Population Cycles in Small Rodents

Demographic and genetic events are closely coupled in fluctuating populations of field mice.

Charles J. Krebs, Michael S. Gaines, Barry L. Keller,
Judith H. Myers, Robert H. Tamarin

Outbreaks of small rodents were recorded in the Old Testament, in Aristotle's writings, and in the pages of European history. Charles Elton (*1*) summarized the colorful history of rodent plagues, and described the general sequence of outbreaks, from rapid multiplication to the destruction of crops and pastures, and the decline of the plague into scarcity. This cycle of abundance and scarcity is a continuing rhythm in many small rodents, although not all high populations reach plague proportions. The population cycles of small rodents have always been a classic problem in population ecology, and speculation on the possible causes of rodent outbreaks has long outstripped the available scientific data. Both for practical reasons and because of our innate curiosity we would like to understand the mechanisms behind the rise and fall of these rodent populations.

Population cycles present an ideal situation in which to study population regulation. One question that has occupied biologists since the time of Malthus and Darwin has been this: What stops population increase? Cyclic populations, which follow a four-step pattern of increase–peak–decline–low, are thus useful in presenting a sequence of contrasting phases and then repeating the phases again and again. In small rodents the period of this cycle (*2*) is usually 3 to 4 years, although 2-year and 5-year cycles sometimes occur.

Since Charles Elton first kindled interest in population cycles in 1924 (*3*), a great amount of effort has been expended in trying to describe and to explain these fluctuations. Two general facts have emerged from this work. First, many species of microtine rodents (lemmings and voles) in many different genera fluctuate in numbers. These species have not all been studied for long time-periods, but it is striking that in no instance has a population been studied in detail and found to be stable in numbers from year to year. Second, these cycles are found in a variety of ecological communities: lemmings on the tundras of North America and Eurasia, red-backed voles in the boreal forests of North America and Scandinavia, meadow voles in New York, field voles in coastal California, New Mexico, Indiana, Britain, Germany, and France. The list grows long and includes rodents from north temperate to arctic areas. No cyclic fluctuations have been described for tropical rodents or for South American species, but almost no population studies have been done on these species.

The phenomenon of population cycles is widespread but there is disagreement about whether we should seek a single explanation for the variety of situations in which it occurs. We adopt here the simplest hypothesis, that a single mechanism underlies all rodent cycles, from lemming cycles in Alaska to field vole cycles in southern Indiana. The only empirical justification we can give for this approach is that demographic events are similar in a variety of species living in different climates and in different plant communities; but the expectation of a single explanation for rodent cycles is only an article of faith.

There are two opposing schools of thought about what stops population increase in small rodents. One school looks to extrinsic agents such as food supply, predators, or disease to stop populations from increasing. The other looks to intrinsic effects, the effects of one individual upon another. We have abandoned a search for extrinsic agents of control for reasons discussed elsewhere (*4*, *5*). This is not to say that extrinsic factors such as weather and food are not influencing microtine populations to varying degrees, but we believe that more important than the variable effects of extrinsic factors are the intrinsic factors which act in a common way in cycling rodents. We have turned our attention toward intrinsic effects, particularly those of behavior and genetics hypothesized by Chitty (*6*). Two essential elements of Chitty's hypothesis are (i) that the genetic composition of the population changes markedly during a cycle in numbers; and (ii) that spacing behavior (or hostility) is the variable which drives the demographic machinery through a cycle.

The suggestion that genetical mechanisms might be involved in the short-term changes in rodent populations has opened a new area of investigation. Population ecologists have traditionally been concerned with quantity rather than quality, and have only recently begun to realize the importance of individual variation (*7*). The genetical basis of the control of population size was discussed as early as 1931 by Ford (*8*) but most geneticists have assumed that population control is an ecological problem and not a genetic one. Lerner (*9*) attempted to bridge the gap between genetics and ecology by showing how the solution of ecological problems might be helped by genetical insights. Although population genetics and population ecology have developed as separate disciplines, we have tried to utilize both these disciplines in our attempts to determine the causes of population cycles in rodents.

From 1965 to 1970 we studied the relationships among population dynamics, aggressive behavior, and genetic composition of field vole populations in southern Indiana. The two species of *Microtus* (*M. pennsylvanicus* and *M. ochrogaster*) that we studied fluctuate strongly in numbers with peak densities recurring at intervals of 2 or 3 years (*10*). The purposes of our investigations were to (i) describe the mechanics of the fluctuations in population size, (ii) monitor genetic changes with polymorphic marker loci, and (iii) measure changes in male aggressive behavior

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Dr. Krebs is at the University of British Columbia, Vancouver 8; Dr. Gaines is at the University of Kansas; Dr. Keller is at Idaho State University; Dr. Myers is at the University of British Columbia; and Dr. Tamarin is at Boston University.

during a population fluctuation. We here synthesize our findings on the demography and genetic composition of *Microtus* populations, and summarize the results of our behavioral studies that have been reported elsewhere (*11*).

Demographic Changes

In *M. pennsylvanicus* changes in population size can be grouped into three phases, each of which lasts several months or more: an increase phase in which the rate of population increase (r) is greater than 0.03 per week (maximum observed, 0.13 per week); a peak phase in which the rate of increase is zero (between −0.02 and +0.03 per week) and density is high and essentially constant; and a decline phase in which r is negative (−0.03 per week or less; maximum observed, −0.12 per week). Figure 1 illustrates these phases for one population of *M. pennsylvanicus*.

Detailed information on changes in population size is available for several species of voles (*4, 5, 10, 12*). The increase phase is typically the most constant phase of the cycle, and once begun may continue through the winter, as shown in Fig. 1. The peak phase often begins with a spring decline in numbers, and a summer or fall increase restores the population to its former level. The decline phase is most variable. It may begin in the fall of the peak year or be delayed until the next spring. The decline may be very rapid, so that most of the population disappears over 1 to 2 months, but often the decline is gradual and prolonged over a year or more. A phase of low numbers may or may not follow the decline, and little is known of this period, which may last a year or longer.

Table 1. Components of population fluctuations in *M. pennsylvanicus* in southern Indiana, 1965 to 1970. The data are expressed as mean values for more than 2000 individuals from four populations. The survival of early juvenile animals is determined from the number of unmarked young per lactating female; survival of subadults and adults is measured as a probability of survival per 14 days.

Phase	Birth rate (% lactating females)	Survival		
		Early juveniles	Subadult and adult	
			Males	Females
Increase	45	1.31	0.78	0.86
Peak	29	0.96	0.79	0.85
Decline	27	0.88	0.71	0.72

Changes in birth and death rates are the immediate cause of the population fluctuations in *M. pennsylvanicus* in Indiana. Table 1 shows that the birth rate, measured by the percentage of adult females captured that are visibly lactating, is reduced both in the peak phase and in the decline phase. The principal reason for this is that the breeding season is shortened in the peak and decline phases. Changes in weight at sexual maturity also contribute to a reduced birth rate in peak populations (*13*).

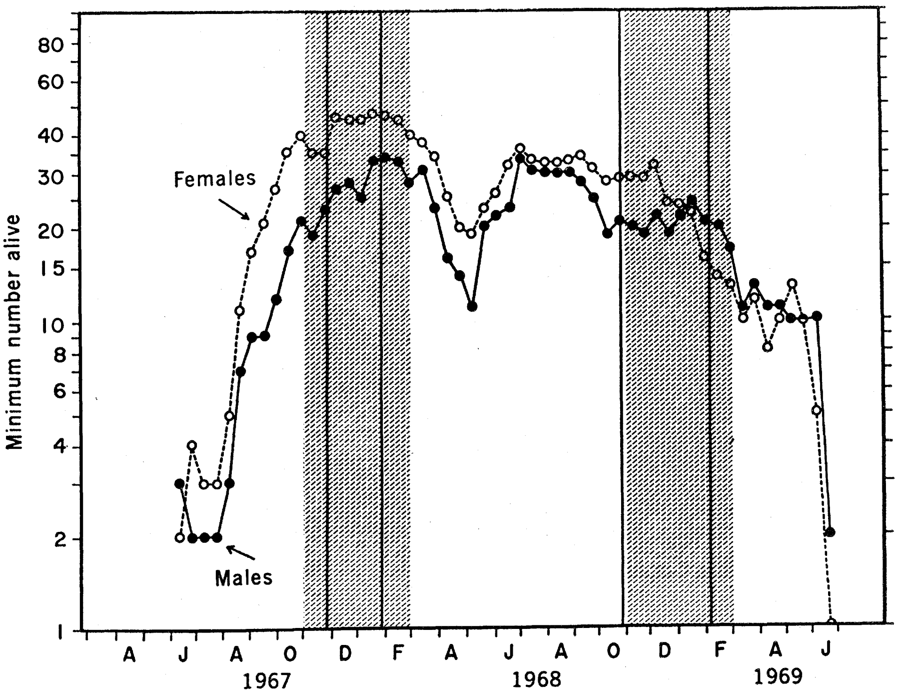


Fig. 1. Population density changes in *Microtus pennsylvanicus* on one grassland area in southern Indiana. Winter months are shaded. Vertical lines separate "summer" breeding period from "winter" period. An increase phase occurred from June to October 1967, and a decline phase from November 1968 to June 1969. [By permission of the Society for the Study of Evolution]

The death rate of small juvenile animals seems to increase dramatically in the peak and declining populations of *M. pennsylvanicus*. By contrast, the death rate of subadult and adult animals is not increased in peak populations but is increased in declining populations. Thus, in a peak population, if an animal is able to survive through the early juvenile stage, it has a high survival rate as an adult. Declining populations suffer from a low birth rate and a high mortality rate of both juveniles and adults (*14*).

What is the nature of the mortality factor acting during the population decline? The older animals seem to bear the brunt of the increased mortality during the population decline. Also, periods of high mortality during the decline are not always synchronous in males and females (see Fig. 2). Therefore, the mortality factors that are affecting the age groups and sexes during the population decline can be very selective, which argues against the overwhelming influence of extrinsic agents such as predation and disease.

These changes in birth and death rates are not unique to *M. pennsylvanicus*. Birth rates are higher in the phase of increase for all vole and lemming populations that have been studied (*5, 12, 13, 15*). The most common method of increasing the reproductive rate is by extending the breeding season, which may continue through the winter in both lemmings and voles. Extended breeding seasons are also accompanied by lowered ages at sexual maturity in some species. These trends are reversed in peak and declining populations, and the breeding season may be particularly short in some peak populations. Litter sizes seem to be essentially the same during the increase phase and the decline phase.

Death rate measurements are available for relatively few small rodent populations (*4, 5, 10, 16*). Juvenile losses are often high in peak populations and especially high in declining populations. Adult death rates are not unusually high in dense populations, but may be very high during the decline phase. The demographic changes which cause these rodent populations to fluctuate are thus a syndrome of reproduc-

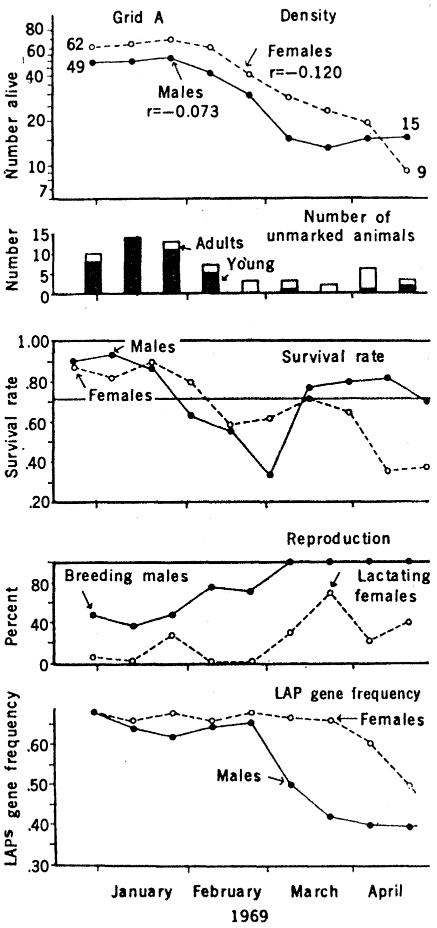


Fig. 2. Detailed breakdown of a population decline in *M. pennsylvanicus* during the spring of 1969. The critical observation is the difference in timing of male losses (highest in early March) and female losses (highest in mid-April). This timing is reflected in the gene frequency changes shown on the lowest graph (*r* is the instantaneous rate of population increase).

tive shifts and mortality changes. Reproduction and early juvenile survival seem to deteriorate first, and only later is adult survival impaired. This syndrome of changes is common to situations as diverse as lemmings in northern Canada, voles in England, and field voles in Indiana.

Growth rates of individual animals are also affected by the population fluctuations. Both males and females in increasing populations grow more rapidly than individuals in peak populations, who in turn grow more rapidly than individuals in declining populations. Figure 3 illustrates this change in growth for *M. pennsylvanicus* from southern Indiana. The higher growth rates of individuals in increasing populations, coupled with higher survival rates, produce animals of larger than average body size in increasing and peak populations (*10*). These large animals are characteristic of all peak populations of small rodents.

Fencing Experiments

The first hint we obtained about how the demographic changes are brought about in field populations came from an experiment designed to answer the question: Does fencing a population of *Microtus* effect its dynamics? We constructed three mouse-proof enclosures in the field, each measuring 2 acres (0.8 hectare), and used these to study populations constrained by the fence, which allowed no immigration or emigration of *Microtus*. Figure 4 shows population changes on two adjacent fields, one of which was fenced in July 1965. Both populations increased in size but diverged sharply in the early peak phase. The fenced population (grid B) continued to increase in the summer of 1966 to 310 animals on the 2-acre plot, a density about three times as high as that on control grid A. The overpopulation of the fenced *M. pennsylvanicus* on grid B resulted in habitat destruction and overgrazing, and led to a sharp decline with symptoms of starvation. The result was the same with enclosed populations of *M. ochrogaster* (*10*), and during the course of our studies four introductions of *M. ochrogaster* to the fenced areas resulted in abnormally high densities. Thus we conclude that fencing a *Microtus* population destroys the regulatory machinery which normally prevents overgrazing and starvation.

Dispersal (immigration and emigration) is the obvious process which is prevented by a fence, and we suggested that dispersal is necessary for normal population regulation in *Microtus*. We could see no indication that predation pressure was changed by the small fence around the large areas we studied. Foxes, cats, weasels, and snakes were known to have entered the fenced areas, and hawks and owls were not deterred.

Dispersal Experiments

If dispersal is important for population regulation, how might it operate? We could envisage two possible ways. First, dispersal might be related to population density, so that more animals would emigrate from an area in the peak phase and especially in the decline phase. These emigrants we would presume to be at a great disadvantage from environmental hazards such as other voles, predators, and bad weather. Second, the number of dispersers might not be as important as the quality of the dispersers. If only animals of a certain genetic type are able to tolerate high densities, dispersal may be one mechanism for sorting out these individuals.

We measured dispersal by maintaining two areas free of *Microtus* by trapping and removing all animals caught for 2 days every second week. Voles were free to colonize the areas for 12 days between each episode of trapping. We defined dispersers as those animals colonizing these vacant habitats (*17*). We thus determined the loss rate of individuals from control populations and the number of colonizers entering the trapped areas, and could calculate the

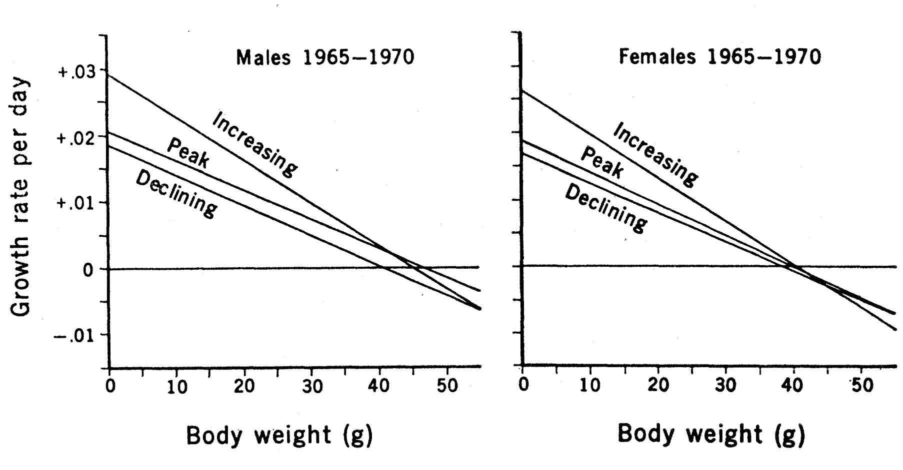


Fig. 3. Instantaneous relative growth rates for *M. pennsylvanicus* in southern Indiana. Regression lines for increase, peak, and decline phases are based on 691, 1898, and 333 observations for males and 776, 1696, and 322 observations for females (all pregnant animals excluded). Slope and elevation of the three regression lines are significantly different ($P < .01$) in both sexes.

fraction, in control populations, of losses attributable to dispersal.

Dispersal was most common in the increase phase of a population fluctuation and least common in the decline phase (Fig. 5). Most of the loss rate in increasing populations seems to be due to emigration (Table 2). Conversely, little of the heavy loss in declining populations is due to animals dispersing into adjacent areas, and hence most losses must be deaths in situ.

Thus dispersal losses from *Microtus* populations were not heaviest during the peak and decline, which supports the view that the role of dispersal is related to the quality of dispersing animals. We present evidence of genetic differences in dispersing *Microtus* in the next section.

Genetic Changes

Polymorphic serum proteins have been used as genetic markers to study the possible role of natural selection in population fluctuations of *Microtus*. We have used the genes *Tf* (transferrin) and *LAP* (leucine aminopeptidase) as markers (*18, 19*). The electrophoretically distinguishable forms of the products of these genes are inherited as if controlled by alleles of single autosomal loci.

We have found evidence of large changes in gene frequency at these two loci in association with population changes. Some of these changes are repetitive and have been observed in several populations (*18, 19*). Figure 2 shows the details of one decline in *M. pennsylvanicus* in the late winter and early spring of 1969. The survival rate of males during this decline dropped to a minimum in early March; female survival dropped 6 weeks later. These periods of poor survival coincided with the onset of sexual maturity in many of the adult males and the approximate dates of weaning first litters in adult females. The frequency of the LAP^S allele (distinguished by slow electrophoretic mobility) dropped about 25 percent in the males beginning at the time of high losses, and 4 to 6 weeks later declined an equal amount in the females. This type of observation supports strongly the hypothesis that demographic events in *Microtus* are genetically selective and that losses are not distributed equally over all genotypes. Because we may be studying linkage effects and because we do not know how selection is acting, we cannot describe the mechanisms which would explain the associations shown in Fig. 2. We cannot therefore assign cause and effect to the observations.

Table 2. Percentage of losses known to be due to dispersal for control populations of *M. pennsylvanicus* in southern Indiana. Total numbers lost are shown in parentheses.

Population phase	Males (%)	Females (%)
Increase	56(32)	69(16)
Peak	33(157)	25(127)
Decline	15(53)	12(42)

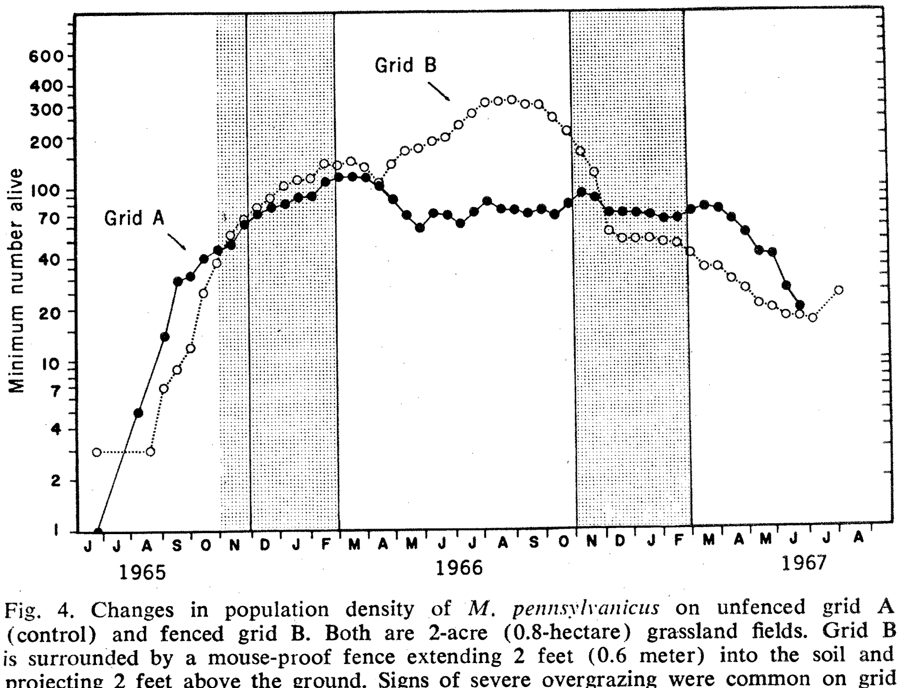


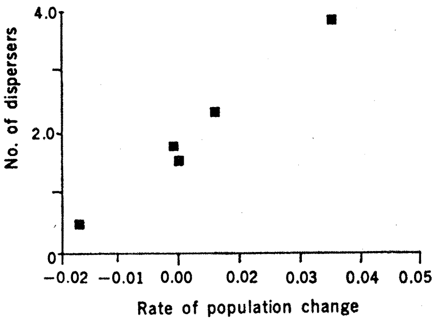
Fig. 4. Changes in population density of *M. pennsylvanicus* on unfenced grid A (control) and fenced grid B. Both are 2-acre (0.8-hectare) grassland fields. Grid B is surrounded by a mouse-proof fence extending 2 feet (0.6 meter) into the soil and projecting 2 feet above the ground. Signs of severe overgrazing were common on grid B by August 1966. [By permission of the Ecological Society of America]

We have also used the *Tf* and *LAP* variation to investigate possible qualitative differences between dispersing *Microtus* and resident animals. Since dispersal is particularly important in the increase phase (Fig. 5, Table 2), we looked for qualitative differences at this time. Figure 6 shows the genotypic frequencies at the locus of the *Tf* gene for control populations and dispersing animals during an increase phase. Heterozygous females (Tf^C/Tf^E, where *C* and *E* are alleles of the *Tf* gene) are much more common in dispersing *Microtus* than in resident populations and 89 percent of the loss of heterozygous females from the control populations during the population increase was due to dispersal. Certain genotypes thus show a tendency to disperse, a possibility suggested by several authors (*20*) but not previously demonstrated in natural populations.

The polymorphic genes that we have used for markers in *M. pennsylvanicus* and *M. ochrogaster* are subject to intensive selection pressure, but we have not been able to determine how these polymorphisms are maintained. For example, let us consider the *Tf* polymorphism in *M. ochrogaster*. Two alleles are found in Indiana populations. The common allele Tf^E has a frequency of 97 percent in female and 93 percent in male *M. ochrogaster*. This polymorphism does not seem to be maintained by heterosis. We have not found any component of fitness (survival, reproduction, or growth) in which heterozygote voles (Tf^E/Tf^F) are superior to homozygote voles (Tf^E/Tf^E), except in declining and low populations when the male heterozygotes survive better than the homozygotes. Increasing populations are always associated with strong selection for the Tf^E allele (*18, 19*). Homozygote Tf^F/Tf^F females had higher prenatal mortality in field experiments (*21*). We do not know if this *Tf* polymorphism is maintained by density-related changes in fitness or by frequency-dependent variations in fitness (*22*).

An alternative explanation for the associations we have described between population density changes and gene frequency changes has been provided by Charlesworth and Giesel (*23*). Population fluctuations result in continual shifts in age structure. Genotypes with differing ages at sexual maturity and differing survival rates will thus change in frequency as a result of population fluctuations, and genetic changes could

Fig. 5. Rate of population change in *M. pennsylvanicus* control population from southern Indiana in relation to dispersal rate from that population, 1968 to 1970. Rate of population change is the instantaneous rate of change per week, averaged over "summer" and "winter" periods shown in Fig. 1. Dispersal rate is the mean number of voles dispersing from the control population to the trapped grid per 2 weeks, averaged over the same time periods. Populations increasing rapidly show the highest dispersal rates.



thus be the side effect of population cycles caused by any mechanism. We do not know whether the genetic changes we have described are causally related to population changes or merely side effects, but we question whether they are adequately explained by the Charlesworth and Giesel model. The size of the changes in gene frequency we observed (for example, Fig. 2) is several times larger than the size of the changes obtained in the Charlesworth and Giesel model (1 to 9 percent). Also, Charlesworth and Giesel obtained relatively little effect on gene frequencies by changing death rates in their model; we have found that changes in death rates of different genotypes are a major component of shifts in gene frequency (*11*, *19*). We suggest that field perturbation experiments may help to resolve these alternative explanations (*24*).

Behavioral Changes

If behavioral interactions among individual voles are the primary mechanism behind population cycles, the behavioral characteristics of individuals would change over the cycle. We have tested this hypothesis only for male *M. pennsylvanicus* and *M. ochrogaster* in our Indiana populations. Males were tested by paired round-robin encounters in a neutral arena in the laboratory. Males of both species showed significant changes in aggressive behavior during the population cycle, so that individuals in peak populations were most aggressive (*11*). Male *M. pennsylvanicus* which dispersed during periods of peak population density tended to be even more aggressive than the residents on control areas (*17*).

Laboratory measurements of behavior can be criticized because we have no way of knowing how such measures might apply to the field situation. There is no doubt that aggression does go on in field populations of voles and lemmings because skin wounds are found, particularly in males (*5*, *25*). Field experiments could be designed to test the effects of aggression on mortality and growth rates, but none has been done yet on lemmings or voles. In the deer mouse (*Peromyscus maniculatus*) field experiments have demonstrated that aggressive adult mice can prevent the recruitment of juveniles into the population (*26*).

Conclusions

We conclude that population fluctuations in *Microtus* in southern Indiana are produced by a syndrome of changes in birth and death rates similar to that found in other species of voles and lemmings. The mechanisms which cause the changes in birth and death rates are demolished by fencing the population so that no dispersal can occur. Dispersal thus seems critical for population regulation in *Microtus*. Because most dispersal occurs during the increase phase of the population cycle and there is little dispersal during the decline phase, dispersal is not directly related to population density. Hence the quality of dispersing animals must be important, and we have found one case of increased dispersal tendency by one genotype.

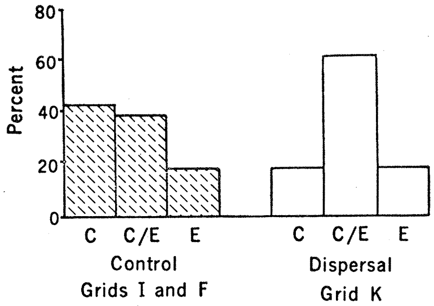


Fig. 6. The increase phase of *M. pennsylvanicus*, fall 1969. Transferrin genotype frequencies of dispersing females on trapped grid K ($N = 39$) compared with those of resident females on control grids immediately adjacent in the same grassland ($N = 224$). Dispersing voles in the increase phase are not a random sample from the control population. C, CIE, and E represent the three transferrin genotypes.

The failure of population regulation of *Microtus* in enclosed areas requires an explanation by any hypothesis attempting to explain population cycles in small rodents. It might be suggested that the fence changed the predation pressure on the enclosed populations. However, the fence was only 2 feet (0.6 meter) high and did not stop the entrance of foxes, weasels, shrews, or avian predators. A striking feature was that the habitat in the enclosures quickly recovered from complete devastation by the start of the spring growing season. Obviously the habitat and food quality were sufficient to support *Microtus* populations of abnormally high densities, and recovery of the habitat was sufficiently quick that the introduction of new animals to these enclosed areas resulted in another population explosion. Finally, hypotheses of population regulation by social stress must account for the finding that *Microtus* can exist at densities several times greater than normal without "stress" taking an obvious toll.

We hypothesize that the prevention of dispersal changes the quality of the populations in the enclosures in comparison to those outside the fence. Voles forced to remain in an overcrowded fenced population do not suffer high mortality rates and continue to reproduce at abnormally high densities until starvation overtakes them. The initial behavioral interactions associated with crowding do not seem sufficient to cause voles to die in situ.

What happens to animals during the population decline? Our studies have not answered this question. The animals did not appear to disperse, but it is possible that the method we used to measure dispersal (movement into a vacant habitat) missed a large segment of dispersing voles which did not remain in the vacant area but kept on moving. Perhaps the dispersal during the increase phase of the population cycle is a colonization type of dispersal, and the animals taking part in it are likely to stay in a new habitat, while during the population decline dispersal is a pathological response to high density, and the animals are not attracted to settling even in a vacant habitat. The alternative to this suggestion is that animals are dying in situ during the decline because of physiological or genetically determined behavioral stress.

Thus the fencing of a population prevents the change in rates of survival and reproduction, from high rates in the increase phase to low rates in the decline phase, and the fenced populations resemble "mouse plagues." A possible explanation is that the differential dispersal of animals during the phase of increase causes the quality of the voles remaining at peak densities in wild populations to be different from the quality of voles at much higher densities in enclosures. Increased sensitivity to density in *Microtus* could cause the decline of wild populations at densities lower than those reached by fenced populations in which selection through dispersal has been prevented. Fencing might also alter the social interactions among *Microtus* in other ways that are not understood.

The analysis of colonizing species by MacArthur and Wilson (*27*) can be applied to our studies of dispersal in populations of *Microtus*. Groups of organisms with good dispersal and colonizing ability are called *r* strategists because they have high reproductive potential and are able to exploit a new environment rapidly. Dispersing voles seem to be *r* strategists. Young females in breeding condition were over-represented in dispersing female *Microtus* (*17*). The Tf^C/Tf^E females, which were more common among dispersers during the phase of population increase (Fig. 6), also have a slight reproductive advantage over the other *Tf* genotypes (*19*). Thus in *Microtus* populations the animals with the highest reproductive potential, the *r* strategists, are dispersing. The segment of the population which remains behind after the selection-via-dispersal are those individuals which are less influenced by increasing population densities. These are the individuals which maximize use of the habitat, the *K* strategists in MacArthur and Wilson's terminology, or voles selected for spacing behavior. Thus we can describe population cycles in *Microtus* in the same theoretical framework as colonizing species on islands.

Our work on *Microtus* is consistent with the hypothesis of genetic and behavioral effects proposed by Chitty (*6*) (Fig. 7) in that it shows both behavioral differences in males during the phases of population fluctuation and periods of strong genetic selection. The greatest gaps in our knowledge are in the area of genetic-behavioral interactions which are most difficult to measure. We have no information on the heritability of aggressive behavior in voles. The pathways by which behavioral events are translated into physiological changes which affect reproduction and growth have been carefully analyzed by Christian and his associates (*28*) for rodents in laboratory situations, but the application of these findings to the complex field events described above remains to be done.

Several experiments are suggested by our work. First, other populations of other rodent species should increase to abnormal densities if enclosed in a large fenced area (*29*). We need to find situations in which this prediction is not fulfilled. Island populations may be an important source of material for such an experiment (*30*). Second, if one-way exit doors were provided from a fenced area, normal population regulation through dispersal should occur. This experiment would provide another method by which dispersers could be identified. Third, if dispersal were prevented after a population reached peak densities, a normal decline phase should occur. This prediction is based on the assumption that dispersal during the increase phase is sufficient to ensure the decline phase 1 or 2 years later. All these experiments are concerned with the dispersal factor, and our work on *Microtus* can be summarized by the admonition: study dispersal.

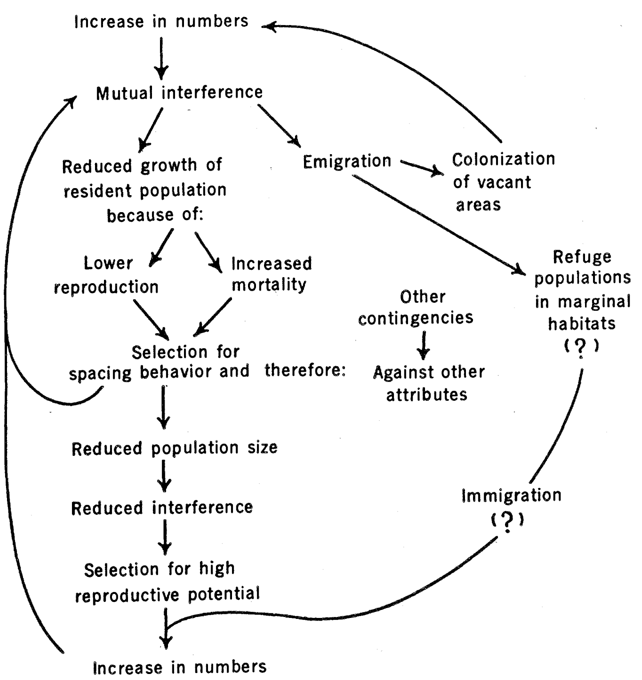


Fig. 7. Modified version of Chitty's hypothesis to explain population fluctuations in small rodents. Density-related changes in natural selection are central to this hypothesis. Our data indicate that selection through dispersal is more important than originally proposed by Chitty.

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31. This research was conducted when all of us were at Indiana University. We thank the National Science Foundation and the Public Health Service for financial support of the research.

The Revolution in Birth Control Practices of U.S. Roman Catholics

Charles F. Westoff and Larry Bumpass

Ever since the 1968 papal encyclical ended the period of ambiguity and speculation about the Roman Catholic Church's position on birth control, there has been considerable interest in how American Catholics would respond to the reaffirmation of the traditional ban on methods of contraception other than the rhythm method. The trend toward nonconformity documented in the 1965 National Fertility Study (NFS) (*1*), the observed reduction in the rate of unwanted births among all groups, including Catholics, during the 1960's (*2*), and the sharp decline in U.S. fertility rates all combine to enhance the plausibility of the hypothesis that Catholic couples have increasingly adopted unapproved methods of contraception. An analysis based on reinterviews with Catholic women in the 1965 study supports this view (*3*).

This report presents data from the 1970 NFS, a probability sample survey in which 6752 ever-married women of reproductive age (under 45) were interviewed across the nation (*4*). Our analysis is based on currently married, white Catholic women living with their husbands. Data for our subsample of the 1970 NFS are analyzed in conjunction with data on comparable women in the 1965 NFS and two earlier U.S. fertility surveys conducted in 1955 (*5*) and 1960 (*6*).

Concepts and Measures

While "current use" is appropriate for describing the contraceptive practices of the population at any given time, "most recent use" is more appropriate for measuring the *usual* contraceptive practices. At any given time, many couples are not using any method simply because the wife is pregnant, trying to become pregnant, or in postpartum. Others may not be practicing birth control for a variety of reasons such as illness, involuntary sterility, or temporary separation. It is a couple's usual method of contraception that is most relevant to the issue of Catholic conformity. Of course, some couples who are not currently using any method will change methods when they begin practicing birth control again; and to the extent that such changes are away from conformity, as seems most likely, our measure understates the level of nonconformity.

The concept of "conformity" is defined here as following the traditional teaching of the Roman Catholic Church, reaffirmed by the encyclical, which prohibits the use of any method of fertility control other than periodic continence, the so-called rhythm method. There are two categories of Catholic women who are classified as conforming to Church doctrine: those who have never used any method of contraception and those whose most recent practice was the rhythm method (*7*).

Trend in Catholic Conformity

By linking the 1955 and 1960 Growth of American Family studies with the 1965 and 1970 National Fertility studies, we can observe the trend in Catholic conformity over a 15-year period. Table 1 shows a dramatic change in the adherence of Catholic women to their Church's teaching on birth control. The proportion of Catholic women between the ages of 18 and 39 (*8*) who use methods of contraception other than rhythm has increased from 30 percent in 1955 to 68 percent in 1970, with the greatest changes occurring in the last 5 years. Between 1965 and 1970, the percentage of Catholic women deviating from official teaching on birth control has risen from 51 to 68 percent. It seems clear that the papal encyclical has not retarded the increasing defection of Catholic women from this teaching.

This trend is even more apparent when the data are examined by the women's age and year of birth across all four studies (vertical comparisons, Table 2). There has been a spectacular increase in nonconformity among Catholic women in the youngest age groups. Among women aged 20 to 24 in the year of each study, the proportion not conforming was 30 percent in 1955, 43 percent 5 years later, 51 percent by 1965, and 78 percent by 1970. The increase from 1955 to 1970 was almost as great for the next two age groups: from 37 to 74 percent for ages 25 to 29 and from 30 to 68 percent for ages 30 to 34.

Dr. Westoff is professor of sociology and associate director of the Office of Population Research, Princeton University, Princeton, New Jersey 08540. Dr. Bumpass is associate professor of sociology, Center for Demography and Ecology, University of Wisconsin, Madison 53706.