The Explanatory Power of Evolution

A casual view of a school of minnows or a flock of blackbirds might suggest that they are all exactly alike, like molecules of water in the ocean. But they are not; many, many minor differences in size, form, or behavior will reward a closer examination. These *microevolutionary* differences accumulate in a population's gene pool over time. A few vanish, when their owners fail to reproduce, but most remain, with greater or lesser effect on the population as a whole. These differences continue to build up when populations of the same species become geographically isolated. Eventually, enough differences accumulate (through natural selection operating on the heritable variations in the gene pool) that interbreeding between the separate isolated populations is no longer possible; what had been separate populations of the same species can become two different species. This situation may go on to produce *macroevolutionary* change.

Drug Resistance in Bacteria

Why Should I Take All My Pills?

When your physician gives you a prescription for an antibiotic, he or she will caution you to take the entire seven-day regimen—all the pills in the pill bottle. The physician's insistence that you not stop taking the medication after three or four days is based on good evolutionary biology. After a few days of the drug therapy you may feel better, because the antibiotic will have killed a great many of the most susceptible bacteria. But if you discontinue the drug prematurely, *some* of the bacteria will survive, and these, which are the more *resistant* bacteria, will be the ones contributing their genes to the next generation. (Bacteria, of course, produce whole new generations on a time scale of minutes and hours.) In a few more days you could be much sicker than before, and a new prescription, this time of a more potent drug, may not do as well as the first one would have. By using the entire initial prescription, then, you have a good chance of killing all of the target organisms; by using just part of it, you are, in effect, selecting for drug-resistant bacteria.

The same sort of evolutionary reasoning is applied when flu or cold sufferers, knowing only that they feel sick, beg their doctors for an antibiotic, such as tetracycline. Influenza and the common cold are caused by viruses, and whereas antibiotics kill bacteria, they are ineffective against viruses. By unnecessarily taking an antibiotic, the patient does nothing to combat the flu or cold, and instead risks selecting for a resistant strain of bacteria that is already present in the body—a strain that in the future may require even stronger drugs or greater doses to control. It should be emphasized that the antibiotic does not *cause* the bacteria to become resistant; it simply sets up the conditions that encourage the microevolutionary shift to a new strain. The genetic mutations that confer resistance on certain individuals of the bacterial colony occur independently of exposure to the drug. But it is only in the presence of the drug that the resistance shows itself, for when confronted with the drug the resistant individuals inevitably yield more descendants than the others can. That the mutations occur independently of the drug has been confirmed repeatedly in the laboratory: a number of colonies of the same original bacteria are cultured in isolation from each other, and in time some of the isolated colonies show themselves to be drug-resistant even though they were never exposed to antibiotics. More often than not, fortunately, no resistant variations are present, and the bacterial infections are totally eliminated by the drug therapy. Organisms like bacteria, which have very short reproductive cycles and huge populations, have an enormous advantage over, say, the peregrine falcon in adapting to lethal environmental changes before they are driven to extinction. Bacteria experience far more mutations because there are so many more individuals and generations. This and the short reproductive cycle allow beneficial mutations to be exploited by natural selection rapidly.

The evolution of pests that are resistant to a particular pesticide works in the same fashion. The repeated wholesale spraying of the pesticide kills the most susceptible insects and leaves the most resistant individuals to breed the next generation. The chemical companies then introduce newer, more potent poisons in an attempt to stay half a jump behind the bugs—and like bacteria, insects reproduce rapidly, in huge quantities.

Rabbits and Myxomatosis
What Are the Limits of Biological Control?

The story of rabbits in Australia is another fascinating case history in evolutionary biology. Rabbits are not native to Australia. Before their introduction, their ecological niche was filled by a great variety of small kangaroos called wallabies. In 1859, 12 wild European rabbits, *Oryctolagus cuniculus*, were imported from England. By 1886 their descendants were colonizing new areas of southeastern Australia at the rate of 66 miles a year in all directions. By 1907 the rabbits had reached both the west and east coasts of Australia, roughly the distance between California and New York. Nothing could stop the plague of rabbits. Thousands of miles of "rabbit-proof fences" failed to stem the tide. Certainly the wallabies had offered no competitive resistance, and the few native predators made scarcely a dent in the rabbit populations. Hunting, trapping, and poisoning were to no avail. The rabbits were eating much of the sparse vegetation that supported Australia's huge sheep and cattle industry, and the graziers were suffering enormous financial losses.

The only solution was biological control. After much testing, government biologists introduced a mosquito-borne viral disease called myxomatosis. This virus caused a nonlethal disease in its natural host, a South American rabbit, but the disease was deadly for the European rabbit and completely harmless to all other Australian wildlife, domestic animals, and humans. To all indications, the solution had been found.

The disease did indeed take hold in 1950, and by 1952 it had produced a nationwide epidemic in the rabbit population. The mortality rate reached 99.9 percent, *but a good evolutionary biologist could predict what would happen next*. A parasite that invariably kills its hosts before ensuring its own survival would be selected against (all of its individuals would die). And that is what inevitably happened with the myxomatosis disease. The viruses had been randomly mutating, and the mutations that produced less virulence were selected (because the more virulent strains died with their hosts). The rabbits too, were mutating, and they were being selected for greater resistance to the disease. The result was a milder disease and stronger rabbits—therefore more rabbits. Today the mortality rate is down to about 40 percent. There are still annual outbreaks of myxomatosis in Australia, but the disease is less effective in controlling the rabbits. This is evolution in action, instigated and observed by humans, and occurring through natural evolutionary forces: *it is not explainable by any other concept*. Meanwhile, the government biologists are trying to develop more virulent strains of myxomatosis. And so it goes.

The Peppered Moth and Industrial Melanism
Can Even Air Pollution Drive Evolution?

One of the most celebrated cases of evolution via natural selection is the shifting fortunes of the peppered moth, *Biston betularia*, observed steadily by scientists for 140 years. This English moth exists in two distinct color phases, light and dark (Figure 13), and individuals of the one phase routinely and successfully mate with individuals of the other phase (thus they are not separate species). Only one pair of genes is involved in the color differences, and dark is dominant to light. (A dominant gene manifests its full effect despite the presence of a contrasted (recessive) gene whose expression for the character is blocked). The moths typically rest on lichen-covered tree trunks and branches, and their main predators are birds. Museum collections made in 1848 (prior to the Industrial Revolution) indicate that the frequency of the dark form was at that time less than 1 percent of the total of peppered moths in Manchester. While resting on the lichens, which are light, the dark variant was clearly visible and was consequently easily spotted by the birds that feed on the moths. The light variety of the moth, far better camouflaged against the lichens, was much less noticeable to the birds, much less often eaten, and far more often passed its genes along.

Fifty years later, the Industrial Revolution, with its sooty air pollution, had blackened and killed the lichens growing on the trees. The dark-colored moths now made up about 95 percent of the population. Films of feeding birds show them selectively eating the conspicuous lighter-colored moths. But in the 1950's, stringent anti-pollution laws were passed in Britain, and since then the air quality has greatly improved and soot has been reduced. *As predicted by evolutionary theory*, the white form is increasing its numbers once again. Natural selection (differential reproduction), brought about by the birds' inevitable concentration on the more visible form, resulted in a change in gene frequency (evolution). Speciation is not involved, since both light and dark forms are the same species. The existence of a dark-colored variant of a species is called **melanism** (there are black-squirrel variants of gray squirrels, and many other such examples); the shift to dark variants of the peppered moth at the height of the Industrial Revolution has been called industrial melanism.