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Physiological Mechanisms of Degradation of Fish Populations in Acidified Water Bodies

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Abstract—Disturbances in fish that result from water acidification and related factors are analyzed in the context of generalizations based on the results of experimental and field studies. It is shown that low pH, monomeric aluminum, and other metal ions have an adverse effect on physiological processes in fish and are a major cause of their death in acidified lakes and rivers. The vulnerability of fish to low water pH at different stages of their life cycle and mechanisms of delayed mortality are discussed.

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Key words: water acidification, hydrogen ions, aluminum, fish, physiological systems, stages of development.

The catastrophic reduction of fish stocks in thousands of lakes in Scandinavia (Henriksen et al., 1989; Muniz, 1991), Europe, and North America (Dillon et al., 1984) has occurred because of water acidification. Freshwater species, evolutionarily adapted to oligotrophic and ultrafresh water bodies, have proved to be very sensitive to acidification. The adverse effect of water acidification in rivers manifests itself in the decreasing abundance of anadromous species. In particular, the drastic reduction of Atlantic salmon stocks in Norwegian waters is explained by acidification of the rivers to which the fish migrate for spawning (Krogland et al., 2002). In Russia, water acidification is observed throughout the humid zone, being especially significant in the Kola Peninsula (Moiseenko, 2002), Karelia, and Vologda and Tver oblasts (Lozovik and Basova, 1994; Komov et al., 1997), the regions rich in valuable salmon and whitefish species. However, although the papers dealing with the phenomenon of water acidification are many, its effect on fish under natural conditions has been studied insufficiently.

In acidified water bodies, changes occur at all trophic levels of the ecosystem (Moiseenko, 2004), but fish populations (especially in the case of salmon) deteriorate mainly because of the direct impact of adverse environmental factors on fish themselves, which causes biochemical and physiological disturbances in the fish body. Knowledge of these disturbances is of great significance for predicting possible changes in fish populations under conditions of decreasing or increasing input of acid-forming substances and for developing the strategy and methods of the conservation and restoration of fish resources.

There are many experimental studies dealing with the effect of low pH on fish. However, fish in acidified waters are exposed to a number of adverse factors, and such conditions are difficult to simulate in the laboratory. Today, it is considered that hydrogen ions (pH), aluminum (Al^{3+}), and calcium deficiency are factors to which fish are especially sensitive (Wood and McDonald, 1987; Rosseland et al., 1990; Rosseland and Staurnes, 1994; Havas and Rosseland, 1995). In addition, increasing concentrations of many other metal ions can have a deleterious effect on fish (Moiseenko, 1999). In acidified waters, the calcium concentration is so low that it approaches the limit of tolerance even for salmonid species adapted to soft water. To understand the causes of physiological disturbances in fish living in acidified waters, it is necessary to have exhaustive information about hydrochemical parameters of the water body, on the one hand, and about specific physiological, biochemical, and cellular processes in the fish body, on the other hand.

Under natural conditions, it is difficult to identify the effect of a certain factor among a variety of interrelated adverse factors. During floods, for example, qualitatively different water masses mix up, which entails considerable fluctuations of water pH and the concentrations of calcium, organic matter, different forms of aluminum, toxic metals, etc. When the purpose is to reveal the effect of a particular factor on physiological mechanisms, an experimental approach is more relevant. Hence, to understand the influence of different abiotic factors affecting fish in acidified water bodies, it is necessary to combine field studies and laboratory experiments.

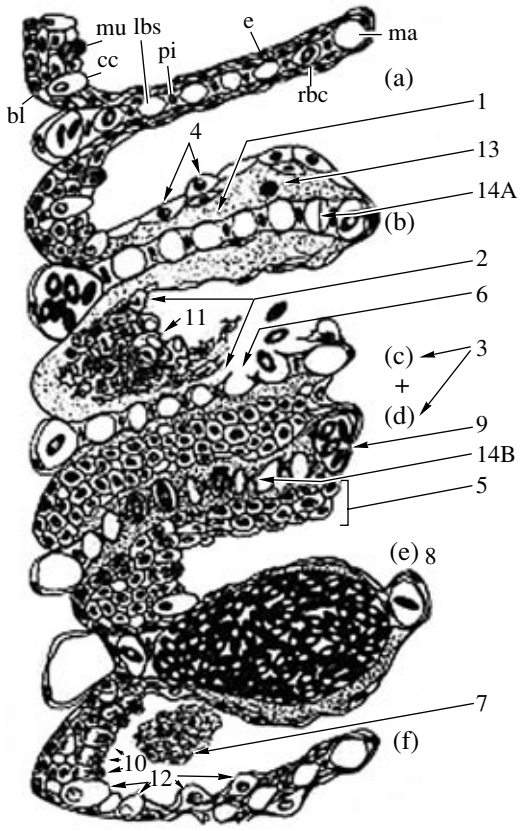

Fig. 1. Comprehensive scheme of gill injuries resulting from the action of H^+ and Al^{3+}. The upper lamella (a) is normal (an example of *Oncorhynchus mykiss*); other lamellae (b–f) are damaged: 1, the height of epithelial cells is increased; 2, necrosis; 3, lysis of lamellae (c and d); 4, cell hypertrophy; 5, hyperplasia; 6, destruction of gill epithelium with hemorrhage into parenchyma; 7, mucus secretion; 8, aneurysms (e); 9, obstruction of blood vessels (hyperemia); 10, proliferation of mucus-secreting cells; 11, early stages of damage to chloride cells; 12, proliferation of chloride cells; 13, epithelium infiltration by leukocytes; 14A, dilatation of blood vessels in the lamella; and 14B, constriction of blood vessels in the lamella. Designations: (bl) basal lamina, (cc) chloride cells, (e) typical epithelial cells of the lamella, (lbs) lamellar blood sinus, (ma) marginal blood vessel, (mu) mucus-secreting cells, (pi) pillar cells, and (rbc) reed blood cells (Rosseland and Staurnes, 1944).

The purpose of this study is to formulate a concept concerning the basic mechanisms of physiological disturbances causing the death of fish in acidified water bodies. In one way or another, these disturbances manifest themselves in many regions of Russia where the problem of water acidification has arisen.

SENSORY ORGANS AND GILLS

In fish, which respire by gills and skin, sensory organs and gills are the primary target for adverse factors acting in acidified water bodies. The external sensory organs of fish, such as the olfactory and gustatory organs, have no "protective barriers" and are vulnerable to the influence of low-pH water and accompanying factors. Toxic substances can disturb the chemosensory functions of fish by masking or counteracting biologically natural chemical signals or by causing direct morphological and physiological damage to receptors (Rosseland and Staurnes, 1994). The impaired functioning of olfactory and gustatory organs is apparently responsible for disturbances in the following forms of behavior: (a) avoidance of dangerous zones where deposition of chemical substances is possible (primary defense mechanisms); (b) social interactions (e.g., during spawning); (c) learned behavior, hiding, and avoidance of predators; (d) searching for food and feeding activity; and (d) orientation during smolt migration in anadromous species.

As the organs of smell are especially important in fish, it is assumed that they largely account for homing in Atlantic salmon. Hence, the impairment of imprinting in these fish because of acidification or water mass mixing may increase the probability of deviation from the correct course of their spawning migration to the river.

Many physiological disturbances in fish exposed to water acidification (e.g., impaired osmoregulation, acid–base disbalance, and dysfunction of the respiratory and circulatory systems) are often directly related to the impact of deleterious agents on gills (Mazeaud and Mazeaud, 1981; Rosseland and Staurnes, 1994). In acid waters, these agents, including Al^{3+}, cause structural changes and disturb biochemical and physiological processes in this organ. Contact with low-pH water elicits a protective response from gill epithelium, which is manifested in intensification of mucus secretion (Segner et al., 1988). In addition to hyperfunction and proliferation of secretory cells, structural changes are observed, which include hypertrophy of the epithelium of respiratory lamellae; necrosis, hyperplasia, and destruction of the epithelium; lysis of lamellae and formation of flask-shaped lamellae; manifestations of telangiectasia; changes in the number and size of chloride cells; and vascularization of the gills (Mallat, 1985). Figure 1 schematically shows main disturbances in the gills of fish exposed in acid waters with a high Al^{3+} content in the laboratory or in natural water bodies. In such conditions, chloride cells are the main target for Al^{3+} and H^+.

The acute toxic effect of H^+ in acid waters is generally explained by an increase in the solubility of metal salts and, hence, the loss of calcium by the gill epithelium (McDonald, 1983). This impairs regulation of membrane permeability and upsets ion balance in the cells, leading to Na^+ and Cl^- outflow. In addition, H^+ ions cause acidosis. At very high H^+ concentrations (never occurring in nature), the mucus may obstruct the interlamellar space, which leads to hypoxia and various gill injuries. However, only in rare cases is high acidity itself responsible for the reduction of fish stocks: it is

accepted that Al^{3+} is the main toxic agent (Rosseland and Staurnes, 1994).

HORMONAL OSMOREGULATION AND METABOLISM

Prolactin and cortisol are hormones responsible for osmoregulation. Prolactin decreases membrane permeability to ions and intensifies mucus secretion, while cortisol stimulates cell proliferation and differentiation of the primary gill epithelium and enhances Na,K-ATPase activity (Staurnes et al., 1984). In acidified waters, cortisol and prolactin levels in fish blood plasma increase (Pottinger and Pickering, 1992) in response to decrease in plasma electrolytes (Wendelaar et al., 1987). This is one of the factors providing for fish resistance to chronic exposure in acid waters with a high aluminum content. The increasing cortisol level has an adverse effect on the immune system (Mazeaud and Mazeaud, 1981; Pickering and Pottinger, 1985) and, therefore, on the health of fish living in acid waters or sporadically exposed to acidification. Thus, primary sublethal physiological stress (problems related to osmoregulation) is combined with secondary stress manifested in the inhibition of immunity by cortisol. This combination may lead to increased fish mortality in water bodies.

Intensified respiratory and cardiac activities in fish exposed to water acidification lead to an increase in metabolic rate and energy expenditures, which reflects activation of internal compensatory mechanisms providing for restoration of homeostasis. Hyperventilation of gills in acidified waters is probably a specific response to the influence of aluminum, as the addition of chelating compounds that bind aluminum ions (citric or humic acid) attenuates this response (Wood and McDonald, 1987; Rosseland et al., 1990).

Thus, the integrated effect of water acidification on fish is not limited to their high mortality during episodes of acidification or in the zones of water mixing (Rosseland and Staurnes, 1994). This effect may also account for postspawning mortality and changes in populations that lead to increased elimination of fish after the first spawning. The results of long-term experiments show that even moderate concentrations of aluminum suppress the growth of fish (Sadler and Turnpenny, 1986).

VULNERABILITY OF FISH AT DIFFERENT STAGES OF THE LIFE CYCLE

Physiological mechanisms and vulnerability to water acidification may change at different stages of the life cycle.

Oogenesis and fertilization. These stages are extremely sensitive to low-pH water. Female fish from acidified water bodies have a decreased calcium level in blood serum and plasma, and this leads to the formation of unviable eggs (Roy et al., 1990). The level of vitellogenin in these fish is also reduced (Mount et al., 1988).

Embryos. All stages of embryonic development are sensitive to acidification (Rosseland, 1986, 1990). In many embryos, the thickening of the chorion is observed, which is probably a result of protein denaturation due to at low pH of the water and perivitelline fluid. Aluminum reduces both egg membrane permeability to ions and Na,K-ATPase activity in the embryo. The mechanism of aluminum permeation through the basal membrane has not yet been studied, but it may be similar to that in the gill epithelium.

Parr. The adverse effect of Al^{3+} on juveniles increases as they grow (Wood and McDonald, 1987), which may be due to changes in the respiratory system. The vulnerability of gills as the main target organ manifests itself at this stage. It is known that Al^{3+} and pH have an effect on the mineral composition of the entire fish body, including calcification of the skeleton and fins (Rosseland and Staurnes, 1994).

A decrease in the abundance of lake and river salmon is accounted for by processes that take place in the critical periods of their life cycle (reproduction and development), which is manifested in the embryos, parr, and in the periods of parr–smolt transformation and spawning. Reproductive failure (reduced fecundity and unviable eggs or larvae) is the main cause of fish population decline in acidified water systems.

Smolt. The mechanisms responsible for a decrease in the abundance of anadromous salmonid fishes deserve special attention. In these species, the parr–smolt transformation is regarded as the most sensitive period (Rosseland et al., 1990; Staurnes et al., 1993). During this short period of transition from the benthic to pelagic way of life, the gill epithelium of juveniles gradually transforms into the tissue characteristic of sea fishes: membrane permeability increases, the number of chloride cells changes, and the structure of gills becomes more complex; Na,K-ATPase activity also changes (Hoar, 1988). All these transformations make the gill epithelium of smolt extremely sensitive to the adverse influence of hydrogen and aluminum ions.

When the smolt of Atlantic salmon develops in acidified waters, the effect of low-pH water manifests itself in the inhibited Na,K-ATPase activity in the gills, disturbed regulation of ion balance, and the complete loss of seawater tolerance. Hydrogen and aluminum ions inhibit enzymes (the biochemical level) that are responsible for the functioning of chloride cells (the cell level); hence, gills (the organ level) become incapable of regulating ion concentrations in the blood plasma when fish migrate from sea to fresh water or vice versa (Hoar, 1988; Staurnes et al., 1993). In the aggregate, these factors make the process of transformation into smolt less effective. As a consequence, the survival of fish in the sea is low and the pattern of their migrations is disturbed, which eventually leads to degradation of salmon populations.

Adult fish. In anadromous species, adult individuals during spawning migration have low tolerance for water acidification. The gill epithelium of spawners migrating from the sea to the river, being adapted to the marine environment, is very sensitive to the influence of low pH and Al^{3+} (Hoar, 1988; Rosseland and Staurnes, 1994).

It should be noted that fish living in lakes have a better chance of avoiding exposure to adverse factors than fish living in rivers. However, fish of the majority of salmon populations live in rivers at early stages of the life cycle, and mass mortality is primarily observed in acidified river waters. River species more frequently enter the zones of mixing of neutral (brackish) and acid waters with a high content of aluminum, which coagulates on the gills. They are also exposed to extreme influences of "pH shock" and high concentrations of metal ions during episodes of acidification related to floods or storms.

SPECIFIC EFFECTS OF Al^{3+}

High concentrations of monomeric aluminum are typical of acidified water bodies, and its effects on fish physiology have been extensively studied (Ganrot et al., 1986; Wood and McDonald, 1987; Exley et al., 1991; Rosseland et al., 1990; Rosseland and Staurnes, 1994; Havas and Rosseland, 1995). It has been shown that aluminum coagulates on the surface of gill epithelium and is incorporated by epithelial cells. The main consequences of its action are as follows: (a) disturbances of the respiratory function because of obstruction of the interlamellar space with the mucus, aluminum coagulation on the gill surface, and decrease in membrane fluidity; (b) disturbances of ion balance and osmoregulation and increasing loss of ions (Na^+, Cl^-, and Ca^{2+}) because of aluminum coagulation on the gill surface, its accumulation in the intercellular space, and inhibition of mechanisms increasing membrane permeability; and (c) circulatory disturbances resulting from a high hematocrit value conditioned by a decrease in the volume of blood plasma and swelling of red blood cells (along with an increase in blood protein concentration and viscosity).

Some general effects of Al^{3+} are due to changes in enzyme activities, calcium metabolism, and the functions of cell nuclei and membrane lipids (Ganrot, 1986). The mucus acts as a hydrodynamic barrier and maintains the gradient of ions between the gill epithelium and ambient water (Exley et al., 1991). An important property of the gill epithelium as a boundary layer is that it can maintain neutral intracellular pH even at low values of water pH, which protects fish living in acid waters (McDonald, 1983).

Aluminum is toxic when it is in the inorganic (ionic) monomeric form. Hence, its toxicity decreases in the presence of Al-binding ligands other than OH^-, such as fluoride, silicic acid, or organic humic acids (Rosseland et al., 1990). At pH 5, silicic acid in the mucus initially has a negative charge and can bind positively charged aluminum hydroxide. Polymerization of Al may cause irritation of the gill epithelium and stimulate secretion of the mucus, which, along with coagulating Al, obstructs the interlamellar space; it may also cause some injuries of the gills.

Since polymerization is a temperature-dependent process, the toxicity of aluminum decreases at low temperatures (Poleo et al., 1991).

According to Exley et al. (1991), aluminum is bound on membranes when it is in the form of a trivalent cation ($A1(H_2O)^{3+}_{6(aq)}$), which interacts with small, negatively charged particles bearing phosphate, carboxyl, and hydroxyl groups. Thus, aluminum can bind to, and neutralize the electric charge of, both the phosphate groups of membrane phospholipids and the carboxyl groups of membrane proteins, thereby decreasing membrane fluidity. In addition, aluminum may possibly serve as a cofactor that binds to functional domains during transport of proteins and the above particles. Both these effects, which can be observed on the apical cell surface, may provide a basis for predicting relationships between important transcellular processes such as the flow of ions (Na^+, Cl^-, Ca^{2+}), the removal of metabolic products (NH_4^+, HCO_3^-), and the diffusion of gases involved in respiration (O_2, CO_2).

Aluminum substitutes calcium in the intercellular space. As shown by the results of in vitro experiments (Exley et al., 1991), its entry into the cell occurs with the involvement of phospholipids. Apparently, aluminum changes membrane permeability, and this facilitates its accumulation in the intercellular space. Within the cell, citric acid is the aluminum-binding ligand, which serves as an intermediary that brings aluminum to high-affinity groups. The activities of anhydrase and N,K-ATPase in the gills is inhibited in salmonid fishes exposed in acidified waters containing aluminum (Staurnes et al., 1984; Rosseland et al., 1990, 1992). The interaction of ATP and Al results in the formation of complexes that are stronger than ATP–magnesium complexes. Therefore, aluminum can affect many enzymatic reactions (with ATP as a substrate), thereby causing various disturbances in cell metabolism (Ganrot, 1986). The involvement of aluminum in basic metabolic processes taking place in the gill epithelium may affect intercellular transport and provide for increased cell mortality (Exley et al., 1991).

BIOACCUMULATION OF METALS

The increased permeability of cell membranes in fish under conditions of acidification facilitates the input and accumulation of not only aluminum but also of many other metals, including toxic elements such as Hg, Cd, Zn, and Pb (Spry and Wiener, 1991; Komov et al., 1997; Haines et al., 1995; Manio, 2001; Moi-

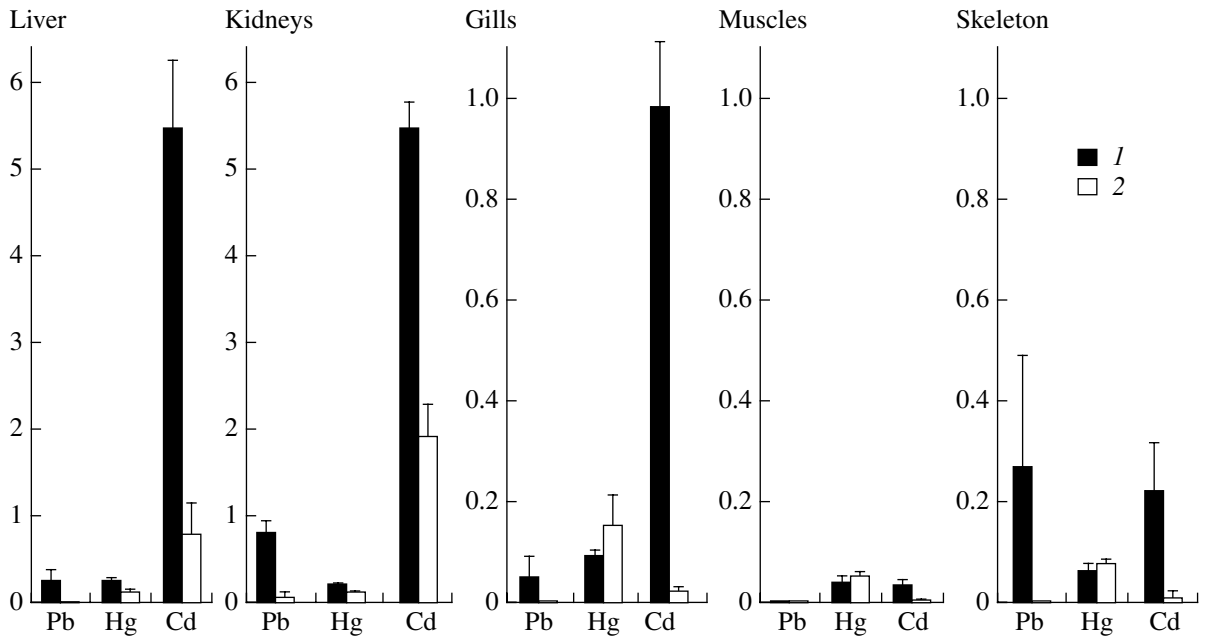

Fig. 2. Contents of Hg, Cd, and Pb (μg/g dry weight) in organs and tissues of brown trout (*Salmo trutta*) from (*1*) acidified lakes (water pH < 6) and (*2*) neutral lakes (water pH ≅ 7).

seenko and Kudryavtseva, 2002). This phenomenon is accounted for by the low calcium content in acid waters.

Studies on the accumulation of some elements in perch and char from acidified and neutral lakes showed that the concentrations of aluminum and some other metals (Ni, Mn, Co, and Zn) were significantly higher in the fish from acidified lakes, although these concentrations in the water were similar (Moiseenko and Kudryavtseva, 2002). The difference in metal accumulation was especially distinct in functionally important organs, namely, the liver and kidneys.

Disturbances of mineral metabolism may entail the development of various pathological processes in fish. For example, we revealed similar pathological changes in the cellular structure of the kidney, diagnosed as nephrocalcinosis and fibroelastosis, in fish from lakes that contained extremely high nickel concentrations, on the one hand, and from mountain tundra lakes in which nickel concentrations were low (1–2 μg/l) but waters were acidified, on the other hand (Moiseenko and Kudryavtseva, 1999).

In fish exposed to low-pH waters, calcium in skeletal elements is substituted by aluminum and strontium. Strontium accumulation deserves special attention. Acidified waters contain increased concentrations of strontium in the ionic form, which is highly labile, is involved in metabolism of the bone tissue and epiphyseal cartilage (Koval'skii, 1974), and can substitute calcium in the fish body under conditions of calcium deficiency in the aquatic environment, which is characteristic of acidified water bodies. Strontium accumulation against the background of decreasing calcium content leads to bone tissue pathology and, in particular, to scoliosis and osteoporosis, which manifest themselves in an abnormal curvature of the spine and impaired ossification of the braincase. Thus, substitution of calcium by strontium or, possibly, some other trace elements is the main etiological factor of these diseases (Moiseenko and Kudryavtseva, 2002).

In recent years, specialists has devoted special attention to the toxic properties of cadmium, lead, mercury, and some other metals. Published data (Scheuhammer, 1991) provide evidence that these elements, which present a major ecological hazard to the environment, are accumulated in large amounts in fish from acidified water bodies and in birds and mammals that live near them. Figure 2 shows data on the contents of Cd, Pb, and Hg in the organs and tissues of the brown trout (*Salmo trutta*) from lakes with water pH ≤ 6 and ≅7. Cadmium is highly labile, which provides for its leaching by acid precipitation, input into water bodies, and accumulation in fish. Significant amounts of cadmium are accumulated in organs characterized by high metabolic activity, namely, the liver, kidneys, and gills. Lead is also accumulated mainly in the kidneys and liver, and its contents are markedly higher in fish from acidified lakes than in fish from neutral waters (Jagon et al., 1993; Haines et al., 1995; Manio, 2001; Moiseenko and Kudryavtseva, 2002).

Therefore, the organs and tissues of fish in low-pH waters actively accumulate toxic metals (Cd, Pb, Hg, etc.), which leads to disorders etiologically related to the contents of trace elements. On the other hand, calcium and several biophilic elements in fish tissues are substituted by aluminum or strontium. These factors

account for the development of osteoporosis, nephrocalcinosis, adipose degeneration of the liver, and other pathologies. The same physiological mechanism may be responsible for the decreasing resistance and survival of fish in acidified water bodies.

The impact of water acidification on the physiological systems of fish has resulted in the reduction of fish stocks in many lakes and rivers. The degradation of fish populations is explained primarily by the direct combined influence of low pH and aluminum ions, which causes biochemical and physiological disturbances. The gills and sensory organs are the primary targets for these factors in acidified waters, and the larvae and juveniles (parr) are the most vulnerable stages of fish ontogeny. A serious hazard to anadromous fish is presented by zones in which acid river waters mix with seawater: under such conditions, dissolved aluminum coagulates on the gill surface, which is often lethal for adult fish.

When both water pH and calcium content are low, fish more actively accumulate highly toxic Hg, Pb, Cd, and other heavy metals from the water in which their concentrations are below the sensitivity threshold of analytical methods. This leads to the development of certain pathologies.

Identification of these pathologies and dysfunctions of physiological systems is important for revealing the factors responsible for fish population decline or extinction, predicting possible population dynamics under conditions of decreasing or increasing water acidity, and developing the strategy and methods of fish stock conservation and restoration.

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