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REVIEW/SYNTHESE

A review of the Chitty Hypothesis of population regulation[1]

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Populations do not increase without limit, and one of the central problems of population biology is to explain why. The self-regulation hypothesis states that indefinite increase in population density is prevented by a change in the quality of the population. Changes in quality may be physiological or behavioural, genotypic or phenotypic, and three different mechanisms of self-regulation have been proposed: (1) the Stress Hypothesis suggests that mutual interactions lead to physiological changes, phenotypic in origin, that reduce births and increase deaths. (2) The Behaviour Hypothesis suggests that mutual interactions involving spacing behaviour prevent unlimited increase and that spacing behaviour is not an inherited trait. (3) The Chitty Hypothesis, or polymorphic behaviour hypothesis, postulates that spacing behaviour limits population density and that individual differences in spacing behaviour have a genetic basis and respond to rapid natural selection.

The testability of the Chitty Hypothesis is examined with regard to 13 predictions that are explicit in Chitty's writings or derived by subsequent workers. Many of the predictions are not unique to the Chitty Hypothesis and only a few difficult manipulations adequately test Chitty's proposed mechanism to the exclusion of all others.

Four population studies are reviewed with reference to the Chitty Hypothesis. While the detailed mechanism proposed by Chitty is not yet adequately tested in any population, his general belief that both behaviour and genetics are relevant to understanding population problems is now assumed by the new generation of population biologists.

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> "Perhaps ecologists of the next generation will be more successful. . . . They might start by doubting the truth of everything that has so far been written on the subject, including the ideas of the present reviewer." (Chitty, D. Ecology, 48: 701. 1967*a*.)

[1]This paper is dedicated to Professor Dennis Chitty upon his retirement from teaching at the Department of Zoology, University of British Columbia, June 1978.

Introduction

Population biology rests on two fundamental observations. First, populations do not increase without limit and seldom achieve their potential rate of increase. Second, animals are common in some areas and scarce in others. Aristotle, Malthus, and Darwin all knew these truths, but only in the present century have we tried to understand the mechanisms behind these simple observations of nature. Conceptual advance is limited by the framework we use to analyze a problem, and population biology has provided a series of conceptual frameworks that have been the subject of bitter arguments (Tamarin 1978*a*). This situation is typical of the preparadigm state of science (Kuhn 1962) and ecologists can be encouraged that at least this part of their science is continuing the traditions established during the 16th-century debates on the nature of the solar system.

The polemic on population regulation probably peaked at the 1957 Cold Spring Harbor Symposium on Population Studies. The controversy had been undermined by a series of studies on natural populations which had proven difficult to fit into the older conceptual frameworks. Chitty's studies on the population fluctuations of the vole *Microtus agrestis* (Chitty 1952) were a significant turning point which helped change the conceptual framework of population biology. In this review I will discuss the current hypotheses of populations regulation and compare these with the Chitty Hypothesis. I will examine the predictions that the Chitty Hypothesis makes and discuss the application of these ideas to four current population problems.

Definition of the Problem

Simple mathematical models have been used by most authors to analyze why no population increases without limit. Population density will increase until the birth rate and the death rate are equal (Fig. 1). For this to happen either the birth rate must fall as density rises, or the death rate must increase as density rises. This type of relationship, idealized in Fig. 1 as linear, is called *density dependent*, and this simple model illustrates the first rule of population regulation: *no population stops increasing unless either the birth rate or the death rate is density dependent*.

A model of this sort is included in all the introductory ecology textbooks (e.g., Krebs 1978, Chap. 15). Note that this model is not a hypothesis but a definition of logical relationships; it cannot be tested and it cannot be doubted (Lack 1966, p. 291). One small qualification needs to be made: 'birth rate' should be births + immigration and 'death rate' should include deaths + emigration. The problem with the model in Fig. 1 is how to apply it to the real world, and it is here that hypotheses need to be introduced. Two hypotheses are widely assumed: H_1: the birth rate and death rate curves in Fig. 1 can be subdivided into components, some of which retain the same graphical relationship with population density and thus are called *density dependent*; H_2: these density-dependent components prevent unlimited increase or regulate population density. The job of the population ecologist, as defined by the density-dependent school, thus became to identify the density-dependent components for his particular population.

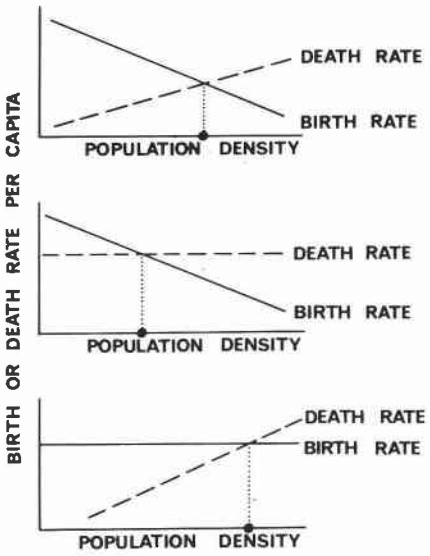

FIG. 1. The problem of population regulation: the theoretical view. Population density comes to an equilibrium only when the per capita birth rate equals the per capita death rate, and this is possible only if birth or death rates are *density dependent*. Many ecologists have tried to apply this density-dependent concept directly to real populations and this has resulted in a prolonged sterile controversy.

But hypothesis H_1 contains the hidden assumption that birth and death rates can be disaggregated into simple additive pieces with no interaction. For example, one might assume that the total death rate operating on an insect population at a certain density is composed of deaths due to (1) parasites, (2) insect predators, (3) vertebrate predators, (4) disease, (5) malnutrition, and (6) losses due to emigration. This assumption was no longer tenable after Errington (1945) discovered *compensatory mortality*, and many field workers found interactions among mortality factors. The strength of the belief about additive mortality, however, is plainly evident in some current practices. First, human mor-

tality data are catalogued under impressive lists of 'cause of death' even in the face of all the information about how smoking or old age predisposes people to death from a variety of causes. Ecologists perhaps have attempted to emulate medical science in trying to list the causes of death of wild animals and plants in the mistaken belief that this form of precision is necessary for understanding mortality patterns. Second, the belief that death rates can be broken down into additive pieces has been mathematically enshrined in *key factor analysis* (Varley et al. 1973), and a generation of insect ecologists continues to use this assumption uncritically (Manly 1977).

The assumption that birth rates can be broken down into pieces in a similar way has never been an attractive idea. Birth rates are often assumed to be constant and independent of population density, so that all attention is focused on mortality rates (Varley et al. 1973).

An alternative approach to the whole problem of population regulation was promoted by Andrewartha and Birch (1954), Chitty (1955), and others. They replaced the model of Fig. 1 by an empirical model of populations in the real world: *observe population changes, and test causal hypotheses experimentally*. The essence of this approach has been adopted by most ecologists since the 1950's, and the tiresome arguments about density dependence are gradually being replaced by more fruitful arguments about empirical relationships.

Chitty (1955) added an important conceptual advance to the empirical approach advocated by Andrewartha and Birch (1954). He provided a simple description of *self-regulation*. Suppose, Chitty argues, that we observe a population at two times, i and n, and that at time n there is a death rate (D_n) higher than the death rate at time i (D_i). This death rate is the result of the interaction of the organisms (O) with their mortality factors (M). Our problem now is to determine why D_n is greater than D_i. The first hypothesis to be explored is that on both occasions we are dealing with organisms whose biological properties are identical. In this case we must look for a difference between the mortality factors at the two times. In other words, we might expect to find at time n that there are more predators or parasites or that the weather is less favourable. Some population changes can certainly be explained in this manner, but in other cases this method has failed to turn up the right clues. We must look at the matter from another angle.

Consider, Chitty continues, the possibility that the environmental conditions are much the same at all times, that there is no important difference between the mortality factors at times i and n. In this case any change in the death rate must be due to a change in the nature of the organisms, a change such that they become less resistant to their normal mortality factors. For example, the animals might die in cold weather at time n, weather that they might have survived at time i. These ideas can be summarized as follows:

	First hypothesis	Second hypothesis
Time	$i \quad n$	$i \quad n$
Death rate	$D_i < D_n$	$D_i < D_n$
Organisms	$O_i = O_n$	$O_i \neq O_n$
Environment	$M_i \neq M_n$	$M_i = M_n$

The first hypothesis describes the classical approach to population regulation used, for example, by Lotka and Volterra, Nicholson, Thompson, Uvarov, and many others. The second hypothesis describes an ideal self-regulatory approach to population regulation. It is unlikely in nature that this second situation would occur in such an ideal form, but more likely that some mixture of these two situations would be found in self-regulatory populations. Note that the concept of density dependence becomes ambiguous under the second hypothesis. The idea that the environment can be subdivided into density-dependent and density-independent factors has meaning only insofar as the properties of the individuals in the population are constant. Self-regulatory systems have added an additional degree of freedom to the system, the individual with variable properties.

There has never been agreement on whether theories of population regulation should take the individual organism or the population as the central unit (Bakker 1964). By emphasizing the individual, Andrewartha and Birch, and Chitty, have pointed out the fallacy of dealing with *population density* as a sufficient measure. The elementary truth that we do in fact care whether a population of 40 humans contains all infants under 1 year or all middle-aged men was somehow lost in all the semantic arguments about density dependence (Bakker 1964). Lotka (1925) pointed out that two populations of equal size may have different properties, if the age structure is different. Leslie and Ranson (1940) recognized this for the vole *Microtus agrestis* and used it as a possible explanation of why cyclic declines might occur. Chitty (1960) generalized this point to include factors other than age in individual "quality," a central concept in all theories of self-regulation.

Figure 2 summarizes the definition of the prob-

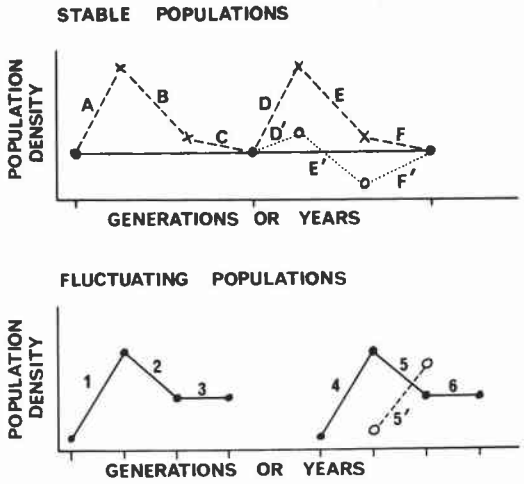

FIG. 2. The problem of population regulation: the pragmatic view. The emphasis is on observing real populations and on experimental manipulation. Stable populations are more difficult to study than fluctuating ones and must be experimentally perturbed to analyze their regulation. This approach has produced more fruitful insight into population dynamics than the approach shown in Fig. 1.

lem of regulation as seen by Chitty (1960) and by Andrewartha and Birch (1954). Two classes of populations are shown: *stable* and *fluctuating*. Consider first the fluctuating population, which is easiest to analyze. Increasing populations (1, 4, 5′) are defined to be the controls, or 'unregulated' populations, and all comparisons are with these. We can ask, for example: How does the population at time 2 differ from that at time 1? How does the population at time 3 differ from that at time 1? We answer these questions by first determining the demographic mechanisms behind the population changes. For example, the population at time 2 may have a lower birth rate than the population at time 1, or a higher death rate, or less immigration, or more emigration (or any combination). Next, we associate some factors as possible causes of the changed demographic rates. For example, food shortage might reduce births and increase deaths at time 2.

If the population fluctuation is singular, we are not able to test any hypotheses about its causes and we can only state the observed associations. There is remarkable disagreement among ecologists about what proportion of ecological events is unique. To assume that everything is unique is scientifically self-defeating, and I assume that many population fluctuations are repeated events. Thus the fluctuation 4, 5, 6 in Fig. 2 is postulated to have the same causes as the fluctuation 1, 2, 3. The comparison of time 4 and 5 is thus a statistical replicate of the comparison of times 1 and 2.

There is a serious practical problem in these comparisons of fluctuating populations. Because these are time series, and years differ, the comparisons of the population at times 1 and 2, for example, are confounded by all differences between times 1 and 2. This produces spurious correlations. Thus year 2 might be cold and wet relative to year 1, and we might postulate that cold and wet weather reduces survival rates. There are two ways to eliminate this problem. First, observe many different sequences like 1, 2, 3 and see if the correlation holds in other cases. Second, manipulate part of the population to produce the comparison of 5 and 5′ in Fig. 2. Many year-to-year differences can be eliminated by this experimental manipulation, and we can compare directly the factors causing the difference between 5 and 5′.

Most population biologists might agree that the scheme outlined in Fig. 2 does indeed represent a sound approach to the study of fluctuating populations. But the next step in the analysis can go two rather different ways. Chitty (1967*b*) suggested that we should determine the *necessary* and *sufficient conditions* associated with population fluctuations. This approach looks for invariant relationships and tests them experimentally; for example, is food shortage always associated with the change from time 1 to 2 (Fig. 2)? An alternative approach is to assume multiple causation and either catalogue a series of fluctuations (e.g., 75% of fluctuations have associated food shortage, 42% have epidemic disease, 27% have serious parasite mortality, etc.) or treat it as a problem in multiple regression with the Y variable being population growth rate. The second or statistical approach has been favored by some (e.g., Lidicker 1973). It has the disadvantage of requiring several lifetimes of work before enough data would be available for rigorous statistical testing, and it still leaves one at the stage of describing mere associations of events. For the present I think it easiest to adopt the Chitty approach, which is straightforward and rigorous and allows testing a hypothesis by studying only one fluctuation. But it is important to recognize that this methodological decision may be wrong and should be reevaluated after it has been tried (Popper 1972).

In a stable population: births + immigration = deaths + emigration, and nothing can be learned about population regulation by the methods we have just discussed unless we change our time frame. Figure 2 shows that we must shift our focus from changes *between* years (or generations) to changes *within* years, if we are to analyze stable populations. Figure 2 shows an artificially simplified life cycle of three stages: reproduction ('eggs'), juvenile, adult. If we observe a series of

years (e.g., the 2 years A, B, C; D, E, F), we can measure the relative variability of gains and losses in each stage. But to gain insight into a stable population, we must manipulate numbers. Figure 2 illustrates one experiment, reducing the number of 'eggs' (D′ vs. D) and following the subsequent two stages. Alternatively, we could reduce population density at the start of the year and follow its return to the stable density. Most of the problems of answering questions about population regulation in stable populations are the same as those in fluctuating populations, and the major need in analyzing stable populations is for an experimental outlook emphasizing population manipulations. Key-factor analysis (Varley et al. 1973) will tell us nothing about a stable population, but an experimental approach can. A splendid case history is now available for the great tit (*Parus major*). Compare the controversy over population regulation in the great tit (Lack 1966) with the precision of the experimental results of J. R. Krebs (1971) and Kluijver (1971).

To summarize: two conceptual approaches have been advanced as modes of explaining why no population increases without limit. The first approach uses the concept of density dependence and has been rendered less useful by the discovery of compensatory mortality and changes in the quality of individuals with density. The second approach uses observed changes in population density and experimental manipulations to determine the necessary and sufficient conditions for changes in numbers. The second framework has proven more fruitful than the first for analyzing real populations.

Hypotheses of Population Regulation

Some components of the environment must prevent population increase, and four broad schools of thought can be recognized (Tamarin 1978*a*). The *biotic school* led by Nicholson (1933) believes that unlimited increase is prevented by parasitism, predation, disease, and food shortage. The *climate school* led by Andrewartha and Birch (1954) believes that weather prevents increase. The *comprehensive school*, originated by Thompson (1929), believes that all environmental factors are involved. The *self-regulation school* believes that changes in individual 'quality' prevent unlimited increase. An overview of these schools is given in Krebs (1978) and will not be repeated here. I am concerned here with the self-regulation school.

The central hypothesis of the self-regulation school was stated first by Chitty (1960): ". . . all species are capable of limiting their own population densities without either destroying the food resources to which they are adapted, or depending on enemies or climatic accidents to prevent them from doing so" (p. 111), and ". . . under appropriate circumstances, indefinite increase in population density is prevented through a deterioration in the quality of the population" (p. 99). Populations stop growing, according to Chitty, because they decay in 'quality' as density goes up. But what is 'quality'? On this point Chitty (1960) is not explicit. *Quality* may be defined as any individual morphological, physiological, or behavioural attribute which influences population fitness. This seems to be Chitty's implicit definition of quality. Quality in this sense must be measured in a specified environment, just as the innate capacity for increase (r_m) as defined by Andrewartha and Birch (1954). Quality, for example, might be measured by viability under standard conditions (e.g., Wellington 1957).

The problem of defining how to measure 'quality' of individuals led to a more restrictive formulation of the hypothesis (Chitty 1967*b*). One problem was that 'quality' might change for several reasons not related to self-regulation. For example, food shortage might reduce fat reserves and lead to reduced viability in high-density populations, and this would not be an example of self-regulation. The important point Chitty (1958, 1960) emphasized was that the interactions between individuals in the population had to be operative in causing the changes in 'quality.' But even if we are careful and restrict our discussion to changes in 'quality' that are the result of mutual interactions, we still must face the problem that 'quality' is environment specific. For example, large-sized adults are characteristic of high-density populations of the vole *Microtus californicus* (Krebs 1966). These large voles may be more aggressive than smaller voles, and they survive better in high-density populations and worse in low-density populations when compared with smaller individuals. In this example, 'quality' (= body size) has changed as density changed, but it is difficult to say that quality has *deteriorated* since the large animals would do *better* if tested in a crowded situation but *worse* if tested in an uncrowded situation.

The general hypothesis of self-regulation does not specify the mechanisms by which self-regulation is achieved. Four types of self-regulation mechanisms are possible because changes may be *physiological* or *behavioural* and may be *genotypic* or *phenotypic*. Of the four possible hypotheses, three are currently used to explain population changes.

(1) Stress hypothesis—This hypothesis suggests that mutual interactions lead to *physiological* changes, *phenotypic* in origin, that reduce births

and increase deaths. Calhoun (1949) and Christian (1950) were early proponents of this hypothesis, which has its roots in the early work of Hans Selye on stress (Selye 1956) and its effects on the endocrine system. Christian and his co-workers have provided a detailed analytical model of this mechanism and how it might apply to mammalian populations (Christian and Davis 1964; Christian 1971).

(2) Behaviour hypothesis—This hypothesis suggests that mutual interactions involving *spacing behaviour* produce changes in birth, death, and dispersal rates and thereby prevent unlimited increase. Individual differences in spacing behaviour are not inherited and are strongly affected by environmental features. This is part of the hypothesis of Wynne-Edwards (1962), and it has a long history in the bird literature in the argument whether territorial behaviour could limit breeding density (Brown 1969).

(3) Polymorphic behaviour hypothesis—This is the *Chitty Hypothesis* described by Chitty (1967*b*) as follows: ". . . all species of animals have a form of behaviour that can prevent unlimited increase in population density" (p. 51), and "Mechanisms for the self-regulation of animal numbers are thought to be a consequence of selection, under conditions of mutual interference, in favour of genotypes that have a worse effect on their neighbours than *vice versa*" (p. 72).

The Chitty Hypothesis is identical with the behaviour hypothesis in postulating that spacing behaviour limits population density but differs in suggesting that individual differences in spacing behaviour have a genetic basis and respond to natural selection on a short time scale.

Watson and Moss (1970) have provided a critical review of the behaviour and polymorphic behaviour hypotheses and have indicated the experimental approaches needed to test these ideas. Watson and Moss make an important point, often lost in discussions about self-regulation, that spacing behaviour may be only one of several factors limiting population increase and that important interactions may occur between extrinsic factors (weather, food, enemies) and spacing behaviour.

Hypotheses about self-regulation have been fruitful in developing cross bridges between biological disciplines. The stress hypothesis has excited endocrinologists, ecologists, and sociologists and led to an exploration of the area of social stress (Selye 1956) and reproductive physiology (Clarke 1977). The behaviour hypothesis has been particularly fruitful in forging a linkage between social behaviour and population regulation (Wynne-Edwards 1962), and this has led recently to the controversies of sociobiology (Wilson 1975). The polymorphic behaviour hypothesis has helped to bring the discipline of population genetics into the ecological area and has fitted into the wider movement to infuse ecology with natural selection (Birch 1960).

Self-regulation models are commonly thought to be restricted to the higher vertebrates, with perhaps a few insects thrown in. The stress hypothesis in particular has always been tightly tied to mammalian endocrinology (Christian and Davis 1964). The behaviour hypothesis is more general and can be applied to all species that have some method of spacing themselves out by avoiding, threatening, or otherwise influencing the dispersion of other individuals (Chitty 1967*b*). Spacing behaviour has been studied in greatest detail by vertebrate ecologists but there is no need to restrict these concepts to vertebrates.

Testability of the Chitty Hypothesis

The central hypothesis of self-regulation is that all *species* are capable of self-regulation by some mechanism, but Chitty (1960) was quick to point out that not all *populations* are self-regulated. This qualification seriously weakens the self-regulation hypothesis (Kikkawa 1977) and thus needs reconsideration. Chitty (1960) suggests that since any mechanism of self-regulation has been evolved by natural selection, it must be adapted only to a limited range of environments and thus would not operate in unnatural environments. Similarly, Chitty (1967*b*, p. 51) states: "we cannot, in fact, make any statements that can be tested on all populations of all species." But it is abundantly clear in the rest of the discussion by Chitty (1967*b*) that he is attempting to use his ideas to generate testable predictions.

We can resolve this dilemma by considering the plight of the individual scientist studying a single population of a species which shows spacing behaviour. If he is to test any of the predictions of self-regulation hypotheses, he must assume that his single population studied for a few years is an example of a self-regulated population. Thus I think it is a mistake in methodology to qualify the self-regulation hypothesis as Chitty has done, and I prefer to begin with the assumption that all populations are self-regulated, knowing that this cannot be true. An analogy of the problem we face might clarify the situation.

Assume that natural populations of species that show spacing behaviour are marbles in a large urn. Some marbles are black (= self-regulated populations) and some marbles are other colors, representing all other types of population regulation. At

the present time we have only a small handful of marbles that we have been able to look at, and most investigators spend a lifetime trying to see the color of one marble. At the start we postulate that all the marbles are black. Now several scenarios might occur as time passes. If none of the first dozen marbles is black, we would be very suspicious of the hypothesis and conclude that black marbles may exist but they are too rare to be of any practical importance. Or we may find that half of the first dozen marbles are black, and then try to classify the blacks. We might find, hypothetically, that no tropical populations showed self-regulation. What we really want to know at this stage is whether the urn contains many or few black marbles, and I think it premature to make our hypotheses any more sophisticated than that.

Another difficulty with Chitty's qualification is that it is now almost impossible to find a 'natural' environment. The effects of man on the earth are so great that such qualifications can provide a refuge for any theory (e.g., Lack 1966, p. 130). So again it would be better to start without this vague qualification on the hypothesis.

If we assume that all populations are self-regulated, we can go on to consider the predictions made by the polymorphic behaviour hypothesis (Chitty 1967*b*). Note that we cannot test the central hypothesis of self-regulation; we must specify the *mechanism* of self-regulation to be tested before we can apply these ideas to a real population. In particular, to show that the 'quality' of individual animals changes with population density is not sufficient to corroborate the general hypothesis of self-regulation. We can only reject this hypothesis by showing that changes in density are not accompanied by changes in 'quality,' but in order to do this we must specify what 'quality' is, and this means we must indicate the *mechanism* of self-regulation. This can be another source of frustration for a population ecologist because after testing and rejecting one type of mechanism of self-regulation, he can still be told that his population may be self-regulated, but by another mechanism than the one he tested. This source of frustration, however, is common to all hypotheses and not unique to self-regulation (Popper 1972).

The following predictions have been suggested to follow from the Chitty Hypothesis applied to fluctuating populations:

(1) *Spacing behaviours will be less common or less intense in increasing populations than in declining populations* (Chitty 1967*b*). This prediction would also be made by the behaviour hypothesis and is implicit in Wynne-Edwards (1962). It would also follow from the food shortage hypothesis of Lack (1966) who suggests that available resources limit population increase and spacing behaviour adjusts to available resources. Interactions involving spacing behaviour ought to have more severe effects in declining populations than they have in peak populations (Fig. 3).

(2) *The genetic composition of a population differs in increasing, stable, and declining populations* (Chitty 1967*b*). This prediction seemed originally the most risque of the Chitty Hypothesis since it implied a very rapid rate of natural selection. This view has now become commonplace and the same prediction is now made from very general theories linking demography and genetics (Charlesworth and Giesel 1972). Evidence on this prediction can only show that demography affects genetic composition and not the converse (Gaines 1978).

(3) *If animals are prevented from interacting adversely, they should go on increasing until they run out of food* (Chitty 1960). This prediction would also be made by the behaviour hypothesis. It is a difficult hypothesis to test and I know of no one who has attempted it on a field population. The domestication of animals could be interpreted as a verification of this prediction.

(4) *If an increasing population is continuously and severely cropped, a deterioration in 'quality' will be prevented; but in a declining population in which 'quality' has deteriorated, no reduction in density should be sufficient to reduce the downward trend* (Chitty 1960). The first part of this prediction would follow from all self-regulatory hypotheses as well as the food-shortage hypothesis and is not a very specific prediction for testing. The second part of this prediction is no longer believed correct (Chitty 1967*b*) since behavioural interactions are critical in declining populations and these may be partly dependent on density. The second part of this prediction now seems specific to the stress hypothesis but may or may not apply to the two behavioural hypotheses, depending on the exact experimental conditions.

(5) *Numbers should continue to increase if animals from an increasing population are transferred to an area from which a declining population has been removed* (Chitty 1960). This experiment would clearly distinguish the self-regulatory hypotheses from others, but it is a prediction common to the stress hypothesis and the two behavioural hypotheses. It is one of the more elegant experiments that Chitty (1960) suggested.

(6) *Numbers should continue to decline if animals from a declining population are transferred to a new area* (Chitty 1960). This is the converse of the

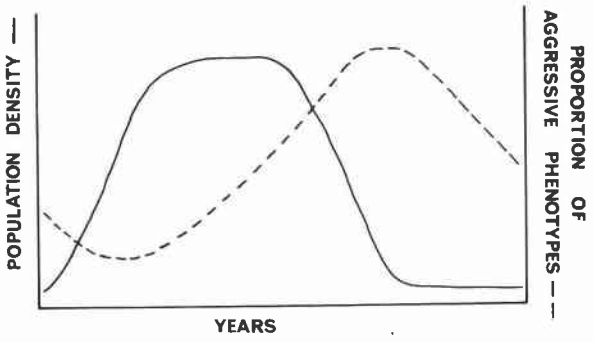

FIG. 3. Postulated changes in the proportion of individuals selected for mutual interference ('aggressive') in relation to a hypothetical population fluctuation, according to my interpretation of the Chitty Hypothesis. The proportion of docile individuals should fluctuate as the mirror image. The effects of mutual interference are postulated to depend on the proportion of aggressive phenotypes in this hypothetical example.

previous experiment and like them is a prediction common to all three self-regulatory hypotheses. Chitty (1967*b*) hedged on this prediction because the social disruption of transfers may reverse the expected result. I think that this prediction would not follow from the stress or the behaviour hypotheses, and that this prediction should hold for the polymorphic behaviour hypothesis.

(7) *Animals reared in isolated cultures should resemble those in expanding, newly introduced, or severely exploited populations, rather than those in stationary or declining populations* (Chitty 1965). This prediction would apply to all of the self-regulatory hypotheses as well as the food, parasite, and disease hypotheses. It does not seem specific enough to test.

(8) *Animals (a) present in stationary or declining populations are selected for their ability to survive the effects of mutual interference. Animals (b) in increasing populations are not so selected but are better fitted to withstand all other hazards of their environment. Mixtures of these animals should produce fitness differences favoring animals (a) at high density and animals (b) at low density* (Chitty and Phipps 1966). This is the most crucial test of the Chitty Hypothesis, according to Chitty (1967*b*). This prediction does seem unique to the Chitty Hypothesis, but it is technically a very difficult test to arrange.

All of the above predictions were derived by a 'black-box' approach to the Chitty Hypothesis, that is by treating the individuals as components of populations defined by density trends. An alternative approach that would allow more precision is to make some predictions about individuals in fluctuating populations (Emlen and Oring 1977). The following predictions can be made from this approach.

(9) *Spacing behaviours must have a high heritability in the broad sense* (Anderson 1975). This prediction is highly specific to the Chitty Hypothesis. The behaviour hypothesis predicts no heritability of spacing behaviours. This prediction can be tested only after the relevant forms of spacing behaviours have been discovered in field populations and a form of measurement of these behaviours perfected.

(10) *All populations contain individuals physiologically and behaviourally organized around two adaptive peaks, one containing increase genotypes adapted for reproduction and colonization and the other containing high-density genotypes adapted for conditions of mutual interference*. This prediction is implicit in Chitty (1967*b*). It is a risky prediction because it specifies just two adaptive peaks and implies that intermediate types of animals have lower fitness than the two extremes. These two types can be identified as *r*-selected and α-selected animals (Stenseth 1978).

(11) *Stationary populations are polymorphic for these two types of individuals* (Chitty 1965). This prediction is an attempt to extend these ideas to nonfluctuating populations.

(12) *Plague populations develop when the individuals of the high-density adaptive peak are lost and only increase genotypes remain in the population*. Sometimes one of the adaptive types postulated in prediction 10 goes extinct, and the resulting increase ends only when food resources are wiped out.

(13) *Natural populations can be manipulated by selection experiments on the increase genotypes or on the high-density genotypes*. This class of experiments ought to be the most powerful test of the Chitty Hypothesis. By introducing or removing certain genotypes we ought to be able to cause the population to rise or fall at will. These experiments are currently prevented by our inability to specify the genotypes of individuals in field populations.

This list of predictions is an impressive testament to the fertility of the Chitty Hypothesis, and it is particularly ironic that this hypothesis is accused of being untestable (Kikkawa 1977). I think one can fault these predictions as being qualitative rather than quantitative, but this fault is common to most ecological science. We should learn to predict qualitative effects before we try to specify numerical results, and one of the problems of modern systems ecology is to think that one can substitute numbers for a proper understanding of the qualita-

tive elements in an ecological system. The Chitty Hypothesis can be boiled down into two simple admonitions: (1) study spacing behaviour, and (2) consider changes in genetic composition in ecological time. We should no longer study populations in the faith that these two qualitative elements are of no concern.

Evolution of Self-regulatory Systems

There are two contrasting views about how to analyze the evolution of self-regulatory mechanisms. One view holds that natural selection acting at the individual or at the group level has actively favored the development of these mechanisms and their maintenance in modern populations. The second view holds that mechanisms of self-regulation did not evolve for that function but for some other functions so that population regulation becomes a fortuitous by-product of natural selection.

The second view is most easily illustrated by discussions in the literature about the evolution of territorial behaviour in birds (Tinbergen 1957; Lack 1966). Brown (1969) has argued cogently against this second view for the evolution of territorial behaviour in birds and has suggested that we approach this problem directly through a study of the effects of self-regulatory mechanisms on gene frequencies.

Wynne-Edwards (1962) argued that spacing behaviour and other adaptations for self-regulation arose through group selection (interpopulational selection of Brown 1966). This view has been almost universally condemned (review in Brown 1969, p. 318) and has now been abandoned by Wynne-Edwards (1977).

We are left with the view that natural selection acting at the individual level has favored the development and maintenance of mechanisms of self-regulation. This view is central to the Chitty Hypothesis and has been argued persuasively by Chitty (1967*b*). The same viewpoint was developed by Brown (1964, 1969) for the evolution of territorial behaviour in birds. The central concept is that selfish behaviour, behaviour which raises one individual's fitness at the expense of another, is adaptive. One of the persistent fallacies in discussions about the evolution of territorial behaviour is that there must be some object of competition that is in short supply, such as nesting places. Wynne-Edwards (1962) typifies this confusion. Brown (1964) pointed out that territorial behaviour can be selected for in situations where its only selective advantage is relative (the exclusion of other individuals from breeding places).

If we suppose that mechanisms of spacing behaviour evolved by individual selection, we still do not know whether variations in spacing behaviour in modern populations are genotypic or phenotypic. The Chitty Hypothesis is unique in postulating that the same natural selection which favored mechanisms of spacing behaviour in the first place can be seen operating today in real populations, i.e., that spacing behaviours have a high heritability. This question is an empirical one, but it is clear that Chitty's view is at variance with the modern theory of a biological response hierarchy (Wilson 1975, p. 145). Individuals must track short-term changes with physiological and behavioural responses, and track long-term changes with genetic shifts (Wilson 1975). This disagreement here is only over time scales: Wilson (1975) separates short and long term (ecological vs. evolutionary time) at 10 generations, while Chitty (1967*b*) would argue for extensive overlap down to one generation.

Physiological changes of the type central to the stress hypothesis are relatively simple to account for because they are clearly adaptive to an individual who must cope with a variety of stressing agents. Physiological deterioration of the type described by Christian and Davis (1964) is thus explained as a pathological by-product of selection favoring the stress mechanisms reported by Selye (1956), but there would seem to be no way of studying this evolutionary effect in real populations.

Examples of Population Studies

I will review four population problems from the viewpoint of the Chitty Hypothesis and try to evaluate the applicability of this idea to natural populations.

(1) Vole and Lemming Fluctuations

Many species of small rodents fluctuate in numbers in a 3- to 4-year cycle. Chitty (1960) summarized early work on the problem of what causes these cycles, and Krebs and Myers (1974) provide a recent review of the detailed demography. Since these fluctuations were the original stimulus to Chitty's ideas, one might hope that these populations at least would fit his hypothesis. I will summarize some of the recent experimental work under the predictions of the Chitty Hypothesis.

The first prediction that interactions involving spacing behaviour increase with population density has been tested explicitly only by Krebs (1970) on *Microtus ochrogaster* and *M. pennsylvanicus*. The results were consistent with the Chitty Hypothesis and with the behaviour hypothesis. In related work, we have tested the prediction that spacing

behaviour limits breeding density in *M. townsendii* with the same affirmative result (Krebs et al. 1978). Rose and Gaines (1976) tested a corollary of this prediction by analyzing the incidence of skin wounds in *M. ochrogaster*. They found that wounding levels in both sexes were higher in declining populations than in increasing or peak populations. Rose (1978) found the same results in *M. pennsylvanicus*. These results are consistent with Chitty's prediction that interactions involving spacing behaviour become progressively more severe as one passes from increasing to peak to declining populations.

The second prediction that genetic composition changes with population density has been verified in several species (see Gaines (1978) for review). Electrophoretic studies of blood protein polymorphisms have established that rapid selection accompanies population fluctuations and that this selection is repeatable in time. But these results do not show that genetic changes can cause demographic changes, as Chitty predicts. Gaines (1978) suggests that multiple locus studies may be productive if demographic parameters can be related to average genic heterozygosity. Smith et al. (1975) and Garten (1976) showed that average heterozygosity was related positively to body weight and to aggressiveness in *Peromyscus polionotus*. If this also applies to voles and lemmings, we could directly relate genetic and demographic variables at the level of the individual. In effect, we could define 'quality' in rigorous genetic terms as average heterozygosity and use some of the predictions above to test Chitty's Hypothesis directly (Smith et al. 1975). These ideas have breathed new life into electrophoretic studies and deserve testing in other rodents.

Boonstra (1977) tested the fourth prediction that cropping a declining population would not improve survival. He did these experiments on *Microtus townsendii* and got conflicting results in the two sexes. In males the prediction held, but in females cropping did improve survival. This example is even more complicated by the fact that *M. townsendii* populations seem to be self-regulated by mechanisms in accord with the behaviour hypothesis, not according to the Chitty Hypothesis. Boonstra's experiment, though interesting, is difficult to interpret and its major merit is to show us how little we understand about differences in social organization and spacing behaviour between the two sexes.

The fifth prediction, moving animals from an increasing population onto an area on which a decline had just occurred and observing that they continue to increase, was verified by Krebs (1966) for one population of *Microtus californicus*. This experiment requires out-of-phase populations, which are not common.

Chitty and Phipps (1966) tried to test prediction 8 in a population of *Microtus agrestis* but were unable to obtain completely satisfactory data. No one else has tried this difficult experiment.

Anderson (1975) tested prediction 9 that spacing behaviours are highly heritable in *Microtus townsendii* and rejected this hypothesis. This is the only attempt I know to measure the heritability of behaviour patterns in wild voles or lemmings. In these tests it is crucial to determine whether the behaviour patterns studied are the ones involved in causing population changes. This problem has scarcely been addressed in rodent studies.

The most important conceptual change that has been made in the Chitty Hypothesis since 1967 has been the recognition of the importance of dispersal in population regulation (Lidicker 1975; Tamarin 1978*b*). The discovery of the fence effect by Krebs et al. (1969) and the subsequent demonstration by Myers and Krebs (1971) that dispersal was related to cyclic phase and was genetically and behaviourally selective made us reconsider the mechanism by which the Chitty Hypothesis might be achieved in small rodent populations. Subsequent work on dispersal (Tamarin 1977; Krebs et al. 1976; Kozakiewicz 1976) has tended to confirm the importance of dispersal during the phase of population increase for population regulation in small rodents. Dispersal seems to be the process by which animals of different 'quality' are sorted out. Dispersal results from spacing behaviour, and the importance of dispersal to small rodents has led to a new series of experiments on island populations (Tamarin 1977; Sullivan 1977).

Studies on fluctuating rodent populations during the last 10 years have been broadly consistent with the predictions of the Chitty Hypothesis. But the hypothesis is far from being corroborated because the most rigorous tests have not yet been achieved and most of the results to date are also consistent with the behaviour hypothesis. Much of the work during the past 10 years has been necessary background work that must be done before rigorous tests can proceed. For example, the experimental designs that are appropriate to a rodent system in which selection operates by mortality in situ are not appropriate to one in which selection operates by emigration. Techniques for measuring spacing behaviour must be developed before one can test hypotheses about spacing behaviour. A considerable shift in viewpoint has also occurred

during the last 10 years. We were formerly enthusiastic about demonstrations of genetic changes (Tamarin and Krebs 1969) but we now accept genetic shifts as common and demand much more specific genetic results to corroborate the Chitty Hypothesis.

Only one experimental test has gone against the Chitty Hypothesis. Spacing behaviours are not inherited in *Microtus townsendii* (Anderson 1975). This vole is unusual in not having 3- to 4-year cycles and seems to be self-regulated by phenotypic changes in spacing behaviour (Krebs et al. 1978). These results are critically dependent on the assumption that laboratory measures of spacing behaviours are relevant to the field situation.

There is still considerable disagreement about the causes of population fluctuations in voles and lemmings. A recent general model of population dynamics of *Microtus agrestis* is based on the assumption that the nutritional condition of individuals is the primary driving force in natural populations and that spacing behaviour and population genetics are irrelevant (Stenseth et al. 1977). Haukioja and Hakala (1975) proposed a hypothesis to explain fluctuations that combines the idea of antiherbivore chemicals in plants with the idea of a genetic polymorphism in herbivore detoxification potential. There is no problem in specifying an alternative explanation for observed demographic changes, as long as the alternatives can be tested, but there is less excuse for ignoring certain experimental results that are at odds with one's hypothesis. Neither of the above hypotheses is consistent with the fence effect (Krebs et al. 1969). Furthermore, almost none of the crucial tests proposed by Stenseth et al. (1977) bears any relationship to the question at issue because they are predictions that flow from very general statements like "Animals require food and are adapted to obtain it." Only 2 of the 11 predictions given by Haukioja and Hakala (1975) are even partly specific to their hypothesis.

The testability of hypotheses is the most central methodological problem in population biology. Every hypothesis must make some risky predictions if we are to test our ideas (Popper 1972). Some predictions about cyclic vole populations, such as "survival rates will be poor during the winter after peak density, relative to the previous winter," are so general that they are predicted by virtually every hypothesis ever proposed to explain cycles, and so they are useless as tests. Some of Chitty's (1967*b*) predictions have suffered from this problem as well. What is important in the future is that we strive for specific predictions to narrow our choices and not try to achieve generality at the price of testability.

(2) Red Grouse Fluctuations

Populations of red grouse (*Lagopus lagopus scoticus*) fluctuate in 6- to 7-year cycles on the moors of eastern Scotland (Mackenzie 1952). Red grouse populations have been studied in more detail than most other bird populations, and they should provide an interesting test case for the predictions of the Chitty Hypothesis. Watson and Moss (1972) have summarized the results of the first decade of work on red grouse.

Populations of red grouse decrease over winter in a characteristic staircase manner (Jenkins et al. 1963). A sudden decrease in autumn is followed by a steady number of grouse over the winter. A second decline in late winter reduces the population to a new level that is constant through the spring. The sudden losses in the fall and late winter are associated with territorial behaviour which divides the birds into two social classes: territorial birds and nonterritorial surplus or transient birds. By a series of shooting experiments Watson and Jenkins (1968) showed that territorial behaviour was limiting breeding density. If a territory owner is removed by shooting during the fall or winter, it is replaced almost immediately by one of the surplus, nonterritorial birds.

Territorial or spacing behaviour in red grouse is not constant from 1 year to the next, and this explains why populations change in size. Watson and Miller (1971) found that in some years grouse were very aggressive, took up large territories, and had low breeding densities. In other years grouse were relatively docile, took up small territories, and had high breeding densities. To understand why red grouse populations fluctuate, we must find out what controls territorial aggressiveness.

Watson and Moss (1972) concluded that nutrition affected territorial aggressiveness in two ways. An indirect link seems to operate via maternal nutrition affecting the quality of the egg, which in turn affects adult aggressiveness. A direct link operates between the nutritive value of summer food and the size of territories taken up in the autumn. Marquiss (1977) attempted to test the direct link by growing red grouse chicks on different planes of nutrition in the laboratory and measuring their agonistic behaviour when adult. He found no relationship between the plane of nutrition and aggressiveness and concluded that good feeding did not lead to reduced aggressiveness and smaller territory sizes. But he found that the plane of nutrition did affect dominance in red grouse. He distinguished two compo-

nents of territorial behaviour: (1) dominance, which he presumed to be equivalent to the ability of a wild cock to get a territory; (2) aggressiveness, presumed to be analogous to cock's ability to defend a territory and related to territory size. Marquiss found no association between the measures of dominance and aggressiveness in grouse on a high nutritive plane but noted a close correlation in grouse on a low nutritive plane. The result from this laboratory study is the prediction that on an area with highly nutritive food the red grouse males that are able to get a territory will defend smaller territories than males from areas with food of lower nutritive value.

Both dominance and aggressiveness in red grouse are inherited traits (R. Moss, personal communication), but nongenetic maternal effects may be more important than genetic effects in causing this inheritance (Moss et al. 1974). Henderson (1977) used three genetic markers to look for evidence of genetic changes over 3 years in two red grouse populations. His populations were at peak density and nearly stationary over the 3 years of study, and he could detect no systematic changes in gene frequencies over time. Henderson's results do not fit prediction 2 of the Chitty Hypothesis, since selection ought to be operating through the peak phase to produce the effects seen in the decline phase (cf. Fig. 3).

Fluctuations in red grouse populations studied during the 1950's and 1960's were explained satisfactorily by a hypothesis which linked changes in territorial behaviour to changes in the food supply (Watson and Moss 1972). These results were the best example showing self-regulation consistent with the behaviour hypothesis, but Watson and Moss (1972, p. 143) pointed out that they had no evidence for or against the Chitty Hypothesis. Recent studies on another population cycle have again shown changes in spacing behaviour with changes in numbers (thus satisfying prediction 1) but no sign of the deterioration in food supply which had accompanied previous population declines (A. Watson, personal communication). Further analysis is necessary to determine whether any of these recent behavioural changes are inherited genetically, as the Chitty Hypothesis predicts, or whether only environmental and social influences are involved. It seems safe to conclude that either the behaviour or the polymorphic behaviour hypothesis will be found to be the basis of population fluctuations in the red grouse.

Given this detailed background for the red grouse, one could test the Chitty Hypothesis directly by analyzing the heritability of spacing behaviours (prediction 9) and by doing selection experiments on local populations (prediction 13). If spacing behaviours are under the joint control of the food supply and the genetic composition of the population one ought to be able to manipulate population changes in ways that might be of economic value. The population biology of the red grouse is probably better understood than that of any other animal and illustrates in a realistic way the complex problem that is hidden in the simple question of what prevents population increase.

(3) Locust Outbreaks

Plagues of locusts are among the most impressive natural reminders of the problem of population regulation. Outbreaks of locusts have been described for at least 3000 years, but it has been possible to analyze these outbreaks only since Uvarov had discovered that locusts were polymorphic (Uvarov 1928). Uvarov found that locusts are polymorphic species, capable of producing a series of forms so strikingly distinct that the extreme forms have often been taken for different species. He named the extreme forms *gregaria* (swarming locust phase) and *solitaria* (solitary grasshopper phase). A great deal of work has gone into the description of the morphological, physiological, and behavioural differences between the locust phases (Ellis 1972).

From an ecological point of view, the locust problem is almost unique because no one disagrees that changes in locust densities are associated with changes in the 'quality' of individuals (Uvarov 1961). The changes in behaviour (swarming) and color alone are so obvious that they have been known for 50 years. Locusts would thus seem to be prime candidates for the self-regulation hypotheses of population regulation but this has not been the case. Most locust workers have sought climatic (rainfall in particular) or biotic (insect parasites, predators) explanations for locust outbreaks (White 1976) and have assumed that changes in locust density cause the changes in phase characteristics, but that there is no feedback as postulated by hypotheses of self-regulation.

What controls phase variation? Uvarov (1966) believed that all phase variation was produced by changes in the social environment, specifically in the degree of crowding. Phase characteristics in locusts were partly inherited, but only nongenetic inheritance (maternal effects) was believed to be important in phase variation (Uvarov 1966, 1977). Gregarious locusts can be produced in the laboratory by rearing locusts crowded in cages, and solitary locusts can be produced by rearing individuals in isolation. Ellis (1972) reviewed the evidence that

crowding was sufficient to explain variations in phase characteristics and concluded that physical factors and genetic variation may be as important as, or more important than, crowding in controlling phase variation.

Lea (1968) proposed a hypothesis to explain outbreaks of the brown locust (*Locusta pardalina*) in South Africa that is virtually identical to the Chitty Hypothesis. Lea suggests that individual locusts differ in a genetically determined quality called *density sensitivity*. This quality is not directly related to phase, and *solitaria* type locusts may be either density sensitive or density insensitive. Density-sensitive individuals avoid one another if possible and are the equivalent of the *r*-selected animals of Stenseth (1978, p. 157). When forced to aggregate because overall population density has risen and there is no empty space, the density-sensitive types produce the best *gregaria* swarms and are thus colonizing genotypes. On the other hand, density-insensitive individuals do not react positively to one another's presence and thus are not easily converted into phase *gregaria*. They should be the analog of the aggressive phenotypes in Fig. 3 and the α-selected animals of Stenseth (1978). The density-insensitive individuals are selected for during a plague by the selective emigration of the density-sensitive types. Swarms of locusts should be predominantly density-sensitive genotypes with a few phase-transformed density-insensitive types from overcrowded areas. The parallelism of Lea's hypothesis with the Chitty Hypothesis and our results with emigrating voles (Krebs and Myers 1974) is striking.

Unfortunately Lea's (1968) hypothesis has not been adequately tested and so remains only a possibility. Lea (1972) found that brown locusts in the solitary phase attain very high densities before even the beginnings of aggregation become apparent. Adult brown locusts must be less than 2 m apart and their density more than 5000 per hectare before the beginning of swarming can be detected (Lea 1972). These observations underscore Uvarov's (1961) point that we should not equate solitary with low density and swarming with high density; what differs is the random-to-uniform distribution of solitary locusts in contrast with the highly clumped distribution of swarming locusts. Lea (1972) also reports a peculiar behaviour pattern of *solitaria* brown locusts. All movement studies of *solitaria* individuals have shown them to be very sedentary with the exception of night flyers. Night flying is relatively uncommon, and night-flying locusts are morphologically closer to *gregaria* than one would expect in a random sample. These dispersing night flyers could be the forbearers of a new plague (Lea 1972).

Conditions for reproduction and survival in locusts are clearly related to rainfall, and rainfall has been used as a predictor of population changes in some species (Symmons 1959; Clark 1972), although this does not apply in other species (Lea 1972; disputed by White 1976). White (1976) has used these climatic correlations as the basis of a hypothesis about the origin of locust plagues. According to White (1976), plagues arise because of variation in the survival of very young locusts, and this survival is determined by changes in the abundance and nitrogen content of their food plants. Any rainfall pattern that stresses the water balance of plants causes them to increase in nitrogen content, and nitrogen is the critical factor in short supply to small, growing locusts. White (1976) dismisses phase transformation as an irrelevant symptom of locust plagues, not a cause. White's views echo the belief of many ecologists that qualitative changes in individuals are irrelevant and that population control is achieved only by weather, food, parasites, predators, or diseases. I would think it more useful to ask how these qualitative changes interact with variations in rainfall or food quality. These two different viewpoints could be tested. White's hypothesis implies that the survival of young locusts on high- and low-nitrogen food is independent of phase state. Chitty (1960) suggested that the survival of animals in any type of constant environment would be strongly affected by the phase state of the original population.

These differences of opinion about the causes of locust plagues may have practical consequences in locust control. Various chemical sprays and baits are now used to kill young locusts and migrating swarms in an attempt to prevent widespread plagues from developing. If you envisage locust outbreaks being generated solely by changes in rainfall, food, or parasites (for example), chemical control presents no ecological problem except pollution. The emphasis is on holding locust numbers down while the environment is 'favorable,' on the understanding that soon the environment will become 'unfavorable' and no control will be needed. But consider the possibility that genetic changes as well as weather (or food) are involved in locust outbreaks, as Lea (1968) and Chitty (1960) would postulate. The effects of chemical control on genetic composition now become critical. If chemicals kill a greater proportion of Lea's density-insensitive genotypes, control may increase the proportion of density-sensitive genotypes and hence the probability of an outbreak, or at least not

change it. Locust plagues may thus increase in frequency if chemical control is superimposed on a population system dependent on qualitative changes for self-regulation. If this situation develops, control efforts would have to intensify, and problems of insecticide resistance would arise. Field evidence on this point is not clear. Lea (1972) states that plagues in the brown locust have increased in frequency since the start of chemical control operations. The important point is that qualitative changes can have some practical effects, and it may be important to evaluate their importance in field populations.

The locust population problem is an instructive one because it shows clearly that qualitative changes accompany density changes and that this is not sufficient evidence for self-regulation. Locust populations cannot be experimentally manipulated for political and economic reasons, and at present we can rely only on natural experiments to test the predictions suggested by the Chitty Hypothesis. There is not sufficient evidence to decide if locust populations are self-regulated or not.

(4) Larch Bud Moth Fluctuations

The larch bud moth (*Zeiraphera diniana*) is a defoliating pest on conifer forests in Europe and Asia. Outbreaks of this moth occur cyclically on larch (*Larix decidua*) in the Alps and on other larch species in Siberia at 8- to 10-year intervals. These cycles have been studied particularly intensively in Switzerland where the amplitude (maximum density divided by minimum density) averages about 30 000 and may reach 100 000! Not all populations are cyclic; some have irregular outbreaks and others are stable. An extensive series of investigations centered on cyclic populations in Switzerland has been summarized by Baltensweiler et al. (1977), and my summary here is based on this review and earlier reviews by Baltensweiler (1968, 1971).

The larch bud moth has one generation a year. There are two color morphs of *Zeiraphera diniana* that are distinguishable in the last larval instar: a dark morph, found mainly on larch, called the larch form; and an orange–yellow morph, found mainly on cembran pine (*Pinus cembra*), called the pine form. When these two morphs are crossed, a full range of intermediate color phases is obtained. The color morphs are good examples of ecotypes adapted to different host plants. This color polymorphism is affected by a complex of environmental and genetic effects, and the ecotypes seem to show typical polygenic inheritance. Field populations are a mixture of color types, which show major differences in fitness. The larch form hatches in the spring earlier than the pine form, and when food and temperature are optimal, the larch form offspring survive better. Nutritional stress selects against the larch form and in favor of intermediate color morphs.

Population cycles in the larch bud moth begin at densities as low as 0.002 larvae per 1 kg larch foliage. Density increases for four or five generations (years), going up about 10-fold each generation. There is typically no pause in the peak phase and densities immediately begin falling for three or four generations at rates which exceed the rates of increase. When densities exceed 100 larvae per kilogram of larch foliage, defoliation becomes visible. Defoliation is common in most outbreaks but is not necessary for a population decline.

Competition for food at high larval densities is believed to be one of the major factors stopping population increase in the larch bud moth (Baltensweiler et al. 1977). Food shortage leads to high larval mortality from direct starvation, reduced weight of pupae and adults, and reduced fecundity. Some of these same effects can be produced by territorial behaviour between individual larvae in the moth *Bupalus piniarius*, even when food is superabundant (Gruys 1971), and Baltensweiler et al. (1977) report that extensive larval emigration also seems to occur at high density in the larch bud moth. The suggestion is that mechanisms of self-regulation through mutual interference do exist in the larch bud moth but that these do not prevent population increase, which is terminated directly by food shortage.

Delayed effects of defoliation may last for 2–4 years after the population peaks. Larch which are defoliated may produce shorter needles of tougher texture, and resins may cover buds and needles. Needles of defoliated trees contain less nitrogen and more secondary plant chemicals that reduce digestibility. These aftereffects of defoliation are augmented by a complex of parasites and predators that cause maximal mortality 2 or 3 years after peak densities. In declining bud moth populations there may be 70–80% larval mortality caused by insect parasites, whereas there is less than 10% mortality in increasing populations.

During the phase of increase, the dark (larch form) color morph increases in frequency, whereas after defoliation the intermediate ecotypes are at a selective advantage for four or five generations. Cycles in population density cause alternating periods of directional selection for two different ecotypes, but there is little or no feedback from the ecotype composition to population density.

Not all larch bud moth populations increase to the point of defoliation, but Baltensweiler et al. (1977) point out that the same type of nutritional stresses may operate in trees only partly damaged by feeding or by weather stresses.

The large scale of larch bud moth infestations and the great dispersal capacity of the adult moths have restricted the application of experimental methods to test these hypotheses. Attempts to manipulate density by chemicals or pathogens have so far not succeeded in altering the cycle fundamentally (Baltensweiler et al. 1977). Further experiments to manipulate low density populations with pheromones are currently under way.

The larch bud moth thus seems to be a contrary instance to the Chitty Hypothesis. Even though these populations have potential mechanisms of self-regulation, population changes are dictated by food shortage and by parasitism. The genetic composition of larch bud moth populations does indeed change systematically with population density (prediction 2) but these genetic changes are not necessary to prevent unlimited increase.

These conclusions are reasonable and consistent with the detail of data summarized by Baltensweiler et al. (1977). I would caution that they have not been tested by experimental manipulations. The Chitty Hypothesis makes some specific predictions about experiments altering color morph frequencies in the larch bud moth. It would be of value to do these experiments to gain further insight into a population which on the surface seems to be a good candidate for self-regulation and yet at present appears to be regulated by conventional biotic mechanisms.

Models of the Chitty Hypothesis

There have been surprisingly few attempts to model the Chitty Hypothesis or any of the self-regulatory hypotheses. Dekker (1975) prepared a simple mathematical model of rodent population cycles from the data summarized in Myers and Krebs (1974). Dekker's model is a differential equation model which uses two discontinuous 'types' of animals to model the cycle. It is a very specific model which mimics the observed population trends but fails to give any further insight into the problem.

Anderson (1975) created a simulation model of the Chitty Hypothesis based on quantitative genetics. She constructed two models, one with nonoverlapping generations and one with overlapping generations. The qualitative results were the same with the two models. Each individual is described by its position on a phenotypic continuum, representing its agonistic behaviour. Any number of phenotypic (behaviour) classes can be used; Anderson found 10 to be sufficient to approximate continuous variation. The habitat was assumed to be homogeneous, and breeding was assumed to be continuous. Four functional relationships were used to specify the model: (1) the number of offspring recruiting per parent was a function of the phenotype (behaviour) of the parent so that very aggressive animals produced fewer recruits relative to docile animals; (2) the relationship between parental phenotype and offspring phenotype was specified by the heritability statistic of quantitative genetics; both the heritability of agonistic behaviour and its within-family phenotypic variance could be varied in different simulations; (3) for each generation the aggressiveness of the entire population was determined by summing the level of aggressiveness of all the animals in the population, as specified by the phenotype class of each individual; and (4) the number of offspring surviving to reproduce from each phenotype class was a function of the total population aggressiveness and an individual's phenotype. Docile animals survived worse than aggressive individuals at high population aggressiveness levels, and this was reversed at low levels. Anderson (1975) found that both her models produced population cycles within realistic parameter ranges when two conditions obtained: (1) relative fitness must be reduced for intermediate phenotypes so that relative fitness is highest at the two ends of the aggressiveness spectrum; (2) when the heritability of aggressiveness fell below 0.8, cycles ceased and the population fluctuated erratically. The value of Anderson's model is that it is the first attempt to quantify some limitations of the Chitty Hypothesis, and while clearly oversimplified it is a useful starting point because it does deal with empirically measurable relationships.

Stenseth (1978) has presented a graphical model for his interpretation of the Chitty Hypothesis. Stenseth (1977) discusses two versions of the Chitty Hypothesis. Version I states that a polymorphic behaviour system will, in the absence of external factors, exhibit cyclic fluctuations. But this is not the Chitty Hypothesis as discussed previously because the Chitty Hypothesis is not specific to cyclic fluctuations but is about self-regulation in stable, fluctuating, and cyclic populations. I can find no statement in Chitty's writings that suggests that all polymorphic behaviour systems must show population cycles. This is a crucial point. If the Chitty Hypothesis really does apply only to cyclic populations, it is of minor academic interest because it would apply to very few species.

Chitty's view that cyclic populations are convenient vehicles for testing his hypothesis must not be confused with the erroneous belief that only cyclic populations can be self-regulated.

Nevertheless, Stenseth (1977) was correct in applying the Chitty Hypothesis to cyclic rodents, and he asks an important question: are changes in extrinsic factors necessary to generate population cycles? According to Stenseth (1977), "Chitty's theory I" states that cycles can be produced in a stable environment. He rejects this possibility by analyzing a Lotka–Volterra type competition model and goes on to postulate "Chitty's theory II" which states that an interaction of extrinsic and intrinsic factors is needed to produce cycles. In particular Stenseth suggests that extrinsic factors such as weather are likely agents triggering a crash in microtine cycles. "Adverse environmental factors" become essential for generating a cycle. Stenseth (1977, 1978) clearly does not subscribe to Chitty's (1967*b*) discussion that the "adverse environmental factors" may be other aggressive individuals of the same species, and not bad weather or abundant predators. A more serious problem arises because Anderson (1975) has clearly constructed a model which produces cycles under the assumptions of what Stenseth labels "Chitty's theory I," and consequently there seems to be no reason at the modelling level to add extrinsic factors to the Chitty Hypothesis to generate cycles.

I would emphasize that these models of the Chitty Hypothesis are no substitute for empirical evidence and experimentation. Models for cyclic populations can be produced from a variety of initial assumptions, some correct and others incorrect. The most important desiderata for any future models of the Chitty Hypothesis would seem to be a clear statement of the assumptions and of the predictions made by the model, so that the field ecologist has some hope of checking the model.

Conclusion

Two major developments have characterized the field of population biology since 1950. One is the development of interest in individual differences as a necessary element in understanding population change. This development was spearheaded by a group of ecologists of diverse background (Calhoun, Christian, Davis, Wellington, and Chitty) and their rallying cry was a simple plea: *consider the possibility that interactions between individuals cause population changes*. Added to this was the development of interest in evolutionary ecology and the adaptive significance of individual behaviour patterns. From the early speculations of Elton and Ford, through the increasingly precise insights of Lack, Birch, Pimentel, and Chitty, to the blossoming of evolutionary ecology in the 1960's and 1970's, the message has become clearer: *consider the possibility that natural selection is operating on an ecological time scale*. Chitty's work and his ideas have played a role in the development of these two lines of thought. Viewpoints have changed such that anyone starting a population study today would be foolish to ignore these two admonitions, whereas 20 years ago you would have been called foolish to worry about them.

The Chitty Hypothesis may be one of the last grand generalizations of population ecology. The conceptual framework of population regulation is now complete. Population biologists of the next few decades must work to apply this conceptual framework to real populations. Detailed analyses of local populations are the order of the day. There are few populations for which we are able to specify the factors preventing increase, but the foundations of natural history have now been laid for many populations so that analytical and experimental work can proceed rapidly. Until this further work has been done, we will not know whether the Chitty Hypothesis applies to many of the species that show spacing behaviour or to few of them. We have accumulated the tools required for testing, and a smaller number of studies have charted the pitfalls of testing the Chitty Hypothesis. In closing I can only echo Chitty's hope:

> These ideas, whether themselves true or false, may suggest further work on behaviour, physiology, and genetics that will contribute to the solution of ecological problems (Chitty 1967*b*, p. 73).

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