CHAPTER 2

THE INVOLVEMENT OF POLLUTION WITH FISH HEALTH

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Abstract: It is generally accepted that pollutants, such as hydrocarbons, enter the aquatic environment by accident or deliberately, and may lead to large-scale and sudden kills of animal life, especially when the compounds are in high quantities. However, more subtle changes to the host may ensue when lesser quantities of pollutants are involved. Here, the resulting damage may include immunosuppression, physical damage to gills and epithelia, and adverse affects on metabolism. Also, there may well be increased susceptibility to various infectious diseases, including lymphocystis and ulceration. Much of the work to date has centered on laboratory studies and also surveys of polluted and clean marine sites, but it is not always possible to make firm conclusions from the data.

Keywords: fish disease; ulceration; fin rot; tail rot; pollutants; pesticides; hydrocarbons; heavy metals

Introduction

It is well established that pollutants of various kinds reach the aquatic environment either accidentally or deliberately. Large-scale releases of hydrocarbons, such as from ocean going tankers e.g. the Exxon Valdez in Alaska and the Braer in the Shetland Islands, have featured prominently on news programmes in many countries when photographs of dead sea birds amid oily beaches greet the viewer. With such examples, it is easy to associate cause, i.e. the pollution event, with effect, namely death of the animal. In addition, there are natural events when, for example, the collapse of algal blooms lead to adverse water quality and large-scale fish kills. Moreover, evidence has been obtained – and will be discussed later – for the presence of specific pollutants within the tissues of aquatic animals. However, there is a dilemma proving a relationship associating the presence of pollutants in tissues with those in the aquatic environment and the concomitant adverse effect on health. A complication is

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that the presence of pollutants, e.g. copper, in tissues is not always correlated to the presence of the compounds in the environment. For example, the accumulation of copper to 1,936 μg/g wet weight was detected in the non-cytosolic fraction of the liver of mullet (*Mugil cephalus*) but without any evidence of environmental contamination insofar as the fish were collected from separate areas with low copper concentrations (Linde et al., 2005). Yet other fish species caught in those same areas did not reveal the presence of high copper concentrations (Linde et al., 2005). This suggests that some species have the innate ability to accumulate heavy metals regardless of whether or not the habitat is contaminated. A previous health scare concerning the presence of mercury in the liver of tuna may also be explained by the inherent ability of the species to accumulate the metal.

Pollutants in the Aquatic Environment

There is a large body of evidence demonstrating the presence of certain specified pollutants in aquatic habitats, and include:

- Heavy metals; these include arsenic, copper, zinc (Gassman et al., 1994; Han et al., 1997), cadmium, lead and mercury which occur in industrial effluents (Gassman et al., 1994; Bernier et al., 1995) and tin, tributyltin and triphenyltin that occur in anti-fouling paints used on the undersurfaces of ships to prevent bioattachment and biofouling (Horiguchi et al., 1995).
- Hydrocarbons; these may result from deliberate spillage during wartime (Evans et al., 1993; Turrell, 1994; Newton and McKenzie, 1995) or accidental discharge from tankers.
- Inorganic nitrogen as ammonia, nitrites and nitrates, which may be derived from agricultural run-off and aquaculture (Ziemann et al., 1992).
- Organic material (Grawinski and Antychowicz, 2001), including faecal debris, from drainage systems. Large numbers of bacteria may be associated with this material (Dudley et al., 1980).
- Pesticides, including dioxin (Guiney et al., 1996), 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD; Grinwis et al., 2000), 1, 1, 1-trichloro-2,2-bisi (*p*-chlorophenyl) ethane (DDT) (Fitzsimons, 1995) and organochlorines (Dethlefsen et al., 1996).
- Plastics (Goldberg, 1995)
- Pulp mill effluents (Lehtinen et al., 1984; Sandstrom, 1994; Couillard and Hodson, 1996; Jeney et al., 2002)
- Toxins, such as from the collapse of dinoflagellate blooms (Noga et al., 1996).

The discharge of these pollutants may be gradual, intermittent pulses or irregular, and may be authorised or unauthorised by national or local authorities. Pollutants may be released in specified areas or dumped in an ad hoc manner. Local or more widespread adverse water quality issues may develop. It is argued that the extent and longevity of pollutants in the aquatic environment is generally unclear, and needs to be firmly clarified through effective monitoring programmes (e.g. Ibe and Kullenberg, 1995).

Evidence for the Presence of Pollutants in the Tissues of Aquatic Animals

The available data have centered largely on laboratory-based in vitro studies and investigations using animals collected from the aquatic environment. Although in vitro studies may produce interesting data, the relevance to explaining the likely outcome of pollution events to biological systems in the aquatic environment is questionable. Nevertheless, there is evidence that a wide range of pollutants, including DDT (Fitzsimons, 1995), thiocyanate (Lanno and Dixon, 1996), didecyldimethylammonium chloride (Wood et al., 1996), polychlorinated-biphenyls (PCBs; Barron et al., 2000) and creosote fractions (Sved et al., 1997) have been researched, and determined to be taken up by aquatic animals from water.

By using animals collected directly from aquatic habitats, there is good agreement between the presence of some pollutants in tissues and the levels in the environment. For example, copper and zinc have been detected in cod (*Gadus morhua*) from coastal waters of Newfoundland, Canada, in invertebrates and vertebrates from Taiwan (Han et al., 1997), and in rabbitfish (*Siganus oramin*) from the polluted waters around Hong Kong (Chan, 1995). Cadmium, lead and mercury were detected albeit in small quantities in fish from the Great Lakes (Bernier et al., 1995). It is interesting to note that the amounts of these metals in cod were below the maximum permitted levels for food (Hellou et al., 1992). Furthermore mercury, in concentrations regarded to be insufficient to cause human health problems, has been found in healthy fish and shellfish collected from the vicinity of discharges from a chlor-alkali plant in India (Joseph and Srivastava, 1993). Crustaceans, molluscs and fish from around a sewage outfall contained pesticides, i.e. chlordane, dieldrin, hexachlorobenzene and DDT (Miskiewicz and Gibbs, 1994). DDT, PCB, organochlorine and 2,3,7,8-tetrachlorobenzo-*p*-dioxin were recorded in fish from Vaike Vain Strait in Estonia (Voigt, 1994), from the North Sea (Dethlefsen et al., 1996) and the Great Lakes (Guiney et al., 1996). However, these publications did not mention any evidence that the compounds had actually harmed the aquatic animals.

Using adult walleye (*Stizostedium vitreum*) caught in polluted and relatively unpolluted sites in Wisconsin, USA during 1996 and 1997, significantly higher PCB concentrations together with more evidence of hepatic neoplasms and tumours (adenomas and carcinomas) were found in fish from the polluted areas (Barron et al., 2000). The authors considered that these deleterious effects were consistent with long-term exposure to tumour promoters, e.g. PCBs, in the environment.

Effect of Pollutants on the Health of Aquatic Animals

This is the crux of the issue – what is the effect of the pollutant on the health of the aquatic animal? It is clear that large quantities of pollutants entering the aquatic environment, such as oil from damaged tankers, over a comparatively short period of time may lead to sudden and often extensive kills. With this scenario, there is a clear link between the pollution event and mortalities among aquatic species. It is the low-level exposure to pollutants that causes the greatest discussion regarding the possible effect on the health of aquatic species. Low-level exposure may well lead to chronic damage, which may not develop for a long time (see Mayer et al., 1993) possibly long after the pollution event has ended. This complicates epidemiological investigation insofar as it is difficult to conclusively associate the pollution with ill health.

The long-term exposure to low-level pollution may lead to the accumulation of the pollutant in animals, which in turn could become weakened/immunocompromised (Vos et al., 1989; Sovenyi and Szakolczai, 1993; Sahoo et al., 2005;) and subsequently colonised by opportunist pathogens leading to clinical disease, for example, gill disease of fish (Austin and Austin, 1999). Anthropogenic factors pose recurring problems, and include:

- Chronic exposure of rainbow trout (*Oncorhynchus mykiss*) to didecyldimethylammonium chloride led to an elevation of stress factors, namely plasma cortisol, glucose and lactate, and a decrease in swimming performance (Wood et al., 1996).
- Chlorinated and aromatic hydrocarbons which accumulate in sediments, and may well lead to a decline in wild fish populations (Arkoosh et al., 1998). Such factors have been considered to be responsible in part for the decline of wild Pacific salmon populations with experimental evidence pointing to the bioaccumulation of these classes of hydrocarbons by juveniles leading to immunosuppression and increased susceptibility to disease (Arkoosh et al., 1998). Also hydrocarbons have been implicated with breakages in the double-stranded DNA, and enzyme induction in common dab (Everaarts et al., 1994).

- Damage to fins, gills, opercula and skin has followed exposure to ammonia, copper and phenol (Kirk and Lewis, 1993), cadmium (Sovenyi and Szakolczai, 1993), creosote (Sved et al., 1997), and chlorine-containing pulp mill effluents (Lindesjoo and Thulin, 1994; Lindesjoo et al., 1994; Sandstrom, 1994). Moreover, ammonia has been reported to cause the development of circular depressions and pitting in the gill epithelium of rainbow trout. Exposure to copper led to fusion of the gill lamellae and swelling of the tips of the filaments and epithelium. In comparison, phenol was observed by electron microscopy to destroy the epithelial layers as far as the cartilage (Kirk and Lewis, 1993).
- Exposure to sewage sludge has affected the growth and protein synthesis in common dab (*Limanda limanda*) (Houlihan et al., 1994) and caused liver damage in fish (Moore et al., 1996).
- Kidney damage has resulted from exposure to cadmium (Sovenyi and Szakolczai, 1993).
- Liver damage may result from contamination with cadmium (Sovenyi and Szakolczai, 1993) and thiocyanate (Lanno and Dixon, 1996).
- Sub-lethal concentrations of the pesticides atrazine and lindane (Cossarini-Dunier, 1987), heavy metals (O'Neill, 1981) including cadmium (Sovenyi and Szakolczai, 1993), sewage components (Secombes et al., 1991; 1992) and aquatic micro-organisms (Robohm et al., 1979; Evans et al., 1997) affect the immune system of fish by either stimulating or retarding antibody production.
- Thiocyanate has been blamed as causing anaemia and interfering with thyroid function (Lanno and Dixon, 1996).
- Winter Stress Syndrome, which is characterised by reduced levels of lipids, may well result from stressors including the presence of various pollutants (Lemly, 1997).

Mill effluents have been the focus of many studies into their association with abnormalities/disease of fish (Lindesjoo and Thulin, 1994; Lindesjoo et al., 1994; Sandstrom, 1994; Jeney et al., 2002). During 1982–1987 in the Baltic Sea, the presence of abnormalities was reported, including gonad malfunction, poor embryo quality and mortalities among coastal fish species, which were considered to have been exposed to mill effluent (Sandstrom, 1994). Of relevance after 1984, the toxicity of effluent was reduced by the substitution of chlorine dioxide for chlorine. Then in 1992, the use of chlorine was eliminated altogether, which reduced the amount of organochlorines in the effluent, and led to a reduction in the level of mortalities in the fish populations (Sandstrom, 1994).

Using roach (*Rutilus rutilus*) caught from two lakes, one of which received bleached kraft mill effluent and the second of which was unpolluted, experimental infection with the digenean parasite *Rhipidocotyle fennica* led to the observation that fish from the polluted site possessed a significantly higher number of parasites after the first 2 days of infection. The explanation was that these fish from the polluted site had decreased resistance to infection. In particular, these roach had a lower leucocrit, and higher alkaline phosphatase and plasma chloride levels compared to those fish from the clean site (Jeney et al., 2002).

Dinoflagellate toxins have been blamed for major fish kills, with evidence describing erosion to the epithelium leading to ulceration (Noga et al., 1996). This observation is especially relevant insofar as ulceration is one of the most common diseases associated with pollution in the marine environment.

Pesticides, namely DDT, have been considered to be responsible for reduced hatching, blue sac disease and swim up syndrome mortalities in lake trout (*Salvelinus namaycush*) (Fitzsimons, 1995) eggs. Yet, these laboratory studies indicated that concentrations of pesticides necessary to affect the eggs were much higher than the levels found in feral fish eggs.

In Dutch coastal and estuarine habitats, flounder (*Platichthys flesus*) have a high prevalence of liver disease (pre-neoplastic) and lymphocystis, and because of this laboratory studies were conducted to determine the effect of xenobiotics, namely TCDD, oral exposure to which led to significantly enhanced immunoreactivity in the hepatocytes and endothelium of various organs and the epithelia of the digestive tract, liver and mesonephros to cytochrome P4501A but without any profound pathology (Grimwis et al., 2000). The conclusion was that flounder was relatively insensitive to the toxic effects of TCDD, but an assumption was made that exposure (to TCDD) could influence the development of tumours (Grimwis et al., 2000).

A polluted environment will inevitably contain a variety of contaminants, and it is difficult to determine which may be the trigger for a disease event. A topical example concerns pigmented salmon syndrome, which is a non-infectious haemolytic anaemia with jaundice, and occurred as an epidemic during the early 1980s in migrating Atlantic salmon (*Salmo salar*) in the River Don, Scotland. The river system received effluent from paper mills, the oil servicing industry and an airport. Ensuing experimentation reproduced the disease signs following exposure of Atlantic salmon to hydrocarbons, namely diesel and resin acids. Interestingly, the syndrome was not detected in the River Don after 1989, and most likely reflected general improvements of water quality therein (Croce et al., 1997).

Of course there is an additional problem where pollution is thought to influence the occurrence of disease but where a firm association between specific pollutants and the incidence if ill health cannot be proven. For

example, there was a strong indication that the presence of proliferative kidney disease in wild brown trout and rainbow trout in southern Germany was correlated with organic pollution. However, this association was not proven (El-Matbouli and Hoffmann, 2002).

Pollution-Related Diseases

The development of measurable disease processes involves an interaction between a host, disease-causing situation, e.g. a pathogen, and inevitably a stressor (Austin and Austin, 1999). Without a stressor, the host will often not develop clinical disease (Austin and Austin, 1999). Here, the argument is that pollutants constitute the stressor. A sizeable proportion of the work correlating fish disease with pollution in the aquatic environment has involved surveys, many of which have been carried out in the North Sea (e.g. Dethlefsen and Watermann, 1980; Dethlefsen et al., 1987; 2000; McVicar et al., 1988; Vethaak and ap Rheinallt, 1992). The basic premise is that fish from polluted sites have a greater incidence of disease than specimens obtained from unpolluted areas (e.g. Dethlefsen et al., 2000). Generally, the evidence supports this premise. For example, a study of epidermal hyperplasia/papilloma, lymphocystis and skin ulcers on female dabs (*Limanda limanda*) of >3 years old from sites in and around the North Sea was carried out between 1992 and 1997. The outcome was evidence for a higher incidence of some diseases in the different sampling sites, and a suggestion by the researchers that further investigation was needed to determine the reasons for the observations and a possible link to anthropogenic factors (Dethlefsen et al., 2000). There is an obvious question concerning water flow and fish migration, i.e. currents will carry water to and from polluted and clean sites. Also, the effects of fish migration need to be considered insofar as specimens caught in polluted areas could have recently arrived from clean sites, and vice versa (Bucke et al., 1992; Vethaak et al., 1992; Jacquez et al., 1994). Notwithstanding these concerns and on the basis of probability, it is generally accepted that disease may be mediated in some way by pollution events (Vethaak and Jol, 1996).

Fish diseases, often linked to pollution, include:

- Carcinomas (Koehler et al., 2004)
- Epidermal papilloma (Dethlefsen and Waterrmann, 1980; Premdas and Metcalfe, 1994)
- Fin/tail erosion (Vethaak, 1992; Vethaak et al., 1996; Bodammer, 2000)
- Gill disease/hyperplasia (Kirk and Lewis, 1993)
- Liver disease (Malins et al., 1980; 1987; Peters et al., 1987; Vethaak et al., 1996)
- Neoplasia (Malins et al., 1980; Bucke and Feist, 1993; Depledge, 1996; Vethaak and Jol, 1996)

- Parasitic diseases (Overstreet and Howse, 1977; Pascoe and Cram, 1977; Das and Shrivastava, 1984; Khan, 1987; Möller, 1987; Khan and Thulin, 1991)
- Skin disease/ulceration (Vethaak, 1992; Vethaak and Jol, 1996) which is often associated with infection by atypical *Aeromonas salmonicida* (Austin and Austin, 1999)
- Viral diseases, principally lymphocystis (Vethaak and Jol, 1996; Vethaak et al., 1996).

The reasons for the occurrence of these conditions have been thought to include contaminated diets (Landsberg, 1995), heavy metals (Rødsaether et al., 1977), nitrogenous compounds such as ammonia (Kirk and Lewis, 1993) and nitrites (Hanson and Grizzle, 1985), pesticides (Voigt, 1994), organic material (Grawinski and Antychowicz, 2001) including sewage (Austin and Stobie, 1992) and/or unspecified pollutants (Vethaak and Jol, 1996; Bodammer, 2000). Of these, fin/tail erosion, gill disease, gill hyperplasia, and ulceration are often linked to bacterial involvement. A possible scenario is that the pollutant stresses or weakens the host allowing colonisation by micro-organisms and thus the development of clinical disease.

Viruses and carcinogens may lead to neoplasias/tumours. In this connection, certain geographical areas have been identified where the occurrence of tumours on fish and shellfish has been correlated with higher concentrations of anthropogenic compounds (Depledge, 1996). Of relevance, Koehler et al. (2004) caught juvenile and adult female flounder (*Platichthys flesus*) from a polluted river, i.e. the River Elbe, and a control site with the data revealing that the young fish had liver damage. The adult females displayed adenomas and carcinomas with a frequency of 70% in the polluted river. Exposure to carcinogens in the river was blamed on the incidence of the tumours (Koehler et al., 2004). Yet, the proof of any correlation between pollution and disease is not always documented. Surveys, such as occur with regularity in the North Sea, point to an association between pollution and disease but do not generally consider the nature or concentration of the pollutant(s). For future studies, it would be relevant to consider

- The possibility of synergism between combinations of pollutants
- The minimum exposure time to a pollutant necessary to initiate deleterious changes to the host.

CONTAMINATED FISH FOOD

Landsberg (1995) considered that toxins, i.e. from macroalgae, e.g. *Caulerpa* spp., and benthic dinoflagellates, e.g. *Gambierdiscus toxicus,* may have been responsible for large-scale mortalities in tropical reef fish in Florida, USA

during 1993–1994. Examination of the fish revealed head lesions, ulcerations, fin and tail rot, and the presence of mucus on the body surface (Note: this may suggest exposure to an irritant; Austin and Austin, 1999). Amoebae, bacteria and turbellarians were found in diseased fish, although it was considered that these organisms were opportunists/secondary invaders rather than the primary cause of illness (Landsberg, 1995). In this connection, it is recognised that many diseases may develop in an already weakened host (Austin and Austin, 1999). Yet, whether or not such weakening results from pollutants is rarely if ever considered.

There is evidence that some serious pathogens may be spread through contaminated fish feed used in aquaculture. Incidences of botulism (Huss et al., 1974), mycobacteriosis (Dulin, 1979), streptococcosis (Minami, 1979) and eye disease caused by *Rhodococcus* (Claveau, 1991) have been linked to feeds containing contaminated fish products.

HEAVY METALS

There is increasing evidence that the presence of heavy metals is linked to the exacerbation of some microbial fish diseases. Copper has been singled out for attention insofar as there is evidence for its role in increasing susceptibility to *Edwardsiella tarda* (Mushiake et al., 1984) and *Vibrio anguillarum* (Rødsaether et al., 1977) infections. It has long been established that exposure to copper resulted in coagulation of the mucus layer of gills, leading to inhibition of oxygen transport and respiratory distress (Westfall, 1945) and to a reduction in the populations of lymphocytes and granulocytes in the blood leading to a reduction in phagocytosis (Mushiake et al., 1985).

Titanium dioxide has been implicated with harm to aquatic animals (Dethlefsen and Watermann, 1980; Lehtinen, 1980; Dethlefsen et al., 1987). Thus, as a result of a survey of 5,942 fish caught in Dutch coastal waters in 1986–1988, a higher incidence of epidermal hyperplasia/papillomas, lymphocystis, liver nodules and infections with *Glugea* was documented in the common dab collected from dump sites receiving titanium dioxide acids in comparison to control sites (Vethaak and van der Meer, 1991). This supported the outcome of an earlier investigation which recorded a link between the incidence of epidermal papillomas in common dabs in relation to the dumping of titanium dioxides wastes (Dethlefsen and Watermann, 1980).

HYDROCARBONS

The presence of hydrocarbons leads to impairment of mucus, defective immune systems, increased incidences of parasitism, the induction of hyperplasia and liver hypertrophy, and mortalities (Fletcher et al., 1982; Haensly

et al., 1982; Khan, 1987; 1991; Lehtinen, 1980). Experimental evidence has shown that fish may develop epidermal lesions and fin erosion, or die following exposure to suspended sediments containing high molecular weight creosote fractions (Sved et al., 1997). On the contrary, low molecular weight creosote fractions led to the appearance of head lesions, specifically around the mouth, nares and opercula (Sved et al., 1997).

NITROGENOUS COMPOUNDS

Data revealed that the presence of nitrites at 6 mg/L of water increased the susceptibility of channel catfish (*Ictalurus punctatus*) to *Aeromonas hydrophila* infection (Hanson and Grizzle, 1985).

PESTICIDES

Pesticides, such as DDT and PCBs, in the aquatic environment have been linked with diseases such as "cauliflower disease", lymphocystis and ulceration (Voigt, 1994) and liver neoplasia (Moore et al., 1996). Malformations in common dab, flounder (*Platichthys flesus*), plaice (*Pleuronectes platessa*) and whiting (*Merlangus* sp.) embryos from the southern North Sea during 1984–1995 were linked to pollution with organochlorines (Dethlefsen et al., 1996). Thus as a direct result of long-term surveys, these workers considered that the malformations resulted from low water temperatures that predisposed the embryos to the effects of organochlorines.

Liver disease, including neoplasia, has been described in winter flounder (*Pleuronectes americanus*) from Boston, USA, particularly in the region of a sewage outfall (Moore et al., 1996). These workers noted that during 1987–1993, there was a reduction in the incidence of neoplasia concomitant with a decline in output of chemicals, particularly DDT and other chlorinated hydrocarbons, into the receiving waters.

ORGANIC MATERIALS/SEWAGE

The presence of some fish diseases has been linked to the presence of unknown components of sewage dumping (Siddall et al., 1994). For example as a result of a survey of 16 sites in the Dutch Wadden Sea, a higher incidence of skin ulcers and fin rot was recorded in fish caught near fresh water drainage sluices than elsewhere (Vethaak, 1992). Pollution by domestic sewage, i.e. leakage from a septic tank, was attributed to a new skin disease, which was characterised by extensive skin lesions and muscle necrosis in rainbow trout (otherwise infected with *Yersinia ruckeri* as enteric redmouth disease for which there may well have been a link with sewage sludge; Dudley et al., 1980) in Scotland

during 1992 (Austin and Stobie, 1992). From diseased fish, *Serratia plymuthica* and *Pseudomonas pseudoalcaligenes* were recovered for the first time as fish pathogens. The skin lesions declined substantially after the leaking septic tank was repaired. Also, organic pollution has been blamed for a high occurrence of *S. plymuthica* infections in salmonid farms in Poland since 1996 (Grawinski and Antychowicz, 2001).

Eutrophic waters associated with faecal pollution and high levels of organic material have been attributed to the cause of disease by enteric bacteria, including *Citrobacter freundii* (Austin and Austin, 1999), *Edwardsiella tarda* (Meyer and Bullock, 1973), *Providencia rettgeri* (Bejerano et al., 1979) and *Serratia marcescens* (Baya et al., 1992). In addition, poultry faeces, which was used to fertilise fish ponds, was blamed for mass mortality in silver carp (*Hypophthalmichthys molitrix*) in Israel (Bejerano et al., 1979).

RADIATION

Despite considerable hype particularly in communities around nuclear power stations and the erratic attention of the press, there is not any hard evidence linking radiation pollution in the aquatic environment with health problems among aquatic organisms.

STRESS

Unspecified stressors have been attributed to oxygen deficiency and a statistically significant increase in the incidence of epidermal papillomas, lymphocystis and skin ulcers especially in female common dab from waters around Denmark during the summers of 1988–1993 (Mellergaard and Nielsen, 1995). Also, stress attributed to unnamed environmental factors has been associated with septicaemia in fish from Nigeria (Oladosu et al., 1994).

UNSPECIFIED CAUSES

Many articles have considered the effect of non-specific pollution on the incidence of disease. For example, Vethaak et al. (1996) discussed disease development in flounder contained in mesocosms with contaminated dredged spoil. Compared to clean systems, fish in the polluted environment displayed a higher incidence of lymphocystis and liver damage leading to neoplasia. Yet, there was no appreciable difference in the development of epidermal disease. These results indicated the health problems associated with long-term exposure to pollutants at levels comparable to those in the aquatic environment. In a parallel study, the polluted waters of the Lower

Lake of Bhopal were blamed on the incidence of tumours, i.e. fibromas, in catfish (*Heteropneustes fossilis*) (Qureshi and Prasad, 1995). Dorsal fin tissues obtained from winter flounder (*Pseudopleuronectes americanus*), which were caught from two polluted sites on the eastern seaboard of the USA, were studied by microscopy. The various pathologies observed included epithelial and mucus cell hyperplasia/hypertrophy, spongiosis and focal necrosis. A view was expressed that hypoxia could be involved in the disease process (Bodammer, 2000).

Conclusions

It is well established that pollutants enter the aquatic environment (e.g. Sved et al., 1997), and may be found in the tissues of aquatic vertebrates and invertebrates (e.g. Han et al., 1997). Moreover, some pollutants are instrumental in damaging aquatic organisms (e.g. Lanno and Dixon, 1996). However, there is only limited evidence that pollutants are actually responsible for the development of disease. Indeed, there is negative evidence that has demonstrated that the incidence of disease diminishes when pollution ceases (Sandstrom, 1994). Certainly, many surveys have reported a higher incidence of diseased animals from polluted rather than clean (=control) sites (Vethaak and ap Rheinallt, 1992). Yet, the weakness in most surveys concerns the absence of good quantitative and qualitative data about the actual pollutants. There remains serious doubt of the accuracy of information concerning the relative level of pollutants in polluted and clean sites. Moreover, it is speculative what influence water movement and fish migration patterns have on the results of the surveys. Notwithstanding, there is accumulating evidence that some pollutants immunosuppress or otherwise weaken fish (Sovenyi and Szakolczai, 1993). These weakened animals are more likely to succumb to disease. Some forms of damage, e.g. to gills and skin (Sved et al., 1997), attributable to pollutants are reminiscent of the signs associated with some infectious diseases, i.e. gill disease and ulceration (Austin and Austin, 1999), respectively. In this connection, it is relevant to mention the increasing recognition of atypical isolates of *Aeromonas salmonicida* as a cause of skin lesions/ulceration in native marine fish (e.g. Wiklund and Bylund, 1993; Nakatsugawa, 1994; Pedersen et al., 1994; Wiklund et al., 1994; Wiklund, 1995; Wiklund and Dalsgaard, 1995; Larsen and Pedersen, 1996; Austin et al., 1998). It remains to be proven whether or not this pathogen, which hitherto has been mostly associated with the disease furunculosis in salmonids (Austin and Austin, 1999), interacts mostly with marine fish that may have been already weakened by pollution.

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