7. E. Corle, *The Gila* (Univ. of Nebraska Press, Lincoln, 1951).
8. C. D. Vanicek, thesis, Utah State University, 1967.
9. R. R. Miller, *Nat. Parks Mag.* **37**, 4 (1963).
10. W. J. Koster, *Southwest Naturalist* **5**, 174 (1960).
11. R. R. Miller, *Copeia* **1963**, 1 (1963).
12. C. L. Hubbs and R. R. Miller, *Papers Mich. Acad. Sci.* **38**, 207 (1953).
13. W. F. Sigler and R. R. Miller, *Fishes of Utah* (Utah State Game and Fish Department, Salt Lake City, 1963).
14. W. H. Dill, *Calif. Fish Game* **30**, 109 (1944); O. L. Wallis, *Trans. Amer. Fish. Soc.* **80**, 84 (1951).
15. R. R. Miller, *Papers Mich. Acad. Sci.* **40**, 125 (1955).
16. W. L. Minckley and N. T. Alger, *Plateau*, in press.
17. W. L. Minckley, in *AAAS Committee on Desert and Arid Lands Research Contrib. No. 8* (1965), p. 48.
18. B. A. Branson *et al.*, *Southwestern Naturalist* **11**, 300 (1966).
19. J. K. Andersen, personal communication.
20. N. Wood, personal communication.
21. P. A. Douglas, *Calif. Fish Game* **38**, 149 (1952).
22. Personal observations; personal communications from personnel of the Arizona Game and Fish Department.
23. R. R. Miller and C. L. Hubbs, *Univ. Mich. Museum Zool. Misc. Pub.* **115**, 1 (1960).
24. W. G. Bradley and J. E. Deacon, *Desert Res. Inst. Univ. Nevada (Las Vegas) Pre-Print No. 9* (1965), p. 1.
25. Personal communication from U.S. Bureau of Reclamation, Boulder City Office.
26. W. E. Barber and W. L. Minckley, *Southwestern Naturalist* **11**, 313 (1966).
27. W. J. Koster, *Guide to the Fishes of New Mexico* (Univ. of New Mexico Press, Albuquerque, 1957).
28. C. L. Hubbs and R. R. Miller, *Univ. Mich. Museum Zool. Occasional Paper No. 433* (1941), p. 1.
29. J. R. Hastings, *J. Arizona Acad. Sci.* **1**, 60 (1959); J. R. Hastings and R. M. Turner, *The Changing Mile* (Univ. of Arizona Press, Tucson, 1965).
30. G. S. Myers, *Tropical Fish Hobbyist* **1965**, (Jan. 1965), p. 31.
31. J. E. Deacon, C. Hubbs, B. J. Zahuranec, *Copeia* **1964**, 384 (1964).
32. G. T. Malmberg, *U.S. Geol. Surv. Water Suppl. Paper 1832* (1967), p. 1.
33. C. L. Hubbs and R. R. Miller, *Univ. Mich. Museum Zool. Occasional Paper No. 507* (1948), p. 1.
34. I. LaRivers, *Fish and Fisheries of Nevada* (Nevada Game and Fish Commission, Reno, 1962).
35. B. L. Wilson, J. E. Deacon, W. G. Bradley, *Trans. California-Nevada Sect. Wildlife Soc.* **1966**, 12 (1966).
36. T. A. Eakin, *U.S. Geol. Surv. Ground-Water Res.-Reconnaissance Ser.* **25**, 1 (1964).
37. F. B. Sumner and U. Lanham, *Biol. Bull.* **82**, 313 (1942); C. Hubbs and W. F. Hettler, *Southwestern Naturalist* **9**, 245 (1964); C. Hubbs, R. C. Baird, J. W. Gerald, *Amer. Midland Naturalist* **77**, 104 (1967); J. E. Deacon and B. L. Wilson, *Southwestern Naturalist* **12**, 31 (1967).
38. C. Hubbs and H. J. Broderick, *Southwestern Naturalist* **8**, 46 (1963).
39. D. Nyquist, thesis, University of Nevada, (1963).
40. W. L. Minckley and L. H. Carufel, *Southwestern Naturalist* **12**, 291 (1967).
41. G. A. Waring, *U.S. Geol. Surv. Water Supply Paper 450* (1920), p. 51.
42. G. B. Maxey and T. W. Robinson, *U.S. Geol. Surv. Water Res. Bull.* **6**, 1 (1947).
43. Personal communication from Nevada State Engineer, Reno.

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Population Regulation and Genetic Feedback

Evolution provides foundation for control of herbivore, parasite, and predator numbers in nature.

David Pimentel

Although within a relatively short period man has learned how to put himself into space, he still is not certain how the numbers of a single plant or animal population are naturally controlled. Aspects of this problem have been investigated since Aristotle's time, they were given important consideration in Darwin's *Origin of Species,* and yet the unknowns far outweigh the discoveries. If we knew more about natural regulation of population, we would be in a better position to devise more effective and safer means of control for important populations of plant and animal pests. We might also be better able to limit the growth of human populations, although that problem is exceedingly complex because of the social activities and nature of man.

Population Characteristics

Before considering how populations in nature are regulated, we should review various characteristics of animals and plants—as individuals and as populations. Do populations of animals in nature fluctuate severely or are they relatively constant? Stability and constancy have been proposed as characteristics of natural populations. Speaking about birds, Lack (*1*) says, "of the species which are familiar to us in England today, most were familiar to our Victorian great-grandparents and many to our medieval ancestors; and the known changes in numbers are largely attributable to man." He continues, "All the available censuses confirm the view that, where conditions are not disturbed, birds fluctuate in numbers between very restricted limits. Thus, among the populations considered above, the highest total recorded was usually between two and six times, rarely as much as ten times, the lowest. This is a negligible range compared with what a geometric rate of increase would allow." Discussing the stability in animal populations in general, MacFadyen (*2*) writes: "it is generally agreed that the same species are usually found in the same habitats at the same seasons for many years in succession, and that they occur in numbers which are of the same order of magnitude."

Further evidence for the thesis that species populations are relatively constant is found in a study of the changes in the fauna of Ontario, Canada (*3*). When Snyder (*4*) evaluated the bird fauna, he found that, over a period of about 70 years, two species became extinct, 23 species increased in number, and six species decreased in number. This represents a total change of only 9 percent of 351 bird species found in Ontario (*5*) and agrees favorably with an 11-percent change (*6*) for 149 species of birds over a 50-year period in Finland. These data suggest that there is relative constancy in the abundance of species populations. The word "relative" must be emphasized because changes in numbers must be related to a species' real potential for fluctuations; to para-

The author is chairman of the department of entomology and limnology, Cornell University, Ithaca, New York.

phrase Lack (*1*), the changes observed are mere "ripples" compared to the possible "waves." Although in geological time 99 percent of all species have become extinct, during periods of 100 years or more constancy is the rule.

There are exceptions to this rule of constancy. What are the population characteristics of plants and animals newly introduced on islands and continents? Typically when a species population enters a new biotic community in which no ecological barrier exists, outbreaks occur in these populations. The following examples of introductions into the United States illustrate this point: Japanese beetle *(Popilla japonica);* European gypsy moth *(Porthetria dispar);* South American fire ant *(Salenopsis saevissima);* Asiatic chestnut blight *(Endothia parasitica)* (fungus); European starling *(Sturnus vulgaris);* and the English sparrow *(Passer domesticus).* Outbreak of chestnut blight was so severe that for all practical purposes it destroyed its host, the American chestnut tree (*Castanea dentata*). After increases in the number of Japanese beetles, a bacterial pathogen epidemic spread through the population and is now effectively controlling the numbers of the beetle.

In nature the numbers of many herbivore, parasitic, and predaceous species are limited by resistant factors inherent in the host. Are resistant factors which limit or prevent pest attack commonly found in plants and animals? Various kinds of resistant factors exist in plants and animals in nature and appear to be quite prevalent. The spines occurring in many kinds of plants, such as cacti, gorse, and hawthorn, prevent feeding by browsing animals. Toxins or growth inhibitors which occur in many kinds of plants limit animal feeding, for example, tannins in oak leaves (*7*), cyanide in bird's-foot trefoil *(Lotus corniculatus)* (*8*), and nepetalactone in catnip (*9*). To prevent predator attack (*10*), poisonous sprays are ejected from many insects and other arthropods, such as acetic acid by whip scorpion (Pedipalpidas), formic acid by ants (Formicidae), and *p*-benzoquinones by flour beetles (Tenebrionidae). Repellent sprays from glands in some vertebrates, such as the skunk *(Mephitis* spp.), the Indian mongoose *(Herpestes auropunctatus),* and the toad *(Bufo marinus),* ward off attacking enemies. Nutritional changes in certain plants prevent the multiplication of attacking insects, for example, aphids on corn (carotene) (*11*) and leafhoppers on beets (linoleic acid) (*12*). A kind of armor plating protects various animals (armadillos, turtles, and certain beetles) from their attackers. Such physiological defense mechanisms as specific antibodies and phagocytosis are present in many kinds of animals (man and other vertebrates) and effectively control pathogen and parasite infections.

When these natural resistant factors in plants and animals are successful, they prevent the uncontrolled increase of the feeding species. Are animal numbers abundant or rare? Rarity, like constancy, is relative. Numbers of a given species can be related to the numbers of another species, to the unit area occupied, or to the food resources of the species. Andrewartha and Birch (*13*) noted that "the truth is that the vast majority of species are rare, by whatever criterion they are judged." In the *Origin of Species,* Darwin wrote, "rarity is the attribute of a vast number of species of all classes, in all countries." In enumerating the number of insects abundant enough to be considered pests, Smith (*14*) warned "that such species form only an insignificant fraction of the total number of phytophagous insects." Of the 240 species of nocturnal Lepidoptera collected by Williams (*15*), 35 species were represented by a single individual each; 85 (including the 35 above) were represented by five or fewer individuals; 115 by ten or fewer; and 205 by 100 or fewer individuals; therefore, there were only 35 species with over 100 individuals. Further data in support of rarity is found in Dunn's (*16*) work with Panamanian snakes; he reports that "about 1/10 of the species make up 1/2 of the individuals in the snake populations."

Many of the abundant species would not be classed as abundant if they were compared to their food source. For example, many species of insects that are easily captured in the field are rare if they are sought on their host plant or if their biomass is compared with the biomass of the plant or animal upon which they feed.

One of the dynamic relationships in the community and ecosystem is the food chain, because animals must seek food to live. Elton (*17*) stated that the "whole structure and activities of the community are dependent upon questions of food-supply." What proportion of animals feed on living, as opposed to nonliving, matter? In nature, the majority of all animals may be classified as herbivore (grazer on living plants), parasite, or predator; few species are truly saprophytic. Though many animals are associated with dead plant matter, these animals are not saprophytes but are herbivores feeding on bacteria, fungi, and other minute organisms in the decaying matter. Jacot (*18*) stated, "I am quite certain that perhaps as much as one half of the Oribatoidea are not saprophytic. Their function is feeding on fungi." Overgaard (*19*) reported that the evidence suggested that nematodes feed not upon humus but upon plant roots, fungi, bacteria, and other animals. Speaking similarly about saprophagous insects, Chapman (*20*) noted that, "These are usually designated as those feeding upon decaying and fermenting matter. It is evident at the start that these insects live in media which may be teeming with microorganisms, and that the decaying material is the medium upon which the microorganisms live." *Drosophila* depend upon yeasts and other microorganisms present in decaying fruit (*21*).

Genetic Feedback

Stability and constancy are characteristics of natural populations; in many hosts there are resistant factors that limit any severe attack of feeding species, and most animals feed on living matter. These seemingly diverse factors are related and are the foundation of the mechanism for population regulation which I termed "genetic feedback" (*22*). Population numbers (herbivore, parasite, or predator) are regulated in this way: high herbivore densities create strong selective pressures on their host-plant populations; selection alters the genetic makeup of the host population to make the host more resistant to attack; this in turn feeds back negatively to limit the feeding pressure of the herbivore. After many such cycles, the numbers of the herbivore populations are ultimately limited, and stability results.

Through the functioning of the genetic feedback mechanism, resistant factors in a given plant can be used to control a parasite which feeds on it. For example, on a susceptible plant genotype the animal population feeding heavily may be reproducing at a rate of two offspring per individual in the population. Under these conditions, the animal population would increase rapidly and would soon cause severe damage to the plant population by overfeeding. On the other hand, if resistant genes were concentrated in the plant population so that only resistant genotypes dominated, animal reproduction might be at a rate of one-half offspring per individual in the population. Then the animal population would decrease, and the damage to the plant population would be kept to a minimum.

An example of this type of change in a plant population took place in the Kansas wheat crop which was susceptible to the Hessian fly (*Phytophaga destructor*). As a result of the low resistance of the wheat to attack, Hessian fly populations increased, and the wheat crop suffered damage. With R. H. Painter's (*23*) development of a resistant variety of wheat, reproduction on the resistant wheat dropped to less than one per individual, and soon the fly population declined to a low level. Thus, by manipulation of the genotypes found in the wheat and not of the quantity of wheat, the fly population was controlled.

Although the interactions of wheat and Hessian flies can be considered to be man-made, we find evidence that under natural conditions biotic communities develop their own controls. In fact, populations in nature are usually regulated by several mechanisms that operate interdependently. These include not only genetic feedback but competition, parasitism, predation, and environmental heterogeneity.

This can be illustrated by a study of what might happen when a new animal species is introduced into a biotic community and becomes established on a plant. At first, the animal increases rapidly on its new plant host and reaches outbreak level. Under these conditions, competition for food among the animals is intense. In addition, the severe feeding pressure tends to eliminate many of the plants; this results in an altered distribution of the plants. With the plant hosts more sparsely distributed, the animal has increased difficulty in locating hosts, and some hosts have time to grow, reproduce, and maintain themselves.

Thus, at the early stages of interaction between animal and plant, competition and environmental heterogeneity along with the pressure from parasites and predators frequently limits the numbers of the animal and prevents the complete destruction of the host. If these factors are successful, then slower-acting genetic change and evolution can take place.

Genetic change in the plant takes several generations because plant response to selective pressure exerted by the animal is slow. When a large animal population exerts severe feeding pressure on the plant population, large numbers of plants are destroyed. The first plants destroyed are primarily those most susceptible to the feeding pressure of the animal; the surviving plants generally carry one or more resistant genes. Under natural conditions, the evolution that occurs in the plant would rarely be caused by mutation but would be due to a recombining and concentrating of the genes already existing at low frequencies in the plant population. Resistance in the host is generally polygenic, and evolution proceeds slowly as genes are recombined in individuals and concentrated in the population. For example, there might be 20 loci (two genes per locus) in the host plants for some resistant character such as hardness. As susceptible genes at each locus are slowly replaced with resistant genes, the amount of resistance gradually increases.

When we look at the problem from a different angle, we find that the change and resistance in the plant can be measured as a response in the survival of the animal; that is, the number of eggs produced, the rate of development and growth, mortality, and longevity of the animal might all be influenced by increasing the concentration of resistant genes in the plant host. At a critical level of resistance in the host, the low birth rate and high death rate in the animal population would result in a significant decrease in numbers, and eventually the population would be sparse. Then with animal numbers rare in relation to those of the plant host, the animal population would only be removing "interest" (excess individuals or energy, or both) from the plant population, and relative equilibrium would exist between plant and animal. The animal would no longer be removing "capital" (those individuals or that energy, or both, needed for maintenance of the plant population). Evolution of this kind with a balance between supply and demand is possible with the genetic feedback mechanism.

Feeding pressure of herbivores, parasites, and predators on their plant or animal host may be limited by various protective mechanisms in their host, but there are examples of subtle genetic changes that significantly affect the survival of the animal that uses the host plant or host animal for food. For instance, when young pea aphids (*Acyrthosiphum pisum*) were placed on a common crop variety of alfalfa (*Medicago sativa*), they produced a mean of 290 offspring in 10 days, whereas the same number of aphids for a similar period on a resistant alfalfa variety produced a mean of only two offspring (*24*). In another example, the mean rate of oviposition (eggs per generation) of the chinch bug (*Blissus leucopterus*) on a susceptible strain of sorghum (*Sorghum vulgare*) was about 100, whereas on a resistant strain the mean oviposition was less than one (*25*). In both, reproduction in the animals feeding on the resistant plant hosts decreased more than 99 percent. This reduced reproduction obviously would have dramatic effects on the population dynamics of the feeding animal populations.

Resistance is effective in limiting animal numbers, and evidence suggests that it plays a dominant role in controlling populations in nature. If so, this would explain why population outbreaks occur frequently in newly introduced species. With little or no resistance, the new species increases rapidly on its susceptible food hosts. Until resistance in the plant host gradually increases, both outbreaks and intense fluctuations will occur. When relative stability is eventually reached and resistance is fully effective, animal numbers will be low. This is one reason why most animals are rare and especially rare relative to their food resource.

In addition, the relative stability and responsiveness of living systems are believed to account in part for the fact that most animals feed on living matter. The interaction between eating and eaten species and genetic feedback within the community form a complex but fully responsive system. Living systems, of course, respond to change and can evolve to provide a functional system whereby careful control of supply and demand can be achieved within the community as a whole.

The adaptation of supply by the plant and demand by the animal evolves and in time attains a state of relative balance. The plant host responds and evolves to its attacking animal only if the numbers of the animal are sufficient to exert some selective pressure on the host. This means that the trophic interactions between herbivore and plant, parasite and host, and predator and prey are important in determining the structure of the community. Based on this knowledge, Elton's statement that "the whole structure and activities of the community are dependent upon questions of food supply" takes on great significance in population control.

Parasite-Host Systems

The validity of genetic feedback functioning as a regulatory mechanism in populations was investigated under controlled laboratory conditions (*26*). The premise of the first experiment was that the numbers of the feeding species would be controlled as genetic resistance evolved in the host population. The housefly (*Musca domestica*) was the host species, and a wasp (*Nasonia vitripennis*) was the parasite or feeding species (Fig. 1). These two species were allowed to interact in the experimental unit for 1004 days while host numbers were kept constant and parasite numbers were allowed to vary. The control unit was similar in design, except that hosts for the parasite population came from a population of houseflies that had not been exposed to the parasite. Hosts that survived exposure to the control parasites were destroyed to prevent the control host population from evolving. In both the control and experimental units, all parasites that emerged from their host types were saved and were returned to their respective population cages.

During the period of study, measurable evolution took place in both the host and parasite populations in the experimental unit. The experimental host population became more resistant to the parasite, as evidenced by a drop in the average reproduction of 135 to 39 progeny per experimental female parasite and a decrease in longevity from about 7 to 4 days. Concurrently, the parasite population evolved some avirulence toward its host. As the experiments progressed, selective pressure on the experimental host population declined, and density of the parasite population declined to about one-half that of the control (about 3700 for the control and 1900 for the experimental). The amplitude of the fluctuations of experimental population (Fig. 2) was significantly less than those experienced by the control.

The ecology and evolution of this same parasite and host were investigated in another experiment during which both parasite and host density were allowed to vary. A specially designed cage, consisting of 30 plastic cells joined together to make a multicelled structure, provided space-time structure for normal parasite-host interactions. With this cage, the population characteristics exhibited by the control or newly associated parasite-host system were compared with those of the first

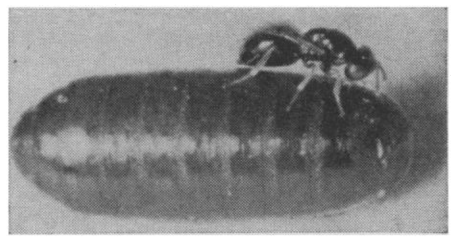

Fig. 1. Wasp parasite and housefly host pupa.

experimental system in which some ecological balance had evolved.

During the 581-day period for the control system and 322-day period for the experimental system, parasite numbers averaged 118 per cell in the control system and only 32 in the experimental system. Host numbers averaged 172 per cell in the control and 462 in the experimental (Fig. 3). Population fluctuations in the control system were severe, whereas in the experimental system they were dampened. The greater stability which the experimental system had already attained enabled it to make efficient use of its environmental resources and in this way increase its chances for survival.

One of the outstanding examples of the genetic feedback functioning in a natural population is the relationship of myxomatosis virus and European rabbits in Australia. After its introduction there in 1859, the European rabbit (*Oryctolagus cuniculus*) population increased to outbreak levels within the following 20 years (*27*). To reduce the density of the rabbit to a harmless level, the myxomatosis virus obtained from South American rabbits was introduced into the rabbit population. In essence, this action was analogous to introducing a new virus species into another community, for the myxomatosis virus and European rabbit had never been associated before. The virus spread rapidly in the rabbit population and immediately reached outbreak levels. During the first epidemic, myxomatosis was fatal to about 98 percent of the rabbits; the second epidemic resulted in about 85 percent mortality; and by the sixth epidemic, mortality was about 25 percent (*28*). Today the virus is less effective than it had been but is still taking its toll of rabbits. Fenner summarized the situation by stating, "We could then envisage a climax association in which myxomatosis still caused moderately severe disease with an appreciable mortality, much as smallpox does in human communities. The reproductive capacity of the rabbit is such that this sort of disease need not seriously interfere with its population size."

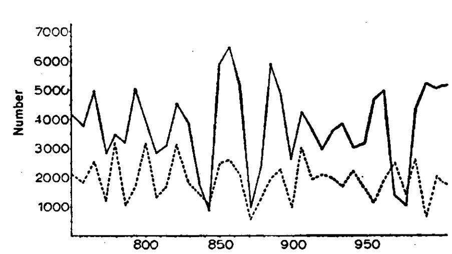

Fig. 2. Population trends of parasite populations for the last 254 days of the 1004-day period of two laboratory parasite-host systems. Solid line, control parasite; dashed line, experimental parasite.

In this adjustment between virus and rabbit, attenuated genetic strains of virus evolved by mutation and tended to replace the virulent strains (*29*). In addition, passive immunity to myxomatosis is conferred to kittens born of immune does (*30*). Finally, a genetic change has occurred in the rabbit population, and this has provided intrinsic resistance to the myxomatosis virus (*31*). This clearly illustrates the alternate functioning of the feedback of density, selection, and genetic change which has in turn altered the density of both populations. There was some similarity between the virus-rabbit relationship, the laboratory wasp-fly relationship, and the type of evolution which took place. In the virus-rabbit association, most of the evolution occurred in the parasite, whereas in the wasp-fly association most of the evolution took place in the host.

Transmission of the myxomatosis virus depends upon mosquitoes (*Aedes* and *Anopheles*) that feed only on living animals (*32*). Rabbits infected with the virulent strain of virus live for a shorter period of time than those infected with the less virulent strain. Because rabbits infected with the less virulent strain live for longer periods of time, mosquitoes have access to that virus for longer periods of time. This gives the avirulent strain a competitive advantage over the virulent strain. In addition, in regions where the avirulent strain is located, rabbits are more abundant, and this allows more total virus to be present than in a comparable region infected with the virulent. Thus, the virus with the greatest rate of increase and density within the rabbit is not the virus selected for, but the virus with demands balanced against supply has survival value in the ecosystem.

Another example of how population regulation evolves from one dominant mechanism to another can be found in

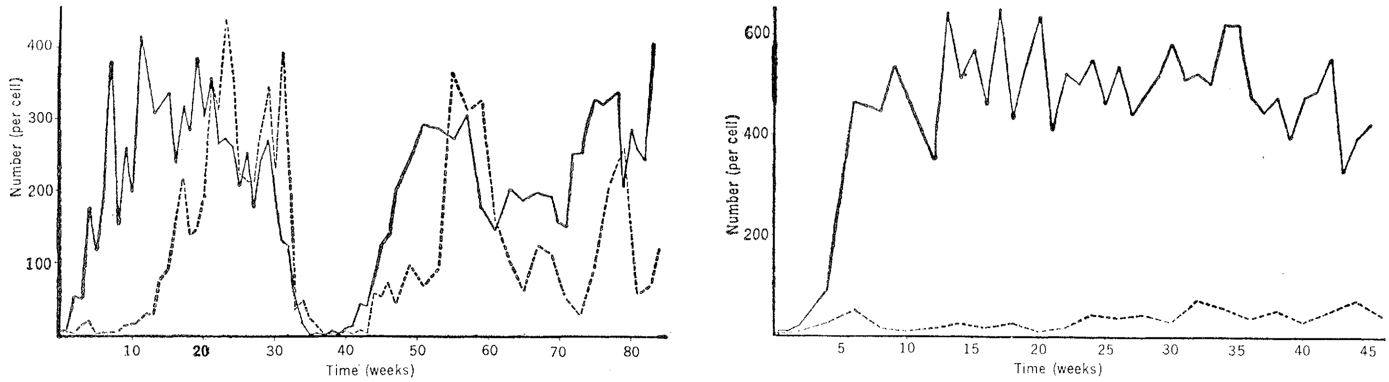

Fig. 3. Population trends of control parasite-host system (left) and experimental system (right) that has evolved some stability or balance between the interacting species. Solid line, mean number of hosts; dashed line, mean number of parasites per cell in 30-cell population cages.

a comparison of the results of the initial interaction of the parasite-host study with the results of these populations after they had interacted for 1004 days (*26*). Initially, parasite density in the experimental system averaged about 3700. Although the density of the parasite population fluctuated, the mean reproduction of a parasite pair at the carrying capacity of the environment would have to be two or a pair with births equaling deaths. Because the experimental parasites produced about 135 progeny per female, 133 of these would have to die each generation to leave a single parasite pair surviving to replace the parent pair. Early in the experimental system, competition was primarily responsible for limiting parasite numbers and causing the death of 133 of the 135 offspring produced per female. The decline from 135 to 39 progeny per female of the experimental parasite meant that the loss of 96 progeny was due to changes brought about by genetic feedback. To maintain the population at this lowered reproductive rate, only 37 of the progeny could be lost to competition. Thus, competition in the beginning was the dominant control mechanism operating in the experimental system, but genetic feedback became dominant with time and through evolution.

Competition and Coexistence

When we consider how the genetic feedback mechanism functions, it seems logical to apply it to situations in which competing species might evolve to occupy the same niche. Competition here refers to species at the same time and place which share the same essential resource in short supply (*2*). Niche is defined as an animal's "place in the biotic environment, its relationship to food and enemies" in the community (*17*).

Competing species seeking the same plant, prey, or host can coexist if their numbers are controlled by genetic feedback. For example, let us assume that two aphid populations feed on sap from the same plant species. The two aphid species can coexist because the more abundant aphid species will eventually be controlled through the processes of genetic feedback. The amount of change that occurs in the characteristics of the plant for protection against the feeding pressure of the animal is dependent on density. Because more plants are selectively destroyed by the abundant aphid, the resistant polygenic factors effective against the abundant aphid would increase in the plant population. This means that the abundant aphid ultimately will be more limited by changes in the plant than the sparse aphid will. Thus, the numbers of both competing aphid populations are controlled by differential evolution of the plant relative to each population. Results of field studies with two aphid species that attack alfalfa (*33*) suggest that two competitive animal species seeking the same food host can be differentially influenced by evolution in the plant.

Genetic feedback may also operate in yet another way to enable two species to coexist and utilize the same thing (food, space, and so on) in the ecosystem. In this case, let us assume that both species are fairly evenly balanced in their competitive ability and that species A is only slightly superior to species B. As the numbers of A are increasing, the numbers of B will be declining and becoming sparse. The abundant individuals of species A must contend principally with intraspecific competitive selection because there is a greater chance for individuals of this species to interact with their own kind. Haldane (*34*) pointed out that intraspecific competitive selection is frequently biologically disadvantageous for the species. At the same time, individuals of species B are contending primarily with interspecific competitive selection. Thus, under this selection species B would evolve and improve its ability to compete with its more abundant cohort species A. As species B improves as a competitor, its numbers increase, and finally B becomes the more abundant species. Then the dominant kind of competition (interspecific or intraspecific) affecting each species is reversed. After many such oscillations and with each oscillation decreasing in intensity, a state of relative stability should result.

This idea—that intraspecific selection on the dominant species and interspecific selection on the sparse species favors the sparse species—was tested successfully with the housefly and blowfly (*Phaenicia sericata*) in a multicelled cage (*35*). In another population system (surviving for 160 weeks or 80 fly generations), there was a persistent alternation of dominance of first the blowfly and then the housefly. A genetic check on the fly populations showed that the currently dominant species remained genetically static, while the sparse species or "underdog" evolved to become the better competitor and dominant species. Although there has been an oscillation in dominance, no damping of the fluctuation has been noted to date.

Conclusion

The importance of the genetic feedback mechanism as a regulatory system in communities is substantiated by its wide application to such diverse interacting population systems as herbivore and plant, parasite and host, predator and prey, and interspecific competitor systems. The real significance of this mechanism for population regulation lies in the fact that it has its foundation in evolution. Population regulation by genetic feedback supports Emerson's (*36*) view that evolution in natural populations is toward homeostasis (balance) within populations, communities, and ecosystems.

Students of population ecology and especially of parasitology and epidemiology generally accept the fact that evolutionary trends in relationships of parasite and host are toward balance. The deductive basis for this generalization rests on the ecological principle that disharmony results in serious losses to both parasite and host. Large numbers of fatal infections in the host population eventually lead to host extinction which in turn brings about the extinction of the parasite. The success of any living population is measured by its relative abundance and distribution as well as its ability to survive in time.

Homeostasis, in herbivore-plant, parasite-host, and predator-prey species and among other community members in general, results in improved survival of the community system. The evolved balance in supply and demand achieved by the feeding species and its host establishes a sound economy for the community. This, of course, enables the community to make effective use of the resources available to it.

Increased species diversity in a community is due in part to community homeostasis. The genetic integration of interspecific competitors which makes possible the use of the same resource by competing species and enables them to occupy the same niche contributes to greater species diversity. The increased network of interactions within the community, resulting from a greater number of species present, further contributes to community homeostasis.

With more knowledge concerning the regulation of natural populations, man will be in a better position to control the pests on his food crops and the parasitic diseases of mankind. This will also help conserve the millions of living species which are vital for the functioning of the vast living system of which he is a part.

References and Notes

1. D. Lack, *The Natural Regulation of Animal Numbers* (Clarendon Press, Oxford, 1954).
2. A. MacFadyen, *Animal Ecology* (Pitman, London, 1957).
3. F. A. Urquhart, *Changes in the Fauna of Ontario* (Univ. of Toronto Press, Toronto, 1957).
4. L. L. Snyder, in *Changes in the Fauna of Ontario*, F. A. Urquhart, Ed. (Univ. of Toronto Press, Toronto 1957), pp. 26–42.
5. ——, *Ontario Birds* (Clarke, Erwin, Toronto, 1951).
6. O. Kalela, *Bird-Banding* **20**, 77 (1949).
7. P. P. Feeny, thesis, Oxford University (1966).
8. J. M. Kingsbury, *Poisonous Plants of United States and Canada* (Prentice-Hall, Englewood Cliffs, N.J., 1964).
9. T. Eisner, *Science* **146**, 1318 (1964).

→ —— and J. Meinwald, *ibid.* **153**, 1341 (1966).

11. B. F. Coon, R. C. Miller, L. W. Aurant, *Pennsylvania Agricultural Experiment Station Report* (1948).
12. J. H. Pepper and E. Hastings, *Montana Agricultural Experiment Station Technical Bulletin 413* (1943).
13. H. G. Andrewartha and L. C. Birch, *The Distribution and Abundance of Animals* (Univ. of Chicago Press, Chicago, 1954).
14. H. S. Smith, *Econ. Entomol.* **28**, 873 (1935).
15. R. A. Fisher, A. S. Corbet, C. B. Williams, *J. Anim. Ecol.* **12**, 42 (1943).
16. E. R. Dunn, *Ecology* **30**, 39 (1949).
17. C. Elton, *Animal Ecology* (Sigwick and Jackson, London, 1927).
18. A. P. Jacot, *Ecology* **17**, 359 (1936).
19. C. Overgaard, *Natura Jutlandica* **2**, 1 (1949).
20. R. N. Chapman, *Animal Ecology* (McGraw-Hill, New York, 1931).
21. M. Demerec, *Biology of Drosophila* (Wiley, New York, 1950).
22. D. Pimentel, *Amer. Natur.* **95**, 65 (1961).
23. R. H. Painter, *Insect Resistance in Crop Plants* (Macmillan, New York, 1951).
24. R. G. Dahms and R. H. Painter, *J. Econ. Entomol.* **33**, 482 (1940).
25. R. G. Dahms, *J. Agr. Res.* **76**, 271 (1948).
26. D. Pimentel and R. Al-Hafidh, *Ann. Entomol. Soc. Amer.* **56**, 676 (1963).
27. D. G. Stead, *The Rabbit of Australia* (Winn, Sydney, Australia, 1935).
28. F. Fenner, in *The Genetics of Colonizing Species*, H. G. Baker and G. L. Stebbins, Eds. (Academic Press, New York, 1965), pp. 485–499.
29. H. V. Thompson, *Ann. Appl. Biol.* **41**, 358 (1954).
30. F. Fenner, *Cold Spring Harbor Symp. Quant. Biol.* **18**, 291 (1953).
31. I. D. Marshall, *J. Hyg.* **56**, 288 (1958).
32. M. F. Day, *J. Australian Inst. Agr. Sci.* **21**, 145 (1955).
33. R. H. Painter, *Proc. Int. Congr. Entomol. 12th* **1964**, 531 (1964).
34. J. B. S. Haldane, *The Causes of Evolution* (Longmans, Green, New York, 1932).
35. D. Pimentel, E. H. Feinberg, P. W. Wood, J. T. Hayes, *Amer. Natur.* **99**, 97 (1965).
36. A. E. Emerson, in *Principles of Animal Ecology*, W. C. Allee, A. E. Emerson, O. Park, T. Park, and K. P. Schmidt, Eds. (Saunders, Philadelphia, 1949), pp. 640–695.

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Air Pollution: Time for Appraisal

Abel Wolman

Some three-quarters of a century ago, Sir Edwin Chadwick of London, England, proposed a project "to draw down air, by machinery, from the upper couches or strata of air and distribute it through great cities, like the Metropolis." He was prompted to suggest this program "on the repeated sight of a great blanket of fog spread over the Metropolis" and even suggested the formation of a "Pure Air Company, which would engage to draw the air from a suitable height . . . and distribute it into houses . . . and do it with a profit, at a very low rate" (*1*).

Needless to report, the company was not formed. London continues to this day to struggle with the fog and its consequences, despite repeated legislative proposals to control it. In the Los Angeles area, however, similar proposals for one form or another of forced drafts have found their way into scientific journals in the 1960's, again without serious attempts at implementation.

The awareness of the air pollution problem has been intensified in the official and public mind by the dramatic episodes in Donora, Pennsylvania, the Meuse Valley in Belgium, in London, and in Los Angeles. In the United States, this dramatic interest was translated into federal legislation in 1963 and further clarified by the Clean Air act of 1967. Simultaneously, official evaluations have come off the press in large numbers. It may therefore be assumed, with ample justification, that the air is variously polluted, that the public is alerted to its significance, and desires that the moves toward cleaner

The author is emeritus professor of sanitary engineering and water resources development at Johns Hopkins University, Baltimore. This article is adapted from the Scientific Research Society of America and Procter prize address given 29 December 1967 at the AAAS meeting in New York.