DISPERSAL IN SMALL ANIMAL POPULATIONS: A MODELING APPROACH TO EXAMINE ALTERNATIVE HYPOTHESES

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ABSTRACT

DISPERSAL IN SMALL ANIMAL POPULATIONS:
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By

Jay Brian Hestbeck

Dispersal mechanisms were classified into two causal categories, demographic and genetic polymorphisms. The demographic hypotheses were composed of the random walk mechanism, the induced emigration hypothesis and the density-dependent hypotheses. The genetic polymorphism hypotheses were further classified into mechanisms that consider temporal variation, increased sensitivity to density changes and dispersal of highly aggressive individuals. A sensitivity analysis was utilized to determine which dispersal mechanism was most logically inferred from the ecological theory on which the model was designed. The individual aggression hypothesis was the most logical demographic hypothesis. The most reasonable genetic polymorphism hypotheses were that presaturation dispersal operates equivalently to saturation dispersal and presaturation dispersal individuals were more sensitive to increasing aggression levels than saturation dispersal individuals.

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INTRODUCTION

Field techniques in small mammal ecology fail to identify dispersers. Consequently, dispersal has commonly been included with mortality to create a gross loss term for a population. Evidence from Lidicker (1962), Pearson (1963), and Krebs, Keller and Tamarin (1969) has shown that dispersal may be a key demographic parameter. Lidicker (1975) has also shown that dispersal could affect gross mortality, age structure, sex ratio, growth rates and social structure of populations. With increasing evidence, dispersal has received much attention as an important demographic parameter, but the actual mechanism controlling dispersal remains highly controversial, and many hypotheses have been proposed to explain possible mechanisms.

I have examined several hypotheses by initially classifying dispersal mechanisms into two causal categories, demographic and genetic polymorphism, following Lidicker (1975). The demographic hypotheses were futher classified into random walk, induced emigration and density-dependent dispersal hypotheses.

1) The random walk mechanism proposes that individuals begin moving in a completely unbiased manner from birth, and a probability of being located in one position at any time is determined by a normal probability distribution.

2) The induced emigration hypothesis states that animals shift their home range as a function of perceiving neighbors.

3) The density-dependent hypotheses propose that the force behind dispersal is directly related to the population density of the area.

The genetic polymorphism dispersal hypotheses were further classified into mechanisms that consider temporal variation, increased sensitivity to density changes, and dispersal of highly aggressive individuals.

1) The temporal variation hypothesis states that dispersal is a direct result of temporal environmental variation.

2) The hypothesis that considers increased sensitivity to density changes as a mechanism of genetic dispersal proposes that genetic dispersers are more sensitive to increasing densities than demographic dispersers.

3) The hypothesis that highly aggressive individuals disperse proposes that genetic dispersers are more aggressive and less tolerant of their neighbors; as a result, these individuals disperse during early population increases.

The nature of these hypotheses make experimental testing very difficult, due to the lack of adequate techniques or to the impossible experimental design. In order to evaluate the contribution of these hypotheses, a design other than field experimentation is necessary. A simulation model allows examination of hypotheses that are otherwise untestable in the field.

Part of the problem in studying dispersal is the lack of a functional definition. Johnston (1961) classified population movements of birds into spacing, dispersal

and migratory movements. Spacing was defined as a result of territorial behavior of adults and was responsible for the dispersal of breeding units (Johnston, 1961). Dispersal was defined as the movement of an individual from its site of birth to its site of breeding; Johnston (1961) stressed that although dispersal and spacing occur simultaneously, they existed as two independent forms of movement.

Separation of movements into spacing and dispersal movements has also been followed in studies of small mammals. Populations of Microtus pennsylvanicus rarely reached densities where territorial behavior would expel members, and thus spacing was not a likely cause for dispersal (Getz, 1961). In a later study, Getz (1962) noted that Microtus ochrogaster displayed little aggressive behavior towards members of its own species, yet M. ochrogaster was found to disperse (Myers and Krebs, 1971). This would indicate that dispersal was not dependent upon aggression or territorial aggression, and hence spacing. This was also noted by King (1973), who stated that all species of small mammals disperse but few express overt aggression; therefore, there must be an alternate mechanism for dispersal.

A distribution of dispersal distances similar to those obtained in natural populations could be simulated solely by considering spacing movements for the procure-

ment of breeding sites (Murray, 1967). This indicated that spacing movements alone could explain dispersal data. This presented a major problem in the study of small mammals in that the general habits of rodents prevented the elucidation of their social organization, such as that accomplished in the study of birds by Johnston (1961). Consequently, the study of dispersal in rodents has also been a study of spacing movements (Krebs and Myers, 1975).

Although dispersal in general was believed to consist of both dispersal movements and spacing movements, the relative contribution of these two to dispersal was unknown; the relative contribution was assumed to vary for different species. Lidicker (1975) supported this assumption by stating that the importance and nature of dispersal varied greatly among different species. Considering the difficulties associated with studying small mammals in the field, a definition of dispersal different from Johnston's was formulated. Dispersal was defined as any movement an individuals organism made from its place of birth but not including temporary exploratory movements. This definition thus incorporated Johnston's ideas on spacing and dispersal movements.

As mentioned above, the random walk hypothesis is one of the demographic hypotheses. Under the random walk hypothesis, all individuals were expected to start moving at birth in a completely unbiased manner (Chapman, 1967;

Pielou, 1969; and Richardson, 1970). The probability of being found at any given location in the future was found to be normally distributed (Chapman, 1967). Since a random walk mechanism for dispersal produced a normal distribution, a test of this hypothesis could be made by comparing a distribution of dispersal distances from the field to the theorized normal distribution. In an early study, the distribution of dispersal distances for Peromyscus maniculatus was non-normal (Dice and Howard, 1951). Smith (1968) also tested this hypothesis for Peromyscus polionotus and found that individuals leaving their homesites would probably move longer distances than would be predicted by a normal distribution. The most recent test of this hypothesis concluded that dispersal movements in Perognathus formosus were not random because too many movements were excessive in length, and too few were short; dispersal was believed to be purposefully directed movements made by certain individuals (French, Togami, and Hayden, 1968). The hypothesis that dispersal operated through a random walk mechanism was thus contradicted in these three studies.

Another hypothesized demographic mechanism for dispersal is that small mammals shift their place of habitation due to a response directly related to the relative intensity and/or frequency of perceiving neighbors (Calhoun and Webb, 1953). They proposed two alternative mechanisms. First: as an animal moves around its home range, it per-

ceives its neighbor. If an avoidance response is associated with this perception, each individual becomes positioned away from its neighbors; when a change occurs in this perception pattern, the animal shifts away from the higher concentration of animals due to the increased level of perception. This mechanism is assumed to produce a uniform distribution of animals since an accumulation of a portion of population in one area could cause the frequency of perceptions to increase. The animals surrounding this area shift their home range away from this increased number of perceptions to maintain the former frequency of perceptions. This shift results in a chain reaction which finally produces equal perception levels on all sides for every individual in the population; thus, a uniform distribution is created. It follows that a reasonable test of this hypothesis is to determine whether a population has a uniform dispersal pattern or not. Mus musculus populations have been found to have extremely high densities in very localized areas where the habitat was relatively homogeneous (Pearson, 1963), and the distribution in this homogeneous area was clumped. The original hypothesis proposed by Calhoun and Webb (1953) was thus contradicted.

The second proposed mechanism to explain induced emigration is based on the fact that each individual may have become conditioned to a certain pattern of perception

from its neighbors. This pattern may be unequal in intensity or frequency of perceptions. When this perception pattern changes, the animal shifts its home range in an attempt to recreate the former perception pattern (Calhoun and Webb, 1953). If the density increases in one area, the perception pattern increases, causing animals to move in the opposite direction in order to find their initial perception level. Density changes only occur after a new generation of individuals are conditioned to a new perception pattern.

From the above hypothesis, one would predict that densities do not change rapidly. Myers and Krebs (1971) found that Microtus ochrogaster had an instantaneous growth rate of 0.231 per week from August to October. Many other species of microtines were also found to have very high population growth rates (Krebs and Myers, 1974). Since the growth rate in these populations was very high, individuals entering the population would increase the perception level. With an increased frequency of perception, animals with the orignal number of perceptions at lower density (adults) would shift their home range. Adults would therefore make up the largest proportion of dispersing individuals. However, field studies show that dispersers are almost invariably young adults (Wilson, 1975). As a result of these studies, the hypothesis of perception-induced emigration as the mechanism by which dispersal

acts was contradicted.

The density-dependent dispersal hypotheses are of three kinds: territorial spacing mechanisms, mechanisms involving intraspecific interaction, and those involving aggression. Dispersal as a function of spacing movements and territorial aggression was explored for Microtus pennsylvanicus (Van Vleck, 1968). When traveling outside an individual's home range, encounters with a resident individual would force the transient individual to move. Absence of an encounter allows the animal to remain. As the density increases, the number of resident animals increases, causing increased numbers of encounters with transient animals; therefore, animals would continue to move and disperse. In this case, dispersal results solely from territorial spacing.

Territorial spacing was also found in studying Spermophilus undulatus (Carl, 1971) and Ochontona princeps (Smith, 1974); females became intolerant of their young shortly after weaning and attempted to exclude them from their territories. The juveniles either filled in vacant territories or dispersed when the preferred habitat became saturated. From these studies, the territorial behavior hypothesis seems a feasible mechanism for dispersal.

As stated earlier, populations of Microtus pennsylvanicus rarely reach densities at which territorial behavior would expel individuals from the population (Getz,

1961). This indicates that dispersal could operate by a mechanism other than territorial behavior. A density-dependent dispersal hypothesis was proposed. This hypothesis sates that as density increases, dispersal increases either by following changing environmental conditions as in Sigmodon hispidus and Reithrodontomys fulvescens (Joule and Cameron, 1975), or by an intraspecific interaction (Strecker, 1954; Archer, 1970; and Grant, 1971). The nature of the interaction was not known, but it was demonstrated with Mus musculus that the interaction was not directly related to available food since dispersal occurred before the food supply was exhausted (Strecker, 1954). This was supported by the experiment by Krebs et al. (1969) with Microtus pennsylvanicus and M. ochrogaster, which showed that by preventing dispersal, the population would grow until the food supply had been exhausted. In the control group, dispersal occurred, and the food supply was not decimated. Dispersal was therefore assumed to act through some density-dependent interaction other than food supply.

This intraspecific density-dependent interaction is hypothesized to be intraspecific aggression (Thiessen, 1966; Delong, 1967; Newsome, 1969; Christian, 1970; and Wilson, 1975). This dispersal mechanism operates by increases in densities causing increased levels of aggression. The rise in aggression then forces the subordinate

individuals to disperse. Therefore, the magnitude of dispersal varies directly with population density and the degree of mutual intolerance.

Another form of dispersal mechanism has been proposed which involves a genetic polymorphism. A genetic basis for dispersal was initially proposed by Dice and Howard (1951). Blair (1953) states that dispersal is caused by either an inherent tendency to disperse activated by physiological changes and/or population pressure working through intraspecific competition, thus suggesting that dispersal could result from two mechanisms in the same population. Howard (1960) then introduced the concept of genetic polymorphisms by stating that dispersal involved two mechanisms, an inherent form and an environmental form which operates simultaneously. Howard also indicated that a heritable characteristic controls the expression of each form. Lidicker (1962) supported the polymorphism hypothesis, but stated that the ultimate mechanism by which inherent dispersal operates is equivalent to the mechanism for environmental dispersal. The difference between the two is that inherent dispersal is more sensitive to increasing densities than environmental dispersal.

The genetic polymorphism hypothesis thus proposes two dispersal forms, inherent dispersal and environmental dispersal. Inherent dispersal (presaturation dispersal) is defined as any movement by an individual from a population before its habitat becomes saturated with its

own species; environmental dispersal (saturation dispersal) is defined as the outward movement of surplus individuals from a population living at or near its carrying capacity (Lidicker, 1975).

From this definition, saturation dispersal utilizes the same mechanism as density-dependent dispersal. Since the mechanisms for density-dependent dispersal exclude a genetic basis, saturation dispersal also operates without a genetic basis. The argument for the existence of a genetic polymorphism mechanism for dispersal then focuses on whether presaturation dispersal could have a genetic basis. It is presumed that if both presaturation and saturation dispersal exist, and if presaturation dispersal has a genetic basis, a genetic polymorphism mechanism for dispersal must also exist.

The existence of a genetic basis for presaturation dispersal was suggested by the fact that individuals seasonally colonizing a habitat differed genetically from the founding population (Anderson, 1970). Further evidence indication dispersers were genetically different from resident individuals was provided by findings that isoenzyme differences existed between Microtus pennsylvanicus and M. ochrogaster (Myers and Krebs, 1971; Pickering, Getz, and Whitt, 1974). Dispersers therefore could be considered to be genetically different from resident individuals, but a genetic locus for dispersal was not established.

Although a genetic basis for controlling dispersal was found in Drosophila melanogaster (Narise, 1962; and Luchnikova, 1966), the strongest evidence for a genetic locus for dispersal was provided by demonstrating that by exerting a selection pressure for and against dispersal in Tribolium, a dispersal phenotype could be selected for (Ogden, 1970).

Since a genetic component of dispersal had been demonstrated, a situation for the evolution of genetic dispersal was investigated. Gadgil (1971) suggested that species which inhabit areas with a variable carrying capacity would develop a sensitive density response for dispersal. Since increasing environmental variability reduced the probability of persistence of a population, the evolution of dispersal would increase the persistence of the population by several orders of magnitude (Roff, 1974). This suggested a possible condition where a dispersal mechanism could have evolved.

Where conditions favor the evolution of a dispersal response, a genetically evolved dispersal mechanism would develop (presaturation dispersal) (Lidicker, 1975). Consequently, presaturation dispersal was expected to evolve in a variable environment. Lidicker (1975) verified this by stating that presaturation dispersal, or genetically-evolved dispersal, occurred in two situations: 1) in species that regularly colonize new habitats and 2) in species which affect their future food supply by feeding

activities. Colonizing behavior would allow the species to more efficiently utilize new habitat; a species which affects its future food supply would have the ability to exploit new suitable habitats. Both of these situations are examples of a variable environment, and therefore support Gadgil's (1971) earlier statement. This concept was supported by the observation that small mammals living in a highly unstable environment seem to be more mobile than mice from stable areas (Sheppe, 1972).

Since environmental variation is composed of temporal and spatial variability, it follows that the level of presaturation dispersal would increase as the temporal variation in the environment increases, and decrease as the average spatial heterogeneity decreases (Levins, 1964). The magnitude of presaturation dispersal is related to temporal variation in the environment and average spatial heterogeneity. Bryant (1974) later analyzed variation in polymorphisms using factor analysis and multiple regression to show that temporal variation of the environment is the major contributor to genetic variation exhibited in the polymorphism, but spatial heterogeneity still provides a minor contribution to the increase in the degree of polymorphism.

A genetic characteristic for presaturation dispersal presumably evolved in areas where temporal variation was large. However, the mechanism for presaturation dispersal

was still unkown. Three major hypothesis as to the mechanism of presaturation dispersal are: temporal environmental variation, individual differences in sensitivity to density-dependent interaction, and individual differences in aggressivity and intolerance of others.

The temporal variation hypothesis is an extension of work by Bryant (1974). Since temporal variation was the main contributor to the creation of a genetic polymorphism as Bryant had concluded, a proposed mechanism for presaturation dispersal is that dispersal results directly from environmental variation. Lidicker (1962) presented the hypothesis that presaturation dispersal operated equivalently to density-dependent dispersal except when individuals differed in sensitivity to increasing densities.

A third hypothesis for presaturation dispersal proposed that highly aggressive individuals are more likely to disperse, and that high levels of aggressiveness are genetically controlled. The intensity of dispersal increased in Drosophila melanogaster as the overall heterozygosity of the population increased (Narise, 1969). This suggested that dispersers are heterozygous. Heterozygotes are assumed to be more aggressive since phenotypic variation in aggression depended on hereditary variation (Lagerspetz, 1964). Therefore, the presaturation disperser is assumed to be a heterozygous individual which disperses due to its aggressive nature and intolerance of other individuals.

An example of this form of dispersal was found in Marmota flaviventris; here, aggressive females occupied small harems or lived as solitary individuals in a surrounding area while social females lived either in large harems or on the periphery of a large colony (Svendsen, 1974).

The purpose of this study was to examine alternate hypotheses of dispersal. By exploring the feasibility of each hypothesis with the use of a simulation model, the controversy surrounding dispersal can be reduced.

THE SIMULATION MODEL

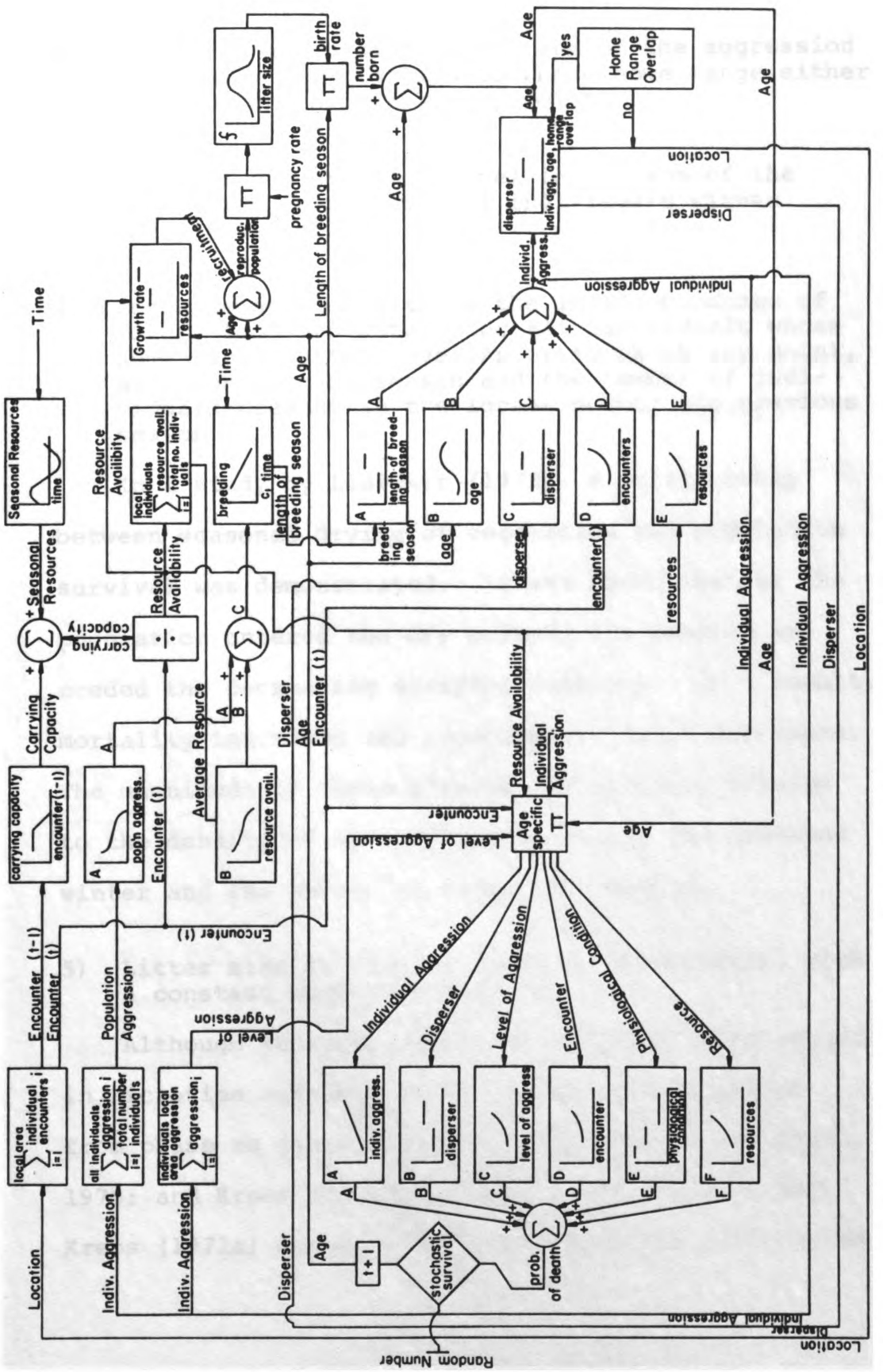
Assumptions and Rationale

A FORTRAN-DYNAMO simulation model of dispersal was constructed (Figure 1; a listing of this program is in Appendix 1). Explicit assumptions inherent in the model are:

1) Individual aggression is a function of breeding season, age, number of encounters with others, available resources, and whether the animals is a resident or non-resident.

The aggressive interactions of most animal species were found to peak during the breeding season (Krebs, 1970; Turner and Iverson, 1973; Lidicker, 1973; and Wilson, 1975). Age was also important in determining dominance and aggression (Christian, 1970). The number of encounters made by an individual was used as a measure of crowding; it was assumed that as animals moved closer, the encounter rate increased exponentially (Wilson, 1975). It was important to determine whether the animal was a resident or non-resident since Wilson indicated that the strongest evoker of aggression was the sight of a stranger. Available resources were included since aggression increased as competition for environmental resources,

Figure 1. Structural representation of the computer model.


Time
Seasonal Resources
time
Seasonal Resources
Resource Availibity
local individuals
resource avail.
total no. individuals
Resource Availability
Growth rate
resources
Carrying Capacity
carrying capacity
encounter(t-1)
Carrying Capacity
Average Resource
resource avail.
Time
breeding
c, time
length of breeding season
Age
reproduc. population
pregnancy rate
litter size
birth rate
number born
Age
Age
yes
Home Range Overlap
no
Age
Location
Disperser
Individual Aggression
Individ. Aggress.
disperser
indiv. aggr., age, home overlap
length of breeding season
age
disperser
encounters
resources
breeding season
age
disperser
encounter(t)
resources
Individual Aggression
Age
Disperser
Location
Resource Availability
Individual Aggression
Age specific
Encounter
Level of Aggression
Age
Encounter (t)
Encounter (t-1)
Population Aggression
Level of Aggression
Individual Aggression
Disperser
Level of Aggression
Encounter
Physiological Condition
Resource
local area individual encounters
all individuals aggression / total number individuals
individuals local area aggression
indiv. aggress.
disperser
level of aggress
encounter
physiological condition
resources
Indiv. Aggression
Indiv. Aggression
Location
Disperser
Age
1 + 1
stochastic survival
prob. of death
Random Number
Location
Disperser
Individual Aggression

notably food and shelter, increased (Wilson, 1975).

2) Localized aggression is the sum of the aggression levels for all individuals whose home range either overlaps or touches at any point.

3) Population aggression level is the sum of the aggression levels for all individuals within the population.

4) Resource availability is the local abundance of cover, food and water, for all individuals whose home ranges either overlap or touch at any point, as a function of season and the number of individuals present in the locale during the previous season.

In a study by Lidicker (1973), a relationship between seasonal drying of vegetation and population survival was demonstrated. It was shown that as the population entered the dry season, its density exceeded the decreasing carrying capacity. As a result, mortality increased and reproductive rates decreased. The magnitude of these effects was directly related to the density of the population during the previous winter and the amount of vegetative drying.

5) Litter size is assumed normally distributed, with a constant mean and variance.

Although seasonal variation in litter size occurred in microtine rodents, it was found to be constant from phase to phase (Hoffman, 1958; Keller and Krebs, 1970; and Krebs and Myers, 1974). Since Myers and Krebs (1971a) demonstrated that dispersal rate varied

from phase to phase, litter size was not considered to have a direct effect on dispersal, and was treated as a constant.

6) Pregnancy rate is assumed to be constant.

Although Southwick (1958) reported that the proportion of fecund mice decreased as the density increased, and Hoffman (1958) found that the prevalence of pregnancy varied inversely with density, pregnancy rate was considered constant. This was justified since Krebs and Myers (1974) found that the pregnancy rate during each population phase of a microtine cycle were equivalent. Since the dispersal rate was significantly different between populations, and since the pregnancy rate did not vary in relation to population phase, pregnancy rate was assumed not to have a direct effect on dispersal and was treated as constant.

7) The length of the breeding season is negatively related to population aggression level and average resource availability for the population.

Length of breeding varied through time (Errington, 1956; Krebs, 1964 and 1966; Keller and Krebs, 1970; Krebs and Myers, 1974), but the mechanism for a varying length in breeding season was unknown. Sadlier (1969) stated that an increased level of aggressive interactions between individuals resulted in a shorter

breeding season. Delong (1967) reported that a reduction in resource availability caused a reduction in breeding activity. Barkalow, Hamilton, and Scoots (1970) found that reproductive parameters were responsive to availability of nutritious food. Therefore, the length of breeding was assumed to be inversely related to population aggression level and average resource availability.

8) Annual recruitment is the product of pregnancy rate, litter size and length of breeding season.

9) The sex ratio is one at birth.

Myers and Krebs (1971b) found that although males were less abundant than females, the sex ratio did not correlate with population density. The sex ratio was therfore treated as unity.

10) Age at sexual maturity is a function of resource availability.

Puberty was reached at an older age in peak-density populations than low-density populations in Microtus (Keller and Krebs, 1970; and Krebs and Myers, 1974). Since there was less competition for available resources in low-density populations, nutrition was assumed to be better for juvenile Microtus. Voles under good nutrition have been found to have a faster growth rate and have subsequently reached puberty at an earlier age (Sadlier, 1969). Growth rate was

directly related to better nutrition, and better nutrition was directly related to abundance of resources. Sexual maturity was thus directly related to resource availability (Delong, 1967).

11) Prenatal mortality is constant.

No significant difference was found in prenatal mortality during a microtine cycle (Hoffman, 1958; and Keller and Krebs, 1970). Prenatal mortality was therefore treated as a constant.

12) Survival is determined as an age-specific function of individual aggression, localized aggression, number of encounters, resource availability and dispersal.

Individual aggression was assumed to have the largest single effect on an individual's behavior. Since differential survival was associated with an individual's behavioral type (Krebs, 1970; and Turner and Iverson, 1973), an individual's aggressiveness was assumed to affect its probability of survival. Survival was also dependent on the aggressiveness of an individual's neighbors (Southwick, 1955); it was assumed that as localized aggression increased, survival decreased. While nestling and juvenile survival was inversely related to density (Whitney, 1976), the model assumed that survival was inversely dependent upon the frequency of individual encounters. Resource availability was considered to be inversely

related to survival. Individuals which dispersed were considered to have a greater probability of death than individuals that did not disperse (Metzgar, 1967; and Ambrose, 1972).

13) Nestlings remain inside the nest and have an insignificant effect on the localized aggression level, population aggression, local resource availability and frequency of social encounters.

14) Confrontations are defined as occurring whenever two individual's home ranges overlap or touch at any point, and whenever one or both individuals exceed an intolerance threshold, measured as individual aggression.

15) Whenever a confrontation occurs, any individuals who exceed a dispersal sensitivity threshold move one fixed step in a random direction. The dispersal sensitivity threshold is that point at which highly aggressive animals disperse. This assumption is applicable only when the evolutionary hypotheses were tested.

16) Whenever a confrontation occurs, the less aggressive animal moves one fixed step in a random direction, provided the more aggressive individual did not move as defined in 15).

17) An individual's home range is constant.

Although home range size decreases as density increases (Sanderson, 1966), home range size was assumed to have an insignificant effect on dispersal.

18) Both sexes are assumed to behave alike.

Behavioral responses for both sexes were similar since male and female Microtus operated under similar

tensions with regard to spatial contacts (Conley, 1976).

Although the following assumptions were not specifically designed into the simulation model, they are implied by the model structure. These implicit assumptions are:

1) The effect of resource availability, localized aggression, and existence of confrontations on an individual by its neighbor is exactly the same whether the neighbor's home range completely overlaps an individual's home range or touches at only a single point.

2) Although resource availability fluctuates as a function of season and previous population density, the environment was considered to be homogeneous and unbounded.

3) All females that enter estrus are fertilized according to the defined pregnancy rate.

After the simulation model was constructed, each variable was examined to ensure that the values produced within the model were biologically realistic.

Methods

At the beginning of each simulation run, a given number of animals were randomly placed onto the grid and their ages were initialized according to a stable age distribution. Each time a new run was made, the same number of animals and initial conditions were defined. After

the initial conditions were defined, the simulation was run in a Monte Carlo mode.

Dispersal was measured by calculating the distance an animal moved from its placement or its place of birth. These distances were calculated for the entire population and were recorded as a frequency distribution of dispersal distances.

A sensitivity analysis was conducted on the following variables, each of which were associated with a specific dispersal hypothesis. Territorial aggression was a measure of territorial spacing. Since the term intraspecific interactions is a broad term that implies no specific mechanism, and since intraspecific interactions were all density-dependent, a measure of importance of density-dependent intraspecific interactions was assumed to be the frequency of encounters. Aggression as a dispersal mechanism could have resulted from two forms of aggression, individual aggression and localized aggression; each form was tested. The temporal variation in the environment was measured by the magnitude of seasonal environmental change. Increased sensitivity to increasing crowding by presaturation individuals was measured by varying the confrontation aggression threshold and varying the density-dependent response through the frequency of encounters. The hypothesis that highly aggressive individuals were more likely to disperse was examined by using an aggression threshold at which highly aggressive animals would

disperse.

In order to provide a basis for comparison, a demographic control and a genetic polymorphism control were calculated by averaging the frequency distribution of dispersal distances for four simulation runs. Each experimental frequency distribution was then determined by taking one variable from above in a stepwise manner and increasing or decreasing its value by 20% from the control value. Again four simulation runs were taken, and the average dispersal distance frequency distribution was calculated. A comparison between the experimental distributions and control distribution was made, and the amount of deviation between these distributions was measured by the Kolmogorov-Smirov goodness-of-fit D statistic (Sokal and Rohlf, 1969). The degree of sensitivity for each variable was then determined by adding the Kolmogorov-Smirov D statistic from the 20% increase frequency distribution to the D statistic of the 20% decrease frequency distribution. The degree of sensitivity is thus a measure of the deviation of the experimental distribution relative to the original steady-state distribution.

The purpose of a sensitivity analysis is to determine which hypotheses merit further testing. A sensitivity value taken by itself is meaningless; the power of the sensitivity analysis comes from the comparison of the sensitivity value of one experimental distribution with that of another distribution, and provides an indication

of the relative importance of each mechanism. Since the relative sensitivity is the total sensitivity for each hypothesis divided by the smallest total sensitivity, the highest relative sensitivity value indicates which hypothesis best explains the mechanism of dispersal.

RESULTS

Comparisons of the dispersal frequency distributions were made within the demographic hypotheses and the genetic polymorphism hypotheses, but not between groups; this comparison was not made since the gentic polymorphism mechanisms described a form of dispersal different from the demographic mechanisms. The genetic polymorphism hypotheses assumed a genetic characteristic was responsible for dispersal, while the demographic hypotheses proposed that dispersal was a function of demographic pressures such as density. A direct comparison between these two sets of hypotheses would therefore not be valid.

The sensitivity analysis of demographic hypotheses indicated that individual aggression was the most sensitive variable in eliciting changes in the dispersal frequency distribution. Individual aggression was followed, in decreasing degree of sensitivity, by local aggression level, territorial aggression and density-dependent interactions (Table 1).

The most sensitive variables in the genetic polymorphism hypotheses were, in decreasing order of sensitivity, aggression threshold for confrontations, density-dependent interactions, environmental variability and dispersal of

highly aggressive individuals (Table 2).

Table 1. Relative sensitivity and total sensitivity values and deviation from control frequency distribution for higher and lower limits of each variable tested for the demographic hypotheses.

	Individual Aggression	Localized Aggression	Territorial Aggression	Intraspecific Interactions
Lower value	.063	.035	.041	.016
Upper value	.037	.026	.019	.023
Total	0.100	0.061	0.060	0.039
Relative sensitivity	2.81	1.56	1.54	1.00

Table 2. Relative sensitivity and total sensitivity values for each variable and deviation from control frequency distribution for higher and lower limits of each variable tested for the genetic polymorphism hypotheses.

	Aggression Threshold	Environmental Variation	Highly Aggressive Individuals	Intraspecific Interactions
Lower value	.031	.014	.015	.027
Upper value	.040	.019	.013	.043
Total	0.071	0.033	0.028	0.070
Relative sensitivity	2.54	1.18	1.00	2.50

DISCUSSION

While many hypotheses for dispersal can be tested in the field, some cannot. Simulation models have been used to avoid this limitation in field research of dispersal and have explored large numbers of hypotheses in order to determine those meriting experimental testing. In this study, several mechanisms for dispersal were examined through computer simulations to determine which dispersal mechanism could be most logically inferred from the ecological theory on which the model was designed.

Through evolutionary time, a mechanism for dispersal evolved; the evolved mechanism was theorized to be more responsive to population pressures and cause a greater dispersive force than other intraspecific processes. I used a sensitivity analysis to evaluate the dispersive force of each possible dispersal mechanism. I assumed that the dispersal mechanism which had the greatest impact on the dispersal distance frequency distribution was the most logical mechanism for dispersal. Although the operation of less sensitive hypotheses for dispersal was not ruled out, they were considered to be of lesser importance.

The sensitivity analysis of the demographic hypotheses indicate that the least sensitive mechanism examined

is the hypothesis that dispersal has a direct relationship to density changes (relative sensitivity 1.00). Analysis also indicates that although the territorial spacing hypothesis as a cause of dispersal and the hypothesis that local aggression levels have a direct relationship to density and have equal relative sensitivities, they both have considerably less sensitivity than the individual aggression hypothesis (relative sensitivity 1.54, 1.56. and 2.81 respectively). The individual aggression hypothesis results in the greatest deviation in the distribution of dispersal distances, which indicates the individual aggression hypothesis as the most feasible demographic dispersal hypothesis. The proposed mechanism for the individual aggression hypothesis is that as density increases, individual aggression levels rise; this rise in aggression increases the frequency of aggressive confrontations. Once a confrontation occurs, the dominant individual in the encounter forces the subordinate animal to disperse. Dispersal then results from subordinate animals losing confrontations to more dominant animals, instead of subordinate animals moving as a result of living in an aggressive neighborhood as proposed by the local aggression level hypothesis. My results support the proposed dispersal mechanism of Thiessen (1966), Delong (1967), Newsome (1969), Christian (1970), and Wilson (1975).

As stated earlier, the genetic polymorphism hypotheses propose two forms of dispersal, a saturation form

and a presaturation form. Saturation dispersal is synonymous with demographic dispersal, and presaturation dispersal results from a genetic characteristic for dispersal that presumedly evolved in variable environments. Since the evolutionary hypotheses assume that a genetic characteristic for dispersal exists, the importance of the evolutionary hypotheses is directly related to the degree in which a species evolved a genetic dispersal locus.

Of the genetic polymorphism hypotheses, the proposed mechanism that highly aggressive individuals are more likely to disperse has the smallest contribution in causing presaturation dispersal (relative sensitivity 1.00). A small contribution to presaturation dispersal also exists for the hypothesis that temporal variation directly causes dispersal (relative sensitivity 1.18), despite the fact that a dispersal polymorphism has been proposed to evolve in a temporal and spatial variable environment.

The two genetic polymorphism hypotheses with the highest sensitivities are presaturation dispersal operates equivalently to saturation dispersal, and presaturation dispersal is more sensitive to increasing aggression (relative sensitivity 2.50 and 2.54 respectively). This indicates that the ultimate mechanism for presaturation dispersal is a density-dependent mechanism, with presaturation dispersal individuals being more sensitive to increased aggression levels than individuals living in conditions

of saturation dispersal. Since individual aggression has the largest contribution to dispersal for the demographic hypotheses, and since the proposed aggression mechanism is density-dependent, I assumed the ultimate mechanism for presaturation dispersal is an increase in individual aggression, causing presaturation individuals to disperse before saturation individuals. Presaturation dispersers are therefore more sensitive to increases in individual aggression. My results support Lidicker's (1962) hypothesis that the dispersal mechanism for innate dispersers is similar to that for environmental dispersers; however, innate dispersers are more sensitive to increasing densities than environmental dispersers.

MANAGEMENT IMPLICATIONS

Although a controversy exists regarding the mechanism of dispersal, the importance of dispersal to the management of wildlife species is unquestioned. This importance lies in the fact that dispersal must be considered in determining management goals in wildlife sanctuaries and refuges, recolonizing ability of species, areas requiring restocking from wildlife refuges, and in predicting the future size and demographic character of wildlife populations.

Dispersal affects the future size and demographic character of wildlife populations by increasing the gross mortality rate and reducing population growth rates. It can also change an area's existing social hierarchy and shift the sex ratio away from a value of unity, since more males disperse than females. As dispersing animals are predominantly composed of juveniles, dispersal is a mechanism which can drive the existing age structure towards a more stable one.

Populations of animals restricted from dispersing have a lower intrinsic rate of increase than unrestricted populations (Mazurkiewicz, 1972; and Lidicker, 1974). By studying dispersal patterns of a species, a more accurate

prediction of future population size and future population surpluses can be made. Hunting limits are based on projected population trends; as a result of dispersal studies, a determination of length of the hunting season and of hunting limit is facilitated.

Dispersal of hoofed animals should be considered when management goals for animal reserves are determined. Since populations of hoofed animals tend to outgrow their available food supplies (Wodszicki, 1950), a force to disperse arises as the carrying capacity of the area is approached. Islands of natural sanctuaries or refuges however, serve as small areas of suitable habitat (Petrides, 1974); dispersal of animals from these areas would not occur. Analogously, where dispersal was prevented in Microtus pennsylvanicus, population levels increased to a point where the habitat was destroyed (Krebs et al., 1969). In order to protect natural reserves from overgrazing, the large herbivore populations must be prevented from reaching a density at which they can destroy their habitat (Petrides, 1974).

An application of dispersal studies occurs in the management of hunting activity. Since the majority of hunting activity occurs in accessible areas, game species in these areas suffer high hunting mortality. Dispersal is then important in determining the species' ability to recolonize vacant areas.

Dispersal ability is an important factor to consider

when determining the value of wildlife refuges in the restocking of local areas. Sanctuaries and refuges are designed to serve as breeding grounds for a population; in evaluating their proposed function, studies of the animal's ability to disperse can indicate whether the refuge can effectively serve as grounds for a breeding population.

Dispersal is an important management and research consideration; by increasing our understanding of the mechanisms behind dispersal, movement patterns can be explained more accurately. From these further studies, improved management plans and goals can be designed for future use.

APPENDIX

Appendix. Dispersal computer model listing.

```
      PROGRAM DSPRSL (OUTPUT)
      DIMENSION ACAX(900),ACAY(900),DEAD(900),ECDEN(900),LAGGRES(900),
     1     AGGRESS(900),ENCOUNT(900),MATURE(900),AGE(900),HOME(900),
     2     CCORE(900),SEX(900),XCORD(900),YCORD(900)
      DIMENSION ITABLE(42),DISPER(26),FDD(26),   AGETAB(9),STABAGE(21)
      REAL LLI, LAGGRES, MATURE
      DATA (DEAD(I),I=1,900)/900*1.0/
      DATA (CCORE(I),I=1,900)/900*0./
      DATA (DISPER(I),I=1,26)/26*0./
      DATA (AGETAB(I),I=1,9)/.0,.04,.15,.45,.65,.75,.75,.7,.6/
      DATA(STABAGE(I),I=1,21)/20.,14.17,11.29,9.35,7.94,6.8,5.857,5.072,
     1  4.38,3.81,3.289,2.83,2.398,2.033,1.668,1.301,1.032,.88,.75,.609,
     2  .5/
      DATA(ITABLE(I),I=1,42)/1,2,3,4,5,6,7,8,9,10,11,12,13,14,15,16,17,
     1  18,19,20,21,22,23,24,25,26,27,28,29,30,31,32,33,34,35,36,37,38,
     2  39,40,41,42/
C
C   SET MODEL INITIAL CONDITIONS FOR EACH RUN
      NINV = 100
      W = 13333.
      CALL RANSET(W)
C   SET SEASONAL COUNTER (SCOUNT)
      SCOUNT = 0.
      KOUNT = 1
      STEP = 1.62
      SR= 10.4976
      XC = 21.5
      YC = 21.5
      GRID = 7.
C   PLACE ANIMALS IN THE GRID FOR THE INITIAL CONDITION
      AVAGG = 0.
      SSURV = FLOAT(NINV)
      X = 1.
      DO  10  I=1,NINV
      RR = RANF(-1) * GRID
      RA = RANF(-1) * 6.283185307
      ACAX(I) = XC + RR * COS(RA)
      ACAY(I) = YC + RR * SIN(RA)
      XCORD(I) = ACAX(I)
      YCORD(I) = ACAY(I)
      ENCOUNT(I) = 10.
      DEAD(I) = 0.
C   AGES INITIALIZED TO STABLE AGE DISTRIBUTION
    3 Y = RANF(-1)
      IAGE = .5 + FNL(STABAGE,0.0,0.05,20,Y)
C   INITIALIZE INDIVIDUALS
      AGE(I) = FLOAT(IAGE)
      MATURE(I) = 0.
      IF(AGE(I) .GT. 5. ) MATURE(I) = 1.
      Z = AGE(I)
      AGGRESS(I) = FNL(AGETAB,0.,2.5,8,Z)
      AVAGG = AVAGG + AGGRESS(I)
      HOME(I) = 0.
      IF(X - 1.) 8,9,9
    8 SEX(I) = 1.
      X = 1.
      GO TO 10
    9 SEX(I) = 0.
      X = 0.
   10 CONTINUE
      AVAGG = AVAGG/SSURV
```

```
      C
      C   SET VALUES FOR MODEL CONTROL PARAMETERS
         25 DT = 1.
65          T = 0.
            DUR = 20.
            NITER=DUR/DT+.01
      C   SET RANDOM NUMBER GENERATOR TO RUN A MONTE CARLO SIMULATION
            W = TIME(Y)
70          CALL RANSET(W)
      C
      C   PROGRAM IS RUN FOR ONE ITERATION
            DO 400 ITER=1,NITER
            CALL MEET(ACAX,ACAY,DEAD,ECDEN  ,LAGGRES,AGGRESS,CCORE,ENCOUNT,
75         1  NINV,SR,AVECDEN,SSURV,SCOUNT,AGE)
            CALL BREED(AVECDEN,AVAGG,SCOUNT,BRDSN,NINV,MATURE,ECDEN,AGE,SEX,
           1  DEAD)
            CALL FIGHT(AGE,HOME,ENCOUNT,AGGRESS,DEAD,NINV,BRDSN,AVAGG,AGETAB,
           1  SSURV,MATURE,ECDEN)
80          CALL MOVE(ACAX,ACAY,HOME,AGGRESS,DEAD,CCORE,NINV,SR,STEP,AGE)
            CALL REPROD(SEX,DEAD,MATURE,LAGGRES,NINV,BRDSN,ACAX,ACAY,AGE,HOME,
           1  AGGRESS,ENCOUNT,XCORD,YCORD,ECDEN)
            IF(KOUNT .NE. 10) GO TO 200
            KOUNT = 0
85    C
      C   COMPUTE OUTPUT
      C   DISPERSAL DISTANCES FOR TOTAL POPULATION
            DO  75  I=1,NINV
            IF(DEAD(I)) 50,50,75
90       50 DDX = XCORD(I) - ACAX(I)
            DDX = DDX * DDX
            DDY = YCORD(I) - ACAY(I)
            DDY = DDY * DDY
            DD = SQRT(DDX + DDY)
95          IDD = DD + 1.5
            IF(IDD . GT. 25) IDD = 26
            DISPER(IDD) = DISPER(IDD) + 1.
         75 CONTINUE
            PRINT 900, T
100         PRINT 901,(ITABLE(I),I=1,25)
            DO  65  II=1,26
      C   FREQUENCY OF DISPERSAL DISTANCES (FDD)
            FDD(II) = DISPER(II)/SSURV
         65 DISPER(II) = 0.
105         PRINT 904,(FDD(I),I=1,26)
        200 KOUNT = KOUNT + 1
      C
      C   UP DATE TIME
            T=T+DT
110         CALL SURVIVE(AGE,DEAD,AGGRESS,ENCOUNT,HOME,MATURE,LAGGRES,NINV,
           1  SSURV,ECDEN,T)
        400 CONTINUE
        900 FORMAT(1H0,*FREQUENCY OF DISPERSAL DISTANCES MEASURED IN TRAP UNIT
           1S AFTER*,F5.0,* TIME PERIODS.*)
115     901 FORMAT(1H0,*   0*,25I5)
        904 FORMAT(1H0,26F5.3)
            STOP
            END
```

```
      SUBROUTINE MEET(ACAX,ACAY,DEAD,ECDEN  ,LAGGRES,AGGRESS,CCORE,
     1  ENCOUNT,NINV,SR,AVECDEN,  SSURV,TIME,AGE)
C   THIS SUBROUTINE CALCULATES LOCAL RESOURCE AVAILABILITY, LOCAL AGGRESSION
C   LEVELS, AND NUMBER OF ENCOUNTERS.
      DIMENSION ACAX(900),ACAY(900),DEAD(900),ECDEN(900),CCORE(900),
     1     ENCOUNT(900),LAGGRES(900),CCORE2(900),AGGRESS(900),AGE(900)
      DIMENSION CCPVAL(4)
      REAL LAGGRES
      DATA (CCORE2(I),I=1,900)/900*0./
      DATA ANGLE/1.256637061/,AMP/6./
      DATA (CCPVAL(I),I=1,4)/20.,20.,20.,0.0/
C   DETERMINE THE NUMBER OF ENCOUNTERS BETWEEN ANIMALS AND THE LOCALIZED
C   ECONOMIC DENSITY
      AVECDEN = 0.
      IF(TIME .EQ. 5.) 1,2
    1 TIME = 1.
      ANG = ANGLE
      GO TO 3
    2 TIME = TIME + 1.
      ANG = TIME * ANGLE
    3 SEASON = AMP * SIN(ANG + .628318531)
      NINVM1 = NINV - 1
      DO  50  I=1,NINVM1
      IF(DEAD(I)) 5,5,50
    5 LAGGRES(I) = 0.
C   CARRYING CAPACITY AS A FUNCTION OF THE NUMBER OF ANIMALS PRESENT
C   DURING THE FORMER TIME PERIOD (ENCTM1)
      ENCTM1 = ENCOUNT(I)
      ENCOUNT(I) = 0.
      A = FNL(CCPVAL,0.0,15.,3,ENCTM1)
      CCP = A + SEASON
C   DISTANCE BETWEEN ANIMALS
      INVP1 = I + 1
      DO  30  J=INVP1,NINV
      IF(AGE(I) .LE. 2.0 .AND. AGE(J) .LE. 2.0) GO TO 30
      IF(DEAD(J)) 10,10,30
   10 DBAX = ACAX(I) - ACAX(J)
      DBAX = DBAX * DBAX
      DBAY = ACAY(I)- ACAY(J)
      DBAY = DBAY * DBAY
      DBA = DBAX + DBAY
C   DETERMINE WHETHER THERE IS A CONFRONTATION
      IF(DBA - SR) 20,20,30
   20 IF(AGE(I) .LE. 2.) 26,21
   21 IF(AGE(J) .LE. 2.) 23,22
   22 ENCOUNT(I) = ENCOUNT(I) + 1.
      LAGGRES(I) = LAGGRES(I) + AGGRESS(J)
   23 CCORE(J) = CCORE(J) + 1.
      CCORE2(J) = CCORE2(J) + AGGRESS(I)
      GO TO 30
C   RECORD ENCOUNTERS AND LOCAL AGGRESSION LEVEL.
   26 ENCOUNT(I) = ENCOUNT(I) + 1.
      LAGGRES(I) = LAGGRES(I) + AGGRESS(J)
   30 CONTINUE
      ENCOUNT(I) = ENCOUNT(I) + CCORE(I)
C   CALCULATE ECONOMIC DENSITY.
      IF(CCP .EQ. 0.) 31,32
   31 ECDEN(I) = 1.5
      GO TO 33
   32 ECDEN(I) = ENCOUNT(I)/CCP
   33 AVECDEN = AVECDEN + ECDEN(I)
      LAGGRES(I) = (LAGGRES(I) + CCORE2(I) + AGGRESS(I))
```

```
      CCORE2(I) = 0.
   50 CCORE(I) = 0.
      IF(DEAD(NINV)) 55,55,70
   55 ENCTM1 = ENCOUNT(NINV)
      ENCOUNT(NINV) = CCORE(NINV)
      A = FNL(CCPVAL,0.0,15.,3,ENCTM1)
      CCP = A * SEASON
      IF(CCP .EQ. 0.) 60,61
   60 ECDEN(NINV) = 1.5
      GO TO 62
   61 ECDEN(NINV) = ENCOUNT(NINV)/CCP
   62 LAGGRES(NINV) = (CCORE2(NINV) * AGGRESS(NINV))
      CCORE(NINV) = 0.
      CCORE2(NINV) = 0.
      AVECDEN = AVECDEN + ECDEN(NINV)
   70 AVECDEN = AVECDEN/SSURV
      RETURN
      END
```

```
      SUBROUTINE BREED(AVECDEN,AVAGG,TIME,BRDSN,NINV,MATURE,ECDEN,AGE,
     1   SEX,DEAD)
C    THIS SUBROUTINE DETERMINES WHETHER BREEDING WILL OCCUR
      DIMENSION MATURE(900),ECDEN(900),AGE(900),SEX(900),DEAD(900)
      DIMENSION EDENVAL(5),AGGVAL(5),CVAL(9)
      REAL MATURE
      DATA (EDENVAL(I),I=1,5)/0.0,4.0,9.0,18.0,30./
      DATA (AGGVAL(I),I=1,5)/0.0,3.5,5.0,6.5,10./
      DATA (CVAL(I),I=1,9)/3.99,3.25,2.75,2.50,2.25,2.0,1.75,1.25,0.0/
      IF(TIME .GT. 3.) 5,10
    5 BRDSN = 0.
      GO TO 15
C   DETERMINE EFFECT OF POPULATION DENSITY (A)
   10 A = FNL(EDENVAL,0.,.30,4,AVECDEN)
C   DETERMINE EFFECT OF POPULATION AGGRESSION LEVEL (B)
      B = FNL(AGGVAL,0.,.75,4,AVAGG)
C   NUMBER OF BREEDING INTERVALS (BRINT)
      C = B * A
      BRINT = FNL(CVAL,0.,5.0,8,C) + 0.5
C   DETERMINE WHETHER BREEDING OCCURS
      BRDSN = 0.
      IF(TIME .LE. BRINT) BRDSN = 1.
   15 CONTINUE
C   MATURITY DELAY AS A FUNCTION OF ECONOMIC DENSITY (ECDEN)
C   DETERMINE LENGTH OF THE DELAY
      DO  50  I=1,NINV
      IF(AGE(I) .LE. 5.) GO TO 50
      IF(DEAD(I) .EQ. 1.) GO TO 50
      IF(MATURE(I) .EQ. 1.) GO TO 50
      IF(ECDEN(I) .LE. .75) 20,25
   20 DELAY = 0.
      GO TO 35
   25 IF(ECDEN(I) .LE. 1.0) 26,30
   26 DELAY = 1.
      GO TO 35
   30 DELAY = 2.
C   TEST INDIVIDUALS
   35 X = AGE(I) - 6.
      IF(X .GE. DELAY) 40,50
   40 MATURE(I) = 1.
   50 CONTINUE
      RETURN
      END
```

```
      SUBROUTINE FIGHT(AGE,HOME,ENCOUNT,AGGRESS,DEAD,NINV,BRDSN,AVAGG,
     1  AGETAB,SSURV,MATURE,ECDEN)
C    THIS SUBROUTINE DETERMINES AN INDIVIDUALS AGGRESSION
      DIMENSION AGE(900),HOME(900),ENCOUNT(900),AGGRESS(900),DEAD(900),
     1     MATURE(900),AGETAB(9),ECDEN(900)
      REAL MATURE
C    DETERMINE AGGRESSION LEVELS FOR EACH ANIMAL
      AVAGG = 0.
      DO  50  I=1,NINV
      IF(DEAD(I)) 4,4,50
C    AGGRESSION AS A FUNCTION OF BREEDING CONDITION
    4 IF(BRDSN) 8,8,5
    5 IF(MATURE(I)) 8,8,6
    6 A = .5
      GO TO 15
    8 A = 0.
C    AGGRESSION AS FUNCTION OF AGE
   15 X = AGE(I)
      B = FNL(AGETAB,0.0,2.5,8,X)
C    AGGRESSION AS FUNCTION OF LOCATION
      IF(HOME(I)) 20,20,25
   20 C = 0.0
      GO TO 30
   25 C = .2
C    AGGRESSION AS FUNCTION OF NUMBER OF ENCOUNTERS PER TIME PERIOD
   30 D = ENCOUNT(I) * (0.02 + 0.001 * ENCOUNT(I))
C    AGGRESSION  AS FUNCTION OF ECONOMIC DENSITY (ECDEN)
      E = ECDEN(I) *(.5 + ECDEN(I))
C    AGGRESSION
      AGGRESS(I) = A + B + C + D + E
      AVAGG = AGGRESS(I) + AVAGG
   50 CONTINUE
      AVAGG = AVAGG/SSURV
      RETURN
      END
```

```
      SUBROUTINE MOVE (ACAX, ACAY, HOME, AGGRESS, DEAD, CCORE, NINV, SR,
     1  STEP,AGE)
C     THIS SUBROUTINE DETERMINES WHICH ANIMALS DISPERSE
      DIMENSION ACAX(900),ACAY(900),HOME(900),DEAD(900),CCORE(900),
     1     AGGRESS(900),AGE(900)
      NINVM1 = NINV - 1
      DO  50  I = 1,NINVM1
      IF(DEAD(I)) 3,3,50
C     NOTE  CCORE IN THIS CASE IS USED TO DESIGNATE ANIMALS WHICH LOST
C     EARLIER CONFRONTATIONS
    3 IF(CCORE(I)) 4,4,30
C     HIGHLY AGGRESSIVE ANIMALS DISPERSE.  THIS ASSUMPTION ONLY APPLIES TO
C     THE GENETIC POLYMORPHISM HYPOTHESES.
    4 IF(AGGRESS(I) .GT. 2.9) GO TO 30
      IF(AGE(I) .LE. 2.) 50,5
C     DISTANCE BETWEEN ANIMALS
    5 INVP1 = I + 1
      DO  20  J=INVP1,NINV
      IF(DEAD(J)) 6,6,20
    6 IF(AGE(I) .LE. 2.) 20,7
    7 DBAX = ACAX(I) - ACAX(J)
      DBAX = DBAX * DBAX
      DBAY = ACAY(I) - ACAY(J)
      DBAY = DBAY * DBAY
      DBA = DBAX + DBAY
C     DETERMINE WHETHER A CONFRONTATION OCCURS
      IF(DBA - SR) 10,20,20
C     INDIVIDUAL DISPERSAL AS A FUNCTION OF INDIVIDUAL AGGRESSION
   10 IF(AGGRESS(I) .LE. 1.5 .AND. AGGRESS(J) .LE. 1.5) GO TO 20
      IF(AGGRESS(I) - AGGRESS(J)) 30,14,18
   14 RN = RANF(-1)
      IF(RN - .5) 18,30,30
   18 CCORE(J) = 1.
   20 CONTINUE
      HOME(I) = 1.0
      GO TO 50
C     ANIMAL I MOVES
   30 A = RANF(-1) * 6.283185307
      ACAX(I) = ACAX(I) + STEP * COS(A)
      ACAY(I) = ACAY(I) + STEP * SIN(A)
      HOME(I) = 0.
      CCORE(I) = 0.
   50 CONTINUE
      IF(CCORE(NINV) .EQ. 1.0) 55,60
   55 A = RANF(-1) * 6.283185307
      ACAX(NINV) = ACAX(NINV) + STEP * COS(A)
      ACAY(NINV) = ACAY(NINV) + STEP * SIN(A)
      HOME(NINV) = 0.
      GO TO 62
   60 HOME(NINV) = 1.
   62 CCORE(NINV) = 0.
      RETURN
      END
```

```
      SUBROUTINE REPROD(SEX,DEAD,MATURE,LAGGRES,NINV,BRDSN,ACAX,ACAY,AGE
     1  ,HOME,AGGRESS,ENCOUNT,XCORD,YCORD,ECDEN)
C     THIS SUBROUTINE DETERMINES THE PREGNANCY RATE OF EACH FEMALE, HER LITTER
C     SIZE, AND PLACES THESE NEW INDIVIDUALS INTO THE POPULATION
      DIMENSION SEX(900),DEAD(900),MATURE(900),LAGGRES(900),ACAX(900),
     1     ACAY(900),AGE(900),HOME(900),ENCOUNT(900),AGGRESS(900),
     2     XCORD(900),YCORD(900),ECDEN(900)
      DIMENSION NMLVAL(41),COUNT(20),MX(20)
      REAL MATURE,LAGGRES,MX
      INTEGER HOLD
      DATA (NMLVAL(I),I=1,41)/-3.5,-1.96,-1.645,-1.439,-1.281,-1.150,
     1 -1.037,-.925,-.841,-.755,-.674,-.598,-.524,-.454,-.386,-.312,
     2 -.253,-.189,-.126,-.056,0.,.056,.126,.189,.253,.312,.386,.454,
     3 .524,.598,.674,.755,.841,.925,1.037,1.150,1.281,1.439,1.645,1.96,
     4 3.5/
C     CHECK WHETHER BREEDING SEASON (BRDSN)
      SLITTER = 0.
      IF(BRDSN .EQ. 0.) RETURN
      LHOLD = 0
      X = 1.
      DO  10  I=1,20
      COUNT(I) = 0.
   10 MX(I) = 0.
C     CHECK TO DETERMINE IF INDIVIDUAL IS REPRODUCTIVE FEMALE
      DO  90  I=1,NINV
      IF(MATURE(I) .EQ. 0.) GO TO 90
      IF(SEX(I) .EQ. 0.) GO TO 90
      IF(DEAD(I) .EQ. 1.) GO TO 90
C     NOTE: COUNT COUNTS THE NUMBER OF FEMALES IN EACH AGE CLASS.
   50 IAGE = AGE(I)
      COUNT(IAGE) = COUNT(IAGE) + 1.
C     PREGNANCY RATE (PREGR)
      PREGR = 90.
C     DETERMINE WHETHER INDIVIDUAL BREEDS
      RANPR = RANF(-1)
      IF(RANPR .GT. PREGR) GO TO 90
C     LITTER SIZE
      R = RANF(-1)
      R = FNL(NMLVAL, 0.,0.025,40,R)
      LITTER = 1.4 * R + 5.5
      SLITTER = SLITTER + FLOAT(LITTER)
C     PLACE NESTLINGS INTO THE POPULATION
      IF(LITTER .EQ. 0) GO TO 90
      DO  80  IND =1,LITTER
      HOLD = LHOLD + 1
      DO  60  J=HOLD,900
      IF(DEAD(J)) 60,60,55
   55 LHOLD = J
      GO TO 61
   60 CONTINUE
C     INITIALIZE NEWBORN INDIVIDUALS
   61 DEAD(J) = 0.
      MATURE(J) = 0.
      AGE(J) = 0.
      HOME(J) = 0.
      AGGRESS(J) = 0.
      ACAX(J) = ACAX(I)
      XCORD(J) = ACAX(J)
      ACAY(J) = ACAY(I)
      YCORD(J) = ACAY(J)
      LAGGRES(J) = LAGGRES(I)
      ENCOUNT(J) = ENCOUNT(I)
```

```
      ECDEN(J) = ECDEN(I)
      IF(X - 1.) 70,75,75
   70 SEX(J) = 1.
65    MX(IAGE) = MX(IAGE) + 1.
      X = 1.
      GO TO 80
   75 SEX(J) = 0.
      X = 0.
70    80 CONTINUE
      90 CONTINUE
      IF(LHOLD .GT. NINV) NINV = LHOLD
      DO  100  I=1,20
      IF(COUNT(I) .EQ. 0.) GO TO 100
75    MX(I) = MX(I)/COUNT(I)
  100 CONTINUE
      PRINT 901, SLITTER
      PRINT 900, (MX(I),I=5,20)
  900 FORMAT(1H0,*MX    *,16F8.4)
80    901 FORMAT(1H0,*THE TOTAL RECRUITMENT FOR THIS TIME PERIOD IS*,F10.0)
      RETURN
      END
```

```
      SUBROUTINE SURVIVE(AGE,DEAD,AGGRESS,ENCOUNT,HOME,MATURE,LAGGRES,
     1 NINV,SSURV,ECDEN,T)
C    THIS SUBROUTINE DETERMINES WHICH INDIVIDUALS SURVIVE
      DIMENSION AGE(900),DEAD(900),AGGRESS(900),ENCOUNT(900),HOME(900),
     1     MATURE(900),LAGGRES(900),ECDEN(900),COUNT(30)
      REAL LAGGRES,MATURE,LAMBDA
      SSURV = 0.
      SDEAD = 0.
      DO  5  I=1,20
    5 COUNT(I) = 0.
      DO  100  I=1,NINV
      IF(DEAD(I)) 10,10,100
C    SURVIVAL AS A FUNCTION OF INDIVIDUAL AGGRESSION (A),DISPERSAL (B),
C    AGGRESSION LEVEL (C),DENSITY (D), AND PHYSIOLOGICAL AGE (E)
   10 IF(AGE(I) .LE. 1.0) 11,20
   11 A = (AGGRESS(I)- .775) * 0.01046
      B = 0.06
      C = LAGGRES(I) * (0.005 + 0.0001 * LAGGRES(I))
      D = ENCOUNT(I) * (0.0065 + 0.0003 * ENCOUNT(I))
      E = 0.04395
      F = 0.18 * ECDEN(I) * (1. + ECDEN(I))
      GO TO 60
   20 IF(MATURE(I) .EQ. 1.) 30,21
   21 A = (AGGRESS(I) - .875) * 0.01214
      IF(HOME(I)) 23,23,25
   23 B = 0.05
      GO TO 26
   25 B = 0.0
   26 C = LAGGRES(I) * (0.0018 + 0.0000337 * LAGGRES(I))
      D = ENCOUNT(I) * (0.0016 + 0.0001 * ENCOUNT(I))
      E = 0.0085
      F = 0.08  * ECDEN(I) * (1. + ECDEN(I))
      GO TO 60
   30 IF(AGE(I) .LE. 19.) 31,85
   31 A = (AGGRESS(I) - 1.85) * 0.00629
      IF(HOME(I)) 33,33,35
   33 B = 0.03
      GO TO 36
   35 B = 0.0
   36 C = LAGGRES(I) * (0.0011 + 0.000023 * LAGGRES(I))
      D = ENCOUNT(I) * (0.0011 + 0.00008 * ENCOUNT(I))
      E = 0.0075
      F = 0.075 * ECDEN(I) * (1. + ECDEN(I))
C    DETERMINE WHETHER ANIMAL SURVIVES
   60 SURV = (1.+A)*(1.-B)*(1.-C)*(1.-D)*(1.-E)*(1.-F)
      PSURV = RANF(-1)
      IF(PSURV - SURV) 80,80,85
   80 AGE(I) = AGE(I) + 1.
      SSURV = SSURV + 1.
      IAGE = AGE(I)
      COUNT(IAGE) = COUNT(IAGE) + 1.
      GO TO 100
   85 DEAD(I) = 1.
   90 SDEAD = SDEAD + 1.
  100 CONTINUE
      PRINT 901, SSURV,SDEAD,LAMBDA
      PRINT 903, (COUNT(I),I=1,20)
  901 FORMAT(1H0,*SSURV = *,F5.0,10X,*SDEAD = *,F5.0)
  903 FORMAT(1H ,*NX    *,20F6.0)
      RETURN
      END
```

```
      FUNCTION FNL(DVAL,XS,DX,N,X)
C     THIS FUNCTION PROVIDES TABLE VALUES
      DIMENSION DVAL(1)
      IF(X -XS) 3,3,2
    2 IF(X - XS - N * DX) 7,5,5
    3 FNL = DVAL (1)
      GO TO 10
    5  FNL = DVAL(N + 1)
      GO TO 10
    7 XD = X - XS
      I = 1.0 + XD/DX
      FNL = (XD - FLOAT(I-1) * DX) * (DVAL(I+1) - DVAL(I))/DX + DVAL(I)
   10 RETURN
      END
```

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