly efficient mechanisms for avoiding injury.". The presence of damage-avoidance mechanisms proves nothing regarding the higher perception of pain - animals even further removed from mammals than fish, such as insects, show some damage-avoidance mechanisms.

It has been proposed that there are several possible criteria, which, if fulfilled together might be grounds for accepting that a species can experience pain (Smith and Boyd, 1991). These are:

1. Possession of receptors sensitive to noxious stimuli, located in functionally useful positions on or in the body, and connected by nervous pathways to the lower parts of the central nervous system.
2. Possession of higher brain centres (especially a structure analogous to the human cerebral cortex).
3. Possession of nervous pathways connecting the nociceptive system to the higher brain centres.
4. Receptors for opioid substances found in the central nervous, especially the brain.
5. Analgesics modify the animal's response to stimuli that would be painful for a human; the animal chooses to take analgesics in "painful" situations.
6. The animal's response to stimuli that would be painful for a human is functionally similar to the human response (that is, the animal responds so as to avoid or minimize damage to its body).
7. The animal's behavioural response persists and it shows an unwillingness to resubmit to a painful procedure; the animal can learn to associate apparently non-painful with apparently

painful events.

The authors suggest that mammals conform to all these criteria, whereas fish can be demonstrated to conform to only numbers 2, 4, and 7. Insects also conform to criteria 4 and 7. The authors cite evidence for the existence of nervous pathways from thalamus to the cerebral cortex in amphibia, reptiles and birds, as well as mammals, but note that although similar pathways have been found in shark (cartilaginous fish), the situation in teleost (bony) fish is not known.

Assuming that sensory information can reach the higher brain centres, the authors consider whether this necessarily equates with the capacity to experience pain proper. The fish forebrain possesses "space" for higher-level processes other than the olfactory role it has been considered to be largely concerned with. The authors cite Walker (1983) as suggesting that higher psychological functions are present in animals roughly in proportion to the extent of development of the forebrain. He suggests that fish, amphibians and reptiles have small cerebral hemispheres and therefore little cognition.

The authors cited above might be interpreted as taking a conservative view on pain perception; assuming fish do not possess a reflective consciousness, and that the area of the brain in fish which is known to be involved with the processing of pain signals in higher vertebrates is small, the likelihood that fish experience pain as we do is minimal. However, some authors take a different stance. Stoskopf (1994) highlights the dearth of work on pain in fish but emphasises the similarities between fish and mammals. He notes the presence of various nociceptors, substance P, and antinociceptive mechanisms and suggests that the presence of these in fish argues in favour of the existence of nonmammalian pain perception. This conclusion could be queried on the grounds raised by Bateson (1991). Although mechanisms known to be involved with the conscious perception of pain in mammals are present in non-mammals, could they not equally well be involved in "automatic" damage avoidance systems involving no conscious perception of painful sensation?

This conscious or unconscious confusing of nociception and pain as being equivalent is highlighted by Kavaliers (1988; 1989). He points out that "descriptions of postures, movements

and vocalizations that are associated with nociception can be quantitative and objective, but interpretations of these behaviours are in words with connotations of human experience". This author also suggests that comparative studies of nervous systems have "rendered it unreasonable to assume that all animals should have conscious feelings and suffering comparable to that of humans in either degree or kind". However, in common with Bateson (1991), he points out that many animals have some level of "feeling" and "awareness" and that there is probably a gradation of sentience between species. In an examination of the evolutionary and comparative aspects of nociception, Kavaliers (1988) reports that even primitive, single celled, animals display "behavioural" changes in response to adverse changes in their environment and cites data which suggest that true nociceptive responses probably evolved in the Cnidaria; anemones in particular display sophisticated behaviours strongly suggestive of the presence of a functional nociceptive system. Insects possess complex nervous systems and also show avoidance and escape responses to aversive stimuli but these are probably pre-programmed responses, and not indicative of "pain" (Eisemann *et al*., 1986). Kavaliers (1988) also cites a number of studies suggesting that molluscs possess functional nociceptive mechanisms and that neuromodulation of nociceptive responses can be shown in many diverse animal groups. It has recently been demonstrated (Woolf and Walters, 1991) that the mollusc *Aplysia* displays many similarities with mammals in the manner in which noxious stimuli generate persistent changes in the nervous system. These authors suggest that mechanisms fundamental to injury-induced behavioural modifications are widespread in the animal kingdom. Kavaliers (1988) overall conclusion is that most animals display nociceptive responses and that the expression of such responses is modulated by the anatomical, morphological, physiological, neural and behavioural characteristics of the animal.

3.4.2 Specific evidence for pain perception in fish. While the evidence above suggests that mechanisms for detecting tissue damage and avoidance of such damage are widely distributed throughout the animal kingdom, there are very few studies to have examined nociception and pain perception in fish, in a scientific and rational manner.

3.4.2.1 Anatomical. It is well established that fish possess complex nervous systems (Northcutt and Davis, 1983; Davis and Northcutt, 1983) but most studies on sensory function have focused on olfaction and gustation (Finger, 1983; Tucker, 1983; Hara, 1992), vision (Nicol, 1989;

Fernald, 1993), and the lateral line system (Popper and Platt, 1993). Nociception has not been considered and does not appear even in the most recent summaries of the fish sensory and nervous systems (Bone *et al*., 1995). The extent to which pain perception in fish has been ignored or sidestepped is exemplified by the complete absence of the subject from a detailed review of the brain and sensory systems of cyprinid fish (Kotrschal *et al*., 1991).

The search methods employed for this review found only *one* publication that deals specifically with nociceptive apparatus in fish (Snow *et al*., 1993). These authors assessed the ability of sharks and rays (cartilaginous fish; elasmobranchs) to sense pain (their definition) by examining the proportion of myelinated and unmyelinated sensory fibres in the dorsal roots of the spinal cord. Their rational was that in mammals most unmyelinated axons represent nociceptors that are only excited by stimuli intense enough to cause tissue damage. The authors found that unmyelinated fibres were virtually absent from the dorsal roots of long-tailed stingray (*Himantura sp.*) and large shovelnose ray (*Rhinobattus battilum*). In the smaller specimens of shovelnose ray a substantial proportion of the fibres were unmyelinated. However, the authors suggest that these may represent fibres destined to become myelinated, as they were all found to be associated with a single Schwann cell, a characteristic of mammalian myelinated cells during early development. The authors conclude that it is likely that mature sharks and rays lack unmyelinated sensory fibres and thus have a very rudimentary system for encoding mechanical stimuli that are potentially damaging. They also note that elasmobranchs, although possessing substance P, lack the ascending system that in mammals is responsible for transmitting nociceptive information to the brain (Cameron *et al*., 1990). They suggest that "perhaps, in the aquatic environment, a well-developed system for perceiving specific forms of pain is of little adaptive value, particularly when one considers that in lower vertebrates, fixed action patterns such as swimming, feeding and flight more completely specify the organisms entire behavioural repertoire than do similar activities in higher vertebrates" and go on to elaborate that to marine fish, being able to perceive and evade potential threats of injury is more important than being aware of injury once it occurs.

It should be emphasised that the cartilaginous fishes, or elasmobranchs, are taxonomically distinct from the bony fishes (teleosts) that comprise most species sought by anglers. There have been no similar studies carried out on teleost fish.

3.4.2.2 Biochemical. Three substances known to be involved in nociceptive perception in mammals are also found in both bony and cartilaginous fishes.

Substance P has been isolated from rainbow trout brain and intestine (Jensen *et al*., 1992, 1993a). The compound has also been detected indirectly via immunoreactivity with antibodies raised against the mammalian form, or specific receptors have been identified, in the brain of the electric fish (*Apteronotus leptorhynchus*; Dulka *et al*., 1995; Weld *et al*., 1994) in areas of the brain concerned with intraspecific electrocommunication (Weld and Maler, 1992) and in the brain of dogfish (*Scyliorhinus canicula*; Rodriguez *et al*., 1993) sea bass (*Dicentrarchus labrax*; Moons *et al*., 1992) and green molly (*Poecilia latipinna*; Batten *et al*., 1990), in neuromasts and electroreceptors of some teleosts (Zaccone *et al*., 1994), in cardiac nerves, lung and gut of the lungfish (*Neoceratodus forsteri*; Holmgren *et al*., 1994) and in the cardiac axons and nerves supplying the stomach of Atlantic cod (Jensen *et al*., 1993b; Davies *et al*., 1994). Distension of the stomach in rainbow trout results in the release of substance P-like material into the gastric vasculature (Jensen and Holmgren, 1992). Substance P has been demonstrated to have effects on the abundance of eosinophilic granule cells in rainbow trout intestine (Powell *et al*., 1993), on the electrical activity of ganglion cells in roach retina (Downing and Djamgoz, 1993) and on dorsal aortic pressure, heart rate and vascular resistance in the dogfish (Holmgren *et al*., 1992). From these results it is clear that substance P is found in many fish tissues and its physiological role may be varied. For example, substance P has been found in areas of the brain concerned with sexual behaviour and endocrine function in mammals and fish (Weld and Maler, 1992). Considerable weight was attached to the presence of substance P in fish in evidence presented to the Medway Inquiry into Shooting and Angling (Medway, 1980) in which it was asserted that "it is now possible to ascertain, with a higher degree of probability than hitherto, whether or not an animal possesses the capacity to feel pain". However, at the time the Medway Report was prepared, the extent of involvement of substance P in systems unrelated to pain perception was not apparent. There has been no work specifically demonstrating a role for substance P in nociceptive processes in fish and its presence alone does not attest to such a function.

There has been less work carried out on the opioid peptides. Leu- and met-enkephalin have been identified in the brain of the dogfish (Vallarino *et al*., 1994) and enkephalin-like

immunoreactivity has been found in the central nervous system of rainbow trout (Vecino *et al*., 1992) and in the visceral sensory area of various elasmobranchs (Stuesse *et al*., 1992). It is suggested that the distribution of enkephalins in specific hypothalamic nuclei, visual areas, and in the brainstem of rainbow trout indicate a role for these peptides in neuroendocrine modulation, and visual and somatosensory functions (Vecino *et al*., 1992).

The opioid peptide β-endorphin is implicated in a wide range of actions in mammals, including pain modulation (analgesic effects), behaviour, and memory (Dalayeun *et al*., 1993). In fish, little is known of the role of β-endorphin except that its levels in the blood are altered by stress. In trout, β-endorphin levels have been shown to both increase (Sumpter *et al*., 1985), remain constant (Sumpter *et al*., 1985) or decline (Balm and Pottinger, 1995) following stress. The implications of these results have yet to be understood.

3.4.2.3 Behavioural. It is generally accepted that the well-being of mammals can be assessed by examination of observed behaviour. Sanford (1992), in a review of methods for the detection and assessment of pain and distress in laboratory animals, identifies four particular responses that might be observed in an animal experiencing pain. These are (from the Association of Veterinary Teachers and Research Workers guidelines):

1. Modification of behaviour
2. A protective response
3. A response designed to minimize pain
4. A response designed to convey the experience to others.

The author notes that the assessment of pain in an animal also requires that a range of contributory factors be taken into account. These include (from the Association of Veterinary Teachers and Research Workers guidelines):

1. Details of the animal
2. History of the animal and its environment
3. Clinical examination
4. Physiological measurements
5. Biochemical measurements

6. "Mental" status
7. Activity
8. Posture, gait, facial expression
9. Reaction to handling
10. Vocalization
11. Response to analgesics

It is clear that the application of most of these criteria to fish is difficult or likely to be unenlightening. The author notes that "for the common domesticated animals and for primates, for example, there seem to be some well-recognised signs associated with pain, but for reptiles and fish, the association is much less certain. It is not clear whether these differences are solely attributable to our lack of knowledge of the behaviour of these lower vertebrates or whether pain sensations are quantitatively or even qualitatively different." (Sanford, 1992). As is implied by this statement, although much research is carried out into the behaviour of fishes (see Pitcher, 1986) there has been no research explicitly designed to identify behavioural correlates of "pain" in fish, and little which has examined behaviours which might be considered related; for example, fear and avoidance (Colgan, 1986).

There is a single study that has been quoted, and perhaps misrepresented, to justify the assertion that fish feel pain (Verheijen and Buwalda, 1988). As there are aspects of this study that are unsatisfactory, and because its conclusions have been given prominence in some quarters, it will be discussed in detail. It should be noted that this work was not published in a peer-reviewed journal, in contrast to the majority of work cited in this review. The study was designed to "find responses indicative of pain and fear, to differentiate responses indicative of pain from responses indicative of fear, and to investigate whether fish find pain or fear the more unpleasant experience" (Verheijen and Buwalda, 1988). In addition, the authors undertook "to estimate whether or not pain and/or fear make the fish suffer according to recent definitions of suffering (in mammals)....".

The experiments were carried out using a number of cyprinid fish. Fish were caught by hook and line in aquaria and were treated as follows:

1. Hooking, followed by immediate relaxation of the line
2. Electrical stimuli administered via an electrode implanted within the roof of the mouth
3. Hooking followed by maintained line tension
4. The introduction of alarm substance from damaged skin of conspecifics into the aquaria
5. Confinement within a small jar

The authors identify procedures 1 and 2 as causing "pain" and procedures 3, 4 and 5 as invoking "fear". However, these descriptions appear to have been decided arbitrarily. It is clear that, in the absence of other information, there are no physiological or behavioural grounds for classifying these procedures in this way.

The authors describe the response of carp to being hooked as including "rapid darts", "spitting" and "head-shaking". They also noted that some carp on slack lines almost immediately resumed feeding, despite the continued presence of the hook. The fish held on taught lines after hooking are described as showing behaviour classed as "fleeing", "spitting of gas", "sinking" and "lying". The fish which were exposed to alarm substance, and which were confined in jars showed all these behaviours, except "spitting" and head-shaking". The authors suggest that most of these observed responses correspond to the natural alarm reaction of fish, but that "spitting" and "head-shaking" constitute a response to possible "painful" stimuli. However, they also note that these two responses are observed in fish that inhale innocuous but unwanted material while ventilating, or feeding. They conclude "that *spit* and *shake head* do indicate low-level pain unless, of course, these responses are also reflexes.".

The authors go on to describe the results of experiments in which local electrical stimulation was employed. In this case, they observed responses that were similar to those noted following hooking and playing of carp. These responses they conclude, could be a response to intense pain, or the result of fear associated with the expected continuation of the strong (electrical) stimulus. The authors observe that the level of electrical stimulation required to evoke a pronounced behavioural response in fish, and that detected by a human subject as painful, is similar. The authors suggest that because the fish show a distinction in the response to weak or strong electrical stimulation, this is evidence for a non-reflex pain-based reaction. The validity of comparing the conscious perceptions of a human subject with the behavioural response of a fish

to stimuli in anatomically unrelated tissues might be questioned.

The authors go on to suggest that the pain, if any, "produced by impalement on the hook contributes less to the unpleasantness of the catching procedure than fear". They conclude that the pain level is low and that the carp *cannot be considered to be suffering.*

The main criticism that may be levelled at this study is that the authors have attributed qualities (pain, fear) to the procedures used, and to the response of the fish, on what amounts to anthropomorphic grounds. The response of the fish employed in their experiments to the stimuli applied can be described purely in terms of reflex behavioural actions with no necessity to invoke the terms "fear" and "pain". Even if the stimuli are processed at a higher level of consciousness, it is plausible to suppose that this could occur without the sensations and emotions associated with pain in mammals. Fear is a complex emotional state and although the terms "arousal" and "fright" are frequently employed in behavioural studies involving fish (e.g. Laming and Ebbesson, 1984) these are used to describe behaviour patterns, not to ascribe a "state of mind" to the fish. The results of this study do not prove the absence of pain perception in fish, neither do they prove the existence of such a system.

In a subsequent publication (Verheijen and Flight, 1992) the authors summarised the results of their previous study as follows:

1. The reactions of fish to impalement by the hook are not indicative of severe pain.
2. The hooked fish in play shows the natural cyprinid-type alarm reaction, which includes disturbance of the hydrostatic equilibrium (buoyancy) as a result of the spitting of gas from the swim bladder.
3. After being unhooked and returned to the water the fish terminates the alarm reaction by sinking, then calms down while lying on the bottom; this behaviour is in accordance with the schedule of the natural alarm reaction.
4. Under favourable conditions the fish does not suffer; the moderate unpleasantness of being caught is the result of fear rather than pain.

The authors also indicate that a similar study carried out on trout (in preparation) produced

similar conclusions.

In summary, a number of points regarding pain perception in fish can be made:

1. It is an advantage for the animal to be aware of damage to its body and to be able to avoid potentially damaging situations. A system that alerts the animal to damage has survival value.
2. Several of the anatomical and biochemical components involved in pain perception in mammals are present in fish. However, some key elements (e.g. unmyelinated nerve fibres) are absent from certain species, other elements (e.g. the forebrain/cerebral cortex) are considerably less well developed in fish than in mammals.
3. The neurophysiological mechanisms underlying pain perception in man and other mammals are complex and not fully understood. Many components of the nociceptive/pain perception system have other, unrelated, functions. Identification of such components in fish cannot be considered proof that fish experience "pain".
4. It is the opinion of experts in the field that animals may possess mechanisms allowing them to avoid damage and facilitate recuperation without conscious perception of pain, in human terms.
5. Our understanding of pain perception in fish is hampered by the lack of research on this subject, and by the difficulties inherent in interpreting the behavioural and physiological responses of an animal taxonomically far removed from mammals.
6. There is no information available in the literature at present that provides firm evidence that fish perceive pain as mammals apparently do, or that they cannot perceive pain as mammals do. The problem of assessing what a fish perceives when exposed to stimuli considered to be noxious or unpleasant in human terms may prove to be intractable.

4. CONCLUSIONS

Capture of fish by angling methods undoubtedly causes significant physiological disruption and may be accompanied by behavioural effects following release. Some mortality may be expected, associated with a small proportion of fish that are not hooked cleanly in the jaw. However, there is no evidence for chronic, long-term, adverse effects of capture and release. Fish appear to recover from the effects of capture within 24 - 72 h.

There is no substantive evidence within the literature that suggests that fish experience pain as mammals appear to. However, the possibility that fish "suffer" as a result of angling practices cannot be categorically discounted. Because of difficulties in understanding the subjective perceptions of a taxonomically distant animal, irrespective of physiological similarities and dissimilarities, it is possibly a question that will never be satisfactorily answered in scientific terms.

Much of the research assimilated for this review concerns salmonid fish, or species that are not native to the UK. There is a requirement for fundamental research on the impact of angling procedures on those cyprinid fish that comprise the quarry of the UK freshwater angler.

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by

T. G. Pottinger BSc PhD

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The Institute of Freshwater Ecology

The Windermere Laboratory

The Ferry House, Far Sawrey

Ambleside, Cumbria LA22 0LP