ISSN 1067-4136, Russian Journal of Ecology, 2009, Vol. 40, No. 5, pp. 305–313. © Pleiades Publishing, Ltd., 2009.
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Anthropogenic Evolution of Animals: Facts and Their Interpretation

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Received March 26, 2009

Abstract—Problems concerning microevolutionary transformations in animal populations are considered. It is shown that genetic variation is the main factor providing the basis for adaptation to environmental changes, including toxic pollution. The selection pressure of a toxic factor gives an advantage in survival to more resistant genotypes in animal populations, which eventually leads to the reduction of their genetic diversity and potential for adaptation to other natural or anthropogenic stress factors. Microevolutionary transformations follow the pattern of *r*-selection, i.e., occur in favor of smaller, earlier maturing individuals capable of expending a greater proportion of their energy resources for reproduction.

Key words: microevolution, population, genetic diversity.

DOI: 10.1134/S1067413609050014

Environmental pollution is now a major factor affecting animal populations. Examples showing that animals respond to anthropogenic pollution by active adaptive and microevolutionary transformations have become increasingly frequent in recent publications (Braginskii et al., 1971; Kolchinskii, 1990; Bol'shakov et al., 1991; Bezel' et al., 1994; Newman, 1995; Chesser and Sugg, 1996; Staton et al., 2001; Hauser et al., 2003; Bezel', 2006; and others). Thus, environmental pollution provides us with a unique model for studying the evolutionary process at work (Dobzhansky, 1970).

Bol'shakov (1991) noted that, although economic activities change the conditions of animal life, they are by mo means aimed at altering the pattern of natural evolution; nevertheless, they have a considerable effect on many aspects of the evolutionary process, primarily at the level of microevolution. The evolution of populations under changing environmental conditions may proceed rapidly and dramatically (Malvey and Diamond, 1996). However, no generalizations on this problem have been made in the international literature devoted to anthropogenic evolution. Industrial melanism and acquired tolerance to pesticides in insects are the most popular examples cited as evidence of anthropogenic evolution. However, the literature on genetic adaptation in living organisms and the evolution of their tolerance to toxic exposure is abundant and difficult to review in a single paper.

A number of studies have been performed to analyze and predict how changes occurring in populations under the impact of toxic agents may reflect on their genetic diversity (Bol'shakov et al., 1991; Newman, 1995; Bickham et al., 2000; Staton et al., 2001; Hauser et al., 2003; and others). The main conclusion is that toxic pollution will lead to a gradual loss of genetic diversity, on the one hand, and that the toxic agent will serve as a factor of selection in favor of individuals with the highest level of tolerance to toxic exposure.

The purpose of this study is to formulate the idea of how changes in the population exposed to a toxic factor may reflect on its genetic diversity and what consequences the selective pressure of environmental pollution factor may have.

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 (Chesser and Sugg, 1996). 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 (1990) 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 (Hauser et al., 2003). In chironomids *Chironomus riparius* from metal-polluted habitats, LC_{50} proved to be 13–250 times higher than in those from a "clean" laboratory culture (Newman, 1995).

Predatory centipedes (Chilopoda) from a population inhabiting areas polluted with Zn, Cd, and Cu were more tolerant to these metals, compared to centipedes from unpolluted habitats, which was confirmed in subsequent experiments on adding metals to the food for arthropods from both populations. This result is indicative of probable adaptation to pollution and increased individual tolerance to metals in the first population. 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 (Bezel' et al., 1994).

Similar examples were described for small mammals. Korosov and Pavlov (1988) compared two populations of forest voles: one inhabited the slopes of the Khamar-Daban Range, which had long been exposed to emissions from a pulp and paper mill, and the other (control) population was from unpolluted habitats. Long-term technogenic impact (over more than 18 years) caused changes in the genotypic structure of the first population, in which many generations had survived under conditions of toxic environmental pollution. Consequently, when kept on the diet supplemented with toxic substances, these animals showed a higher survival rate than did voles from unpolluted habitats.

In the 1970s, specialists obtained data on remote consequences of DDT action on fish that had survived at its concentrations that proved to be lethal for the majority of animals tested. It is known that DDT-tolerant individuals appear in populations of rapidly reproducing fish species. According to Braginskii et al. (1971), this tolerance develops only in individuals surviving exposure to high, almost lethal concentrations of the toxic agent, whereas small concentrations produce no such effect. Selection for this character made it possible to produce a stock of DDT-tolerant mosquito fish (*Gambusia affinis*). 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 (Klerks, 1990; Hauser et al., 2003) maintain that tolerance to a toxic agent may be achieved by means of 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, be inherited. These differences between physiological and genetic tolerance account for genetic selection of physiologically flexible individuals.

Hauser et al. (2003) 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 (Moiseenko, 2002).

Nekrasova (1989) studied the development of tolerance to chlorofos (trichlorfon) in *Anopheles dorsalis* mosquito larvae collected in copper-polluted and relatively clean water bodies. A higher survival rate proved to be characteristic of larvae from technogenically 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 (1998) 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. (2003) revealed changes in glutathione S-transferase and mixed-functional oxidase activities in chironomids *Chironomus riparius* after their exposure to DDT. Jerneloev (1988) 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. Experiments of Cuvin–Aralar and Aralar (1995) with the fish *Oreochromis niloticus* exposed to a mixture of metals (Hg, Zn, and Cd) showed that individuals sensitive to their impact were eliminated at the stages of maturation and spawning. More tolerant individuals managed to adapt to these metals and transmit this ability to their progeny, which proved to be less vulnerable to other metals as well.

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 (Chesser and Sugg, 1996).

Thus, the response of animal 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, or mutations, 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 (Hauser et al., 2003). 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. 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 (Walker et al., 2001). Izyumov et al. (2002) revealed an elevated level of chromosome aberrations in gametes and somatic cells of juvenile roach (*Rutilus rutilus* L.) experimentally exposed to toxicants with mutagenic effects (chlorofos, phenol, MNNG, 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 (Izyumov et al., 2003).

Cajaraville et al. (2003) 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 neoplasms, including malignant tumors), whereas mutations in gametes can be transmitted to the progeny, Shugart and Theodorakis (1994) 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 (Altukhov, 2003).

CHANGES IN GENETIC POOL

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: (1) directed selection for tolerant genotypes and (2) 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 (Bol'shakov et al., 1991).

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 (Shilova and Shatunovskii, 2005). Munkittrick and Dixon (1989) 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 (Shvarts, 1980; Bol'shakov et al., 1991). 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. 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 (Bol'shakov et al., 1991).

It is known that the mutation process and selection in combination account for directed changes in a population (Altukhov, 2003). The response of genetically heterogeneous populations to an environmental stress factor is manifested in changes in the distribution of population parameters (Glotov and Tarakanov, 1985). 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. Shilova and Shatunovskii (2005) describe an illustrative example: reproduction of frogs in areas polluted with chemical waste ceased completely, but their population abundance in these habitats was maintained due to regular recruitment of immigrants,

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. Bezel' (1987) confirmed this conclusion in his model calculations.

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.

Shvarts (1980) noted that colonization of areas with drastically reduced population density leads to the formation of a new ecological system on the basis of interbreeding between immigrants and surviving members of the initial population, redistribution of immigrants, and emigration of surviving individuals. 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-l 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.

Wies et al. (1999) identified genetic differences in the mummichog (*Fundulus heteroclitus*) from a New Jersey estuary polluted with heavy metals. Hauser et al. (2003) 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 (Depledge, 1996). Thus, Walker et al. (2001) generalized published data and, on this basis, described selection for insecticide tolerance in insects, distinguishing the following mechanisms: (1) behavioral (increasing sensitivity and avoidance); (2) enhancement of functions related to detoxification (of DDT by dihydrochlorinase, of carbomates by microsomal monooxidases, and of organophosphate compounds by glutathione transferases and esterases); (3) reduction of sensitivity against adverse factors and (4) reduction of cell permeability. 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 (Gilyarov, 2003). 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 (Bezel', 2006). On the other hand, numerous facts confirm the possibility of long-term, sustainable existence of populations under considerable toxic stress. Some authors (McHeily, 1968; Bezel', 2006) consider that in plant cenopopulations tolerant to metal pollution, polymorphism characteristic of the initial (background) populations should be restored every year in new generations of plants. In the course of their subsequent

ontogeny, plants tolerant to toxic agents and adapted to current weather conditions survive and ensure the necessary level of population reproduction.

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. (1999) experimented with two populations of the mummichog (*F. heteroclitus*): 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.

Heithaus and Laushman (1997) 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 *Campostoma anomalus* (the species with a broad ecological valency). Moreover, *C. anomalus* populations from the most polluted habitats showed the lowest level of intrapopulation genetic diversity. The authors note 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 (Hauser et al., 2003). Bezel' (2006) refers to the example of improvement in population tolerance to a toxic factor accompanied by the impairment of adaptation to other environmental factors that was obtained in studies on the common bent grass (*Agrostis tenuis*). Therefore, this phenomenon is universal for both animal and plant kingdoms.

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.

THE RANGE AND DIRECTION OF ADAPTIVE TRANSFORMATIONS

Considering the main directions of anthropogenic microevolutionary transformations in animal populations, Kolchinskii (1990) puts emphasis on decrease in the size of populations and their transition to a short reproductive cycle. Genetic adaptation in short-cycle populations with a high growth rate proceeds more rapidly. Homozygosity for tolerance provides for its strong expression. In heterozygotes, tolerance depends on the degree of dominance of alleles responsible for this trait, which, in turn, accounts for the rate at which these alleles are spread within the population (naturally, dominant alleles spread more rapidly than recessive).

On the premise of general evolution laws, it may be assumed that certain factors will promote and accelerate selection for tolerance, whereas other factors will interfere with acquisition of tolerance by species exposed to pollution. Let us consider some of them.

(1) Genetic factors: Mutations: a source of genetic variation, which provides the raw material for selection (mutation rate 10^{-4} to 10^{-6} events per gene per generation).

Dominance: selection for tolerance controlled by dominant alleles proceeds more rapidly.

Mono- and polygenicity: selection is more rapid when tolerance is under monogenic control.

Fitness: selection is more rapid when differences in fitness between tolerant and sensitive individuals are significant.

(2) Ecological factors: Generation time and reproduction rate: short-cycle species with a high population growth rate respond to selection pressure more rapidly.

Population size: genetic variation in a small population may be low.

Mating: provides for recombination of the tolerant genotype.

Emigration/immigration: if migrants are of nontolerant genotype, the population evolves slowly.

Living conditions (microenvironment): selection is retarded when sensitive species can find protection from toxic impact (appropriate microhabitats, shelters, etc.).

Stage of development: Selection efficiency depends on sensitivity of individual developmental stages.

The rate of elimination or adaptation of a population to anthropogenic stress depends on the momentum and severity of the stress factor and the adaptation potential of the population. The polygenomic system allows rapid changes in gene frequencies and, therefore, allows species to undergo rapid evolutionary transformations in response to changes in the environment.

Changes in the life strategy of fishes under the impact of toxic water pollution are generalized in the concept of life cycle types formed by *r*- and *K*-selection (Pianka, 1981). *K*-selection operates under stable, barely changeable conditions and favors the survival of fish with a long life span, large body size, late maturation, and polycyclic reproduction. Conversely, *r*-selection operates in populations living under variable conditions and favors the survival of fish characterized by a relatively small body size, early maturation, high mortality in older age groups, and, therefore, monocyclicity (or a few spawning cycles) and greater energy expenditures for reproduction. Changes in the life cycle strategy resulting from *r*-selection acquire special adaptive significance under unfavorable environmental conditions, including toxic exposure. The most significant transformations of ecological population structure take place under the impact of anthropogenic factors (Shvarts, 1980; Bol'shakov et al., 1991).

For the evolutionarily young and "flexible" species *Coregonus lavaretus* inhabiting the subarctic lake Imandra, which has been polluted over more than 70 years, changes in population parameters manifested themselves in a reduced growth rate, increased variation in the time of maturation (which may be accelerated as well as retarded), a shorter life span, and a reduced number of spawnings. The "lag" in body size can be traced beginning from the first years of life, which is explained by an increased metabolic level and redistribution of the energy budget to satisfy requirements for energy-intensive detoxification processes (Moiseenko, 2002). In long-term experiments with the sheepshead minnow (*Cyprinodon variegatus*), a group of these fish was exposed for 374 days in a tanker vessel containing bottom sediments polluted with a mixture of metals. After the end of exposure, the body size of both female and male fish was markedly smaller than in the control) group kept under normal, unpolluted conditions (Rowe, 2003). In *F. heteroclitus* population inhabiting a polluted New Jersey estuary and differing genetically from those living in clean habitats, very small females proved to be capable of producing eggs, and most embryos developing from these eggs were tolerant to methylmercury.

Shilova and Shatunovskii (2005) describe the enhancement of population fecundity in small rodents and fishes due to activation of compensatory mechanisms providing for rapid restoration and maintenance of population size. They show that the age of sexual maturation in both groups of vertebrates exposed to pollution shifts toward younger age classes. Bezel' et al. (1994) provide data that young of the year in small rodent populations from polluted areas mature rapidly and constitute the best protected part of the population, with their early involvement in reproduction being the main factor accounting for the maintenance of population size under conditions of chronic exposure to toxic agents.

The data described above show that populations living in a toxically polluted environment are subject to *r*-selection, which favors the survival of smaller and earlier maturing individuals, i.e., those that can realize their reproductive potential and produce viable progeny at a younger age. Early maturation ensures an increased rate of population reproduction against the background of high mortality both at early developmental stages and in older age groups, being the leading ecological mechanism of population maintenance under conditions of toxic pollution.

CONCLUSIONS

The problem of anthropogenic evolution of animals is topical in recent biological research. Specialists have accumulated numerous facts concerning genetic changes in animal populations exposed to pollution, which confirm the existence of anthropogenic microevolution.

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 oxidases, 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: (1) directed selection for tolerant genotypes and (2) 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

changes in allele frequencies, which eventually provides a source for genetic variation and microevolutionary processes. 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. Microevolutionary 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.

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