Animal Population Regulation by the Genetic Feed-Back Mechanism
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ANIMAL POPULATION REGULATION BY THE GENETIC FEED-BACK MECHANISM*

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Natural population regulation has its foundation in the process of evolution. One such process described herein is called the genetic feed-back mechanism. This mechanism through genetic evolution integrates herbivore and plant, parasite and host, and predator and prey in the community. It functions as a feed-back system through the dynamics of density pressure, selective pressure, and genetic changes in the interacting populations. Density influences selection; selection influences genetic make-up; and in turn, genetic make-up influences density. The actions and reactions of the interacting populations in the food chain cycling in this mechanism result in the evolution and regulation of animal populations.

E. B. Ford (1930 and 1931) was the first to point out the importance of genetic changes in population dynamics and specifically as a cause of population fluctuations. He proposed that "numerical increase inevitably prepares the way for reduction, and the reverse; so giving rise to fluctuations in numbers, with alternating periods of high and low variability" (Ford, 1931). During mass increases caused by the changing environment variability increases, and many inferior genetic types result. "These are eliminated, and the numbers reduced when conditions become more rigorous again" (Ford, 1956). A genetic system of population regulation suggested by Franz (1949) proposes that population waves are generated both by inbreeding and the fixation of deleterious genes during mass increases. These are followed by declines initiated by the consequent weakening of the population. The relation between density regulation and natural selection was explored by Haldane (1956), who showed that genetic changes and adaptation for some environmental selective factor may cause a population to increase or decrease to a new stable level. Chitty (1957 and 1960) presented evidence to support the fact that changes in the genetics and viability of field mice are responsible for the population cycles of this species. That "the role of individual differences in population dynamics has been relatively neglected" was emphasized by Wellington (1957), and in a later paper

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(1960) he explained how individual differences might influence populations. Recently, Turner (1960) proposed that population outbreaks follow cross-breeding between isolated inbreeding populations. From this evidence, it is clear that genetic changes play some role in population regulation, but the manner in which they function is not known. The genetic feed-back mechanism may provide an insight into the nature of this problem.

When a species population enters a new biotic community in which it is not integrated and in which no ecological barrier exists, it reaches outbreak levels a short while after introduction. The following cases amply illustrate this point: Japanese beetle introduced into the United States (Smith and Hadley, 1926), European rabbit introduced into Australia (Stead, 1935), and European gypsy moth introduced into eastern United States (Forbush and Fernald, 1896). The Hessian fly was still another species to reach high density within a short time after its introduction into the United States. In this instance, through genetic change of wheat plants and the consequent alteration of food quality, the density of the herbivore or Hessian fly population was controlled. Previous to 1942, when only susceptible varieties of wheat were grown in Kansas, the Hessian fly population occurred in large numbers across Kansas. When the flies were fed on certain resistant varieties, they suffered high mortality in the larval populations from time of hatching to pupation. If development did take place, growth of the larvae was slow on these resistant varieties (Painter, 1951). When the resistant varieties of wheat were introduced into Kansas in 1942, the Hessian fly population declined in a few years to the point where the flies were too scarce to be used in resistant wheat tests (Painter, 1954). Thus, by changing the genetic make-up of the plant population type this herbivore was feeding upon, the animal population was significantly reduced. If the resistant wheat were exchanged for the original susceptible variety, an increase in the Hessian fly population could be predicted.

The interaction of oysters and a pathogen found in the waters off Prince Edward Island further demonstrates the importance of genetic mechanisms in natural populations. Before 1915, oysters in the Malpeque Bay abounded. During 1915, however, the oyster population was struck by disease. Nearly 90 per cent of the oyster population was infected with the yellowish-green pustules during the first wave (Needler and Logie, 1947). The population of oysters decreased rapidly, and by 1926 no oysters were harvested (figure 1). Subsequently in 1929, fishermen noticed an increase in oysters, and nearly 500 barrels were removed during 1930. By 1940 production approached the 1915 level of 4000 barrels (figure 1). An investigation to determine if this increase was due to genetic resistance developing in the oyster led Needler and Logie (1947) to import oyster spat from the disease-free Hillsborough River. Over 90 per cent of the Hillsborough spat died of disease whereas the Malpeque Bay spat treated in a similar manner in the bay survived. Malpeque Bay spat were also compared with Enmore River spat. The Enmore River region received the disease about two years previous to this test.
Under the infective conditions at Enmore only 30 per cent of the Enmore River spat survived whereas 98 per cent of the resistant Malpeque Bay spat survived.

Since the pathogen was not isolated, no tests were made of the parasite to determine if any changes in its reproduction and pathogenicity occurred. The oyster itself, however, was well adapted for rapid evolutionary change when it is considered that an oyster produces nearly 60,000,000 young per year (Galtsoff, 1930). This natural event with oysters showed that parasite pressure resulted in genetic change in the oyster-host, and this regulated the density of the parasite itself.

The myxomatosis outbreak in the rabbit population in Australia is another example of the genetic changes functioning in the adjustment of a natural parasite and host population. At Christmas, 1859, the European rabbit (*Oryctolagus cuniculus*) was introduced into Australia and increased rapidly during the next 20 years (Stead, 1935). The extent of the damage caused by these animals prompted the Australian Government to investigate the possibility of introducing some biotic agent which would reduce the density of the rabbit to a less harmful level. The myxomatosis virus obtained from South American rabbits was introduced into the Australian rabbit population. During the first epizootic, myxomatosis was fatal to between 97 and 99 per cent of the rabbits; the second epizootic resulted in 85 to 95 per cent mortality; and the third epizootic resulted in 40 to 60 per cent mortality (Fenner, *et al.*, 1953). The effect on the rabbit population was less severe with each succeeding epizootic, suggesting that the two populations were becoming integrated and adjusted to one another in the ecosystem.

In this adjustment between virus and rabbit, attenuated genetic strains of virus have evolved by mutation and are tending to replace the virulent strains (Thompson, 1954). In addition, passive immunity to myxomatosis is

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**FIGURE 1. Annual yields of oysters from Malpeque Bay and associated regions off Prince Edward Island.** (After Needler and Logie, 1947.)
conferring to kittens born of immune does (Fenner, 1953). Finally, a genetic change has occurred in the rabbit population, and this is providing intrinsic resistance to myxomatosis (Marshall, 1958). Here definitely is illustrated the functioning of the feed-back of density, selection, and genetic change which has in turn altered the density of both populations. In this particular association, genetic change seems to be taking place in both populations. The virulent genetic strain of virus has an apparent advantage in that it reproduces quickly and can rapidly convert rabbit protoplasm into itself. This is an advantage only when this virus is compared to another which has a slower rate of increase within the rabbit. The virus strains, however, must be evaluated in the ecosystem to determine which strain ultimately has the adaptive advantage. As Li (1955) points out, "adaptability is a response of populations rather than of the individual." If the virulent strain were to become 100 per cent effective in attacking the rabbit in the ecosystem, it would destroy itself by destroying its food supply. A less virulent strain in this case would tend to have greater survival value when the strains are separated diversely in space. The non-pathogenic strain observed in Australia has a selective advantage in this region.

Transmission of the myxomatosis virus depends upon mosquitoes (Aedes and Anopheles) which feed only on living animals (Day, 1955). 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 provides the non-virulent strain a competitive advantage over the virulent strain. In addition, in regions where the non-virulent 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 virus. Thus, the virus with the greatest rate of increase and density within the rabbit is not the virus selected for, but it is the virus whose demands are balanced against supply which has survival value in the ecosystem.

The above mentioned natural populations were regulated by genetic changes operating in the feed-back mechanism. The interactions and trends which operated in this mechanism are translated into biomathematics and explained in the following model using a herbivore-plant system. This proposed feed-back mechanism functions through polygenic action, but to illustrate the process two alleles (A, a) at one locus are adequate. The alleles in various proportions \( p^2 + 2pq + q^2 = 1 \) in the diploid plant determine whether an animal population feeding on the plant increases or decreases, where \( p^2 \) is the proportion of homozygous dominants (AA), \( 2pq \) is the proportion of heterozygotes (Aa), and \( q^2 \) is the proportion of homozygous recessives (aa) in the plant population. The animals distribute themselves equally on the individual plants, and animal reproduction (R) varies according to the plant genotype on which they are feeding. For example, on the AA plant animal reproduction \( (R_p) \) is 2, on the Aa plant animal reproduction \( (R_{pq}) \) is 1, and on the aa plant animal reproduction \( (R_q) \) is 0.5. Thus animal
density (N) at an initial time \((N_{t0})\) increases or decreases in the following generation \((N_{t1})\) depending on the proportion of the AA, Aa, and aa plant genotypes. In this case then

\[ N_{t1} = p^2R_pN_{t0} + 2pqR_pqN_{t0} + q^2R_qN_{t0}. \]

Environmental selective pressure \((S)\), or survival \((1 - S)\), and animal density pressure determines the genotypic proportions of the plant population at the next generation. For this example, environmental selective pressure on the AA plant \((S_p)\) is 0.001, on the Aa plant \((S_{pq})\) is 0.2, and on the aa plant \((S_q)\) is 0.6. In a situation without the animal the "relative fitness" (Li, 1955) of the genotypes is such that the AA plants are better than the Aa plants, and the Aa plants are better than the aa plants \((p^2 > 2pq > q^2)\). Plant tolerance for animal density varies according to plant genotype. At one maximal animal density \((b)\) none of the AA plants survive; at another density \((c)\) none of the Aa plants survive; and still at another density \((d)\) none of the aa plants survive. Pressure by the animal on the AA, Aa, and aa plant genotypes is proportional to animal density as \(\frac{b - N}{b}\), \(\frac{c - N}{c}\), and \(\frac{d - N}{d}\), respectively. Thus the selective pressure against the plant genotypes exerted by animal density and the other environmental factors in the ecosystem is

\[ p^2(1 - S_p) \frac{(b - N)}{b} + 2pq(1 - S_{pq}) \frac{(c - N)}{c} + q^2(1 - S_q) \frac{(d - N)}{d} = G, \]

The total proportion of the plant population surviving is \(G\). Following the selection, the proportions of the surviving plant genotypes are

\[ \frac{p^2(1 - S_p)}{G} \frac{(b - N)}{b} + \frac{2pq(1 - S_{pq})}{G} \frac{(c - N)}{c} + \frac{q^2(1 - S_q)}{G} \frac{(d - N)}{d} = 1. \]

The proportion of the A allele \((p)\) surviving is \(p^2 + \frac{1}{2}(2pq) = p\) and the proportion of the surviving a allele \((q)\) is \(q^2 + \frac{1}{2}(2pq) = q\). By random mating the surviving allelic proportions,

\[ (p + q)^2 = 1. \]

The proportions of the plant genotypes present in the next generation are

\[ p^2 + 2pq + q^2 = 1. \]

For the present example, the maximum animal density \((b)\) for survival of the AA plants is 1075; the maximal animal density \((c)\) for the Aa plants is 1500; and the maximal density for survival of the aa plants is 3000. Logically the genotype destroyed by the lowest maximum animal density is the genotype on which the animal has the highest reproduction rate. If we now imagine 150 animals being introduced into the ecosystem and the proportions of the plant genotypes are .360 AA plants, .500 Aa plants, and .140 aa
plants and the numerical example is fed into a digital computer and cycled through equations 1, 2, 3, 4, and 5 for 100 generations, we find that the animal population fluctuations decrease in amplitude with each generation and stability is reached at an animal density of 795 in 72 generations (figure 2). The genotypic plant proportions at stability are .172 AA plants, .485 Aa plants, and .343 aa plants.

The model was revised and made more complex to allow the genetics of the animal population to vary in addition to density. Changes in the plant population remained similar to those in the previous model. The relation of animal genetics in this system is represented by two alleles (Z, z) at one locus in the animal. The proportions of ZZ are indicated by x^2, of Zz by 2xy, and zz by y^2. It is assumed that the three animal genotypes (ZZ, Zz, zz) reproduce differently on each of the three plant genotypes (AA, Aa, aa).

\[
\begin{align*}
N_{t1p} = N_{to}p^2x^2I_x + N_{to}p^22xyI_{xy} + N_{to}p^2y^2I_y
\end{align*}
\]

and on the Aa plants the animals produce
\[ N_{t1p} = N_{to} 2pq x^2 J_x + N_{to} 4pqxy J_{xy} + N_{to} 2pqy^2 J_y \]

and on the aa plants the animals produce
\[ N_{toq} = N_{to} q^2 x^2 L_x + N_{to} q^2 xy L_{xy} + N_{to} q^2 y^2 L_y. \]

Animal density for the following generation is
\[ N_{t1} = N_{t1p} + N_{t1pq} + N_{t1q}. \]

The proportions of the surviving animal genotypes \( x^2 \), \( 2xy \), and \( y^2 \) respectively are
\[ \frac{N_{to} p^2 x^2 I_x + N_{to} 2pq x^2 J_x + N_{to} q^2 x^2 L_x}{N_{t1}} \]
\[ + \frac{N_{to} p^2 2xy I_{xy} + N_{to} 4pqxy J_{xy} + N_{to} q^2 2xy L_{xy}}{N_{t1}} \]
\[ + \frac{N_{to} p^2 y^2 I_y + N_{to} 2pqy^2 J_y + N_{to} q^2 y^2 L_y}{N_{t1}} = 1. \]

The proportion of the \( Z \) allele (\( x \)) of the animals surviving is \( x^2 + \frac{1}{2}(2xy) = x \) and the proportion of the surviving \( z \) allele (\( y \)) is \( y^2 + \frac{1}{2}(2xy) = y \). By random mating the surviving allelic proportions of the animal,
\[ (x + y)^2 = 1 \]

the proportions of the animal genotypes in the next generation are
\[ x^2 + 2xy + y^2 = 1. \]

Then with the animal density (\( N_{t1} \)) we pass through equations 2, 3, 4, and 5 as in the first model. Starting with the conditions listed in figure 3 and cycling these data through equations 6, 7, 8, 9, 2, 3, 4, and 5, population cycles result for 2100 generations. Only 100 generations are graphed in figure 3. During the 2100 generations the amplitude of the cycles decreased by only four individuals. By extrapolation, the population would continue to cycle for about 20,000 generations.

Starting with conditions similar to those listed in figure 3, except that \( S_{pq} = 0.2 \) and \( S_q = 0.6 \), the population stabilizes in 100 generations, and there results a population trend similar to that graphed in figure 2.

Numerous other models were constructed. In one the animal passed through ten generations while in the plant there was one generation; in another, animal reproduction was made density-dependent, and in another, plant density varied in addition to its genetic changes. The feed-back mechanism regulated in all cases.

A longer time is required for the model to stabilize when the starting animal density is relatively far from the stable value, when the starting genotypic proportions are far from the stable values, and if the differences in either the environmental selective pressure or the maximum animal densities
FIGURE 3. Animal population cycles resulting from the interaction of plants and animals in the genetic feed-back mechanism. Starting conditions: \( p^2 \) (.172), \( 2pq \) (.485), \( q^2 \) (.343), \( x^2 \) (.250), \( 2xy \) (.500), \( y^2 \) (.250), \( N_0 \) (400), \( S_p \) (.001), \( S_{pq} \) (.1), \( S_q \) (.2), \( b \) (1075), \( c \) (1500), \( d \) (3000), \( I_x \) (2.8), \( I_{xy} \) (2.2), \( I_y \) (.8), \( J_x \) (1), \( J_{xy} \) (1), \( J_y \) (1), \( L_x \) (.1), \( L_{xy} \) (.4), and \( L_y \) (1.1).

for the survival are small between each plant genotype. For example, using 0.001, 0.100, and 0.200 for environmental selection pressures for the respective genotypes, AA, Aa, and aa, has greater instability than larger differences such as 0.001, 0.200, and 0.600, respectively. Similarly the maximum densities, 1000, 2000, and 3000, contribute less stability than larger differences such as 1000, 3000, and 9000. Any change occurring in the environmental pressure, in the animal reproduction on the plant genotypes, or in the maximum animal density for the survival of each genotype will change the stabilizing density of the animal to a new level or change the stable values for both the animal density and the plant genotypic proportions.

The feed-back mechanism will regulate under any number of combinations of starting conditions if (1) animal reproduction on the AA plant is greater than that on the aa plant, (2) the animal reproduces greater than one on any one of the genotypes and less than one on another, and (3) the AA plants have a greater survival value than the aa plants in the ecosystem without the animal. Conditions with the homozygous plant genotypes, of course, can be reversed.

The biomathematical models allowed simplification and thus a better understanding of the functioning and action of the feed-back mechanism. In nature the feed-back mechanism would be based most likely on a multifactorial system instead of the unifactorial genetic system used in the models. A multifactorial system would have greater flexibility, and, of course, greater complexity. Obviously the evolution of population regulation in nature would be most difficult to follow because of the nearly infinite number...
of changing environmental factors. The addition of these other factors will
not change the course of evolution in population regulation, but these fac-
tors will make the course devious.

With the biomathematics as a base of understanding the function of the
mechanism, further details are needed to relate it to biological systems. In
all cases, the density of the herbivore populations is a function of both
birth rate and death rate, excluding dispersal. Both birth rate and death rate
of the herbivore are a function of the quality of food provided by the plant.
The food quality is a function of the genetic characteristics of the plant
population which in turn are a function of the selective pressure exerted on
the plant by the herbivore. Therefore, herbivore density at an initial time
determines herbivore density at some future time. So the cycle continues
with the density-dependent mechanism regulating the size of the herbivore
population.

It is natural that animal reproduction on the AA plants in the models is
greater than that on the aa plants and at the same time the AA plants pos-
sess a greater survival value than the aa plants in the ecosystem without
the animal. If the animal-resistant aa plant possessed greater survival value
than the animal-susceptible AA plants, then the animal could not have es-
tablished itself because of this initial ecological barrier. Whenever the ani-
mal is absent or at low density, the plant population gradually shifts until
there exists a higher proportion of AA plants than aa plants. In the eco-
system without the animal, the AA plant has an advantage because it has
greater survival value than the aa plant. In contrast, when animal density
is high, the aa plant has greater advantage than the AA plant because it is
more resistant to animal attack.

Man's selection, which operates to produce plants and animals adapted to
artificial environments, has sacrificed characters which are necessary to
the animals if they are to compete and survive with natural forms in the wild
(Srb and Owen, 1953). Because the resistant genes which would allow a
plant or animal to survive in an ecosystem that now includes some attacking
animal were existing at low frequency in the original ecosystem, it must be
assumed that these resistant genes at high frequency are to some extent
disadvantageous—if this were not so, then the genes would have been com-
mon in the population in the original ecosystem (Crow, 1957).

Organisms only maintain characters important to their survival. Degenera-
tion of skin pigmentation and eyes in cave fishes results from decreased
selection for these characters and increased selection for sensory barbels
and other characters which lead to better adaptation to the cave environment
(Hubbs, 1938). An organism adapting itself to some special feature of the
environment, like resistance to animal attack, must sacrifice certain organs
or functions for the greater efficiency of others (Huxley, 1943). This effi-
ciency is basic to the economy of the organism itself. Because the domi-
nant selective pressures receive special attention, they tax the other sys-
tems of the organism for support.
Then with relaxation of the new selective pressure, a reversion to original genetic type will result, following the principle of "genetic homeostasis" (Lerner, 1954). Darwin (1859) was aware that domesticated animals if not under continuous selection tended to revert to original wild stocks. Examples of reversion following the withdrawal of selective pressures on laboratory populations are numerous. Mather and Harrison (1949) and Streams (1960) found that Drosophila selected for high bristle number tended to return to their original low bristle number when selection ceased. Resistance to insecticides by house flies was lost when this selective pressure was removed from the populations (Pimentel et al., 1953; Varzandeh et al., 1954; Barbesgaard and Keiding, 1955). Populations of microorganisms lost their high level of resistance to drugs when cultured under normal conditions without the drug (Cardot and Laugier, 1923; Morgenroth, 1924; Dettwiler and Schmidt, 1940; Schmidt et al., 1942; Davies and Hinshelwood, 1943; Fulton and Yorke, 1943).

The genetic change in the plant necessary to reduce the herbivore population itself is determined by the nature of the environment. When environmental pressure is severe then only slight genetic change is needed in the plant to reduce the herbivore population. However, when the environment is highly favorable for the herbivore, the pressure on the plants becomes more intense, and the change in the plant has to be significant to reduce the herbivore population to a point of balanced supply and demand.

The rabbit-virus model mentioned earlier presents evidence concerning the question of differential rates of evolution between the eaten and eating species—this is also true of the interactions between cynipids and their oak-tree hosts. Because cynipids pass through more generations than oak trees per unit time, a faster rate of evolutionary change in the cynipids is possible than in oak trees. Similarly, the myxomatosis virus has a greater capacity for change in a short period when compared with rabbits. Natural selection does not favor the cynipids or virus which evolves and destroys its host, but favors the cynipids or virus which allows survival of its host. Through individuals and colonies selection favors the balanced supply-demand system. The evolution of the virus-rabbit association substantiates this fact.

In some cases where animal pressure is severe and the animal must contend with competitive and other environmental factors, the plant may evolve faster than the animal. This, however, is not a requisite for the successful operation of the system. Evolution in each animal is toward survival and the leaving of reproductively successful progeny. Beyond a certain point, the animal gains no advantage in overcoming the plant's resistance to leave more progeny. Selection does not favor a high reproductive rate per se but a reproductive rate profitable from the standpoint of producing successful progeny. Fair apportionment of nutriment must be made between that devoted to maintenance of the parent and production of young (Fisher, 1930) and in some cases maintenance of these young upon birth. Therefore, any
increase in fertility is made at the expense of other survival traits (Smith, 1954; Cole, 1957). Selection for lower reproductive rates has been documented by Lack (1956). He found that in "Switzerland, the alpine swift (Apus melba) normally lays three eggs"; the number of young which fly from broods initially consisting of one, two, three, and four young was respectively, 1.0, 1.7, 2.4, and 2.2. Thus, the largest brood (four) produced fewer successful young than the smaller brood of three.

Selection for the most productive birth rates, not necessarily the highest, occurs in family units (Birch, 1960; Emerson, 1960), colonies (Emerson, 1958), and individuals (Root, 1960). Fundamental to optimal reproduction is the supply and demand economy which exists between individuals or colonies of the herbivores and plants, parasites and hosts, and predators and prey. On partially isolated trees an insect colony which evolves to reproduce at a high rate by converting the trees into insect protoplasm at a rapid rate may destroy the trees and in the end produce fewer successful young than another colony with a lower reproductive rate (Wright, 1960). Viruses and bacteria, which attack relatively long-lived plants and animals, do not evolve rapidly to high reproductive types and greater pathogenicity. Thus, the drift of evolution is toward optimal reproductive types which will provide a balanced economy of supply and demand between the eating and eaten species.

Population birth rate of the animal is influenced either by alterations in the quality of the food or alterations in the intrinsic nature of the eating individuals themselves. Beadle (1945) has experimentally developed genetic strains of Neurospora which were unable or inefficiently able to synthesize various necessary vitamins and amino acids from a minimal medium. Some of the genetic strains were unable to grow while others grew at reduced rates on such medium. Thus, by altering the genetic make-up of the individual and correspondingly the biochemical structure of the organism, its functional rate of growth can be changed, and, of course, this will influence the dynamics of such a population.

The feed-back mechanism is in accord with the fact that the majority of animal species are rare in nature (Darwin, 1859; Andrewartha and Birch, 1954; Milne, 1960). The herbivore, parasite, or predator population cannot be abundant compared with its respective plant, host, or prey population. In the balanced economy, the eating species feed on only the interest (excess individuals of the eaten population) and do not touch the capital (those individuals necessary for the maintenance of the population of the eaten species).

That the genetic feed-back mechanism functions as a regulatory system in herbivore-plant and parasite-host relationships is supported by evidence from the biomathematics of population dynamics and studies of natural populations. The principles of the mechanism also apply to predator-prey systems. The importance of the feed-back mechanism as a regulatory system is substantiated by its wide application to such diverse interacting sys-
tems; however, the significance of the mechanism lies in the fact that it has its foundation in evolution. The mechanism follows Dr. A. E. Emerson’s view that evolution is toward increased homeostasis within populations and the ecosystem (Emerson, 1960).

The task which lies ahead is to determine how the feed-back mechanism functions in regulating natural populations and what its relationship is to the other regulatory theories like “competition” (Nicholson, 1933; Varley, 1947; Solomon, 1949; DeBach, 1958; Morris, 1959; Holling, 1959; Watt, 1959), and “environmental randomness” (Thompson, 1929; Andrewartha and Birch, 1954; Milne, 1957). I do not propose that the feed-back mechanism is the only means of population regulation, nor that this mechanism is independent of the “competition” and “environmental randomness” ideas.

The three are interdependent, and I suspect that upon the introduction of a new animal type into a new ecosystem there is an evolution of regulation from both the “competition” and “environmental randomness” conditions to the feed-back mechanism. That is, before sufficient change takes place in the eating population and eaten population, the principal means of regulation is through “competition” and “environmental randomness.”

In well-designed experiments lie the challenge and necessary evidence concerning the various control mechanisms. The validity of the feed-back mechanism is under experimental investigation both in the laboratory and field by myself and students of ecology at Cornell University.

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SUMMARY

That a genetic feed-back mechanism functions to regulate populations of herbivores, parasites, and predators is supported by evidence from the biomathematics of population dynamics and studies of natural populations. The mechanism functions as a feed-back system through the dynamics of density pressure, selective pressure, and genetic changes in interacting populations. In a herbivore-plant system, animal density influences selective pressure on plants; this selection influences genetic make-up of plant; and in turn, the genetic make-up of plant influences animal density. The actions and reactions of interacting populations in the food chain cycling in the genetic feed-back mechanism result in the evolution and regulation of animal populations.
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