Journal of the Minnesota Academy of Science

Volume 36
Number 2 *Volume 36, Nos. 2 and 3*

Article 3

1969

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ABSTRACT — Three examples, industrial melanism in moths, insecticide resistance in insects, and myxomatosis in Australian rabbits, are used to illustrate the concept that evolution may result from tampering with the physical or biotic environment of a species. Hence, concern about the effects of pollution must encompass not only the possibility of extinction but the possibility of evolution. In the case of harmful species, biological or chemical control measures may result in populations more refractory to control than the original untreated populations.

The word evolution usually brings to mind such things as the dinosaurs or other extinct groups whose fossil record extends over millions of years. However, evolution is a continuous process. It has occurred in the immediate past and is taking place at the present time. Some of the best-studied cases of present-day evolution have resulted from environmental pollution, which has produced new selection pressures on existing species. These affected populations have really only two alternatives under such circumstances: They adapt to the changed conditions or they die. In discussions of the effects of pollution such as Rachel Carson's "Silent Spring," attention has been centered on the elimination of entire populations of birds or other species. But the cases in which species have adapted to changed environmental conditions are of considerable biological interest and also pose new and different problems. The best studied examples of this have resulted when the pollutants consisted of unusual amounts of familiar substances or else were new synthetic substances such as the insecticides.

Industrial melanism in moths

The Industrial Revolution began in England about 1800. Coal was the primary source of energy in the factories, and consequently the entire countryside in the industrial areas came to be blanketed under a coat of soot and dirt from the numerous smokestacks. A number of species of moths in England normally rested in the open on tree trunks or rocks during the day but were inconspicuous because their pigment patterns blended in so well with the bark of trees or with the lichens growing on the trees and rocks. As industrial soot killed the lichens and darkened the trees, these light colored moths became conspicuous against their new background and no longer had protective, cryptic coloration.

In the course of a century and a half since then, the light-colored typical form in more than 80 species of moths has been replaced in British industrial areas by a darker melanistic form of moth that blends with the new type of background (Ford, 1964). Similar changes have been observed in still other species in industrial areas of continental Europe and the United States. In the early 1800's, the dark form was a rare aberrant type of moth, found only occasionally in the populations of light moths. Today some populations consist of close to 100% of the dark type, and the light form is the rare aberrant type.

Evolution has been defined as "descent with modification"; in other words, an existing population has descended with some hereditary changes from an ancestral population. In this case, the existing populations of dark moths are known to have descended from the ancestral populations of light moths so that, by definition, this transition represents an example of evolution.

In nearly all cases, the melanism has turned out to be due to a single, simple dominant gene. Thus, the change is hereditary, not due to a direct environmental effect. The dominant gene has increased in frequency from being rare to being very common. It is the new "wild type" gene in these populations.

The mechanism by which this change was produced is natural selection. Birds have been found to prey more heavily on dark moths resting on a light background than on light moths, but they capture proportionately more of the light moths resting on a dark background. Thus, in the new environment created by industrial soot the dark moths survived better by being less conspicuous and left more progeny than the light moths. The frequency of the gene causing melanism increased very rapidly as a result of this differential predation and differential reproduction. The evolutionary change was relatively rapid, and it was adaptive in that the population was better able to survive and reproduce in its new polluted environment (Kettlewell, 1961).

Insecticide resistance

Organic insecticides, of which DDT is the most familiar, started to come into use in the early 1940's. The first resistant house flies were reported in 1946. Thereafter, the number of species in which insecticide resistance was reported increased rapidly (Crow, 1966).

YEAR	Reported number of resistant species
1948	12
1951	16
1954	25
1957	76
1960	137
1963	157
1966	165 +

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Not only house flies, but mosquitoes, lice, fruit flies, and many other insect species also were found to have become resistant to insecticides. Probably many additional species have become resistant but have never been studied and reported. Nearly every species exposed to an insecticide has been able to develop some degree of resistance, and there seems to be no insecticide among the great variety tested to which some species has not become resistant. Furthermore, the resistance is due to an evolutionary change, to a change in the genetic composition of the exposed populations as a result of natural selection. The resistance is inherited, and is not some sort of physiological tolerance acquired by an exposed individual. This can be shown by taking parents from a resistant population and rearing their progeny without any contact with DDT. When first exposed to DDT, they are resistant, unlike the progeny of susceptible individuals.

The mechanism responsible for the difference in resistance is different in different populations, in some cases in different populations of the same species. Differences in DDT resistance have been found to be due to differences in:

Absorption of DDT through the insect cuticle.

Lipid content (DDT is fat-soluble, and is thus stored in the fat bodies where it does little harm).

Rate of detoxification by enzymes.

Sensitivity of the nervous system.

Behavior, the insects having behavior patterns that tend to minimize their exposure to DDT.

All of these differences are demonstrably hereditary. As might be expected, different genetic mechanisms are responsible for the different modes of resistance; dominant and recessive, autosomal and sex-linked, single factors and multiple factors, all have been reported. The diversity of paths taken illustrates another aspect of evolution, its opportunism. Natural populations of insects contain a great deal of genetic variability in what is often called their "gene pool," but the variability in the gene pool of one population is different from the variability in the gene pool of another. When the populations are confronted by the lethal stress of DDT, any kind of gene that in some way permits an individual to survive in the presence of DDT will be passed on to subsequent generations and will increase in frequency because individuals lacking such genes will die, and their non-resistant genes will be eliminated from the population. When all susceptible individuals die, it is not surprising that resistance has evolved so rapidly in insects — in as little as a year or two. Furthermore, the opportunism of evolution under these conditions should not be surprising, for the populations can only work within the framework of their existing variability. The insecticides are not mutagenic and do not cause mutations to resistance to occur. Thus, there is no Lamarckian type of response to the new environmental stress.

Although evolution is not usually thought to be a subject adapted to study in the laboratory, nevertheless it is possible to study micro-evolutionary changes such as the development of insecticide resistance in experimental populations. Exposure of genetically variable populations of fruit flies, *Drosophila melanogaster,* to DDT in the laboratory has resulted in a hundred-fold increase in DDT resistance as compared with the unexposed controls, as shown below (Merrell, 1960).

POPULATION	ED_{50}(mg)	Relative resistance
731 C	0.04	1
91 C	0.16	4
731 R	4.2	105
91 R	11.7	293

The resistance rose rapidly at first and has been maintained in these experimental populations for more than a decade. The original dosage in the populations was 0.2 or 0.5 mg with 75 percent to 90 percent mortality. Now dosage in the populations is 150 mg with minimal mortality. The flies have become so resistant that they walk about on visible deposits of DDT crystals during testing, and it has become increasingly difficult to get good estimates of the effective lethal dose. In these populations, it is possible to study a number of aspects of the way in which evolutionary changes actually occur rather than to speculate by inference from the fossil record.

In contrast to the genetically variable populations, highly inbred populations did not develop increased resistance when exposed to DDT (Merrell and Underhill, 1956). This result has certain implications. First, favorable spontaneous mutations did not accumulate fast enough to permit increased resistance to develop. Moreover, there was no indication that DDT was able to induce adaptive mutations. Finally, there was no sign that physiological adaptation of some sort was involved.

In addition, genetically variable exposed and unexposed groups from the same original population diverged from one another not only in DDT resistance, but also in other respects. In general, the unexposed controls showed higher fecundity and fertility, but the resistant flies had a longer life span (Underhill and Merrell, 1966). The environmental conditions for control and exposed populations differed only in the presence of DDT impregnated strips in the exposed populations, but this difference put a selective premium on fecundity and fertility in the first flies of the unexposed group to reach fresh food. On the other hand, the selective pressure in the other populations favored not only DDT resistance but also an extended life span and the ability to produce eggs over a long period of time. Thus, at least some of the eggs would be laid under conditions favorable to their development after the DDT strip had become covered by food and debris. Hence, natural selection seems to operate in subtle as well as more direct and obvious ways.

The rabbit in Australia

Before Europeans reached Australia, the only placental mammals there were bats, several species of rats, and the

Australian aborigines and their dogs. The mammalian fauna consisted almost exclusively of marsupials, the kangaroos, the wallabies, and a number of other unique and unusual species of pouched mammals. When Europeans colonized the world during the 16th, 17th, and 18th centuries, they brought with them, intentionally and unintentionally, many species of plants and animals, both domesticated and wild. One species introduced into Australia with dire consequences was the European rabbit, *Oryctolagus cuniculus* (Fenner, 1965).

In 1859 about two dozen wild English rabbits were deliberately released on a sheep station in Victoria. By 1865, 20,000 rabbits had been killed on that one ranch alone, and they were spreading rapidly, eventually covering all of southern Australia despite desperate efforts to halt their spread by such devices as barrier fences that covered thousands of miles. This represents a new and different sort of pollution. In the course of a century the rabbit population of Australia increased from 24 to several hundred million individuals. Their effect on agriculture and ranching was devastating. Efforts at control were were futile, and there were no natural enemies or predators.

The myxoma virus, a member of the poxvirus group, was originally found only in the Americas. It caused a relatively benign disease (lesions called fibromas) in South American rabbits (*Sylvilagus brasiliensis*) that was transferred by mosquitoes. In European rabbits, however, the virus caused a very severe generalized infection that was almost always fatal. The suggestion was made as early as 1918 by the Brazilian Dr. Aragão that the myxoma virus be used to control the rabbit pests in Australia, but the idea was not tested until 1950.

At that time virulent Brazilian-type myxoma virus was inoculated into rabbits at trial sites in the Murray Valley of Australia. The disease escaped from the test sites, and by 1954 it had spread throughout the rabbit population of Australia. The initial death rate was estimated to be 99.8% of the rabbits infected, but the death rate after a year or so was found to have fallen to about 90%. Thus, the virus was losing its effectiveness as a means of controlling the rabbit population.

As you should now expect, the rabbits, upon test, were found to have evolved an increased level of resistance to the virus through natural selection of the genetically more resistant individuals as the progenitors of the next generation.

Perhaps more surprising is the fact that the virus itself was found to have evolved from the original highly virulent strain to a less virulent type of virus. Why should this be? A parasite that is too virulent kills its host rapidly and this in turn leads to its own demise. Thus, it is to the advantage of the parasite to reach some sort of accommodation with its host that permits it to survive and spread to other hosts. Therefore, mutant viral strains of reduced virulence permitted infected rabbits to survive longer and to serve as reservoirs of virus that could be picked up by mosquitoes and transmitted to other hosts. Thus, natural selection acted to increase the frequency of the less virulent viruses, and both the rabbit and the myxoma virus have evolved in relation to one another. The consequence of this evolution, hand-in-hand, by rabbit and virus, is that, given the reproductive potential of the rabbit (a five-fold to twelve-fold annual increase), the virus no longer is completely effective in keeping the pest under control.

Because pollution may have such far-reaching and unforeseen consequences, the indiscriminate pollution of the environment has been widely condemned. However, most attention has been directed toward the deleterious or lethal effects of the pollutant on existing species. The lethal effects of pesticides on the target species are welcome, but their harmful effects on other species are cause for concern. Because death results when environmental stresses are beyond the ability of organisms to respond adaptively, mortality is usually the most conspicuous effect of pollution. Hence, the death of individuals and the possible extinction of species have been the major source of concern about pollution.

That species might evolve in response to changing environmental conditions was at first quite unexpected. The evidence now clearly indicates that whenever a new environmental stress on a genetically variable population permits differential survival and reproduction, the population will evolve adaptively to the stress. Thus, there are now reports not only of insects resistant to DDT, but of rats resistant to rat poison. Consequently, new and even more difficult problems of control may arise because of the ability of pest species to evolve. While there may be some comfort in the realization that other species also may have the ability to adapt in the face of new and rigorous selection pressures, this is a weak rationale for permitting unabated pollution.

Biological control is usually regarded as more desirable than control by pesticides because its effects are more specific and restricted. However, the evolutionary changes in the rabbit and the myxoma virus suggest that even biological control may not provide a permanent panacea. Furthermore, just as the introduction of a new pesticide may have unforeseen consequences, the introduction of a new predator, or parasite, or pathogen also may have unexpected biological ramifications.

In our present state of knowledge, there are no experts on pollution. However, it is becoming clear that in finding new and unusual ways to foul or disrupt his environment, man runs the risk of affecting not only the evolution of other species but his own as well. The great present need is for additional reliable information. Until such data are available, past experience suggests that we should proceed with caution and circumspection before further polluting our environment.

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Corrigenda to

MERRELL, DAVID J. 1969. Natural selection in a leopard frog population. J. Minn. Acad. Sci. 35:86-89.

P. 86. col. 1. par. 1. Full quotation from Waddington, "Further, in a certain number of cases, though unfortunately not in very many, the actual occurrence of natural selection can be demonstrated."

P. 88. col. 2. par. 3 line 4. . . . If phenotypic selection operates within a homozygous population . . . line 7 . . . Thus, whether or not gene frequency changes resulted from the death of the deformed frogs is irrelevant to . . . par. 4 line 11 . . . seems applicable in this case, with the deformed frogs carrying fewer of the modifiers stabilizing normal development than the normal frogs. P. 5 line 3 . . . Despite the wealth of genetic variability they carry, . . .

P. 89. col. 1. par. 1 . . . probably occurred as the result of the more or less continuous predation by the garter snakes.

NASA-NSTA Schedule 12 Youth Science Congresses

The National Science Teachers Association and the National Aeronautics and Space Administration have scheduled twelve regional Youth Science Congresses for the spring of 1971. This is the seventh year the congresses have been sponsored by the two groups.

All students in grades 10 to 12 in any United States high school are eligible to apply for participation.

Up to 20 students will be selected from each region to participate in a two-day, expense-paid trip to a NASA center or a city having aerospace research facilities. Participants also will receive a medallion commemorating their attendance.

At the congress, the participants will present oral reports on their project or investigation. They also will take part in question-and-answer periods and informal sessions with fellow students, participating scientists, and science teacher guests; and they will visit NASA laboratories or related research installations.

Congresses will be held at Minneapolis, Minn.; St. Louis, Mo.; Colorado Springs, Colo.; and at the following NASA centers:

The Lewis Research Center, Cleveland, Ohio; Goddard Space Flight Center, Greenbelt, Maryland (2 congresses); Langley Research Center, Langley, Virginia; Marshall Space Flight Center, Huntsville, Alabama; John F. Kennedy Space Center, Cape Kennedy, Florida; Manned Spacecraft Center, Houston, Texas; Pasadena Office (Jet Propulsion Laboratory), Pasadena, California; and NASA Ames Research Center, Moffett Field, California.

Entry materials may be obtained from NSTA. The deadline for application is February 10, 1971.