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Endocrine, Physiological and Histopathological Responses of Fish and Their Larvae to Stress with Emphasis on Exposure to Crude Oil and Various Petroleum Hydrocarbons

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الاستجابات الهرمونية والفيزيولوجية والتركيبية للأسماك ويرقاتها للضغوط البيئية مع التركيز على آثار تعرضها للبترول الخام ومشتقاته العديدة.

عبدالعزيز بن يحيى الكندي وآن براون وكولن وارنج

خلاصة: تم مراجعة الاستجابات الهرمونية والفيزيولوجية للأسماك التي تتعرض لأنواع عديدة من الضغوط والعوامل الفيزيائية والكيميائية مع التركيز على أثر البترول الخام ومشتقاته في هذا الصدد، وقد تم تشخيص كيميائية الهيدروكربونات البترولية وقدرتها على تسميم الأسماك ووسائل تعرض هذه الحيوانات لهذه المشتقات البترولية، كما تم استقصاء أثر البترول الخام ومشتقاته على عوامل هرمونية عديدة في الدم كالتغيرات في تركيز الأدرينالين والنورأدرينالين والكورتيزولات والثيرويدات وهذه هرمونات مهمة في ابراز أثر الضغوط المحيطة عامة على الأسماك والكيمائية منها خاصة. كما تم استقصاء أثر مشتقات البترول على سكر الدم والوظائف الأيونية والأسموزية وأكسجين الدم ونسبة الخلايا الحمراء والهيموجلوبين بالإضافة إلى التغيرات التركيبية الناتجة عن تعرض الأسماك لأنواع عديدة من البترول الخام ومشتقاتها المختلفة.

ABSTRACT: Various endocrine and physiological responses of fish exposed to forceful physical and chemical stimuli are reviewed with emphasis on the effects of crude oils and their hydrocarbon constituents. The chemistry and toxicity of petroleum hydrocarbons are examined and methods for experimental exposure of fish to crude oil and petroleum hydrocarbons are considered. A variety of blood-borne parameters recognized as reliable tools in determining the relative severity of stress in fish are reviewed. The effects of stress and petroleum hydrocarbons on endocrine responses including changes in plasma

catecholamines, corticosteroids, and thyroid hormones are reviewed. The physiological responses: changes in plasma glucose, osmotic and ionic regulation, blood oxygen, hematocrit and hemoglobin concentration are explored, and histopathological effects of crude oil on fish are reviewed. Recent studies of the effects of petroleum hydrocarbons on fish larvae are considered and the increased sensitivity of the early life stages of fish are highlighted.

CONTENTS

1. Introduction

1.1 The Fish Stress Response

Developing an accurate knowledge of the stress responses in fish is a crucial element for better understanding of the problems related to the well-being and survival of fish when exposed to forceful physical and chemical stimuli. A variety of blood-borne parameters have been recognized as reliable tools in determining the relative severity of stress in fish (Mazeaud and Mazeaud, 1981; Barton, 1988; Brown *et al* 1990; Waring *et al* 1992; Pickering, 1993a,b; AlKindi *et al* 1996). These physiological indicators have been measured in a range of fish species to evaluate the trauma inflicted on fish by various stressors (Mazeaud *et al* 1977; Giesy *et al* 1988; Folmar, 1993; Pickering, 1993a,b). Identifying stress responses can be achieved by determining the changes in plasma catecholamines (Mazeaud *et al* 1977; Mazeaud and Mazeaud, 1981; Barton, 1988; Tang and Boutilier, 1988; Gingerich and Drottar, 1989; Aota *et al* 1990) and plasma corticosteroids (Donaldson, 1981; Sumpter *et al* 1986; Thomas and Rice, 1987; Pickering and Pottinger, 1989; Whitehead and Brown 1989; Waring *et al* 1992; Foo and Lam, 1993; Mazur and Iwama, 1993; AlKindi *et al* 1996). These primary (endocrine) responses to stress are more immediate than the induced secondary (metabolic) responses (Barton and Toth, 1980;

Mazeaud and Mazeaud, 1981). The stress responses of fish have been reviewed recently by Wendelaar Bonga (1997). This review will highlight aspects of the fish stress response applicable in understanding the effects of petroleum hydrocarbons and then review the literature describing histopathological effects of hydrocarbons in fish.

1.2 Petroleum Hydrocarbons

Crude oils and their water-soluble fractions are a major source of pollution in the marine environment. Studies have estimated that the quantity of crude oils and their constituent hydrocarbons, which enter the marine environment, is in the range 2 to 20 million tons per annum (Solangi, 1980; Neff, 1990). The main sources of petroleum hydrocarbon pollution are oil spills, oil-tanker washings and offshore production which generates produced waters (Dey *et al* 1983; Neff, 1990; Steinhauer *et al* 1994; Syvertsen, 1996). Massive volumes of crude oil have been released into the marine environment as a result of large oil spills. For example, the Exxon Valdez spill released 258,000 barrels (Maki, 1991), Ixtoc I 475,000 metric tons (Jernelov and Linden, 1981), the Iraqi-Iranian War is estimated to have resulted in the release of 6 million barrels, the Gulf War, 7-11 million barrels (Alam, 1993) and the Braer oil spill, 85,000 tons (Ritchie and O'Sullivan, 1994).

Various changes occur when oil is spilled at sea. These encompass bacterial degradation, photooxidation, evaporation, emulsification, dissolution, dilution by spreading, clustering to form tar-balls and formation of the water-soluble fraction (WSF) and water-accommodated fraction (WAF) (Dey *et al* 1983; Neff, 1990; Ehrhardt *et al* 1992). Ultimately, much of the spilled oil penetrates into the bottom sediment. It appears that the attraction between hydrocarbons and sediment is greater than the separation forces and the loss of hydrocarbons is significantly reduced after attachment to sediment (Moore and Dwyer, 1974; Hyland and Schneider, 1976; Neff, 1990; I.C.E.S. 1991; Steinhauer *et al* 1994). Petroleum residues may thus persist in sediment of beaches for 20 years after an oil spill (Vandermeulen and Singh, 1994).

Crude oils are complex mixtures containing literally thousands of organic and a few inorganic compounds. Most crude oils contain the same major classes of compounds but the amounts of each compound are different (Neff, 1990). Aromatic hydrocarbons based on the benzene ring may constitute 20% of the total hydrocarbon content of crude oil. Crude oils usually have higher concentrations of monocyclic aromatic hydrocarbons such as benzene and toluene than dicyclic aromatics such as naphthalenes or polycyclic aromatic hydrocarbons (Anderson *et al* 1974). The abundance of aromatic hydrocarbons in crude oil is usually inversely related to their molecular weight; one-ring (benzene) to the three-ring (phenanthrene) compounds comprise at least 90% of the total aromatic hydrocarbons present in petroleum (Neff, 1979, 1985, 1990).

Aromatic hydrocarbons with methyl substitutions of one or more rings are more toxic than less substituted compounds, but tend to be less water-soluble, and thus less abundant in the WSF of crude oil (Anderson *et al* 1974; Neff, 1990).

There are at least two pathways by which petroleum hydrocarbons can be taken into fish, firstly via the gills (Evans, 1987) and secondly via the gut with food or with the seawater drunk for volume regulation (Lee *et al* 1972; Stegeman, 1977). The rate of hydrocarbon uptake will be affected by exposure concentration, the molecular weight of the hydrocarbons and the amount of lipid in the fish, which depends on the species, age, season and reproductive state as well as feeding rate and oxygen uptake (Neff *et al* 1976; Falk-Petersen *et al* 1982; Rice, 1985). Retention of hydrocarbons in the animal tissues will depend upon the partitioning of hydrocarbons between the exposure water and the tissue lipids (Neely *et al* 1974). Binding of hydrocarbons to tissue lipids is probably by lipophilic interaction (Stone, 1975). Thus, hydrocarbons remain exchangeable and when the animals are returned to hydrocarbon-free seawater, the lipid/water partition coefficients for the hydrocarbons will permit their gradual release from the tissues to the water (Neff *et al* 1976). The longer tissue retention time and the more toxic effects have been indicated for aromatic hydrocarbons with more than one ring and methyl-substitutions (i.e. naphthalene, methylnaphthalenes and higher molecular weight PAHs) (Anderson *et al* 1974; Morrow, 1974; Morrow *et al* 1975; Neff *et al* 1976; Falk-Petersen *et al* 1982; Neff, 1990) are probably due to their lipophilic interaction with animal tissue. Tetramethyl-benzene and 1- and 2-methylnaphthalene were absorbed in the highest concentrations by English sole exposed to crude oil in sediment

(McCain *et al* 1978). Lipophilic interaction of hydrocarbons with lipids could lead to the accumulation of aromatic hydrocarbons in the structural lipids of membranes and disturb the membrane function (Payne *et al* 1978; McKeown and March, 1978; Solangi, 1980; Zbanyszek and Smith, 1984).

1.3 Exposure of Fish to Petroleum Hydrocarbons

During an oil spill, transient high levels of petroleum hydrocarbons in the seawater phase are followed by a decline as natural processes such as evaporation, photooxidation and biodegradation occur. The acute toxic effects of an oil spill are most likely caused by soluble low molecular weight aromatic hydrocarbons such as benzenes, toluene and naphthalene. Acute toxicity has been reported to be inversely related to the molecular weight of aromatic hydrocarbons (McAuliffe, 1977; Neff, 1990), but chronic effects are always attributed to four and five-ring polycyclic aromatic hydrocarbons (Neff, 1990). Compared to monocyclic aromatics, naphthalene appeared to be accumulated to a greater extent by fish (FalkPetersen *et al* 1982) and retained for longer periods of time following exposure, thus making naphthalenes probably the most acutely toxic aromatic hydrocarbon (Morrow, 1974; Morrow *et al* 1975; Neff *et al* 1976; FalkPetersen *et al* 1983, Falk-Petersen and Kjorsvik, 1987; Neff, 1990).

In oil-polluted marine waters, concentrations of total hydrocarbons are usually substantially less than 1 ppm (Malins and Hodgins 1981; Neff 1990). Acute short-term laboratory studies (1-96h) have investigated the effects of high concentrations of petroleum hydrocarbons, generally above those ordinarily encountered after most major oil spills (Mazmanidi and Kovaleva, 1975; Thomas *et al* 1980; Kiceniuk and Khan, 1987; Davison *et al* 1992; AlKindi *et al* 1996) while chronic studies, lasting for several months have used lower exposure concentrations of petroleum hydrocarbons, generally in the sublethal ppb range (Mazmanidi and Kovaleva, 1975; Hawkes, 1977; Payne *et al* 1978; Whipple *et al* 1978; Khan *et al* 1981; Woodward *et al* 1981; Fletcher *et al* 1982; Khan, 1987; Davison *et al* 1993; Stephens *et al* 2000).

The heavy fractions (i.e. polycyclic aromatic hydrocarbons and n-alkanes) of crude oil persist in sediment for long periods of time and benthic fish will be particularly exposed to these hydrocarbons (Steinhauer *et al* 1994).

1.4 Experimental Exposure Methods

There are several routes through which fish may be exposed to crude oils and their products. Experimental approaches have included exposure of fish to a dietary intake of petroleum hydrocarbons via contaminated food (Gruger *et al* 1977; Leatherland and Sonstegard, 1978; Carls and Rice, 1987). Sediment contaminated with crude oil has been employed for chronic exposure of fish to petroleum hydrocarbons for up to several months in some cases (McCain *et al* 1978; Fletcher *et al* 1981, 1982; Khan, 1991a; Truscott *et al* 1992; Tahir *et al* 1993; Khan *et al* 1994).

Fish exposed to the water-accommodated fraction (WAF) of crude oils and their constituents have been the subject of several investigations (Anderson *et al* 1974; McKeown and March, 1977, 1978, Prasad, 1987, 1988, 1991), but the most widely used experimental approach has involved exposure of fish to the water-soluble fraction of crude oils or their products (Thomas and Rice, 1975, 1987; Hawkes, 1977; Smith and Cameron, 1979; Korolev *et al* 1980; Solangi, 1980; Thomas *et al* 1980; Engelhardt *et al* 1981; Eurell and Haensly, 1981; Solangi and Overstreet, 1982; Khan and Kiceniuk, 1984, 1988; Tilseth *et al* 1984; Zbanyszek and Smith, 1984; Moles *et al* 1985; Hellou *et al* 1986; Carls and Rice, 1987; Hellou and Payne, 1987; Kiceniuk and Khan, 1987; Rice *et al* 1987; Khan, 1990; Ehrhardt *et al* 1992; Davison *et al* 1992, 1993). Generally, the preparation of the WSF of crude oil is achieved by low energy mechanical mixing of crude oil with water followed by a period of separation of oil from the water phase (Anderson *et al* 1974; Shaw, 1977). The difference between water-accommodated and water-soluble fractions is mainly dependent on their preparation. A high energy vigorous mixing of oil with water may form an emulsion that will not separate and which will yield a water-accommodated fraction rather than a water-soluble fraction of crude oil, but with low energy gentle mixing, emulsions are less likely to form and the WSF of crude oil will not contain the water-accommodated fraction.

Several studies have employed the water-soluble fraction of crude oil that has been prepared by a variety of flow-through systems (Hellou *et al* 1986; Kiceniuk and Khan, 1987; Khan and Kiceniuk, 1988; Khan, 1990) or prepared in a contained system to supply a static test system (Anderson *et al* 1974; Rice *et al* 1976; Gruger *et al* 1977; Tamra and Karinen, 1977; Moles *et al* 1979; Solangi and Overstreet, 1982; Thomas and Rice, 1987). The preparation of a WSF using a flow-through system is usually accomplished by dropping or spraying water onto a layer of crude oil on top of a water layer, and water, containing the WSF of crude oil, is then drawn from the bottom of the water layer (Moles and Rice, 1983). The advantage of a flow-through system is that it can achieve a constant concentration of petroleum hydrocarbons in the prepared WSF. Another advantage of this system is that spraying provides some form of aeration, which maintains a constant supply of oxygenated water containing the WSF of crude oil (Moles and Rice, 1983; Moles *et al* 1985). However, after an oil spill, the immediate transient high concentrations of petroleum hydrocarbons will probably decline, subsequently so that a flow-through system of producing a WSF of crude oil with constant hydrocarbon levels does not mimic an oil spill. Nevertheless, oil in water was detectable one month after the Braer oil incident, particularly in areas near the wreck (Ritchie and O'Sullivan, 1994).

For a static testing system of the effects of the WSF of crude oil, low energy mixing of crude oil with water is achieved by gently mixing, followed by a period of total separation of the oil phase from the water phase which will be used in the static test system (Anderson *et al* 1974; Gruger *et al* 1977; Moles *et al* 1979; Thomas and Rice, 1987; AlKindi *et al* 1996). The main advantage of this approach is the ease of producing the WSF of crude oil. However, changes in the composition of the WSF during the experimental exposures are unavoidable. Furthermore, it is usually impractical to produce large volumes of the WSF of crude oil by this method.

2. Endocrine Responses to Stress and Their Induction in Fish Exposed to Petroleum Hydrocarbons

2.1 Plasma Catecholamines

The chromaffin tissue of the head kidney, the homologue of the adrenal medulla of higher vertebrates, synthesizes, stores and secretes three hormones: adrenaline, noradrenaline and dopamine (Mazeaud and Mazeaud, 1981; Randall and Perry, 1992). The three hormones exist in different proportions depending on the species (Mazeaud *et al* 1977; Mazeaud and Mazeaud, 1981; Folmar, 1993; Pickering, 1993b). The relative amounts of circulating adrenaline and noradrenaline, the main catecholamines involved in fish responses to stress, vary from one species to another (Mazeaud *et al* 1977; Mazeaud and Mazeaud, 1981; Gingerich and Drottar, 1989; Hathaway and Epple, 1989).

Catecholamine synthesis involves hydroxylation of a precursor molecule (tyrosine) into L-dopa then deamination of L-dopa to form dopamine. Noradrenaline is produced by further hydroxylation of dopamine and adrenaline is then produced by methylation of noradrenaline (Jonsson and Nilsson, 1983).

Catecholamines are stored within granules in chromaffin cells. The chromaffin cells are innervated by preganglionic cholinergic fibres of the sympathetic nervous system which stimulate catecholamine release into circulation (Nilsson *et al* 1976). A rapid and dramatic increase in circulating catecholamines can occur in response to a variety of physical stressors such as forced swimming and handling (Mazeaud *et al* 1977; Hughes *et al* 1988; Vijayan and Moon, 1994) and forceful exercise (Butler *et al* 1986; Primmett *et al* 1986; Tang and Boutilier, 1988; Wood *et al* 1990). These responses are likely to be a reflection of the oxygen status of the fish. Plasma catecholamines are elevated in response to hypoxia (Mazeaud *et al* 1977; Ristori and Laurent, 1989; Aota *et al* 1990; Perry *et al* 1991; Perry *et al* 1993), and this may explain the similar rise caused by chemical stressors such as anaesthetics (Gingerich and Drottar, 1989; Hathaway and Epple, 1989; Iwama *et al* 1989).

While the above stressors are known to cause various physiological changes, few studies have investigated the effects of petroleum hydrocarbons on endocrine pathways responsive to stress. However, AlKindi *et al* (1996) found that WSF-exposed flounders had elevated plasma noradrenaline concentrations while plasma

adrenaline concentrations were unaffected. This was most likely a response to the profound decline in the blood oxygen content of these flounder (AlKindi *et al* 1996).

When released in response to stressful conditions, catecholamines enhance the survival of fish by exerting a variety of beneficial effects such as mobilizing energy reserves and increasing oxygen carrying capacity. Catecholamines stimulate glycogenolysis and gluconeogenesis, and consequently increase blood sugar providing an animal with a readily available energy source to deal with stressful conditions (Mazeaud and Mazeaud, 1981; Morata *et al* 1982; Ristori and Laurent, 1985; Suarez and Mommsen, 1987; Sheridan and Mur, 1988; Wright *et al* 1989; Pickering 1990; Wood *et al* 1990; Wendelaar Bonga, 1997). Adrenaline stimulates glycogenolysis by increasing levels of intracellular cyclic AMP, which then activates glycogen phosphorylase and causes the breakdown of glycogen to glucose (Morata *et al* 1982; Van Raaij *et al* 1995). There is also evidence to suggest the involvement of catecholamines in the mobilization of free fatty acids (Waring *et al* 1996b) though results are inconsistent (see review by Wendelaar Bonga, 1997)

Catecholamines have a stimulatory action on cardiovascular function (Randall and Perry, 1992; Perry and Bernier, 1999), increasing heart rate (Wahlqvist and Nilsson, 1977; Satchell, 1991) stroke volume (Wood and Shelton, 1980; Mazeaud and Mazeaud, 1981; Pennec, 1987), blood pressure (Wahlqvist and Nilsson, 1977; Wood and Shelton, 1980) and branchial blood flow (Capra and Satchell, 1977; Butler and Metcalfe, 1983; Stagg and Shuttleworth, 1984). Elevated branchial blood flow has been suggested to increase gaseous exchange at the branchial surface (Booth, 1979), although this adaptive response needs to be balanced against a non-adaptive increase in water permeability of the gills (Bennett and Rankin, 1987).

Catecholamines have been reported to have a beneficial effect on the oxygen carrying capacity of fish by causing various physiological responses. Catecholamines stimulate a rapid release of red blood cells from the spleen into the bloodstream (Kita and Itazawa, 1989; Perry and Kinkead, 1989). Furthermore, catecholamines have been reported to affect red blood cell pH of some species of fish (Nikinmaa, 1986; Primmett *et al* 1986) and cause red blood cell swelling (Nikinmaa, 1983; Nikinmaa *et al* 1984; Ling and Wells, 1985; Fuchs and Albers, 1988; Wood, 1991; Perry and Thomas, 1993). Under conditions of forceful exercise, anaerobic metabolism of the white muscle will cause metabolic acidosis that results in the decrease of plasma pH which will reduce red blood cell pH causing a decrease in oxygen carrying capacity of the hemoglobin by means of the Root effect. Catecholamines can counteract this effect, particularly in salmonid fish, by stimulating H^+ excretion from red blood cells, hence restoring intracellular pH which results in maintenance of the oxygen carrying capacity of the cells. Simultaneously, Na^+ and Cl^- move into the cells, causing osmotic swelling, which ultimately decreases the organic phosphate concentration of the red blood cells, and consequently, again increases the affinity of hemoglobin for oxygen (Paajaste and Nikinmaa, 1991; Wood, 1991; Perry and Thomas, 1993). In contrast to the salmonids it seems that fish with high hypoxia tolerance (eg carp, tench, eels) do not rely on this mechanism (see review by Jensen *et al* 1993). In addition, in the rainbow trout, *Oncorhynchus mykiss*, adrenaline may directly affect the oxygen affinity of at least one of the hemoglobin components of red blood cells (Falcioni *et al* 1991). In contrast to the salmonids it seems that fish with high hypoxia tolerance (eg carp, tench, eels) do not rely on this mechanism

2.2 Plasma Cortisol

Cortisol is the major corticosteroid hormone secreted by the interrenal tissue found in the 'head kidneys' of fish (Chester Jones *et al* 1969; Donaldson, 1981; Fryer, 1975; Idler and Truscott, 1972; Matty, 1985; Nichols and Weisbart, 1984). During stress, a corticotrophin releasing factor (CRF) released from the hypothalamus stimulates the anterior pituitary, p*ars distalis*, to secrete adrenocorticotrophic hormone, ACTH (Fryer and Peter, 1977). Subsequently, ACTH stimulates the synthesis and release of cortisol from the interrenal tissue.

Cortisol has been recognized as a reliable indicator of stress in fish (Mazeaud *et al* 1977; Barton and Toth, 1980; Donaldson, 1981; Barton, 1988; Waring *et al* 1992; Waring *et al* 1996a: McDonald *et al* 1993; Pickering, 1993b). Cortisol is released in fish in response to a wide variety of stress stimuli. Cortisol has been reported to increase in response to physical stressors such as handling (Sumpter *et al* 1986; Thomas and Robertson, 1991; Melotti *et al* 1992; Waring *et al* 1992; Barry *et al* 1993; Foo and Lam, 1993), physical disturbance and exercise

(Zelnik and Goldspink, 1981; Barton *et al* 1986; Flos *et al* 1988; White and Fletcher, 1989; McDonald *et al* 1993) and capture or confinement (Davis and Parker, 1986; Sumpter *et al* 1986; Davis and Parker, 1990; Melotti *et al* 1992). Elevation of plasma cortisol has also been reported in response to environmental chemical stressors such as acid (Brown *et al* 1984; Barton *et al* 1985; Jones *et al* 1987; Edwards *et al* 1987; Goss and Wood, 1988; Whitehead and Brown, 1989; Brown *et al* 1989), aluminum (Goss and Wood, 1988; Whitehead and Brown, 1989; Brown and Whitehead, 1995; Waring *et al* 1996a), and many other pollutants (Swift, 1981; Gluth and Hanke, 1984) and prolonged exposure to anaesthesia (Strange and Schreck, 1978). However, exposure of rainbow trout and chinook salmon to brief anaesthesia did not change the plasma cortisol (Strange and Schreck, 1978; Iwama *et al* 1989).

Cortisol has been widely used to assess the state of health in fish exposed to stress (Mazeaud and Mazeaud, 1981; Pickering, 1993b). Changes in the concentration of plasma cortisol, however, depend upon the nature of the stress stimuli, the duration of the stress, the magnitude and severity of the stress (Barton and Toth, 1980; Pickering *et al* 1982; Davis and Parker, 1983; Gluth and Hanke, 1984) and the species under investigation (Davis and Parker, 1983, 1986; Waring *et al* 1992). Nevertheless, since increased concentrations of plasma cortisol constitute part of the generalized stress response (Mazeaud *et al* 1977), a release of cortisol in fish exposed to petroleum hydrocarbons might be expected. So far only a few studies have examined plasma cortisol in fish exposed to petroleum hydrocarbons. Generally, exposure of fish to crude oils and their derivatives caused elevated plasma cortisol concentrations (DiMichele and Taylor, 1978; Thomas *et al* 1980; Lopez *et al* 1981; Thomas and Rice 1987). The WSF of crude oil increased plasma cortisol in juvenile coho salmon after 48 h exposure (Thomas and Rice, 1987), and the WSF of fuel oil caused a 50-fold increase in the circulating cortisol concentration of stripped mullet after 1 h followed by a recovery to resting levels 6 h after oil addition (Thomas *et al* 1980). Exposure to individual petroleum hydrocarbons and other oil products similarly increased plasma cortisol; fish exposed to naphthalene exhibited an increase in serum cortisol (DiMichele and Taylor, 1978). Exposure of rainbow trout to crude oil caused an initial elevation of plasma cortisol (Engelhardt *et al* 1981). An elevated concentration of plasma cortisol in wild eels, caught at field sites, persisted for at least 8 months after an oil spill (Lopez *et al* 1981). The WSF of Omani crude oil caused a significant elevation in plasma cortisol concentrations in exposed flounders, *Pleuronectes flesus* (AlKindi *et al* 1996).

Increased cortisol secretion in response to acute stress presumably enhances survival. An adaptive role of cortisol has been suggested to be linked to mobilization of energy reserves through its catabolic functions (Mazeaud *et al* 1977; Barton *et al* 1985; Jones *et al* 1987; Goss and Wood, 1988; Brown *et al* 1989; Whitehead and Brown, 1989; Pickering, 1990; Thomas and Robertson, 1991; Waring *et al* 1992; Barry *et al* 1993; Bollard *et al* 1993). Cortisol administration to fish has been commonly reported to cause hyperglycemia (Leach and Taylor, 1980; Davis *et al* 1985; Barton *et al* 1987; Van der Boon *et al* 1991; Vijayan *et al* 1991). Similarly, administration of cortisol to hypophysectomised eels resulted in a dramatic increase in plasma glucose (Chan and Woo, 1978a). In contrast to these observations, however, cortisol treatment caused hypoglycemia in American eels (Foster and Moon, 1986) and administration of cortisol to rainbow trout to achieve plasma concentrations similar to those occurring during stress did not induce hyperglycemia (Andersen *et al* 1991).

The hyperglycaemic effects of administered cortisol have been suggested to result from the inhibitory effects of the hormone on glucose oxidation and utilization in peripheral tissues (Gill and Khanna, 1975; Van der Boon *et al* 1991). This may be accompanied by stimulated gluconeogenesis. Cortisol can directly provide the free amino acids for gluconeogenesis via stimulation of protein catabolism (Lidman *et al* 1979; Marshall Adams *et al* 1985) with elevation of circulating amino acids in some species (Chan and Woo, 1978a), but not apparently in *Fundulus heteroclitus* (Leach and Taylor, 1982), or may act indirectly by inhibiting protein synthesis (reviewed by Van der Boon *et al* 1991). In gluconeogenesis, free amino acids act as precursors for glucose synthesis and the regulation of this process in the liver is suggested to be mediated by cortisol (Lidman *et al* 1979; Murat *et al* 1981). The gluconeogenic action of cortisol was supported in studies by Vijayan *et al* (1994) when a corticosteroid antagonist inhibited the increased alanine gluconeogenesis in cortisol-treated fish.

Contradictory results have also been reported concerning the effects of cortisol on liver glycogen. Administration of cortisol caused an increase in liver glycogen content (Chan and Woo, 1978a; Lidman *et al*

1979; Leach and Taylor, 1982). On the other hand, decreased glycogen content in the liver in response to elevated cortisol has been reported (Barton *et al* 1986; Foster and Moon, 1986; Vijayan and Leatherland, 1989; Soengas *et al* 1992).

Increased blood fatty acid concentrations in parallel with an elevation in plasma cortisol have been reported (Dave *et al* 1979; White and Fletcher, 1989; Waring *et al* 1992) and a role of cortisol in lipid-mobilization has been proposed (Lidman *et al* 1979; Sheridan, 1987). Changes in blood glucose and free fatty acid compositions are probably significant actions by which cortisol increases the availability of energy and the readiness of fish to survive stress. However, there are significant species differences in the mechanisms involved in achieving this and the mode of action of catecholamines and cortisol in individual species (see Wendelaar Bonga, 1997).

Cortisol appears to have beneficial effects on ionoregulatory balance in both freshwater and seawater fish (Parwez and Goswami, 1985; Laurent and Perry, 1990; Madsen, 1990; Barton and Iwama, 1991; Bisbal and Specker, 1991). The mechanism by which cortisol influences ion balance and its interaction with other hormones has been the subject of many investigations (McCormick 1995). Cortisol stimulation of salt uptake is thought to be via increased gill Na^+-K^+-ATPase activity (Pickford *et al* 1970; Forrest *et al* 1973; Dange, 1986; Langhorne and Simpson, 1986; Madsen, 1990; Bisbal and Specker, 1991). However, in some experiments cortisol had no apparent effect on this enzyme (Langdon *et al* 1984; Redding *et al* 1984). Linked to the elevated Na^+-K^+-ATPase, cortisol has been shown to cause chloride cell proliferation (Perry and Wood, 1985; Laurent and Perry, 1990; Madsen, 1990) and increase the area of the chloride cell, which is in contact with the external media and therefore concerned with ion transport (Laurent and Perry, 1990). Cortisol could thus play a role in the reduction of ionic imbalance in stressed fish.

2.3 Plasma Thyroid Hormones

Thyroxine (T_4) is synthesized in the thyroid follicles by iodination of tyrosine (Matty, 1985). The thyroid gland primarily secretes L-thyroxine in response to thyroid stimulating hormone (TSH) released from the pituitary (Eales and Brown, 1993). TSH release from the pituitary is regulated by a hypothalamic hormone, thyrotrophin releasing hormone (TRH) and thyrotrophin release inhibiting factor (TIF) and /or negative feedback inhibition by T_4 (Leatherland, 1982; Matty, 1985; Eales, 1990; Eales and Brown, 1993).

Over 99% of T_4 binds to plasma proteins called thyroglobulins (Eales, 1990; Rousset and Mornex, 1991). Free T_4 enters cells of peripheral tissue (brain, liver, gill, kidney, heart and muscle) and is then deiodinated to form triidothyronine, (T_3) by L-thyroxine 5'monodeiodinase (MDI), the enzyme responsible for peripheral conversion of T_4 to T_3 (Eales, 1990; Leatherland *et al* 1990; Byamungu *et al* 1992; MacLatchy and Eales, 1990, 1992; Eales *et al* 1993a,b). Eales and Brown (1993) suggest that in teleosts the 'peripheral model' of plasma T_3 regulation by MDI activity predominates over the 'central model' via the hypothalamic-hypophyseal-thyroidal axis. Thus, for example, exogenous T_4 or TSH will raise plasma T_3 concentrations despite massive increases in plasma T_4. Similar trends also occur in nature during parr-smolt transformation (Eales and Brown, 1993). A physiologically viable model is that the thyroidally-secreted T_4 is a relatively inactive prohormone converted peripherally via MDI and that the hypothalamic-hypophyseal-thyroidal axis merely ensures adequate T_4 secretion (Eales, 1985, 1990; Eales and Brown, 1993). Thus all T_3 in teleosts may be generated peripherally and T_3 represents the active thyroid hormone at the level of target cells (Plisetskaya *et al* 1983; Eales, 1990; Eales and Brown, 1993).

Stressors such as physical disturbances and environmental toxicants including petroleum hydrocarbons have been reported to affect thyroid function but responses have been inconsistent (Osborn and Simpson, 1972; Brown *et al* 1978; Leatherland and Sonstegard, 1978; Pickering *et al* 1982; Plisetskaya *et al* 1983; Brown *et al* 1984; Leatherland, 1985; Brown *et al* 1989; Whitehead and Brown, 1989; Brown *et al* 1990; Sinha *et al* 1991; AlKindi *et al* 1996; Johnston *et al* 1996; Waring *et al* 1996a; Waring and Brown, 1997). Reduced plasma T_4 concentrations were observed in flounder exposed to the WSF of crude oil but plasma T_3 concentrations were unaffected (AlKindi *et al* 1996). Stress has been reported to alter the T_3:T_4 ratio (Osborn and Simpson, 1972; Brown *et al* 1978). If thyroid hormones have biological significance in stressed fish, then potential physiological benefits lie in their potential roles in regulating osmoregulation, growth and metabolism.

Thyroid hormones have been suggested to have significant hypo-osmoregulatory effects on fish adapted to seawater. T_4 treatment reduced the elevation of plasma Na^+ in fish transferred from freshwater to seawater, whereas treatment with thiourea (a thyroid hormone antagonist) resulted in a significant increase in plasma Na^+ concentrations in fish transferred to seawater (Knoeppel *et al* 1982; Leatherland, 1985). Subsequent studies suggested that conversion of T_4 to T_3 was essential for the osmoregulatory action of thyroid hormones (LeLoup and Lebel, 1993). Potential iono and osmo-regulatory effects of thyroid hormones reflect their possible effects on gill Na^+-K^+-ATPase activity. Gill Na^+-K^+-ATPase activity increased concomitantly with an elevation in plasma T_4 (Dickhoff *et al* 1977; Folmar and Dickhoff, 1979; Sullivan *et al* 1983; Boeuf and Prunet, 1985). However, in other experiments administration of thyroid hormones did not affect the gill Na^+-K^+-ATPase activity (Dickhoff *et al* 1977; Saunders *et al* 1985; Dange, 1986) and T_3 treatment decreased gill Na^+-K^+-ATPase activity of freshwater rainbow trout (Omeljaniuk and Eales, 1986). Part of the explanation for such differences may reflect the interaction of thyroid hormones with other hormones, such as cortisol and growth hormone, in determining Na^+-K^+-ATPase activity (Dange, 1986; see review by McCormick, 1995). T_4 administration did not increase Na^+-K^+-ATPase activity in seawater-acclimated tilapia by itself but did act synergistically when administered with cortisol (Dange, 1986). However, there was no apparent synergistic action of T_4 with cortisol in increasing chloride cell number or gill Na^+-K^+-ATPase activity in rainbow trout (Madsen, 1990).

Thyroid hormones affect various aspects of metabolism. Effects of thyroid hormones on carbohydrate and lipid metabolism have been reported (Leatherland, 1982; Plisetskaya *et al* 1983) and thyroid hormones may play a role in a stress-related mobilization of glucose in fish. Thyroxine treatment caused hyperglycemic effects in some cases (Chan and Woo, 1978b). However, other studies observed hypoglycemic responses to thyroid treatment (Murat and Serfaty, 1970). Closely related significant elevations of plasma T_4 and glucose induced by disturbance stress were reported (Himick and Eales, 1990). It has also been shown that injection of glucose caused elevation of both plasma glucose and T_4 (Himick and Eales, 1990).

Treatment of fish with low doses of thyroid hormones has been reported to stimulate protein synthesis whereas, treatment with high doses of thyroid hormones appeared to cause catabolic effects (Medda and Ray, 1979; Matty *et al* 1982). The outcome of the various metabolic impacts of thyroid hormones in determining the growth of fish has varied in studies so far. Thyroid hormone administration has been reported to accelerate fish growth (Higgs *et al* 1979; Saunders *et al* 1985; Woo *et al* 1991) and, in support of this concept, elevated concentrations of plasma T_3 were found in periods of rapid growth (McKenzie *et al* 1993). However, in other studies thyroid supplementation did not affect growth (Leatherland *et al* 1987; Gannam and Lovell, 1991; Soengas *et al* 1992). The apparent inconsistencies in these experiments may partially reflect the interaction of thyroid hormones with other hormones such as growth hormone and cortisol. Thyroid hormones treatment was found to promote the anabolic effects of growth hormone (e.g. lower liver glycogen and higher serum cholesterol) in rainbow trout (Fagerlund *et al* 1980; Farbridge and Leatherland, 1988). On the other hand, growth hormone treatment significantly elevated the plasma T_3 concentration of rainbow trout (Farbridge and Leatherland, 1988; MacLatchy and Eales, 1990) indicating an increased peripheral conversion of T_4 to T_3.

Thyroid hormones may play a significant role in fish behaviour. For example, increased concentrations of plasma thyroxine were associated with migration of juvenile yellow American eels (Castonguay and Dutil, 1990). The level of motor activity of migrant Atlantic salmon was also suggested to be determined by thyroidal status (Youngson and Webb, 1993). Sublethal exposure of coho salmon to arsenic decreased thyroxine concentrations which coincide with decreased migration of smolts (Nichols *et al* 1984).

2.4 Endocrine Interactions During Stress

Stress-induced increases in plasma cortisol in rainbow trout were closely correlated with elevations in growth hormone (Pickering *et al* 1991). Stress was also reported to increase plasma growth hormone in immature rainbow trout (Takahashi *et al* 1991). This stimulation of both cortisol and growth hormone release is important as cortisol has been found to facilitate the actions of other hormones, for example, growth hormone and thyroid hormones (Dange, 1986; Madsen, 1990). Thus, growth hormone stimulation of gill Na^+-K^+-ATPase activity and chloride cell number was enhanced by cortisol treatment (Madsen, 1990).

Chronic stress in fish is often associated with a suppression of growth (reviewed by Wendelaar Bonga, 1997). For toxic stress the distinction of direct toxic effects and those resulting from the integrated hormonal stress responses may be difficult to determine. The influence of hormonal factors is further complicated by the multiplicity of endocrine systems affecting growth. Pickering (1993a) reviewed the integrated hormonal control of growth (gonadal steroids, the thyroid axis, the HPI axis, growth hormone and catecholamines) in relation to stress-induced growth suppression.

It has become clear in recent years that the aspects of the two components of the primary endocrine responses to stress (hypothalamic-pituitary-interrenal (HPI) axis and the hypothalamic-sympathetic-chromaffin cell axis) are in reality components of an integrated response to stress and these two components influence one another. Activation of the HPI axis, ACTH treatment and increased plasma cortisol resulting from cortisol implants have each been found to result in increased chromaffin stores of catecholamines and their release into circulation (Reid *et al* 1996). This implies that chronically stressed fish may be more able to generate a catecholamine response to stress. Furthermore, elevated plasma cortisol concentrations have been reported to elevate the internal population of adrenoreceptors (Reid *et al* 1992) potentially enhancing physiological responses to the circulating catecholamines.

3. Physiological Responses to Stress in Fish

3.1 Carbohydrate Metabolism and Plasma Glucose

Many teleost fish rely primarily on protein and lipid sources for energy and they are considered to possess poor enzyme system for utilization of carbohydrates (Cowey and Sargent, 1979; Walton and Cowey, 1982). In these species, amino acids such as arginine and lysine have been shown to be more effective than glucose in stimulating insulin release (Higuera and Cardenas, 1986; Petersen *et al* 1987; Suarez and Mommsen, 1987). However, carbohydrate metabolism increases in states of high energy demand such as stress. In stress, blood glucose is elevated as a result of both glycogenolysis and gluconeogenesis (Suarez and Mommsen, 1987; WendelaarBonga, 1997).

Stress has been described as an energy drain with energy that might be utilized in growth being diverted to catabolic utilization (Barton and Schreck, 1987; Pickering, 1990; McDonald *et al* 1993). Mobilization of readily available energy in the form of glucose is suggested to enhance the survival of fish (Barton and Iwama, 1991; Pickering, 1993b). It is perhaps not surprising, therefore, that elevation of plasma glucose has been recognized as part of a generalized stress response in fish (Mazeaud *et al* 1977). Increased plasma glucose has been widely reported in fish exposed to physical stressors such as handling and confinement (Mazeaud *et al* 1977; Davis and Parker, 1990; Melotti *et al* 1992; Waring *et al* 1992; Barry *et al* 1993; Foo and Lam, 1993), exercised fish (Zelnik and Goldspink, 1981; Barton *et al* 1986; Flos *et al* 1988; White and Fletcher, 1989; Waring *et al* 1992; McDonald *et al* 1993) and fish exposed to pollutants (Gluth and Hanke, 1984; Edwards *et al* 1987; Jones *et al* 1987; Goss and Wood, 1988; Whitehead and Brown, 1989; AlKindi *et al* 1996).

Petroleum hydrocarbons have been reported to increase plasma glucose in various fish species (DiMichele and Taylor, 1978; Thomas *et al* 1980; Zbanyszek and Smith, 1984; Aabel and Jarvi, 1990; AlKindi *et al* 1996), with dose-related responses in mullets exposed to the WSF of fuel oil (Thomas *et al* 1980). However, rainbow trout exposed to bunker C oil showed an unusual decrease in blood glucose concentrations which was attributed to a malfunction of glucose reabsorption by the renal tubules (McKeown and March, 1977, 1978).

3.2 Osmotic and Ionic Regulation

The regulation of the internal body fluid composition is essential for normal cellular functions in all organisms. In both seawater and freshwater there is a large osmotic gradient between the extracellular fluid of an aquatic organism and its environment (Eddy, 1981; Giesy *et al* 1988). Maintaining a relatively constant internal environment, independent of the external environment, is achieved via the combined actions of the gills, gut and kidneys (Eddy, 1981; McCormick, 1995; Fuentes and Eddy, 1997). The ionic and osmotic concentrations of the body fluid of marine teleost fish are approximately one third those of seawater. The

continuous outward osmotic movement of water is counteracted by drinking seawater, while the continuous influx of salts (sodium and chloride) is balanced by active efflux of salt via branchial chloride cells (Eddy, 1981). In contrast, the body fluids of freshwater fish are more concentrated than the surrounding environment and consequently there will be an influx of water and an outward salt diffusion. Freshwater fish excrete this excess water by production of copious volumes of hypotonic urine and salt losses are compensated for by active uptake via the gills (Eddy, 1981; McDonald and Rogano, 1986). Although highly simplified, this description of the ionic and osmotic-regulatory processes in both marine and freshwater fish may provide the basis for discussion of the osmoregulatory imbalances in stressed fish.

Disturbed plasma osmolarity and electrolyte concentrations have been recognised as a significant effect of stress in fish (Mazeaud *et al* 1977; Thomas *et al* 1980; Engelhardt *et al* 1981; Haux *et al* 1985; Robertson *et al* 1987; Goss and Wood, 1988; Brown *et al* 1990; Waring *et al* 1992; Barry *et al* 1993; Wendelaar Bonga, 1997). Failure to maintain ionic and osmotic balance within certain limits may cause the death of stressed fish (Stegeman and Sabo, 1976; McDonald, 1983). Altered plasma osmotic and ionic concentrations have been reported in response to physical stressors such as handling and transport (Barton *et al* 1985; Robertson *et al* 1987; Davis and Parker, 1990; Waring *et al* 1992; Barry *et al* 1993; Waring *et al* 1996b), and pollutant stress (Swift 1981; Stagg and Shuttleworth, 1982; Gluth and Hanke, 1984; Edwards *et al* 1987; Jones *et al* 1987; Goss and Wood, 1988; Allen, 1994) including exposure to petroleum hydrocarbons.

Petroleum hydrocarbons have been reported to have profound effects on plasma Na^+, K^+ and Cl^- concentrations with increases in fish exposed to crude oil in seawater, while concentrations of these ions decreased in fish exposed to crude oil in freshwater (Morrow, 1974; Engelhardt *et al* 1981; Baklien *et al* 1986). However, decreased plasma chloride concentration in marine fish have also been reported (Payne *et al* 1978; Fletcher *et al* 1979; Kiceniuk *et al* 1980) while no change in plasma Cl^- occurred in the Antarctic fish, *Pagothenia borchgrevinki*, exposed to the WSF of fuel oil (Davison *et al* 1992, 1993). Moreover, exposure to the WSF of crude oil had no effect on plasma Na^+ and Cl^- concentrations of flounders (AlKindi *et al* 1996). Sediment containing aliphatic hydrocarbons had no effect on plasma Cl^- concentrations of winter flounder *Pleuronectes americanus* (Payne *et al* 1995). Interestingly, Brauner *et al* (1999) found that WSF contaminated water had no effect on plasma Na^+ and K^+ concentrations in *Hoplosternum littorale* (a teleost from the Amazon), whereas crude oil exposure via the diet caused significant reductions in the plasma concentrations of these ions.

Reported changes in concentrations of plasma divalent ions in response to petroleum hydrocarbons are also inconsistent. Plasma Ca^{2+} concentrations were decreased in rainbow trout exposed to bunker C oil solution in seawater (McKeown and March, 1977), but flounders exposed to dispersed crude oil in seawater showed an elevation of plasma concentrations of both Ca^{2+} and Mg^{2+} (Baklien *et al* 1986) while no significant changes in plasma Ca^{2+} and Mg^{2+} were observed in marine cunners, *Tautogolabrus adspersus*, chronically exposed to weathered crude oil (Fletcher *et al* 1979).

Changes in plasma osmolality of fish exposed to crude oil were generally in line with those to be expected from the change in monovalent ions in the particular group of experimental fish (McKeown and March, 1977, 1978; Engelhardt *et al* 1981). Thus, osmolality was increased in seawater mullets acutely exposed to the WSF of fuel oil (Thomas *et al* 1980), however, there was no change in the plasma osmolarity of an Antarctic fish *Pagothenia borchgrevinki* in response to the WSF of fuel oil (Davison *et al* 1993) or in flounders exposed to the WSF of crude oil (AlKindi *et al* 1996).

Ionic and osmotic disturbances in fish exposed to petroleum hydrocarbons suggest specific types of gill damage such as increased permeability to ions or a deceased activity of ion pumps/exchange mechanisms (McKeown and March, 1977, 1978; Wong and Engelhardt, 1982). In line with this concept, gill Na^+-K^+-ATPase activity was reduced in Pacific staghorn sculpin exposed to refinery wastewater (Boese *et al* 1982).

3.3 Endocrine Systems and Ionic and Osmotic Regulation During Stress

Catecholamines, released as part of the generalized stress response, have been reported to increase branchial water permeability and cause branchial vasodilation, and may, partially explain stress-related weight loss in seawater fish and a gain in weight by stressed freshwater fish (Mazeaud *et al* 1977). The branchial effects

of catecholamines would probably lead to disturbances in plasma ions with elevated plasma catecholamines in freshwater fish, leading to a decrease in the concentration of plasma ions and elevated catecholamines in seawater fish, increasing the concentration of plasma ions (Mazeaud *et al* 1977; Mazeaud and Mazeaud, 1981; Isaia, 1984; Wendelaar Bonga, 1997). However, injection of freshwater-adapted eels with adrenaline, noradrenaline or dopamine did not significantly change plasma osmolarity, Na^+ or Cl^- (Epple and Kahn, 1985).

Cortisol is a major stress-related hormone and is reputed to directly affect osmoregulation. Since stress frequently causes changes in plasma osmolarity in fish, the role of cortisol is of interest. Cortisol is suggested to stimulate ion movement by increasing gill Na^+-K^+-ATPase activity (Pickford *et al* 1970; Forrest *et al* 1973; Dange, 1986; Madsen, 1990; Bern and Madsen, 1992; McCormick, 1995). In this connection, cortisol has been observed to increase the number of chloride cells (Perry and Wood, 1985; Laurent and Perry, 1990) and increase their apical area (Laurent and Perry, 1990). Thyroid hormones may also affect the osmoregulation of teleost fish via their effects on Na^+-K^+-ATPase activity, but this is still far from conclusive.

3.4 Blood Oxygen

The internal respiratory status of fish is determined by the combined processes of branchial gas transfer and blood gas transport. Ventilatory water flow and lamellar blood perfusion, together with the diffusive properties of the gill epithelium collectively, determine branchial gas transfer. Blood gas transport is affected by numerous factors including the blood oxygen carrying capacity and homodynamic variables such as cardiac output and regional blood flow distribution (Perry and Wood, 1989).

During stress, plasma catecholamines may be elevated and the increase in catecholamines generally enhances the oxygen carrying capacity of fish. This is particularly evident during hypoxia (Mazeaud *et al* 1977; Ristori and Laurent, 1989; Aota *et al* 1990; Perry *et al* 1991, 1993) and hypercapnia (Perry *et al* 1989). For example, catecholamines stimulate a rapid release of red blood cells from the spleen into the bloodstream (Kita and Itazawa, 1989; Perry and Kinkead, 1989). Furthermore, catecholamines have been reported to increase red blood cell pH (Nikinmaa, 1986; Primmett *et al* 1986) and to increase the oxygen affinity of hemoglobin. Catecholamines may also enhance the oxygen carrying capacity via their homodynamic effects, stimulation of hyperventilation and by increasing branchial gaseous exchange.

The respiratory status of fish has been an area of major interest in studying the effects of pollutants on fish (Patten, 1977; Johnstone and Hawkins, 1980; Gluth and Hanke, 1984; Malte, 1986; Malte and Weber, 1988; Davison *et al* 1993). Blood oxygen decreased in rainbow trout exposed to aluminum (Malte, 1986), acid (Malte and Weber, 1988) or copper (Wilson and Taylor, 1993). AlKindi *et al* (1996) found reduced blood oxygen content in WSF-exposed flounders. This hypoxia may have been the cause of the elevated plasma noradrenaline concentrations.

The Antarctic fish, *Pagothenia borchgrevinki*, exposed to the WSF of fuel oil were much less tolerant of low levels of ambient oxygen than non-exposed fish (Davison *et al* 1993) and markedly elevated their rate of oxygen consumption during pollutant exposure (Davison *et al* 1992). Similarly, juvenile American shad exposed to the WSF of crude oil were less tolerant of lower levels of dissolved oxygen (Tagatz, 1961). Reduction in oxygen uptake has been reported as a serious symptom of petroleum toxicity in a variety of fish species (DeVries, 1977; Prasad, 1987; Davison *et al* 1993). Reduced oxygen uptake was observed in sculpin exposed to naphthalene (DeVries, 1977) and in carp exposed to crude oil (Prasad 1987). However, increased oxygen uptake was observed in cod exposed to a simulated oil slick (Johnstone and Hawkins, 1980). Brauner *et al* (1999) found elevated breathing frequency in the air breathing teleost *Hoplosternum littorale* exposed to crude oil.

The respiratory effects of petroleum hydrocarbon may be related to their histopathological effects on gills, which are discussed later in this review. Interestingly, Gagnon and Holdway (1999) found altered gill metabolic enzymes in Atlantic salmon (*Salmo salar*) exposed to the water accommodated fraction of crude oil and dispersed crude oil. Also, some recent work has shown that crude oil exposure or exposure to its WSF reduces the growth rate of fish (Christiansen and George, 1995; Gundersen *et al* 1996).

3.5 Hematocrit and Blood Hemoglobin Concentration

Changes in hematocrit (% packed erythrocytes) and blood hemoglobin concentrations have been widely reported as indicators of stress in fish (Fletcher, 1975; Thomas *et al* 1980; Engelhardt *et al* 1981; Haux *et al* 1985; Goss and Wood, 1988; Aabel and Jarvi, 1990; Davison *et al* 1992, 1993). Physical stressors such as handling and transport and pollutant stressors have been reported to increase hematocrit and blood hemoglobin concentrations (Fletcher, 1975; Yamamoto *et al* 1980; Swift, 1981; Barton *et al* 1985; Jones *et al* 1987; Goss and Wood, 1988; AlKindi *et al* 1996), although some studies of stressed fish have reported decreased hematocrit and blood hemoglobin concentrations (Hattingh and Van Pletzen, 1974; Davis and Parker, 1990; Mazur and Iwama, 1993) or no significant change in these parameters (Haux *et al* 1985).

Crude oils and their products have been reported to affect blood hemoglobin concentration and hematocrit. Hematocrit initially increased after exposure of flounder to a 50% dilution of the WSF of Omani crude oil (AlKindi *et al* 1996), dab and English sole to oil-contaminated sediment (McCain *et al* 1978; Tahir *et al* 1993), Pacific staghorn sculpin to petroleum refinery wastewater (Boese *et al* 1982) and Antarctic fish (*Pagothenia borchgrevinki)* to fuel oil (Davison *et al* 1992, 1993). Similarly, hemoglobin concentrations were increased in rainbow trout exposed to the WSF of a mixture of aromatic hydrocarbons (Zbanyszek and Smith, 1984), and in Atlantic salmon exposed to an oil slick, and in English sole exposed to oil-contaminated sediment (McCain *et al* 1978). However, hematocrit was decreased in sculpin exposed to petroleum hydrocarbons (DeVries, 1977). Hematocrit and blood hemoglobin concentrations were also dramatically reduced (by ~50%) in flounder exposed to a 50% solution of the WSF of crude oil (resulting in exposure to approximately 6ppm aromatic hydrocarbons) (AlKindi *et al* 1996).

Adding Further confusion to the discussion of the haematological effects of petroleum hydrocarbons, there have been several studies in which no significant changes in either hematocrit and/or hemoglobin concentrations were found. The hematocrit of cunners, *Tautogolabrus adspersus* (Payne *et al* 1978; Kiceniuk *et al* 1980) and Atlantic salmon (Aabel and Jarvi, 1990) and the hematocrit and hemoglobin of longhorn sculpin (Khan, 1991a) were unaffected after chronic exposure to crude oil. Hematocrits and blood hemoglobin concentrations were also unchanged in *Hoplosternum littorale* exposed to the WSF of crude oil (Brauner *et al* 1999) and hematocrit was not affected in winter flounder exposed to sediment contaminated with oil-base mud (Payne *et al* 1995). The apparent contradictions in haematological effects of petroleum hydrocarbons are likely to reflect a combination of differences in the level of petroleum hydrocarbons to which the fish have been exposed, any resultant gill damage, the length of time of exposure and species differences in sensitivity and catecholamine responses. In relation to the last of these factors, it is clear that fish possess a splenic reservoir of erythrocytes which when released (under catecholamine stimulation) significantly elevates hematocrit and blood hemoglobin concentrations (Yamamoto *et al* 1980; Kita and Itazawa, 1989; Yamamoto and Itazawa, 1989; Pearson and Stevens, 1991). Reduction of splenic weight, an indication of the release of erythrocytes into general circulation by splenic contraction, has been recognized as a useful index of stress in fish. Decreased splenic weight has been reported in fish exposed to crude oils and their derivatives (Payne *et al* 1978; Kiceniuk *et al* 1980). However, splenic weight was unchanged in other studies of fish exposed to petroleum hydrocarbons (Khan, 1991a; Davison *et al* 1993; Payne *et al* 1995).

4. Histopathological Effects of Crude Oil and Petroleum Hydrocarbons on Fish

Petroleum hydrocarbons have been reputed to cause a variety of histopathological effects in fish. Malins (1982) reviewed the structural effects of petroleum hydrocarbons on marine fish and reported hepatocellular vacuolisation, increased liver rough endoplasmic reticulum (RER), gill damage (for example, epithelial lifting, chloride cell damage and fusion of the secondary lamellae), hyperplasia of the olfactory epithelium, degeneration of olfactory mucosal tissue and development abnormalities (for example, misfit of the lower jaw into the upper jaw, missing premaxillary bones and failure of the jaw to fully differentiate, absence of the branchiostegal membranes and reduced cephalization).

4.1 Gill Histopathology

Copious mucous secretion from the gills with hyperplasia and hypertrophy of mucus-producing epithelial cells are widely reported histopathological effects of crude oils and their products (Hawkes, 1977; Solangi, 1980; Engelhardt *et al* 1981; Lopez *et al* 1981 Haensly *et al* 1982; Solangi and Overstreet 1982; Khan and Kiceniuk, 1984, 1988; Khan, 1987; Prasad, 1988, 1991; Davison *et al* 1993).

Aside from increased mucous secretion, petroleum hydrocarbons have been reputed to have several other pathological effects on the gills. These include separation of respiratory epithelium, lamellar curling and hyperplasia, fusion of adjacent secondary lamellae and hemorrhaging of gill filaments (Blanton and Robinson, 1973; Hawkes, 1977; Solangi, 1980; Engelhardt *et al* 1981; Malins and Hodgins, 1981; Woodward *et al* 1981; Haensly *et al* 1982; Solangi and Overstreet, 1982; Khan and Kiceniuk, 1984; AlKindi, 1995). In some studies, gill damage and hemorrhaging from gill filaments could account for the decline in the number of circulating red blood cells (AlKindi *et al* 1996), but few studies have included both physiological and histological approaches. A further change in gill histology reported in some studies was a proliferation of chloride cells in fish exposed to crude oil and its various fractions (Solangi, 1980; Engelhardt *et al* 1981; Lopez, 1981). Such an event could result from endocrine responses which have already been discussed.

Gills have been observed to be sensitive to crude oil or its WSF even at relatively low concentrations. Coho salmon and starry flounder exposed to about 100 ppb of the WSF of crude oil for five days developed gill lesions which included loss of surface cells or the first two or three layers of the mucous cells, exposing immature mucous cells (Hawkes, 1977). In addition, increased levels of parasite infection were observed in the gills of these fish. The extent of parasitic infection of the gills of oil-treated sculpin and cod was about 17-fold greater than that of control fish (Khan, 1990) and chronic exposure of longhorn sculpin (Khan, 1991a) and flounder (Khan, 1991b) to oil-contaminated sediment increased parasite infection. An increased number of monogenoid parasites in the gills of cod exposed to the WSF of crude oil (approximately 30-80 ppb) occurred alongside other changes in gill morphology such as epithelial hyperplasia at the interlamellar bases and an increased number of mucus-secreting cells (Khan and Kiceniuk, 1988). Such events could reflect the well-known immunosuppressive action of an activated corticosteroid response (see Wendelaar Bonga, 1997). At higher concentrations, 70% of the exposed fish showed lesions such as epithelial hyperplasia and lamellar curling. Similarly, lamellar curling and swelling (edema) with ruptured afferent and efferent blood vessels, increased necrotic debris and extensive deterioration of the gill structure were all observed in rainbow trout exposed to bunker C oil mixed in seawater (McKeown and March, 1977, 1978) or the WSF of crude oil (Prasad 1988; Khan and Kiceniuk, 1988). However, Payne *et al* (1995) reported no gill histopathological changes in winter flounder exposed to sediment contaminated with oil-base mud, apart from a very mild hyperplasia of the gill tips in fish exposed to the lowest hydrocarbon level.

Chronic exposure to oil-shale water caused a graded increase in the diffusion distance of the gills of rainbow trout, which was proportional to the pollutant concentration. Increased diffusion distance was attributed to swelling of the lamellae and epithelial cell hypertrophy (Johnson, 1983). Increased branchial diffusion distance has also been attributed to elevated mucous secretion in the Antarctic fish, *Pagothenia borchgrevinki,* exposed to the WSF fuel oil (Davison *et al* 1993).

Fish living in the vicinity of drilling platforms which may be exposed to high levels of petroleum hydrocarbons were reported to show hyperplasia of gill epithelium (Grizzle, 1986). Once into production, coastal and marine platforms in many areas of the world currently discharge the waste ‘produced water’, contaminated with ppm levels of hydrocarbons, into the marine environment. Here it will be heavily diluted and subject to rapid degradation, but effects on fish living in the vicinity of the platforms is of interest. In a recent study, turbot juveniles were exposed for up to 6 weeks to dilutions of a North Sea oil platform ‘produced water’ using dilutions to mimic the likely concentration close to the discharge point to the likely concentration at several kilometers from discharge (Stephens *et al* 2000). In turbot exposed to 1% or 0.1% produced water (likely to occur close to the discharge point) for 6 weeks ~50% gill lamellae were fused at their tips which appeared to develop after a curling and sticking at their tips. As a result, respiratory and/or ionoregulatory processes were likely to be disturbed in these fish. The higher concentration of produced water (1%) resulted in a similar

proportion of fused lamellae within 3 weeks. Lower concentrations, however, did not cause a significant development of fusion.

4.2 Liver Histopathology

Increased cellular vacuolization, RER proliferation and glycogen depletion have been widely reported in the liver of fish exposed to petroleum hydrocarbons (Hawkes 1977; DiMichelle and Taylor, 1978; McCain *et al* 1978; Sabo *et al* 1978; Whipple *et al* 1978; Solangi, 1980; Solangi, and Overstreet, 1982; Kiceniuk and Khan, 1983; Khan and Kiceniuk, 1984). According to Sindermann (1982), increased vacuolization in liver cells reflects an increased hepatic lipid content in fish exposed to petroleum hydrocarbons. However, Eurell and Haensly (1981) suggested that vacuole formation was not directly related to lipid accumulation; after 14-21 days of exposing Atlantic croaker, *Micropogon undualatus* to 5% WSF of crude oil, liver lipids were lower in exposed fish than in controls, although at 10% WSF, lipid content was increased. Since vacuolization in liver cells was a consistent response, it did not appear to be closely related to an increased lipid content. Also, exposure of sculpin to naphthalene did not appear to cause histological damage in the liver (DeVries, 1977). Marty *et al* (1999) found evidence of increased incidence of hepatic necrosis in Pacific herring (*Clupea pallasi*) sampled from oiled sites in Alaska and suggested that this may have resulted from a crude-oil associated increase in viral disease prevalence.

Vacuolization and increased RER have been observed not only in the liver but also in a variety of other organs. The intestine of petroleum-exposed chinook salmon revealed a presence of irregular cellular inclusions in the columnar cells of the intestinal mucosa, increased vesicle density in the cytoplasm near the luminal surface of the columnar cells and cytoplasmic changes of the basal cells of the mucosa. Moreover, membrane bound vesicles containing fine granular material filled the luminal half of the intestine columnar cells and increased RER, as in the liver, has also been observed (Hawkes *et al* 1980; Haensly *et al* 1982).

4.3 Kidney Histopathology

There have been relatively few histopathological studies of fish exposed to the WSF of crude oil (Solangi, 1980; Haensly *et al* 1982). In two studies shrinkage of renal tubules and epithelial separation from basement membranes suggested loss of functional renal tubules in fish exposed to the WSF of crude oil (Solangi, 1980), while dilation of the Bowman's space and hypertrophy of the glomeruli were observed in kidneys of the plaice (*Pleuronectes platessa*) collected from oil spill areas (Haensly *et al* 1982).

5. Effects of Petroleum Hydrocarbons on Fish Larvae

The early life stages of marine fish appear to be more sensitive to hydrocarbons than adult fish (Stene and Lonning, 1984; Carls and Rice, 1988; Marchini *et al* 1992). The greatest embryonic sensitivity to pollutants such as the WSF of crude oil may be very early in the development of an embryo when damage to a few precursor cells could potentially result in extensive damage as the embryo develops (Moore and Dwyer, 1974; Rosenthal and Alderdice, 1976; Carls and Rice, 1988). However, studies often indicate that fish eggs are, in fact ,more resistant to dissolved petroleum hydrocarbons than larvae (see Carls and Rice, 1988). These variations in resistance to hydrocarbons may reflect differences in the bioaccumulation of hydrocarbons by eggs and larvae: embryonic tissues may accumulate substantially less hydrocarbons (which may tend to partition in the yolk) than accumulation by larval tissues (Carls and Rice, 1988).

Moore and Dwyer (1974) estimated that fish larvae may be ten times more sensitive to soluble aromatic hydrocarbons (ranging from 0.1 ppm to 1.0 ppm) than adult fish. This high sensitivity has been suggested to reflect a possible reduction in detoxifying capacity, their high surface area in contact with the environment and their limited mobility (Rice, 1985).

Petroleum hydrocarbons have been reported to affect the survival of fish larvae (Linden, 1976; Kuhnhold *et al* 1978; Falk-Petersen *et al* 1985; Tilseth *et al* 1984; Carls, 1987; Edsall, 1991; Marchini *et al* 1992; AlKindi *et al* 1996). Exposure to weathered crude oil caused high mortality in larvae of the Baltic herring

(Linden, 1976). Similarly, petroleum hydrocarbons such as polycyclic aromatic hydrocarbons (i.e. naphthalene and 1,3-dimethylnaphthalene) and cyclic alkanes were found to be toxic to rainbow trout alevins (Edsall, 1991) and exposure of cod larvae or Pacific herring larvae to the WSF of crude oil affected survival (Falk-Petersen *et al* 1983; Carls, 1987). Exposure to benzene derivatives also reduced the survival of larval and juvenile fathead minnow with larvae being more sensitive than juveniles (Marchini *et al* 1992).

Crude oils and their hydrocarbon derivatives have been reported to cause morphological and developmental abnormalities in fish larvae (Lindcn, 1976; Lonning, 1977; Smith and Cameron, 1977, 1979; Cameron and Smith, 1980). Exposure of Pacific herring larvae to crude oil caused enlargement of the pericardial cavity, failure in development of branchiostegal membranes and erosion of pectoral fins (Smith and Cameron, 1977), and abnormal mouth development (Smith and Cameron, 1977, 1979). Exposure to crude oil caused cellular disruption manifested in intercellular membrane breakdown, irregular and non-membrane bound intracellular spaces in brain and muscle tissues and swollen mitochondria 'some with damaged cristae' in Pacific herring larvae (Cameron and Smith, 1980). Exposure of three marine species of larvae to crude oil and aromatic hydrocarbons caused abnormally bent notochords, poor differentiation of the head region and protruding eye lenses (Lonning, 1977). An ultrastructural study of plaice larvae exposed to crude oil showed irregular and degenerating muscle tissue (Falk-Petersen and Kjorsvik, 1987). These morphological abnormalities can exert dramatic impacts on larval fitness. A completely normal mouth and normal swimming are essential for feeding while well developed fins are important for swimming and avoidance of predators (Smith and Cameron, 1977).

Exposure of fish larvae to petroleum hydrocarbons has been reported to affect their swimming ability (Linden, 1975; Smith and Cameron, 1977; Tilseth *et al* 1984; Carls, 1987). Rice (1985) described the pattern of behaviour of larvae exposed to high levels of the WSF of crude oil as a brief burst in activity followed by deep narcosis and, ultimately, death. This pattern of rapidly reduced swimming ability was confirmed in turbot larvae exposed to 25% to 50% of the WSF of crude oil, although there was good survival (over a 6h experimental period) in 25% and 33% WSF (AlKindi, 1995; Stephens *et al* 1997). Reduced swimming ability is a widely reported effect of oil hydrocarbons on fish larvae (Linden, 1975; Smith and Cameron, 1977; Tilseth *et al* 1984; Carls, 1987). Reduction in swimming ability and deep narcosis of larvae will seriously cripple their ability to feed and to avoid predators. Baltic herring larvae exposed to crude oil showed abnormal swimming behaviour characterized by vigorous swimming up to the surface followed by a slow sinking to the bottom (Linden, 1975).

Petroleum hydrocarbons have also been reported to affect the metabolism of fish larvae (Eldridge *et al* 1977; Serigstad and Adoff, 1985). Depressed oxygen uptake may cause impaired growth and development (Eldridge *et* al 1977; Kuhnhold *et al* 1978; Serigstad and Addof, 1985; Tilseth *et al* 1984; Carls, 1987; Marchini *et al* 1992). Moles *et al* (1981) found that the growth rate of coho salmon fry was inversely related to increasing concentrations of naphthalene and toluene. Direct exposure to crude oil caused a reduction in the length of larvae of the Baltic herring (Linden, 1976), Pacific herring (Smith and Cameron, 1977; Carls, 1987) and cod (Tilseth *et al* 1984). Ingestion of crude oil in the diet also resulted in a reduced body length of Pacific herring larvae (Carls, 1987). Exposure to benzene derivatives also reduced the growth of fathead minnow larvae (Marchini *et al* 1992) and post-yolk sac larvae of Pacific herring exposed to benzene showed decreased growth even though they ingested more food than unexposed larvae (Eldridge *et al* 1977). However, exposure to the WSF of crude oil caused a rapid reduction in feeding of Pacific herring larvae (Carls, 1987). Oil-induced reduction in growth may represent the result of a diversion of energy to the well-established detoxification processes (Eldridge *et al* 1977; Kuhnhold *et al* 1978) as well as effects on feeding and oxygen uptake (see above).

Very few studies have investigated the resultant endocrine stress responses of larvae exposed to petroleum hydrocarbons. A concentration and time-related increase of whole body T_4 and T_3 content of turbot larvae exposed to the WSF of crude oil was reported (Stephens *et al* 1997). Increased whole body cortisol content (a measure of *de novo* synthesis and release of cortisol) has also been observed in fish larvae exposed to petroleum hydrocarbons (Ramsay, 1991; AlKindi, 1995; Stephens *et al* 1997).

6. Conclusions and Future Research

Endocrine responses of fish exposed to petroleum and related hydrocarbons are not well established. Cortisol has been investigated more than any other stress-related hormone but so far responses are not well established (AlKindi *et al* 1996). There is a need for further dose-related studies on the effects of petroleum hydrocarbons on plasma cortisol and other hormones. There is also a need to establish the ability of fish to adapt to chronic exposure to petroleum hydrocarbons. Chronic exposures may 'down-regulate' the corticosteroid response, which has been observed in response to other chronic stressors (Wendelaar Bonga, 1997).

Crude oil and their products have been reported to affect osmotic and ionic balances of a wide variety of fish species but the responses have not been entirely consistent. Analysis of the iono- and osmo-regulatory processes are required to more clearly determine the impact of petroleum hydrocarbons.

Morphological alterations caused by exposure of fish to individual petroleum hydrocarbons and to model hydrocarbon mixtures or to whole crude oil have been observed by investigators both in laboratory and field studies, but investigators often report the structural effects of toxicants without investigating potential functional abnormalities. There are relatively few integrated studies of morphological and physiological events.

The evaluation of the hypothalamic neurosecretory hormones during petroleum-induced stress should be explored in future studies. We can obtain a more complete picture by examining not only the peripheral aspects but also the central control as well. Investigations at a molecular level are needed to understand the full mechanism of stress.

Finally, most of the research on the effects of stress and pollutants on fish has been carried out in cold waters of the northern latitudinal regions of the World, and therefore, there is a need to carry out similar studies in the warm waters. This is especially vital in areas like the Arabian Gulf, which is probably one of the largest oil trafficking and highly polluted regions in the world.

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