Current Trends in Ecology
Vol. 2, 2011

Review

Fish ecology change under long-term toxic impacts: A review

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ABSTRACT

The article is devoted to current trend in fish ecology under long-term chronic toxic impacts. It is shown that fish exposed to sublethal doses of toxic substances for a long time have a smaller body size, the period of their sexual maturation becomes more variable, and the frequency of spawning and life span decrease. Changeability factors for population under toxic impacts are distinguished. Regularities of fish populations associated with selective effect of toxic agents and additional energy supply for detoxification are detailed in this work. The biochemical and bioenergetic mechanisms, responsible for changeability of populations are analyzed. The ecological expediency of change in the life cycle strategy corresponding to r-selection (conversion to a short reproductive cycle) is substantiated. Role of evolutionary pre-adaptation and biological mechanisms in maintenance of population homeostasis are considered. It is shown, that adaptation develops through a number of broods due to selection pressure, eliminating the least resistant individuals. Selection improves population's resistance to toxic pollution, but at a later stage it may result in reduction of the genetic diversity, decrease of its adaptive capacities to other environmental factors along with all negative consequences, arising under it.

KEYWORDS: fish population, toxic pollution, adaptation, mutations, tolerance gene, life cycle strategy

INTRODUCTION

Environmental pollution has become a factor of rapid (in the evolutionary aspect) change in life conditions. In this respect, aquatic systems are especially vulnerable, as they eventually accumulate all kinds of pollutants. Aquatic ecosystems are stressed in all levels, ranging from individual and up to the population and community levels [1, 2, 3, 4]. Environmental and fishery scientists continually strive to develop and improve methods that provide information both on the effects of environmental stressors on aquatic organisms and on the mechanisms responsible for these effects [5, 6, 7]. There are more and more examples showing that the organism's response to anthropogenic pollution by active adaptive modifications including fish population [8, 9, 10, 11, 12, 13].

The current focus on the collection of chronic toxicity data provides insight into the biochemical and physiological response of organisms to contaminants but it is often difficult to extrapolate from these data to ecologically relevant endpoints, particularly changes in population structure [14]. Studies on the consequences of long-term environmental pollution for animal populations are still rare. This is primarily explained by the absence of long series of observations concerning

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The purpose of this works was: To reveal current trends in fish ecology under long-term toxic impacts and to establish a change in the life cycle strategy of fish; to explain the ecological mechanisms and their adaptive significance for maintaining population size under chronic toxic pollution.

the characteristics of natural populations before and after exposure to chronic pollution.

We note that complicating the interpretation of fish population's response to stressors, however, is the complexity of the environment itself. For example, growth and other integrative measures of fish health are influenced not only by contaminants but other factors such as food availability, competition, and fluctuating physical-chemical and nutrient factors. Because of these complicating environmental factors and the complex nature of biological responses to stressors, it is unlikely that single variables can accurately reflect an organism's response to environmental stress.

The combined impact of many factors changes conditions in water bodies in such a way that they markedly deviate from natural conditions of the aquatic environment. This environment acquires a new property, namely, toxicity. Conditions that existed in the past provided for the formation of characters and their combinations enabling the species to survive within a certain range of fluctuations of abiotic factors. At the same time, the life cycle is not rigid: any cycle is flexible and depends on the interaction of hereditary and environmental factors [15, 16]. It is reasonable to suppose that the populations respond to anthropogenic changes of the environment, including toxic pollution, in accordance with the evolutionarily established self-regulation mechanisms responsible for the maintenance of population size under extreme conditions. The self-regulation mechanisms, which are among general properties acquired by animals in the course of historical development, are aimed at counterbalancing environmental changes rather than maintaining stability [5, 17, 18].

Biochemical and bioenergetics mechanisms of regulation

Recent biochemical studies on the effect of long-term toxic stress on fish show that the response is formed at two interconnected levels. The primary (endocrine) response is aimed at increasing the rate of energy metabolism and involves stimulation of adrenergic systems (an increase in catecholamine, epinephrine, and norepinephrine production) and hypothalamic-hypophyseal-adrenal centres (an increase in ATP and corticosterone production). The secondary response concerns metabolic adaptation to permanently high energy expenditures and involves the increased production of catecholamines (energy supply via glycogenolysis) and cortisol (inhibition of protein synthesis and energy supply via catabolism of glycogen, lipids, and proteins) [19, 20]. The increase in the rate of energy metabolism in organisms exposed to extreme conditions, which apparently include environmental pollution, is an established fact confirmed by numerous investigations and regarded as a law of organism response on stress [7, 16, 19, 21, 22, 23].

The biochemical data on intensification of catabolic processes under conditions of long-term toxic stress explain the mechanism of additional energy expenditures for detoxification. Let us consider how this mechanism can control variation in the parameters of fish populations.

The energy budget of an organism may be represented as follows [20]:

$$GE = FE + UE + ME,$$

where GE is gross energy, FE and UE are energy losses with wastes, and ME is the energy expended for metabolism. In turn, ME is subdivided into two parts: MEm is the energy necessary for maintaining life activities, and MEp is the energy providing for fish growth (the energy deposited, in particular, in generative organs). In this context, growth potential may be expressed as MEp/MEm. Investigations of Seyr and Reader [21] demonstrated that long-term exposure to low toxic concentrations increases the energy expenses of organisms for maintaining metabolism. In majority of situations the growth reduction is explained by activation of the energy-consuming processes of toxicity elimination in fish organisms. There are examples of maintaining physiological homeostasis by means of the specific protein-metallothioneins induction under the toxic contamination conditions, that is the energy-consuming process [23].

In terms of bioenergetics, the additional energy expenditure for detoxification changes this ratio toward an increase in the proportion of energy involved in metabolism (MEm) and an decrease in the proportion of assimilated energy MEp, which eventually results in the reduced potential for growth and reproduction. Indication of adenyl energy charge is often used for estimation of the pollution process and toxic stress effects on the

energy state of an organism. Energy charge of the organism's state shows the quantity of chemically banded energy, stored in a pool of adenine nucleotides and available for providing metabolism processes at the given moment. Exposure to stress conditions, including toxic effect leads to persistent decrease of the adenyl energy charge, defining the domination of the catabolic processes over the synthetic ones [24]. Numerous experimental investigations on fish, exposed to the influence of various pesticide concentrations (endosulfan, carbofuran, fenvalerate, lindane, DDT, atrazine, HCB), toxic agents (nickel, zinc, cadmium), complex industrial pollution (hydrocarbons, industrial effluents, pulp-and-paper waste), demonstrated persistent decrease of energy charge of test species, indicating intensification of catabolic processes under the influence of toxic agents and flexibility of response of this factor at different taxonomical groups of water animals [13, 20, 23, 24].

In case energy consumption for maintaining the main metabolism, owing to mobilization of toxicity elimination mechanism, is increased, some energy, accumulated in tissues for growth gain and reproduction, is also decreased. Fish growth retardation is considered to be a tertiary effect of the long-term stress (under conditions of contamination), when primary response comes from hormonal regulation displacement (increase of cortisone) and secondary response stems from growth of catabolic processes in an organism, that was approved by investigations in the long-term experiments.

Ecological mechanisms of regulation and adaptation

If the dose of a toxic agent and the period of exposure do not exceed the detoxification capacity of an organism and its ability to maintain a high rate of energy metabolism, this organism survives and produces the progeny; otherwise, it develops various pathologies and dysfunctions and eventually dies. From the ecological standpoint, the population under chronic toxic conditions is affected by two factors:

1) the elimination of organisms due to higher mortality caused by toxicoses;
2) the selection of individuals which can provide additional energy expenditure for detoxification and life support.

Under conditions of these factors, rearrangement of the fish's life cycle provides the main function - reproduction, compensating its depletion in the number in a varying degree. It is known that early maturation provides replenishments in case of high rate of elimination of individuals. Joining of younger females to the spawning population provides compensation of the depletion in numbers that approves the ecological expediency of early maturation for maintenance of the population size. Early maturation of fish under conditions of long-term toxic contamination - is a well-known fact [4, 5, 7, 13]. Whitefish (*Coregonus lavaretus*, L) of a polymorphic and evolutionarily young species, from an arctic lake Imandra that has been polluted for more than 70 years is an example of their earlier sexual maturation, and the frequency of spawning and life span decrease [12]. The following processes of drifting of reproductive age to earlier age periods and growth retardation were ascertained for a number of species, abiding in the Baltic Sea: salmon (*Salmo salar*), cod (*Gadus morhua*) in the Baltic Sea and freshwater cod (*Lota lota*) in lakes of Finland demonstrated the clearest characteristics of such process [25]. Though precise reasons were not defined, authors relate this phenomenon to aquatic habitat contamination. According to Elliott *et al.* [7], investigations with *Zoarces viviparous* demonstrated that females mature early and as well as their female brood, abiding in basins, relatively contaminated with mercury. Comparatively high level of mercury accumulation in female brood organisms was detected.

Contamination influences sizes and quantity of spawned eggs. Experiments with gudgeon *Cyprinodon variegate*, that had been kept in contaminated conditions for about an year, demonstrated, that the quantity of maturing females reduced in comparison with the control group, while quantity of smaller eggs increased among females survived [26]. Landahl *et al.* [6] adduced results of investigations by the example of the population of English sol (*Pleuronectes vetulus*), abiding in contaminated conditions. According to the results of this investigation, scientists concluded that fertility of individuals reduced by 30 %, but it didn't influence the population size. It was demonstrated that females matured in younger age, their gonads were characterized by higher quantity of small eggs, that increased fertility of the fish population,

surviving in conditions of toxic contamination. In case of toxic chronic impacts the big number of small sized eggs is increased in maturing females. This process increases population breeding power of fish, surviving in conditions of toxic contamination, and maintains the fish population.

Increase of the female proportion in the population is another important mechanism of maintenance of the population number and breeding power in subtoxic environment conditions. Such effects occur among fish populations and under the effect of natural stress factors, such as unfavorable climate, or intensive commercial fishing [27]. The influence of a number of toxic agents leads to feminization of the population of fish, that increases population fertility. Goksor *et al.* [18], referring to the bulk of generalizations, concluded that the majority of fish is capable for sex reversal on the early stage of its development under the influence of low doses of certain organic toxicants. In case full-grown fish of certain sensitive species during the period of its gonadogenesis or its brood in the juvenile stage are exposed to certain toxicants during the period of 10 days, then predomination of females in brood genotype occurs [28]. Feminization of the population increases the population breeding power and may be considered as yet another mechanism of support of its number, therefore sex ration under toxic load is "shifted" toward the female domination.

Selection for tolerance

Despite living in a polluted environment, many organisms have proved to be capable of successful reproduction, development, and growth. Tolerance may be defined as the ability of certain individuals to withstand exposure to the concentrations of toxic agents that are harmful or lethal to other individuals [8]. Selection will proceed in favor of most tolerant individuals, i.e., those capable of activating their intrinsic mechanisms of survival, including detoxification systems.

The phenomenon of adaptation to living in a toxic environment has been observed in many studies on aquatic and terrestrial animals. Reviewing data on the development of metal tolerance in aquatic organisms, Klerks [29] concludes that many specialists fail to discriminate between acclimation and genetic adaptation. As shown in his own studies, benthic organisms succeeded in populating metal polluted biotopes due to both acclimation and genetic adaptation. On the other hand, the increased tolerance of benthic communities in estuaries exposed to pollution with cadmium more than 30 years ago is attributed to genetic adaptation alone. The improvement of genetic tolerance to Cu and Pb was also observed in isopods (*Asellus meridianus*) living in rivers polluted with mining waste [11]. In chironomids (*Chironomus riparius*) from metal polluted habitats, LC50 proved to be 13–250 times higher than in those from a "clean" laboratory culture [30]. Due to the initial qualitative heterogeneity of natural populations, this population became "enriched" with tolerant individuals. This is why it was characterized by an increased survival rate, compared to the control, upon exposure at high concentrations of toxic elements.

In the 1970s, specialists obtained data on remote consequences of DDT action on fish (*Gambusia affinis*) that had survived at its concentrations that proved to be lethal for the majority of tested fish. It is known that tolerant individuals to DDT appear in populations of rapidly reproducing fish species. This tolerance develops only in individuals surviving exposure to high, almost lethal concentrations of the toxic agent, whereas small concentrations produce no such effect [13]. Selection of this character made it possible to produce a stock of tolerant mosquito fish. These fish proved to be toxic for predators feeding on them. Moreover, they released appreciable amounts of DDT into ambient water, making it toxic for other fish. However, their tolerance to other (organophosphate) pesticides was very low.

Other specialists [11, 29] maintain that tolerance to a toxic agent may be achieved by means of fish acclimation to it at low concentrations. The phenotypic response to heavy metal exposure may involve the increased induction of metallothioneins, low molecular weight sulfur containing proteins that bind metals and facilitate their removal, as well as the formation of intercellular granules in which metals are encapsulated. These types of response are not inherited and do not manifest themselves in the progeny transferred to an unpolluted environment. Alternatively, tolerance may develop due to genetic mechanisms and, therefore, can be inherited. These differences

between physiological and genetic tolerance account for genetic selection of physiologically flexible individuals.

Hauser *et al.* [11] distinguished two genetic bases for selection: (1) preadaptive, or selection of genes that took place in the population before exposure to toxic pollution, and (2) postadaptive, obtained from natural variability via selection induced by toxic agents. The ability to survive under pollution stress is regulated by different detoxification mechanisms. For example, the mechanism of tolerance to metals depends on the ability to regulate the rate of their intake or release, induce the synthesis of metallothioneins, and bind or sequester metals, as well as on differences in the sensitivity of enzymes to the inhibitory action of metals [13].

Nekrasova [31] studied the development of tolerance to chlorofos in mosquito larvae (*Anopheles dorsalis*) collected in copper polluted and relatively clean water bodies. A higher survival rate proved to be characteristic of larvae from polluted habitats. The author explained the development of unspecific tolerance to different toxic agents by selection of individuals with lower filtering activity, which provided for a reduced intake of these agents. Other mechanisms of selection also cannot be excluded. In any case, however, the fact is that surviving larvae and their populations formed as a result of selection under heavy metal pollution showed increased tolerance to other toxic factors (in this case, chlorofos). Moore and Willson [32] suggested that individual lysosomal activity accounts for species specific sensitivity or tolerance of animals to toxic substances. In black gobies (*Gobius niger*) from a port water area polluted with a mixture of toxicants, they revealed a high activity of mixed functional oxidases. Exposure to pollutants can result in selection for such a mechanism on condition of the survival of individuals potentially capable of its activation. However, the enhancement of oxidase activity in a polluted environment may be a consequence of not only selection but also of acclimation, with latent protective mechanisms being activated in the presence of toxic factors. Hauser *et al.* [11] revealed changes in glutathione S transferase and mixed functional oxidase activities in chironomids (*Chironomus riparius*) after their exposure to DDT.

Jerneloev [33] studied Japanese dace (*Tribolodon hakoniensis*) that lived in acid water with a high humus content over 15 generations and found that these fish developed high tolerance to acidification due to activation of cellular excretion as a component of the osmoregulatory system responsible for the removal of acids. Selection under such conditions also resulted in an increased red blood count of these fish. It should be noted, however, that the evolution of tolerance does not always take place, and weakened individuals from polluted areas are often no less, or even more, sensitive to toxic factors than their counterparts from unpolluted habitats [8].

Thus, the response of fish and invertebrate populations to selection pressure induced by pollution results in the survival of certain genotypes and elimination of some other genotypes. Long-term selection pressure of this type may provide for the formation of local ecotypes. The enhancement of functions related to detoxification and removal of toxic pollutants may be used as a biological marker for evaluating long-term effects of toxic exposure on aquatic animal populations.

Mutations

Heritable changes in genomic DNA, are the source of genetic variation in natural populations. Mutations can occur spontaneously or develop gradually under the influence of an altered environment. Genotoxic agents induce spontaneous mutations manifested in replication, recombination, and instability of chemical bonds in DNA, which, in turn, may result in phenomena such as structural isomerization, loss of amino groups, and incompatibility of bases during DNA replication. Genetic disturbances occur either under direct impact of toxic agents or after their biological transformation (i.e., under the effect of their derivatives). In addition, intracellular disturbances may also reflect on genetic processes. A toxic agent may affect the DNA molecule directly, by interacting with nucleotides, or indirectly, by initiating its replication. Both toxic chemical compounds and their derivatives can inflict damage to DNA. The latter can interact with DNA, thereby modifying its structure, initiating repair, or producing chromosomal mutations [11].

Active forms of various polycyclic aromatic hydrocarbons can covalently bind to DNA and disturb its native structure. Analysis by the method of alkaline hydrolysis provided evidence for an increased frequency of DNA breaks in freshwater fishes exposed to benz[a]pyrene and in turtles from polluted areas, compared to their counterparts from clean habitats [9].

Izyumov *et al.* [34] revealed an elevated level of chromosome aberrations in gametes and somatic cells of juvenile roach (*Rutilus rutilus*) experimentally exposed to toxicants with mutagenic effects (chlorofos, phenol, etc.). An increased frequency of micronuclei was detected in peripheral erythrocytes of roach and bream from natural populations living in a polluted pool of the Rybinsk Reservoir. Most probably, this was due to the mutagenic action of polluted aquatic environment [35]. Cajaraville *et al.* [36] showed that disturbances of DNA structure in fish, especially in marine species, occur under the effect of oil spills, mercury poisoning, or contact with other metals and organic xenobiotics. Such structural disturbances have far-reaching consequences, including rearrangements in the functioning of enzyme systems and protein metabolism, production of cell damaging toxins, inhibition of cell growth, accelerated tissue aging, suppression of immune response, impairment of fitness, and increased morbidity, including the frequency of malignant tumors. Mutations occurring in somatic cells are not inherited and do not impair the genetic pool (e.g., mutations causing various neoplasm, including malignant tumors), whereas mutations in gametes can be transmitted to the progeny. Shugart and Theodorakis [37] showed that mutations in gametes lead to developmental abnormalities, embryonic mortality, and hereditary mutations.

Thus, environmental pollution increases the mutational load on populations. Although the consequences of mutations are difficult to estimate, it is obvious that the mutation process leads to changes in the pattern of allele frequencies within the gene pool, thereby inevitably affecting the fitness of individuals and whole populations. Harmful mutations are rapidly eliminated by selection, but recessive mutations, being concealed from selection, may accumulate in a series of generations. Such mutations create a genetic load, which always reduces the viability of populations to a certain extent [38].

The state of an organism is a function of its genetic features and environmental factors. As follows from the data discussed above, two key factors have influence on the genetic pool of a population living in a toxic environment: (i) directed selection for tolerant genotypes and (ii) accumulation of recessive mutations.

Some animals-carriers of rare mutations and genotypes-are subject to the impact of eliminating factors, while others remain beyond their reach. As a result, the former decrease in numbers and may even disappear from the genetic pool, but the latter remain in the population. Thus, the total population size decreases under the effect of a toxic agent, but the frequency of tolerant genotypes increases. It is these genotypes that will provide the material for natural selection in the course of population recovery and subsequent dynamics.

Differing in tolerance to the toxic factor, individual subpopulation groups differently respond to its action, which may lead to serious structural changes. A critical population decline upon a major pollution incident switches on ecophysiological compensatory mechanisms, which provide for restoration of population density and stabilization of intrapopulation processes. Munkittrick and Dixon [1] proved that the response of a population to pollution is identical to its response to any other factor affecting its density. Therefore, high mortality caused by toxic pollution will be counterbalanced by well-known mechanisms of population maintenance.

In any case, whether the toxic impact is permanent or periodic, the significance of stable (tolerant) genotypes in the population will increase, since they represent the sole genetic variant ensuring selective success under such conditions. Mechanisms intensifying reproductive processes in residual populations (in our case, those consisting of tolerant individuals) are well known [13, 16, 39]. Thus, population decline upon a major discharge of toxic pollutants takes place within a short period of time, and only individuals whose genotype ensures high resistance to the toxic factor can survive in the population. Model calculations of animal survival in a population comprising N genotypes were made at the

Institute of Plant and Animal Ecology, Ural Division, Russian Academy of Sciences [39]. At 97% mortality caused by pesticide application, 1% of animals survive due to natural tolerance to pesticides and 2% survive accidentally; when the population recovers its initial size, the proportion of tolerant animals in it reaches 25%. Upon the second pesticide application, this proportion increases to 77%; upon the third application, to 98% and subsequently remains at this level. In nature, however, pesticide tolerance will be accumulated in the population more slowly, since the initial proportion of tolerant individuals may be markedly smaller and population decline after toxic exposure may be less drastic.

It is known that the mutation process and selection in combination account for directed changes in a population. The response of genetically heterogeneous populations to an environmental stress factor is manifested in changes in the distribution of population parameters. As a result, the population may acquire new alleles or undergo changes in the occurrence frequencies of certain alleles, which eventually will provide a source for genetic variation and microevolutionary processes. Allele frequencies may change due to mutations, selection, migration, and genetic drift. Changes due to migration and genetic drift may be indicative of perturbations in the environment. Migration and genetic drift may have a dual significance for a local population exposed to pollution.

Due to immigrants from unpolluted areas and their successful interbreeding with population members, genetic variability within the population may increase more rapidly than under the effect of selection or the mutation process, with the inflow of new genes reducing the efficiency of population adaptation to toxic agents. Gene flow is also of major significance when the local population is incapable of adaptation and is maintained due to recruitment of immigrants or stable genotypes favored by selection. Adverse effects of pollution on the population may manifest themselves beyond the area directly affected by pollutants. Individuals with different genotypes (stable or carrying a mutation load) may disperse and interbreed with individuals from other populations.

Thus, the range of effects resulting from the life of the population in a polluted environment may be significantly broader. In populations of a small size (and so become most populations exposed to chronic pollution), the role of genetic drift in the evolutionary process may be more important than that of natural selection, especially in the case of intermittent pollution. The reduction of population size because of increasing mortality and decreasing fecundity is a typical consequence of pollution exposure. This leads to changes in the genetic pool that make the population more vulnerable to adverse factors. Genetic drift will initially result in fluctuations of the frequencies of alleles (accounting for high viability or detoxification) between generations and, eventually, in complete fixation of these alleles in the population. The probability of inbreeding in a small population is higher. When the effective population size decreases, e.g., due to a major pollution incident, inbreeding also contributes to selection for pollution tolerance. These processes inevitably lead to new genetic combinations, i.e., to significant and irreversible changes in the genetic pool and, therefore, to microevolution.

Identification of tolerance gene

Specialists are still arguing whether or not a special gene accounting for pollution tolerance exists in natural populations. Electrophoretic studies have provided many examples of differences in genetic structure between invertebrates and fishes from polluted and unpolluted habitats. The results of original studies by Newman (1995) provide evidence for some genetic differences between tolerant individuals and individuals from the initial population. For example, using electrophoretic analysis, he revealed a specific allozyme in the eastern mosquito fish *Gambusia holbrooki* of tolerant genotype, which live in metal polluted waters. This allozyme subsequently proved to be common to many fish species from polluted habitats. In another study, several generations of mosquito fish were reared in 7250-liters mesocosms either under near natural (control) conditions or in the presence of relatively low concentrations of inorganic mercury. In the second (test) variant, unlike in the control, segregation of three allozymes at the PGI-2 locus was revealed. When the progeny of fish with the altered PGI-2 locus were exposed to higher mercury concentrations in 96-h

experiments, they survived for longer periods of time, showing higher tolerance to the toxic agent. Allozymes identified in this tolerant genotype proved to be similar to those in other fish species from polluted habitats [30].

Wies *et al.* [40] identified genetic differences in the mummichog (*Fundulus heteroclitus*) from a New Jersey estuary polluted with heavy metals. Hauser *et al.* [11] refer to the example of increased tolerance in mosquito fish and mollusk populations from ponds polluted with a mixture of metals near a thermal power plant, compared to populations from relatively clean water bodies. Studies on their genetic features provided evidence for probable selection for pollution tolerance in both species, with the surviving genotypes being of smaller body size.

Identification of a gene or gene complex accounting for tolerance to toxic agents is a very difficult task, since detoxification is accomplished by different mechanisms and different genes may be responsible for it [9, 41]. Thus, the enhancement of tolerance to the toxic effect of heavy metals may be achieved by different mechanisms, including the induction of metallothioneins, activation of mixed-functional oxidases, reduction of cell permeability for metals, and their sequestering or active excretion. As follows from these examples, tolerance to a toxic agent may be accounted for by different mechanisms. Therefore, this trait is polygenic, and identification of some definite "tolerance locus" is unlikely.

The cost of adaptation

Environmental pollution creates extreme conditions for living organisms, with their tolerance serving a protective function against adverse environmental factors. This protection helps to survive longer but is often achieved at the expense of impairment in other functions. Invariances in the life cycle and rigid connections between individual parameters account for the situation that the improvement of fitness via changes in some character must be paid for by modifications in other characters and a decline in the fitness of the same organisms upon even a slight change of conditions [42]. The survival of a population in a "subtoxic" environment may result in the impairment of adaptation in general, creating the risk of its extinction upon exposure to extreme conditions of some other kind. In other words, selection for a genotype tolerant to certain extreme environmental factors reduces the potential for adaptation to other such factors.

Tolerance may be characteristic of a narrow, specific range of phenotypes and be accompanied by the loss of genetic diversity. The reduction of genetic variability in a population may restrict its potential for adaptation and its ability to respond to changes in "conventional" natural-climatic factors. On the other hand, numerous facts confirm the possibility of long-term, sustainable existence of populations under considerable toxic stress. This means that restoration of the initial heterogeneous population is possible within a short time after the cessation of toxic exposure. Therefore, the process of population adaptation in this case has not yet reached the level of evolutionary transformation. Wies *et al.* [40] experimented with two populations of the mummichog: one from an estuarine area exposed to long-term pollution with oil spills and methylmercury, and the other from an unpolluted (background) water area. Fertilized eggs obtained from these fish were placed in water with different salinities to estimate embryonic survival. Embryos from control fish showed a higher survival rate at high salinities, while embryos tolerant to methylmercury proved to be less adapted to salinity variations. The authors suggested that the population from the polluted water area is adapted to a very narrow salinity range, 15-20‰, whereas the progeny of control fish are capable of surviving at salinities ranging from 10 to 30‰. Thus, the development of tolerance to methylmercury entailed the reduction of genetic diversity within the population and the impairment of fish ability to withstand the impact of natural stress factors and different types of pollution [40].

Heithaus and Laushman [43] used allozyme electrophoresis to study genetic diversity in tree fish species from six streams differing in water quality. The level of genetic variability between populations from different streams proved to be the lowest in *Etheostoma caeruleum* (a highly specialized species), intermediate in *E. blenniodes* (the species with a medium specialization level), and the highest in *Cmpostoma anomalus* (the

species with a broad ecological valency). Moreover, the populations *C. anomalus* from the most polluted habitats showed the lowest level of intrapopulation genetic diversity. The authors noted that a low level of intrapopulation genetic diversity may be a good indicator of long-term exposure to pollution, being a probable consequence of selection for tolerance.

Therefore, the study of tolerance combined with analysis of data on the life cycle is necessary for predicting and preventing the risk of genetic erosion. The data discussed above indicate that metal tolerance develops via selection of individuals capable of reducing metal intake, inducing the synthesis of metallothioneins, binding and excreting metals. These properties place organisms tolerant to toxic metals at risk for deficiency in essential trace elements, which may account for their reduced tolerance to changes in other environmental factors [11].

Thus, selection may improve population tolerance to toxic agents but subsequently result in the reduction of genetic diversity and population adaptation to natural and anthropogenic stress factors.

Change in the life cycle strategy

The aforementioned changes in the ecology of fish under the effect of toxic water are explained by the concept of life cycle types formed by *r*- and K-selection. K-selections act when environmental conditions are stable and favors the survival of organisms with a long life span, a large body size, late maturation, and polycyclic reproduction. Conversely, *r*-selection operates in the populations that live under variable conditions and favors the survival of organisms characterized by a small size, early maturation, and one-time reproduction. In the case of *r*-selection, individuals in the populations of the same species mature earlier, are smaller, and expend more resources for reproduction [15]. Changes in the strategy of life cycle, corresponding to *r*-selection, attain the adaptive value under the effect of unfavorable environmental factors. Indubitably, the toxic factor refers to unfavorable ones as well. Acclimatization of individuals is manifested in activation of the detoxification mechanism and capacity of fish physiological systems to bend more energies to homeostasis maintenance and detoxification of penetrating poisons, as it has been proved above. In case acclimatization to new conditions leads to adaptation through selection, it is obvious, that the population will take on new properties and somewhat different genetic pool.

The example of the study of whitefish population *Coregonus Lavaretus L*, that has been surviving for the period of more than 70 years under conditions of the long-term toxic effect (in Imandra lakes, Russia), and data analysis in the scientific literature show that in conditions of the high elimination rate of individuals by reason of toxicosis and stressful energetic balance, the life cycle strategy of fish is “shifted” toward the corresponding *r*-selection. Persistency and selection occur among individuals preadaptated to distribution of energetic elements for providing more active metabolism, to earlier maturation and spawning, wherein gametogenesis proceeds within the normal features, and the probability of bringing a healthy brood is higher [12].

The mentioned results of the investigations on the lake of Imandra demonstrated that a high compliance of whitefish allowed to implement the ecological mechanisms of population number maintenance under conditions of water pollution (population with new features was created in the water body). We cite a number of additional proving examples. According to the data of Toppin *et al.* [44], under the effect of heavy metals the life cycle of *Fundulus heteroclitus* changed in the following way: fish were smaller, ready for reproduction in the earlier age and had a shorter life cycle. These changes in the life cycle provided the reproductive success of the population, in spite of the high elimination rate on the early stages of development. Similar changes of the life cycle strategy are covered in works of Elliott *et al.* [7] by the example of *Zoarces viviparous*.

Thus, the population responds to chronic water pollution as to extreme conditions, in accordance with the evolutionarily established mechanisms of self-regulation. These mechanisms are as follows: i) the retardation of maturation and the absence from spawning when fish have to maintain a high rate of energy metabolism and, hence, fail to accumulate energy resources sufficient for spawning in due time; ii) early maturation and the

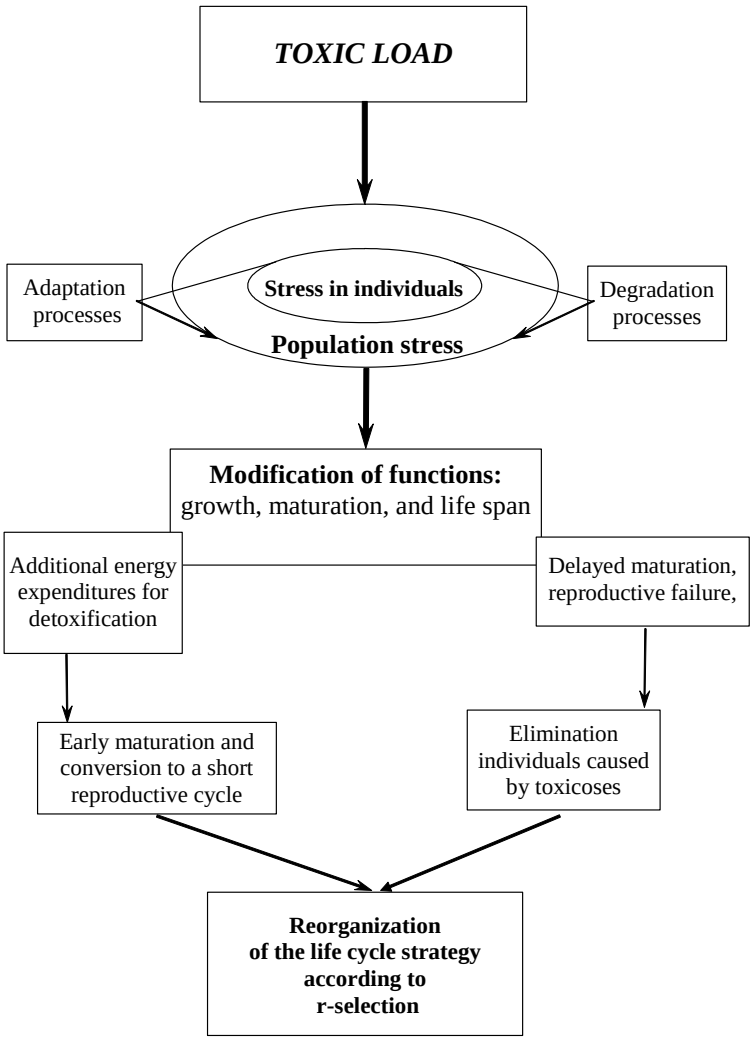

Figure 1. Scheme of reorganization of the life cycle strategy in fish under conditions of long-term toxic pollution of the aquatic environment.

reduction of individual life span; iii) in some cases - feminization of a population and increase in number of small eggs.

The life cycle reflects the response of organisms to the entire complex of conditions, and its strategy depends on reproduction as the central process. Degree of reproductive process suppression under contamination conditions depends first of all on the toxic agents' effect on reproductive organs and particular maturation functions, defined with systemic physiological state of fish and its toxicants' effect resistance (or toxicity elimination capacity). There are scientific evidences, demonstrating that a number of toxicants has a poisonous effect on maturation, ovulation and spawning period, and therefore has an effect on a number of population recruits [23].

The toxic substances put selective pressure upon a population [8, 9]. Exposure to toxic factors leads to a high mortality rate and difficulties in maintaining energy balance. Under such conditions, the life strategy of fish changes in the direction corresponding to the effect of *r*-selection. This type of selection favors the survival of individuals adapted to the redistribution of energy supply for maintaining more active metabolism at the expense of growth and to maturation and spawning at an early age, when gametogenesis proceeds

normally and the probability of producing a normal progeny is higher. The delayed maturation and lower frequency of spawning result from the shortage of energy resources necessary for normal gametogenesis. Under the toxic load, K-strategy is ecologically inexpedient, as the delayed maturation and longevity increase the probability of diseases and disturbances of reproduction. The mechanisms regulating population size are responsible for reorganization of the adaptive system that controls individual differences in the rates of growth and sexual maturation, the frequency of spawning, and longevity (Figure 1). This is reflected in population structure: body size, the proportion of fish in older age groups, and the age of recruitment to the spawning stock decrease.

CONCLUSION

Environmental pollution has become a major stress factor affecting fish. The selection pressure of toxic factors accounts for the survival of more tolerant genotypes. Being determined genetically, this tolerance is inherited. Long-term exposure to this selection pressure may give rise to local ecotypes capable of surviving and reproducing under conditions of toxic pollution. The ability to survive under such conditions is regulated by various mechanisms improving individual viability, such as avoidance, reduction of cell permeability for toxic agents and sensitivity to them, activation of mixed-functional oxidizes, induction of metallothioneins, sequestering and excretion of ecotoxicants, etc. Hence, no definite locus or gene accounting for tolerance has been identified as yet.

Environmental pollution increases the mutational load on populations. Many mutations are not inherited (somatic mutations) and do not affect the genetic pool, whereas mutations in gametes may be inherited. Recessive mutations are invisible for selection and accumulate in a series of generations, creating a genetic load. Two key factors act upon the genetic pool of a population in a toxic environment: i) directed selection for tolerant genotypes and ii) accumulation of recessive mutations. The mutation process combined with selection is known to account for directed changes in the population: it can acquire new alleles or undergo hangers in allele frequencies, which eventually provides a source for genetic variation and current trends in fish ecology. The rate of these processes depends on a series of genetic and ecological factors. The reduction of genetic diversity may improve tolerance of a population to a certain toxic factor, but usually at the expense of impairment in its general potential for adaptation to other stress factors or kinds of pollution.

Individual fish and populations respond to toxic pollution according to the evolutionarily established mechanisms (preadaptations) providing for the improvement of viability and self-regulation of stock abundance under extreme conditions. Under conditions of toxic pollution, the fish population experiences the impact of two factors: i) the additional energy expenditures for detoxification and ii) the increased elimination of individuals caused by toxicoses. Ecologically expedient transformations under such conditions are those attributable to the effect of *r*-selection, when the population consists mostly of relatively small individuals that mature earlier and are capable of realizing their reproductive potential at a younger age, producing a high-quality progeny.

The population changes under toxic impacts occur due to elimination of the least resistant species, individual acclimatization of organisms and selection of the species tolerant to genetic adaptation. Adaptation to toxicants has a wide range of mechanisms of vital capacity rise, such as: toxicity elimination, avoidance, regulation, export and etc. Adaptation develops through a number of broods due to selection pressure, eliminating the least resistant species. Selection improves population's resistance to toxicants, but at a later stage it may result in reduction of the genetic diversity, decrease of its adaptive capacities to other environmental factors along with all negative consequences, arising under it.

There is a debatable question concerning the extent to which the newly acquired properties of the population can be fixed genetically. Assuming that the frequency of alleles responsible for a short reproductive cycle in a population increases, is it possible to discuss the direction of micro-evolutionary changes in this population? The principal directions of anthropogenic micro-evolutionary transformations in animal populations include the decrease of body size and transition to a short reproductive cycle. The results of this and

numerous other studies confirm this idea. Micro-evolutionary transformations follow the pattern of *r*-selection, i.e., in favor of small, early maturing individuals capable of assigning more energy resources for reproduction. Early maturation ensures an increased rate of reproduction and effective maintenance of population size.

ACKNOWLEDGMENTS

The work was supported by the Russian Foundation for Basic Research (Projects no 10-05-00854) and grant of Russian Governments (№ 11G34.31.0036).

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