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A review on water quality and its impact on Fish health

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Abstract
Potentially harmful substances e.g. pesticides, heavy metals and hydrocarbons are often released into the aquatic environment. When large quantities of pollutants are released there may be an immediate impact as measured by large-scale sudden mortalities of aquatic organisms, e.g. fish kills resulting from contamination of water ways with agricultural pesticides. Lower levels of discharge may result in an accumulation of the pollutants in aquatic organisms. The end results, which may occur long after the pollutants have passed through the environment, include immunosuppressant, reduced metabolism, and damage to gills and epithelia. The absorption and accumulation of different pollutants vary among different biological systems. Therefore, the aims of this review article are to highlighten the impact of the water quality on fish health and the possible control methods. There is now a real need to study the interrelationships between the pollution of surface waters by a wide range of chemicals and diseases in natural fish populations, and the processes involved. This represents an important but at present under-developed field of scientific research and fisheries management.

Keywords: aquatic biota, Water Quality, Pollution, Fish health

1. Introduction
The chemistry of natural surface waters is complex, and depends on the equilibrium reached with the normal physical, chemical and biological characteristics of the surrounding environment. Thus, there can never be a normal surface water quality; every natural water will have a different composition (Alabaster and Lloyd, 1980) [1].

Even rainwater varies in composition in different localities and regions. The precipitated water droplets will absorb acidic ions, volatile chemicals and fine particulate matter of natural and anthropogenic origin. A substantial proportion of the nitrogen input to soils comes with the winter rainfall. Recent data have shown that the level of atrazine in rain can be as high as 1 μg l^{-1} in areas where there is widespread use of this herbicide (Alabaster and Lloyd, 1980) [1].

Rain that falls on soil overlying granite rock will tend to remain acidic and the run-off will be a soft water, low in calcium and bicarbonate. Water draining from areas of peat bogs will also be acidic, and can be extremely so with sudden rainfall after a prolonged dry period. Water with a low pH (<5.0) will dissolve naturally occurring metals from the soils and rocks, especially aluminum and in some areas copper, zinc and lead. Soft waters may be clear, or brown with varying amounts of dissolved humid substances (Alabaster and Lloyd, 1980) [1].

Water falling on soil overlying chalk and limestone will become alkaline with hardness depending on the amount of dissolved calcium and bicarbonate that it contains. To some extent the surface water quality will depend on the type of vegetation covering the watershed, since the products of plant decay (as with the peat bogs mentioned above) will find their way into the streams draining the area. Water draining from coniferous forests tends to be acidic (Alabaster and Lloyd, 1980) [1].

These are examples of natural causes of differences in water quality. Some indication has already been given of the added impact that can be caused by man's activities. Metal mining, by increasing the surface area of exposed rock to rainfall, can cause elevated concentrations of metals in drainage water. Commercial forestry can cause an increase in suspended mineral solids in the water after areas have been cleared by cutting and logging. Intensive agriculture can contribute fertilizers and pesticides applied to arable crops, and strong organic liquids can be produced from silage manufacture and from the rearing of poultry and cattle. Weaker organic wastes, but in much greater volumes, can be discharged in the form of untreated or treated sewage (Birge *et al.,* 1977) [2]. As well as these changes in water chemistry, the physical habitat of the watercourse can be altered by man's activities. Rivers can be canalized for transport and flood prevention;

dams are constructed for water storage and power generation; diversions are made to accommodate other land usage projects. These changes can affect the flow and depth of the water. Also, changes in the drainage characteristics of the watershed can lead to a more rapid run-off, leading to greater fluctuations in the river flow rates (Lloyd, 1992) [9].

Mention should be made here of the diurnal and seasonal fluctuations of temperature. These are less susceptible to man's intervention, although changes in water depth and flow rates can affect the rates of diurnal warming and cooling. Obviously, heated discharges from power stations and industry can have a considerable effect on the aquatic biota (Birge *et al.,* 1977).

Apart from affecting the suspended mineral solids content of the water, these physical changes *per se* have little effect on the chemical quality of the water. However, they can affect the natural biological community that can live in equilibrium with the particular chemical and physical characteristics of their environment, and changes in the composition of the aquatic biota can also affect the water quality (Birge *et al.,* 1977) [2].

The most obvious effects are those caused by increased plant growth. Clearly, rooted plants will provide shade and cover for a wide range of aquatic species. But all green plants, including algae, photosynthesize during their period of growth in the daylight hours, and respire at all times. During daylight, plants absorb carbon dioxide from the water and this is converted to carbohydrate; dissolved oxygen is produced and this is released into the water (Lloyd, 1992).

When plant growth is active, these photosynthetic processes are more pronounced than that of respiration, in which dissolved oxygen is absorbed and carbon dioxide is released. As a result, the pH of the water is raised during the day as the amount of carbonic acid is reduced; the dissolved oxygen concentrations are also raised during the day. At night, the level of carbon dioxide increases, leading to a lower pH, and the level of dissolved oxygen falls (Lloyd, 1992) [9].

These fluctuations of dissolved gas concentrations are natural and normal, but they can be accentuated by, for example, an increase in the plant nutrients as a result of fertilizer run-off from arable land which can cause excessive weed growth. Also, the discharge of readily biodegradable organic wastes can increase the amplitude of the fluctuations (Birge *et al.,* 1977) [2].

Therefore, the quality of surface water is never constant; it is constantly changing in response to daily, seasonal and climatic rhythms. Organisms, including fish, in a particular water-body can adapt to these natural fluctuations of water quality (including temperature) as they occur (Bohl, 1989) [3].

2. Causes and Effects of Pollution on Fish

2.1 Harmful variations in natural water quality characteristics

2.1.1 Water temperature

Fish are poikilothermic animals, that is, their body temperature is the same as, or 0.5 to 1 °C above or below, the temperature of the water in which they live. The metabolic rate of fish is closely correlated to the water temperature: the higher the water temperature (i.e. the closer to the optimum values within the normal range), the greater the metabolism. This generalization applies particularly to warm-water fish. Cold-water fish, e.g. salmonids, whitefish, or burbot, have a different type of metabolism: their metabolic rate can continue at comparatively low temperatures, whereas at high water temperatures, usually above 20 °C, they become less active and consume less food. Water temperature also has a great influence on the initiation and course of a number of fish diseases. The immune system of the majority of fish species has an optimum performance at water temperatures of about 15 °C (Bohl, 1989) [3].

In their natural environment, fish can easily tolerate the seasonal changes in temperature, e.g. a decrease to 0 °C in winter and increase to 20-30 °C (depending on species) in summer under Central European conditions. However, these changes should not be abrupt; temperature shock occurs if the fish are put into a new environment where the temperature is 12 °C colder or warmer (8 °C in the case of salmonids) than the original water. Under these conditions fish may die, showing symptoms of paralysis of the respiratory and cardiac muscles. With young fry, problems may arise even where the difference in temperature is as low as 1.5-3 °C. If fish are fed and then abruptly transferred to water colder by 8 °C or more, their digestive processes will slow down or stop. The food will remain undigested or half-digested in the digestive tract and the gases produced can cause the fish to become bloated, lose balance, and finally die. If carp are given a high-nitrogen feed (e.g. natural food or high-protein pellets), abrupt transfer too much colder water will considerably increase the level of ammonia nitrogen in the blood serum because the decrease in metabolic rate reduces the diffusion of ammonia from the gills. This can lead to ammonia autointoxication and death (Bohl, 1989) [3].

2.1.2 Water pH

The optimal pH range for fish is from 6.5 to 8.5. Alkaline pH values above 9.2 and acidity below 4.8 can damage and kill salmonids (e.g. brown and rainbow trout); and pH values above 10.8 and below 5.0 may be rapidly fatal to cyprinids (especially carp and tench). Thus salmonids, in comparison with cyprinids, are more vulnerable to high pH and more resistant to low pH. The American char is especially resistant to acid waters and can tolerate pH levels as low as 4.5-5.0. (Hrbáček *et al.,* 1974) [4].

Low water pH most frequently occurs during the spring, especially when acidified snow melts, and in water draining peat bogs. High alkaline pH can occur in eutrophic reservoirs (ponds) where the green plants (the blue-green algae, green algae and higher aquatic plants) take up considerable amounts of CO_2 during the day for intensive photosynthetic activity. This affects the buffering capacity of the water and the pH can rise to 9.0-10.0 or even higher if bicarbonate is adsorbed from waters of medium alkalinity. Water pH can also be changed when mineral acids and hydroxides, or other acidic or alkaline substances, are discharged or leach into water courses, ponds or lakes (Hrbáček *et al.,* 1974) [4].

As a defence against the effect of a low or high water pH, fish can produce an increased amount of mucus on the skin and on the inner side of the gill covers. Extremely high or low pH values cause damage to fish tissues, especially the gills, and hemorrhages may occur in the gills and on the lower part of the body. Excess amounts of mucus, often containing blood, can be seen in post mortem examination of the skin and gills. The mucus is dull-colored and watery (Hrbáček *et al.,* 1974) [4].

2.1.3 Dissolved oxygen

Different fish species have different requirements for the concentration of oxygen dissolved in water. Salmonids have the more demanding requirements for oxygen in the water; their optimum concentration is 8–10 mg per litre, and if the level declines below 3 mg per litre they begin to show signs of

suffocation. Cyprinids are less demanding; they can thrive in water containing 6-8 mg per litre and show signs of suffocation only, when the oxygen concentration falls to 1.5-2.0 mg per litre (Hrbáček *et al.,* 1974) [4].

The oxygen requirements of fish also depend on a number of other factors, including the temperature, pH, and CO_2 level of the water, and the metabolic rate of the fish. The major criteria for the oxygen requirement of fish include temperature, and the average individual weight and the total weight of fish per unit volume of water. Oxygen requirements increase at a higher temperature (e.g. an increase in water temperature from 10 to 20 °C at least doubles the oxygen demand); a higher total weight of fish per unit volume of water can lead to increased activity and thus increased respiration as a result of overcrowding (Khan and Thulin 1991) [5].

Oxygen requirements per unit weight of fish significantly decline with increasing individual weight. In carp this reduction may be expressed by the following ratios: yearling = 1, two-year-old carp = 0.5-0.7, marketable carp = 0.3-0.4. Significant differences in oxygen demand are also found for different species. Using a coefficient of 1 to express the oxygen requirement of common carp, the comparative values for some other species are as follows: trout 2.83, peeled 2.20, pike perch 1.76, roach 1.51, sturgeon 1.50, perch 1.46, bream 1.41, pike 1.10, eel 0.83, and tench 0.83 (Khan and Thulin 1991) [5].

As stated earlier, the factor most frequently responsible for a significant reduction in the oxygen concentration of the water (oxygen deficiency) is pollution by biodegradable organic substances (including waste waters from agriculture, the food industry, and public sewage). These substances are decomposed by bacteria which use oxygen from the water for this process. A few chemicals may be oxidized in the absence of bacteria. The concentration of organic substances in water in terms of their capacity for taking oxygen from the water can be measured by means of the chemical oxygen demand (COD, which represents a theoretical maximum) and the biochemical oxygen demand within five days (BOD_5, which represents the potential for bacterial degradation). The upper limit of COD, as determined by the Kubela method, for the optimal range for cyprinids in pond or river waters, is 20–30 mg O_2 per litre and the corresponding BOD_5 limit for cyprinids is 8–15 mg O_2 per litre, depending on the intensity of the culture and the rates of reiteration. For salmonids the corresponding levels are up to 10 mg O_2 per litre for COD and up to 5 mg O_2 per litre for BOD (Khan and Thulin 1991) [5].

In winter, fish are commonly killed by suffocation in polluted storage ponds and in summer this often happens in polluted water courses with high temperatures and low flow rates. In severely eutrophicated ponds, oxygen deficiency often occurs during the summer early in morning as a result of the night time oxygen consumption by bacteria for the decomposition of organic substances and the respiration of aquatic plants. In heavily fertilized ponds (e.g. those used for the treatment of sewage) with a constant inflow of degradable organic substances, oxygen deficiency can also be caused by an excessive development of zooplankton; the zooplankton itself requires oxygen for respiration and, in addition, its feeding pressure reduces the phytoplankton population which produces oxygen during the day (Khan and Thulin 1991) [5].

Even in ponds where the oxygen levels have been satisfactory during the summer, when plant growth was vigorous, severe oxygen deficiencies can occur in the autumn when the plants begin to die and decompose. This deficiency can be more pronounced if the sky is heavily overcast during the day, so that the limited oxygen production by photosynthesis is further reduced. In these cases, the maximum oxygen deficiency occurs just before daybreak (Koskivaara *et al.,* 1991) [6].

2.1.4 Super saturation with dissolved gas

Super saturation with dissolved gas occurs when the pressure of the dissolved gas exceeds the atmospheric pressure. It occurs when water is equilibrated with air under pressure, e.g. at the bottom of a lake or reservoir, in ground water, or if air is drawn into a centrifugal water pump. It can also occur if cold air-equilibrated water is warmed up without re-equilibration to the higher temperature. A bottle containing such water will show either minute bubbles forming as a cloudy suspension which will clear from the bottom upwards, or larger bubbles forming on the glass wall. This is analogous to that seen in an opened bottle of carbonated drinking water. (Koskivaara *et al.,* 1991) [6].

If fish are exposed (at a lower atmospheric pressure) to such water, their blood equilibrates with the excess pressure in the water. Bubbles form in the blood and these can block the capillaries; in sub-acute cases the dorsal and caudal fin can be affected, and bubbles may be visible between the fin rays. The epidermal tissue distal to the occlusions then becomes necrotic and cases are known where the dorsal fins of trout have become completely eroded. In severe cases, death occurs rapidly as a result of blockage of the major arteries, and large bubbles are clearly seen between the rays of all the fins. A similar effect of gas bubbles forming in the blood can be experienced by deep-sea divers when they return to the surface (Koskivaara *et al.,* 1991) [6].

2.1.5 Ammonia

Ammonia pollution of water courses, ponds and lakes may be of organic origin (domestic sewage, agricultural wastes, or the reduction of nitrates and nitrites by bacteria in anoxic waters) or of inorganic origin (industrial effluents from gas works, coking plants and power generator stations). In water or in biological fluids, ammonia is present in a molecular (none dissociated) form (NH_3) and in the form of ammonia ion (dissociated) (NH_4^+). The ratio between these two forms depends on the pH and temperature of the water (Table 1). The cell walls of organisms are comparatively impermeable to the ammonia ion (NH_4^+), but molecular ammonia (NH_3) can readily diffuse across the tissue barriers where a concentration gradient exists, and is therefore the potentially toxic form to fish. Also, under normal conditions there is an acid-base balance at the water-tissue interface. If this balance is altered, the side on which the pH is lower will attract additional molecular ammonia. This explains how molecular ammonia passes from water through the epithelium of the gills to the blood and also how it passes from the blood to the tissues. Ammonia has a particular toxic effect on the brain; this is why nervous symptoms are so pronounced in cases of ammonia toxicity to fish (Koskivaara and Valtonen, 1992) [7].

To a lesser extent, the toxicity of ammonia is affected by the amount of free CO_2 in the water. This is because the diffusion of respiratory CO_2 at the gill surface reduces the pH of the water, thus reducing the proportion of none dissociated ammonia there. The extent of the reduction in pH depends on the amount of CO_2 already present in the water (Koskivaara and Valtonen, 1992) [7].

Non-dissociated ammonia is highly toxic to fish. The LC_{50} values, determined in acute toxicity tests, are in the range of 1.0 to 1.5 mg NH_3 per litre for cyprinid fish and 0.5 to 0.8 mg NH_3 per litre for salmonids. The maximum admissible

ammonia (NH_3) concentration is 0.05 mg per litre for cyprinids and 0.0125 mg per litre for salmonids (Koskivaara and Valtonen, 1992) [7].

It should be emphasized here that these standards apply to ammonia as a toxic substance. Other standards for total ammonia are applied to control eutrophication of waters and prevent excessive algal and plant growth that can cause physical problems and affect the oxygen balance. The first signs of ammonia toxicity include a slight restlessness, and increased respiration; the fish congregate close to the water surface. In later stages, cyprinids gasp for air, their restlessness increases with rapid movements and respiration becomes irregular; then follows a stage of intense activity. Finally, the fish react violently to outside stimuli; they lose their balance, leap out of the water, and their muscles twitch in spasms. Affected fish lie on their side and spasmodically open wide their mouths and gill opercula. Then follows a short period of apparent recovery. The fish return to normal swimming and appear slightly restless. This stage is then replaced by another period of high activity; the body surface becomes pale and the fish die (Koskivaara and Valtonen, 1992) [7].

The skin of ammonia poisoned fish is light in color, and covered with a thick or excessive layer of mucus. In some cases small hemorrhages occur, mainly at the base of the pectoral fins and in the anterior part of the ocular cavity. The gills are heavily congested and contain a considerable amount of mucus; fish exposed to high ammonia concentrations may have slight to severe bleeding of the gills. Intense mucus production can be observed on the inner side of the gill opercula, mainly at the posterior end. The organs inside the body cavity are congested and parenchymatous, and show dystrophic changes (Koskivaara and Valtonen, 1992) [7].

In recent years, considerable losses among farmed carp have been caused by the so-called toxic necrosis of the gills. The factors responsible for the occurence of this disease include ammonia poisoning in which the ammonia level in the blood is considerably increased. As stated earlier, ammonia is the final product of nitrogen metabolism in carp (as it is in other species) and most of it is excreted via the gills into the water. If the diffusion rate is reduced for some reason or another (high water pH, oxygen deficit, damaged gills etc.), the ammonia level in the blood will steadily rise, causing a condition known as autointoxication, which may lead to toxic gill necrosis in carp (Lewis and Morris, 1986) [8].

A very interesting case of autointoxication among carp yearlings (C_1) where extremely high ammonia N levels were found in the blood serum occurred after their transfer from a pond to well water in large aquarium tanks. Some of the fish caught and transferred during the morning exhibited typical symptoms of ammonia poisoning the following morning. These symptoms included considerable restlessness, increased respiration, leaping out of the water, uncoordinated activity, and tonic-colonic spasms of the muscles. The skin of the affected fish was light in color; the gills were heavily congested, dark red and showed edematous swellings (particularly severe on the edges of the gill filaments). It is known that ammonia toxicity is accompanied by an increase in the permeability of the fish epithelium to water, as measured by an increase in the flow of urine. If the kidneys cannot cope with the increased water influx, edema is likely to occur. An increased water influx may also occur if the skin or the mucus coating of the fish is damaged by handling and during transport. The histo pathological changes in the gills corresponded with what had been described for toxic necrosis of carp gills. The digestive tract of those fish with severe poisoning symptoms was filled with undigested food. On the other hand, fish that had cleared their gut (feces found on the bottom of the tank, the gut almost empty), were free from symptoms of toxic damage. The average blood serum level of ammonia N in the fish with symptoms of poisoning was 3054 (2400-3600) µg per 100 ml of serum, whereas in the fish free of such symptoms the ammonia level was 825 (750-900) µg per 100 ml of serum. In the affected fish the autointoxication, associated with the considerable increase in the blood serum ammonia N level, was probably due to the persistence and absorption of the gut contents (natural food and high-protein feed pellets) of the carp exposed to environmental stress (confinement and reduced oxygen level during transport, and water temperature reduced by about 5 °C) (Lewis and Morris, 1986) [8].

On the basis of this case of ammonia poisoning of carp, some other unexplained incidents of rapid death among fish may be ascribed to a similar cause. Such events may occur mainly in carp farms where there is an intensive feeding with a high-nitrogen diet, if the fish are also exposed to other stresses caused by e.g. an abrupt oxygen deficit, or sudden changes in water temperature (Lewis and Morris, 1986) [8].

2.1.6 Nitrites and nitrates

Nitrites as a rule are found together with nitrates and ammonia nitrogen in surface waters but their concentrations are usually low because of their instability. They are readily oxidized to nitrate or reduced to ammonia, both chemically and biochemically by bacteria. Nitrates are the final product of the aerobic decomposition of organic nitrogen compounds. They are present in low concentrations in all surface waters. There is almost no nitrate retention in soil, so it is readily leached to watercourses, ponds and lakes. The main sources of nitrate pollution of surface waters is the use of nitrogenous fertilizers and manures on arable land leading to diffuse inputs, and the discharge of sewage effluents from treatment works (Lewis and Morris, 1986) [8].

Nitrite can be associated with ammonia concentrations in the water. In normal aerobic conditions, ammonia is oxidized to nitrite and then to nitrate by two separate bacterial actions. If the second stage of oxidation is inhibited by bactericidal chemicals in the water, nitrite concentrations will increase. This may be important in small ponds or aquaria where water is reticulated through a purification filter; the ammonia-oxidizing bacteria need to become established for the filter to function, and they may be affected by the use of antibiotics to control fish diseases (Lloyd, 1992) [9].

The toxic action of nitrite on fish is incompletely known; it depends on a number of internal and external factors (such as fish species and age, and general water quality). The importance and role of these factors have been frequently studied and reviewed. Different authors often come to contradictory conclusions, and usually fail to offer a definitive explanation of either the mechanism of nitrite toxic action on fish or the modifying effects of different environmental factors (Lloyd, 1992) [9].

It is now clear that nitrite ions are taken up into the fish by the chloride cells of the gills. In the blood, nitrites become bound to hemoglobin, giving rise to methaemoglobin: this then reduces the oxygen transporting capacity of the blood. The increase in the amount of methaemoglobin can be seen as a brown color of the blood and gills. If the amount of methaemoglobin in the blood does not exceed 50% of the total hemoglobin, the fish usually survive. If the fish have more methaemoglobin in their blood (70-80%) they become torpid,

and with a further increase in the methaemoglobin level they lose their orientation and are unable to react to stimuli. Nevertheless, the fish may still be able to survive because the erythrocytes in their blood contain the enzyme reductase which can convert methaemoglobin to hemoglobin. This process can return the hemoglobin to its normal level within 24-48 hours, if the fish are put into nitrite-free water (Lloyd, 1992) [9].

Several authors have shown that nitrite toxicity to fish can be affected by certain water quality characteristics (e.g. Lewis and Morris, 1986) [8]. In this investigation, the 96h LC50 for rainbow trout ranged from 0.24 to 12.20 mg per litre, depending on the chloride content of the dilution water (in this case the chloride content ranged from 0.35 to 40.9 mg per litre). The effect of chloride on nitrite toxicity is so marked that the results of tests made without recording the chloride concentrations in the water cannot be compared with those of other tests.

Nitrite toxicity can be also influenced by bicarbonate, potassium, sodium, calcium and other ions, but their effect is not so great as that of chloride. Among these, potassium is the more significant, and that of sodium and calcium is less. These monovalent ions are also involved in the ionic fluxes across the gill epithelium and so directly or indirectly influence the uptake of nitrite. The pH value has also been considered as important for nitrite toxicity; pH and temperature control dissociation between NO_2 and none dissociated HNO_2 and it was believed that the uptake of nitrites into fish blood plasma depended on the diffusion of none dissociated HNO_2 across the gill epithelium. However, the results of later experiments refuted these theories and showed that within the acidity-alkalinity range encountered in natural waters the pH is of little importance in nitrite toxicity (Mattheis *et al.,* 1984) [10].

Another factor that influences nitrite toxicity is the dissolved oxygen concentration and water temperature. This is associated with the fact that fish need fully oxygenated water when the oxygen-carrying capacity of the blood is reduced by the formation of methaemoglobin, and the oxygen requirement of fish increases with temperature (Mattheis *et al.,* 1984) [10].

Long exposure to sub lethal concentrations of nitrites does not cause much damage to the fish. Concentrations corresponding to 20–40% of the minimum levels having a lethal action on the fish may slightly depress their growth but no serious damage has ever been recorded (Mattheis *et al.,* 1984) [10].

The toxicity of nitrates to fish is very low, and mortalities have only been recorded when concentrations have exceeded 1000 mg per litre; 80 mg per litre is considered to be the maximum admissible nitrate concentration for carp and 20 mg per litre for rainbow trout. In surface waters and in fish farms where the water contains ample oxygen with no danger of de nitrification (i.e. conversion of NO_3^- to NO_2^- and then to elementary nitrogen or N_2O and NO), it is not so necessary to monitor the concentration of nitrates. However, as with ammonia, water quality standards need to be set for nitrate to prevent eutrophication, and the excessive growth of algae and plants, which can have a secondary effect on fish (Mattheis *et al.,* 1984) [10].

2.1.7 Hydrogen sulphide (H_2S)

Hydrogen sulphide occurs in organically polluted waters from the decomposition of proteins. It is also present in industrial effluents including those from metallurgical and chemical works, paper pulp plants, and tanneries. It has a high to very high toxicity to fish; the lethal concentrations for different fish species range from 0.4 mg H_2S per litre (salmonids) to 4 mg per litre (crucian carp, tench and eel). The toxicity of H_2S decreases with increasing water pH, because of a reduction in the ratio of the none dissociated toxic H_2S to the less toxic HS ions). The concentration of none dissociated H_2S can be calculated from the measured total hydrogen sulphide ($HS + H_2S + S^{2-}$) concentration and the pH value of the water, using the formula:

$$H_2S = (HS^- + H_2S + S^2).p\frac{1}{10^{pH-7}+1}$$

Where p = activity coefficient depending on the ionic strength of water. For natural water it is about 0.92 (Pascoe and Cram, 1977) [11].

Hydrogen sulphide can be formed in decomposing rich organic mud, and escapes into the overlying water together with other gases (e.g. methane and carbon dioxide) formed by anaerobic degradation. In aerobic waters the H_2S is rapidly oxidized to sulphate; however, it is possible for fish living close to the surface of such muds to be exposed to hydrogen sulphide (Pascoe and Cram, 1977) [11].

2.1.8 Carbon dioxide

Carbon dioxide is dissolved in water in its molecular gaseous state; only 10 % is in the form of carbonic acid H_2CO_3. These two forms of carbon dioxide together constitute what is termed free CO_2. The ionic forms, i.e. fixed carbon dioxide, are represented by the bicarbonate and carbonate ions (HCO_3^- and CO_3^{2-}respectively). Their presence is important for the buffering capacity of the water. The amounts of CO_2 present in flowing surface waters are typically in the order of a few mg per litre, and seldom rise above 20 to 30 mg per litre. In stagnant surface waters the CO_2 levels are stratified because of photosynthetic assimilation by phytoplankton, the upper strata usually having less free CO_2 than the lower strata. If all the free CO_2 in the surface strata is used for photosynthesis, the pH of the water there may rise above 8.3, and in waters of moderate bicarbonate alkalinity to 10.0 and above during the daylight hours. Ground waters from limestone or chalk strata usually contain several tens of mg of free CO_2 per litre, and this may be important where well water is used for fish culture (Perevozchenko and Davydov, 1974) [12].

The toxic action of carbon dioxide is either direct or indirect. The indirect action of both free and bound CO_2 is exerted on fish through its influence on water pH, especially where, as described earlier, the values rise to toxic levels. Also, changes in pH affect the toxicity of those chemicals which exist in the dissociated and none dissociated forms of which only one is toxic, such as H_2S and ammonia (Perevozchenko and Davydov, 1974) [12].

A direct adverse effect occurs when there is an excess or absence of free CO_2. In waters of low oxygen content, such as where intensive biodegradation is taking place, or where fish are kept or transported in a high density, or when poorly aerated ground waters are used, free CO_2 may reach harmful levels. In such cases the diffusion of CO_2 from the fish blood into the respiratory water is reduced, the blood CO_2 raises and acidosis develops. If the rise in CO_2 concentration is relatively slow (e.g. over 1 day), fish can adapt to the acidosis by increasing the bicarbonate concentration of the blood. Adapted fish can then suffer from alkalinosis if returned to water of low CO_2 content (Perevozchenko and Davydov, 1974) [12].

In water of low O_2 and high CO_2, where gaseous exchange at the respiratory surface is limited, the fish increase their

ventilation rate, become restless, lose equilibrium, and may die. Twenty mg free CO_2 per litre is considered the maximum permissible concentration for trout (higher concentrations can cause kidney problems) and 25 mg free CO_2 per litre is the maximum for carp (if the acid capacity is 0.5 m mol per litre at a pH of up to 4.5). The sensitivity of fish to free carbon dioxide declines with increasing acid capacity of water (Perevozchenko and Davydov, 1974) [12].

However, the more frequent occurrence is a lack of free carbon dioxide in water. Carbon dioxide deficiency occurs when too much free CO_2 is utilized for photosynthetic activity by the phytoplankton, or when the water used in thermal power plants is artificially softened or when water is aerated more vigorously than necessary with CO_2 free air. Free carbon dioxide concentrations below 1 mg per litre affect the acid-base balance in the fish blood and tissues, and cause alkalosis. A lack of free carbon dioxide is particularly harmful to cyprinid fry when they pass from endogenous to exogenous nutrition. Cyprinid fry respire through their body surface and are unable to regulate their acid-base balance by gill respiration. A low partial pressure of free CO_2 in water is conductive to a high CO_2 diffusion rate from the body, leading to alkalosis and finally to death. If the fry of cyprinids suffer from free CO_2 deficiency, they gather close to the water surface and show symptoms of suffocation even though the concentration of oxygen in the water is adequate (Taege, 1982) [18].

2.2 Chemicals in water as a result of man's activities

This section briefly describes the toxicity to fish of chemicals that are likely to occur in surface waters. Where possible, the acute toxic concentrations are given to provide information useful for cases of sporadic discharges where high concentrations may exist for a short time, and maximum admissible concentrations which are relevant for low-level continuous discharges. Clinical and patho-anatomic effects are also described. For more detailed information standard reference works should be consulted (Pitter, 1974) [13].

2.2.1 Chlorine

Active chlorine can be discharged into water courses, lakes and ponds in effluents from textile and paper plants. Chlorine and compounds that release active chlorine into water are used as disinfectants in both public health and veterinary medicine. Thus, chlorine can be discharged in water from public swimming pools and from sterilizing procedures for equipment in dairy farms (Pitter, 1974) [13].

Chlorinated lime is used for a total disinfection of pond bottoms (application rate of 600 kg per ha), fish storage ponds and other facilities for fish culture and transport. If fish suffer from a gill disease, a recommended remedial procedure is to spread chlorinated lime on the surface of the pond at a rate of 10-15 kg per ha (if the average depth of the pond is 1 m). However, over dosage or improper handling of chlorine or chlorine-releasing compounds can damage or kill fish. Marketed fish may also be harmed by chlorine if retailers keep them in tanks supplied with chlorinated tap water which contains 0.05 to 0.3 mg active chlorine per litre. Higher, rapidly lethal, concentrations can occur if the water supply works abstracts water containing a high content of organic matter; excessive chlorine then has to be used to disinfect the water. If chlorinated water from a public supply has to be used, it should be passed through an activated charcoal filter to remove the chlorine; for small-scale use, small amounts (c. 10 mg per litre) of sodium thio sulphate can be added to the water to react with the chlorine. Low concentrations of chlorine can be naturally absorbed by organic matter in the water and in sediments (Pitter, 1974) [13].

Active chlorine is very toxic to fish. Its toxicity largely depends on water temperature: for example, an active chlorine concentration of 3.5 mg per litre has a sub lethal effect on carp at a water temperature of 3-7 °C but when exposed to the same concentration at a temperature of 15–20°C they die in 1 to 2 hours. In general, a prolonged exposure to active chlorine concentrations of 0.04 to 0.2 mg per litre is considered to be toxic to the majority of fish species (Taege, 1982) [18].

Active chlorine may affect specific parts of the fish (e.g. the skin and gills) or the whole body (i.e. when chlorine is absorbed into the blood). The systemic effect manifests itself mainly as nervous disorders. The clinical symptoms of chlorine intoxication include a considerable restlessness, leaping out of the water, muscle tetanus, lying on one side, and spasmic movement of the mouth, fins and tail. The buccal spasms hinder respiration, so that the fish suffocate, and ultimately die. The skin and gills of the poisoned fish are covered with a thick layer of mucus and if the concentration of active chlorine is very high the gills become congested and can hemorrhage. The body surface of such fish becomes pale and the margins of the gill filaments and fins are covered with a grey-white coating. Histopathologically, there is a marked dystrophy and necrobiosis leading to necrosis, with desquamation of the gill respiratory epithelium and of the epidermis of the skin (Taege, 1982) [18].

2.2.2 Cyanides

Cyanides do not occur naturally in waters; they can be discharged in various industrial effluents, particularly from metal plating works and from the thermal processing of coal (e.g. for town gas production). Cyanides may be present in water either as simple compounds (none dissociated HCN, simple CN ions) or as complex compounds (e.g. complexes with iron, cobalt, nickel and other metals). Simple cyanides are very toxic or extremely toxic to fish species; lethal concentrations for the majority of species are in the range of 0.03 to 0.5 mg per litre. Cyanide toxicity is affected by the pH of the water; if the pH is low the proportion of none dissociated HCN increases and so does the toxicity. Cyanide toxicity is also markedly enhanced by an increase in water temperature and a decrease in the concentration of dissolved oxygen in the water (Pitter, 1974) [13].

With complex cyanides, the toxicity varies according to their ability to dissociate into metal and HCN. For example, the complex iron cyanides which do not dissociate are of low to very low toxicity to fish but the complex cyanides of zinc, cadmium, copper and mercury which do are highly toxic. The concentrations of different cyanide compounds proposed as maximum admissible levels for fish culture are in the range of 0.002 to 0.02 mg per litre (Taege, 1982) [18].

2.2.3 Divalent metals and their salts

Trace quantities of metals present in waters may be of natural origin. If waters are polluted with metals at greater concentrations, the source may be traced back to ore mining and processing, to smelting plants, rolling mills plants for the surface treatment of metals, film, textile and leather industries and other sources. Atmospheric precipitation can wash out metals in dust and aerosols generated by the burning of fossil fuels, by the exhaust gases of motor vehicles, and from other sources (Schreckenbach, 1982) [14].

The mechanism of the toxic action of metals on fish is varied.

Most of the metals have a great affinity for amino acids and the SH groups of proteins: as such, they act as enzyme poisons. The toxicity of metals to fish is significantly affected by the form in which they occur in water. The ionic forms of metals or simple inorganic compounds are more toxic than complex inorganic or organic compounds. The toxic action of metals is particularly pronounced in the early stages of development of the fish (Schreckenbach, 1982) [14].

Another potentially harmful property of many metals is their ability to accumulate in the sediments and in the aquatic flora and fauna (bioaccumulation). This property is quantitatively described by the accumulation coefficient (concentration in substrate/ concentration in water) and such values can range from several hundred to many thousands; mercury, selenium and cadmium have a particularly high bioaccumulation capacity. Hence, the concentration of these metals in water does not provide a true indication of the total pollution of the aquatic medium; it is better to use the content of metals in the sediments, and especially also in the bodies of predatory fish which are the final link in the food chain, as an indicator (Schreckenbach, 1982) [14].

The metals found to be of highest importance to fisheries in practice include aluminum, chromium, iron, nickel, copper, zinc, arsenic, cadmium, mercury and lead.

2.2.4 Polychlorinated biphenyls (PCBs)

Polychlorinated biphenyls are recognized as very important environmental pollutants. PCBs are among the most environmentally persistent of organic compounds; although their solubility in water is very low, they are readily soluble in nonpolar solvents and can accumulate in fats. Mixtures of a large number of PCBs isomers are used in the heavy electrical equipment industry (e.g. in power capacitors and high-voltage transformers), mechanical engineering (e.g. as inflammable liquids for heat transfer, in hydraulic fluids and in lubricants for compressors) and in the chemical industry (e.g. the production of synthetic varnishes, dyestuffs and plastics). The world-wide trade names of PCBs include Delor (Czechoslovakia), Aroclor (USA), Clophen (Germany), Kaneclor (Japan), and Savol and Sovtol (USSR). In response to a growing concern about rising levels of PCBs in the environment from diffuse sources, their accumulation in biota, and uncertainty about their toxic effects, the production of polychlorinated biphenyls was restricted in 1971, and successive controls placed on its use and disposal. The main concern is that, once in the natural environment, they cannot be recovered or removed (Schreckenbach, 1982) [14].

PCBs present a very difficult eco toxicological problem; there are 209 individual PCBs, each one with different toxicological properties. Toxicity tests are carried out on commercial formulations which are identified by the extent to which they are chlorinated, and not by the specific PCBs that they contain. This makes it difficult to assess their toxicity in the environment, because differential uptake of the individual compounds leads to a different ratio being found in organisms when compared to that in the tested formulations. Therefore, any assessment of the toxicity of PCBs can be made only in general terms on the basis of tests with commonly used formulations (Schreckenbach, 1982) [14].

Of the various toxic actions of PCBs reported, they have been found to adversely affect the enzyme systems within the micro somal fraction of the liver. If fish are exposed for a long time to low sub lethal PCBs levels, the compounds accumulate in the body and can cause, mainly in the fry, deformities in the skeleton, damage to the skin and fins (the fins disintegrate), to the parenchymatous organs (mainly in the liver where hypertrophy, local dystrophy, and necrobiotic to necrotic changes can occur), and to the gonads. These effects can cause a subsequent mortality during hatching, high mortality of early fry and an increased occurrence of different deformities among the survivors (Svobodová and Faina, 1984) [15].

The maximum admissible PCBs concentrations in water range from 1.10^{-6} to 5.10^{-6} mg per litre for salmonids and from 2.10^{-6} to 1.10^{-5} mg per litre for cyprinids (Mattheis *et al*., 1984) [10]. Lower admissible concentrations are recommended during hatching and rearing of the early stages of the fry. However, analytical measurement of these concentrations in solution may be difficult; also, a significant proportion of the uptake of PCBs will be from the food. Analysis of fish tissue will give an indication of the degree of exposure, but the concentrations found must be correlated with the tissue fat content. Where significant amounts occur, the analysis should identify and quantify a number of key individual PCBs for an expert evaluation of the potential hazard (Svobodová and Faina, 1984) [15].

2.2.5 Surfactants

Surfactants are compounds which, by lowering the surface tension of water, can facilitate the formation of emulsions with otherwise immiscible liquids such as oils and fat. They are widely used domestically and in industry. In recent years, the traditional soaps have been replaced by detergents that contain synthetic surfactants and other ingredients; for domestic washing of garments, these may contain water softeners, optical brighteners, and perfumes (Svobodová and Faina, 1984) [15].

Surfactants are either ionic (liable to electrolytic dissociation) or nonionic (none dissociating in water). Ionic surfactants are subdivided into anionic (dissociating to a surface active anion and an inactive cation), cationic (dissociating to a surface active cation and an inactive anion), and ampholytic (assuming either anionic or cationic properties, depending on ambient conditions). The anionic surfactants are those most widely used in industry (Pascoe and Cram, 1977) [11].

Because of the large number of synthetic surfactants in production, it is not surprising that they span a wide range of chemical toxic actions for aquatic organisms. However, they do have a common physico-chemical effect in that they can damage the lipid components of cell membranes. Because the surface tension of the ambient water is decreased, the lipids are less water repellant and this leads to hydration and enlargement of the cell volume. At low surfactant concentrations this enlargement is reversible. Higher concentration can cause a suppression of metabolic processes in the cells. Long-term exposure may damage the cells which then become necrotic in the later stages. These changes result mainly in an impairment of the gill respiratory epithelium. In addition, the exposure of fish to some surfactants can cause changes in the activity of respiratory enzymes, especially cytochrome oxidase. Surfactants can also damage the protective layer of mucus on the skin; the layer loosens and the resistance of the fish to infection decreases. Sub lethal surfactant concentrations can also damage eggs and sperm (Pascoe and Cram, 1977) [11].

During embryonic and larval development, the sensitivity of fish to surfactants is sometimes greater by an order of magnitude in comparison with the juvenile and adult stages. Of the abiotic factors, the molecular structure of the surfactant and the physico-chemical properties of water exert the greatest influence on their toxicity. The results of investigations into

the relationship between toxicity and molecular structure indicate, for example with linear alkyl benzene sulphonates, that the toxicity to fish is markedly increased with the length of the molecular chain. A similar correlation between toxicity and chain length was observed with other surfactants. Among the physico-chemical properties of water, increasing calcium and magnesium concentrations have the greatest effect on reducing surfactant toxicity and some influence is also exerted by the pH. This may be important where surfactants are incorporated into a detergent containing water softening chemicals (e.g. polyphosphates). Where both cationic and anionic surfactants are present in waste waters their toxicity is much reduced, due to the formation of insoluble complex (Pascoe and Cram, 1977) [11].

The acute toxicity of surfactants varies considerably with the species of fish. Nevertheless, these compounds and the detergents that contain them are highly toxic to fish in the majority of cases, the 48-hour LC_{50} ranging between 1 and 10 mg per litre. A small proportion of surfactants can be classified as having a medium toxicity (48-hour LC_{50} between 10 and 100 mg per litre) and a few have a very low toxicity (48-hour LC_{50} to 10 000 mg per litre). For the majority of surfactants, no significant differences in their toxicity to fish were recorded between the anionic, cationic and nonionic groups (Svobodová and Vykusová, 1991) [16].

As stated above, surfactants can cause damage to the gill respiration epithelium (e.g. enlargement and vacuolation of the cells with dystrophic to necrobiotic changes). Therefore, the clinical signs of poisoning include respiratory disorders (increased respiration rate, and cyprinids gasp for air at the water surface) and later by inactivity. The characteristics in the patho-anatomic examination are an increased amount of mucus on the skin and in the gills, and congestion to edematous swelling of the gill apparatus. The mucus is easily removed from the body surface and gills (Svobodová and Vykusová, 1991) [16].

2.2.6 Pesticides

In recent years, the number of pesticides available and the quantity used has considerably increased. The term "pesticide" is used to include insecticides, acaricides, herbicides, fungicides and algicides, indeed any chemical which is used to control an unwanted organism (except bacteria), even rotenone which is used to kill unwanted fish. Pesticides are chemicals which have a specific toxic action to which the pest species is particularly sensitive. The chemical is then applied at a concentration which kills the pest but does not affect a wide range of non-target organisms. The ideal pesticide is a chemical which is extremely pest-specific; for the pesticide user it should also be persistent in order to avoid the need for repeated applications. However, on environmental grounds, pesticides should be non-persistent to avoid concentrations building up in environmental compartments and causing unsuspected side-effects. For example, the insecticide DDT is very persistent and thus can build up in food chains to ultimately affect the egg-shell thickness of birds of prey (Svobodová *et al.,* 1987) [17].

Because pesticides are designed and used to kill living organisms, and because of the possibility of unsuspected side effects, it is tempting to implicate them in any incident of fish poisoning where there is no other obvious cause of the damage. There are many cases, therefore, where pesticides have been assumed to be the cause of damage but where the real cause was some other factor (Svobodová *et al.,* 1987) [17].

Some cases of pesticide poisoning of fish are obvious; accidental discharges from road accidents, factory disasters, over spraying of water, or careless disposal of unwanted spray and pesticide containers, can be clearly identified as causes of mortality, especially if the concentrations measured or calculated in the water exceed the 96 hour LC_{50} by a significant margin. Less easily identified are cases of long-term leaching of persistent pesticides from fields and forests. Besides these acute and chronic direct effects, an indirect action may be important. Inexpert application of aquatic herbicides or algicides to the water, or the accidental contamination of surface waters with these chemicals, may kill excessive quantities of aquatic plants and algae. The rapid decomposition of this organic matter forms a considerable dissolved oxygen demand on the water. This will lead to an oxygen deficit and the fish may die of suffocation (Svobodová *et al.,* 1987) [17].

Another potentially serious indirect consequence of pesticide contamination of the aquatic biota is the reduction or complete destruction of the natural food supply of the fish. Many of the organisms on which the fish feed are much more sensitive, particularly to insecticides, than the fish themselves. For example, the LC_{50} for the organo-phosphorus insecticide formulation "Soldep" (active ingredient 25% trichlorphon) for common carp is 545 mg per litre of water whereas for *Daphnia magna* it is 0.0002 to 0.001 mg per litre (Svobodová *et al.,* 1987) [17].

When a pesticide enters the aquatic environment, the active ingredient may undergo chemical and biological degradation. In some cases the degradation products may be more toxic to fish than the original active ingredient. For example, parathion is biodegraded to form paradoxes, which is a more toxic compound; similarly, trichlorphon is degraded to form the more toxic compound dichlorvos. It follows that the absence of a specific active ingredient in water cannot guarantee that harmful degradation products are not present (Tamiru Alemayehu, 2000) [19].

Some herbicides are used in fish culture and water management to kill unwanted aquatic plants (e.g. Gramoxone S, Reglone). Trichlorphon based organo-phosphorus insecticides, e.g. Soldep, Masoten, Neguvon, etc. are used to reduce the larger *Daphnia* in the zooplankton to prevent an oxygen deficit in the pond, to kill predatory cyclopids before stocking the pond with fish at the sac fry stage, to control parasites that infest cyprinids, and for other management purposes. Pesticides based on copper oxychloride may be used to control fish parasites, including the control of gastropod intermediate hosts, and to kill excessive growths of algae (Tamiru Alemayehu, 2000) [19].

However, in the majority of cases pesticides have the potential to cause damage to fish. The most toxic pesticides are those based on chloro hydrocarbons (e.g. DDT, dieldrin), organo-phosphorus compounds, carbamates and thiocarbamates, carboxylic acid derivatives, substituted urea, triazines and diazines, synthetic pyrethroids, and metallic compounds.

2.2.7 Oils and refined products

Oils and refined products have been responsible for many of the recently recorded pollution incidents in surface and underground waters. Between 1970 to 1990 these substances were responsible for the majority of water pollution accidents recorded on a worldwide basis. These accidents were not associated with problems in sewage treatment plants; most of them were due to careless storage and handling of oil, transport accidents, and defective equipment, all of which can be ascribed either directly or indirectly to human error

(Telford, 1998) [20].

However, oils and refined products can also be discharged into the aquatic environment with industrial effluents. The petrochemical industry is mainly responsible for such effluents; other important sources of pollution include the engineering and metallurgical industry and car and truck repair and service stations. Most of these sources have discharged polluting effluents for many years. To some extent, the large number of reported oil-related pollution incidents is due to the very visible surface film that is formed; it therefore needs no chemical analysis for its detection. Even very small discharges can produce a large area of "sheen" in which the thickness of the oil is about 1 micron. For this reason, few discharges of oil go unnoticed. The harmful effects of such discharges depend on the physical effects of the surface film, and on the transfer of water soluble products into the water (Telford, 1998) [20].

However, few of the constituent of oil and refined oil products will readily dissolve in water. There are also large differences between oil and its different products as to their toxicity to fish; most of them have 48h LC50 values within the range of 0.5 to 200 mg per litre. The toxicity varies according to the chemical composition of the different products, with the water solubility of the different petroleum hydrocarbons, and with the degree of emulsification of insoluble components in the water. It is generally agreed that the lighter oil fractions (including kerosene, petrol, benzene, toluene and xylene) are much more toxic to fish than the heavy fractions (heavy paraffin's and tars). There are also differences in the sensitivity to oils and refined products between different fish species. The fry of predatory fishes (especially pikeperch and trout) show the greatest sensitivity to refined products (Telford, 1998) [20].

When oils are discharged to rivers or ponds they spread on the surface, thus reducing (especially in stagnant waters) the transfer of oxygen from the air to water. In cases of pollution of flowing turbulent waters the oil does not from an intact layer on the water surface but becomes dispersed as droplets into the water. In such cases, the gills of fish can become mechanically contaminated and their respiratory capacity reduced. Oil products may contain various highly toxic substances, such as benzene, toluene and xylene which are to some extent soluble in water; these penetrate into the fish and can have a direct toxic effect. These toxic components include the naphthenic acids which are acute nerve poisons and are able to kill fish at concentrations as low as 0.03 to 0.1 mg per litre (Telford, 1998) [20].

2.2.8 Dyes

Chemical dyestuffs have also been attracting the attention of toxicologists in recent years. These can be present in the effluents from textile production, food processing and paper mills. Although these colored effluents are, like oils, very conspicuous even at very great dilutions they seldom cause severe damage to the fish. The toxicity of dyes depends on the physico-chemical composition of the water; in water containing considerable amounts of organic matter the dyes are bound to these substances and their toxicity is decreased (Tenalem Ayenew, 1998) [21].

The mechanism of toxic action of effluents containing dyestuffs on fish is not direct in the majority of cases. If the water is heavily polluted with colored organic waste, the increase in the organic content alone can lead to an oxygen deficit. Other dyes may increase or decrease the water pH. Some, e.g. aniline, can act as methaemoglobin poisons and as carcinogenic substances.

There is a considerable variation in the acute toxicity of different dyestuffs to fish. Most of the dyes rank among those substances of low to very low toxicity to fish (48h LC_{50}s in the range of 100 to 10 000 mg per litre). This group includes coloring agents used in the food industry and selected organic dyestuffs. Another group, including e.g. acriflavine, rhodamine and also aniline and to a lesser extent methylene blue, are substances of medium toxicity to fish, with 48h LC_{50}s in the range of 10 to 100 mg per litre. The group of dyes of very high toxicity to fish includes,

2.2.9 Phytoplankton toxins

As described earlier in this publication increasing eutrophication of surface waters can cause a massive development of phytoplankton and higher aquatic plants (Wood, 1974) [24]. This bloom can cause the water pH to rise to levels above 10, and its collapse and subsequent decomposition together with other decaying organic matter can cause an oxygen deficit. Further, some algal species produce substances (toxins) that may affect not only fish but also domestic animals and man if the water is ingested. These species include, in particular, the blue green algae of the genera *Microcystis, Aphanizomenon* and *Anabaena*. An endotoxin, with the properties of cyclic polypeptides, has been isolated from the alga *Microcystis aeruginosa* (Tenalem Ayenew, 1998) [21].

In exposed fish, the action of these blue-green algal toxins is to increase thiaminase activity and reduce thiamine content in organs and tissues; this leads to a vitamin B_1 deficiency. The toxins are released into the water during the period of algal bloom particularly when the cells die and decompose. These toxins can enter the fish through the gills and body surface; some may also be ingested with food. The clinical symptoms of poisoning include damage to the central nervous system. Initially, there is increased activity and respiration, followed by uncoordinated movements; finally the fish lie flat on the bottom and die. The major patho-anatomic signs include hemorrhages on the skin and gills and in the internal organs (Tenalem Ayenew, 1998) [21].

Some phytoplankton species have been found to produce hydroxylamine as a by-product of their metabolism (Wood, 1974) [24]. The occurrence of this hydroxylamine in the heavily eutrophicated waters of some ponds is also accompanied by a high concentration of organic substances; these reduce the oxide reduction potential of the environment, thus allowing the hydroxylamine to accumulate. For this reason, the highest hydroxylamine concentrations are usually recorded in surface waters during the periods when a bloom of blue-green algae is decomposing, and under these conditions, the concentrations may reach toxic levels for a short time. Hydroxylamine is highly toxic to fish, its LC_{50} for acute exposure being less than 10 mg per litre (in sensitive fishes it may be as low as about 1 mg per litre). The toxic action of this substance includes severe methaemoglobinaemia and damage to the central nervous system. Although there are few cases where damage to fish has been attributable to the action of phytoplankton toxins, this possible source of pollution should not be underrated, especially in warm regions (Tenalem Ayenew, 1998) [21].

3. Control of Water Quality

Everyone who manufactures, handles, or uses substances that can pollute the environment should regularly check their equipment and take the appropriate measures to prevent accidents; this is a moral obligation, which is also contained in the legislation of many countries. A strict discipline and a

proper responsibility exercised by all those who are involved in every stage from manufacture to disposal provides the most effective, the most readily available and the cheapest means of pollution prevention (Wood, 1974) [24].

The main method for preventing the chronic pollution of surface water is the installation of treatment plants, as are used for domestic sewage and for industrial effluents. In principle, all waste waters must be treated before they are discharged into the aquatic environment. These treatment processes may be simple; for example, the so-called biological oxidation ponds are used to intercept and biodegrade the organic wastes derived from agricultural production (livestock fattening and rearing facilities, including those for waterfowl) and from the food industries (slaughterhouses, poultry processing plants, dairy products, etc.). To a lesser extent these oxidation ponds may be used to intercept and degrade the sewage from residential areas, so long as such waste waters are not polluted with petroleum products, PCB, pesticides and other dangerous chemicals that are resistant to degradation (Wedemeyer and McLeay, 1981) [23].

However, oxidation ponds need to be extensive because of the large surface area required to allow sufficient oxygen to diffuse into the water. The amount of land required can be reduced by increasing the water surface area either by the use of stones or other media in trickling filters or by artificial aeration (the activated sludge process), both widely used in sewage treatment works. Substances that are not degraded by biological oxidation should be removed from waste waters by specific processes (Wedemeyer and McLeay, 1981) [23].

At industrial sites, especially at those factories where there is a danger of leakage of oils and refined products or other very toxic substances into drains that discharge directly to surface waters, it may be necessary to build special leak-proof pits or containment walls to protect the nearby aquatic environment from contamination (Wedemeyer and McLeay, 1981) [23].

Apart from these direct discharges to water, considerable attention should be paid to some of the technological processes used in agriculture, in particular the spray application of chemicals, including fertilizers and pesticides, onto fields. Any direct drift of the chemicals to water areas during their application should be avoided and in particular precautions should be taken to prevent the subsequent leaching of the chemicals by rainfall into rivers and ponds. Such precautions are incorporated in the regulations set to control such applications (no chemical spraying in wet or windy weather, or 24 hours before rain is expected etc.). They are also reflected in some principles of land management; the use of grassy strips around water reservoirs, the use of appropriate crops (e.g. those not needing excessive pesticide applications) and the proper tillage of sprayed fields. However, the improper disposal of surplus or unused pesticides, or the careless disposal of containers, is probably responsible for more pollution incidents than the proper use of pesticides on land (Vámos and Szöllözy, 1974) [22].

It is required by current regulations that any new chemical must be subjected to a programme of toxicological testing and evaluation before it can be placed on the market. In particular, its potential for biological degradability, acute and chronic toxicity and/or teratogenicity and mutagenicity should be established. For pesticides, priority should be given to chemicals with high target selectivity, a low active concentration, a low toxicity to non-target species and a rapid degradability. As stated earlier, the natural environment should not be allowed to become contaminated with toxic substances of low degradability (Vámos and Szöllözy, 1974) [22].

The toxicity of substances, formulations and effluents to fish depends, first of all, on their chemical properties (e.g. their composition, water solubility, and pH), then on the sensitivity of the fish exposed to these substances. Within species, salmonids are generally the most sensitive, and cyprinids tend to be somewhat more resistant; within life stages, older fish tend to be more resistant than younger fish. Also important is the general state of health of the fish including the state of feeding of early fry and, finally the influence on toxicity of the water quality characteristics of the aquatic medium (temperature, pH, dissolved oxygen concentration, hardness, etc.) (Vámos and Szöllözy, 1974) [22].

4. Conclusion

Substances which contaminate the aquatic environment can be harmful not only by their direct effects on the organisms there. It is well established that some diseases and developmental abnormalities may occur more frequently in fish living in a polluted environment. However, there is only a limited amount of information on this association, which is mainly related to experiences with farmed or cultured fish. The limited number of environmental stressors involved - low dissolved oxygen, extremes of temperature and pH, and ammonia - are probably due to the sitting of fish farms on relatively unpolluted waters. There is now a real need to study the interrelationships between the pollution of surface waters by a wide range of chemicals and diseases in natural fish populations, and the processes involved. This represents an important but at present under-developed field of scientific research and fisheries management.

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