A genetic explanation for ten-year cycles of grouse

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Summary. Chitty's Polymorphic Behavioural Hypothesis (Chitty 1967) was logically reduced to three main assumptions that were mathematically modelled:

1) Level of aggression is genetically determined by simple Mendelian selection.
2) Recruitment is inversely related to female parental level of aggression.
3) Aggressives are completely successful in breeding competition.

The model utilized data from willow ptarmigan populations, but was generalized to other grouse species. Simulation results were indistinguishable from the behaviour of real world grouse populations, lending support to Chitty's hypothesis as the explanation of cyclicity. The model also seems applicable to other species. Eight tests that would falsify the model were identified.

Introduction

With the exception of a few specific cases, there is still no general answer to the question "Why do populations of animals cease growing and not continue to increase without limit?" (Pielou 1977; Krebs 1978). Population ecology as science has no universal paradigm under which it operates (Kuhn 1962; Lakatos and Musgrave 1965).

Dennis Chitty (1958) proposed an encompassing theory, that all animals have the ability to self-regulate their population levels below resource limits. The theory was intuitively appealing, but initially was insufficiently precise to provide much direction for research programmes, or to allow falsification by empirical testing. Pitelka (1958) at the same symposium, criticized the concept on the basis that "It may be a strain on Occam's Razor to suggest genetic hypotheses regarding such fluctuations as long as more directly ecological explanations can be invoked and tested." Since then, no "directly ecological" theory has emerged, yet the entire field of sociobiology has arisen with its explanations of behavioural traits based on just such genetic hypotheses (Wilson 1975).

Chitty's ideas developed further and became more rigidly specified as Chitty's Polymorphic Behavioural Hypothesis (Chitty 1967), henceforth abbreviated to CPBH.

Briefly, CPBH states that, at low densities, passive individuals are selectively favoured that can tolerate crowding, allowing population buildup and an increase in density. At high densities, more aggressive, less viable individuals become favoured. These aggressive individuals, through interference or spacing behaviour, lower breeding density and cause a population decline. The theory was developed to explain changes in abundance of animals that undergo regular periodic fluctuations, commonly called "cycles" (Elton 1942; Keith 1963). It would be beneficial for the reader to be familiar with the review presented by Krebs (1978).

In the present paper, a study is detailed in which the logical consistency and falsifiability of the Chitty Hypothesis are investigated, leading to the definition of the central core of the hypothesis. The results are mathematically represented and simulated with empirical data from willow ptarmigan (Lagopus lagopus L.).

Chitty first suggested that the theory could be "falsified,..., by proving that there are no significant differences between expanding, stationary, and declining populations in the distributions of the properties of the individuals" (Chitty 1960). Such a situation would in fact be a disproof, but it could never be realistically expected to be realized. Only in the impossible situation where variations amongst individuals were totally irrelevant to fitness and were non-adaptive, would changes in density not cause genotypic changes in density-dependent and frequency-dependent alleles (Wright 1968; Roughgarden 1971; Smouse 1976; Poulson 1979). "Differences in the properties of individuals" would always be present. The existence of these genotype changes with density changes does not distinguish genetic variability as either cause or effect.

To the present, testability has been enhanced but no disproofs of CPBH have yet been conceived. Continual experimentation, particularly on microtine rodents, did not seem to significantly support or refute the theory (Krebs and Myers 1974), despite the fact that Chitty himself believed strongly in the Popperian view of a sophisticated falsificationist approach to science (Popper 1959, 1965; Lakatos and Musgrave 1965; Koertge 1979) and favoured attempts to falsify the theory. The situation was that envisioned by Lakatos (1965) wherein the central theory forms a core surrounded by a belt of testable hypotheses. Empirical data do not necessarily impinge on the core theory itself, but if the belt is sufficiently riddled by falsified hypotheses, the belt collapses and the central theory is rejected. All tests of CPBH to date have been in the very outer fringe.
of the belt, due to the great conceptual leap of inferring changes in the genotype frequency from changes in the behavioural phenotype frequency at a population level.

Given the state of affairs, it appeared relevant to attempt a mathematical simulation of CPBH; in hopes of determining which of the potential factors involved were truly necessary or at least sufficient (Krebs and Myers 1974). To mathematically model the theory, we closely investigated its structure and explicitly defined its basic assumptions. By so doing, we developed hypotheses close to the core. The majority of the discussion will deal with cycling animals where CPBH has been most extensively debated. Our emphasis will be on grouse, particularly ptarmigan.

Defining the assumptions

CPBH has been couched in semantic terms that are vague and empirically imprecise. Some problems are obvious. We know that behaviour is under genetic control, but how susceptible is it to environmental and other influences? What did Chitty mean by quality? What is viability? Does intrinsic mean strictly genetic?

A serious logical fallacy is that at high population levels a genotype would be favoured that produces "individuals in a declining population that are intrinsically less viable than their predecessors" (Chitty 1960). If viability and fitness are synonymous, by its very definition selection could not favour less fit individuals. The existence of this paradox in the field data of voles is an artifact of the data collection methodology and will be seen to be due to the confusion of population parameters of the total population with that of the breeding population in the selective arguments.

We would paraphrase Chitty's definition of CPBH for cycling small mammals as follows:

At low densities, individuals of low aggressive levels tolerate crowding such that population densities increase to the point where highly aggressive individuals are favoured and the population declines.

Within this definition are three inherent assumptions that are crucial to CPBH.

1) Level of aggression of an individual is primarily genetically determined.

2) Net population fecundity (recruitment) is inversely related to female parental level of aggression.

3) Aggressive individuals are more successful in breeding competition at high densities.

Assumption 1 is explicitly stated by Chitty and must necessarily be met to consider a genetic explanation for these fluctuations.

Assumption 2 has not been specifically identified previously. Only three methods are available to force population growth to cease and become negative at high densities. Either mortality increases, dispersal increases, or fecundity decreases (Pielou 1977). Measured mortality rates often do increase in the decline phase, but this condition is not necessary. Disappearance from the study site has often been confused with mortality. Adult mortality rates are constant throughout population fluctuations in ptarmigan (Bergerud 1970).

Dispersal has been identified as a major population regulation factor (Wynne-Edwards 1962) and was thought to be critical in voles, but the number of dispersers is greatest during the increase phase and drops significantly before the population begins to decline (Myers and Krebs 1971). There is usually no major dispersal in ptarmigan populations (Bergerud 1970; Myrberget 1972). Dispersal is not sufficient to explain the decline.

The only method remaining to cause the decline is a decrease in net population fecundity. Yet litter sizes in voles and clutch sizes in ptarmigan are not correlated with changes in density (Zedja 1966; Bergerud 1970; Keller and Krebs 1970). The critical factor causing population declines from peak densities must be lack of recruitment to the breeding population. Given that aggression is correlated with population density (Watson and Moss 1980), Assumption 2 is necessary.

Assumption 3 is also implicit and necessary. Aggressives are at a selective advantage at high densities. For their fitness to be higher at high densities, they must pass more genes to the succeeding generations than passives. They must either breed more often, or produce more offspring from each breeding. But given Assumption 2, fecundity is lower, therefore the option of producing more offspring is not available. Aggressives must breed more often than passives at high densities. For all intents, ptarmigan have only a single breeding period per year. Thus, aggressives must increase their fitness by dominating breeding competition such that some passives are excluded from successful recruitment.

Are these three assumptions all that are necessary to explain cyclic fluctuations in abundance of animals? The criteria by which science accepts or rejects scientific explanations are the subject of continual philosophical debate (Lakatos 1965; Salmon 1979; Rombesburg 1981). Predictive value, simplicity and goodness-of-fit to empirical data are all valid criteria by which to judge the worth of a theory; but unless we invoke the "Psychology of Research" (Kuhn 1965), the theory's resistance to falsification is the strongest single criterion scientists possess (Popper 1959; Lakatos 1965; Koertge 1979). If a simple computer simulation incorporating only these three conditions could produce population fluctuations indistinguishable from real-world behaviour, it would be strong evidence that CPBH is valid. Most importantly, the simulation could also help generate empirical falsification tests.

To be represented mathematically, the assumptions must be more precise. It is necessary to restate Assumption 2 in terms of individual selection. Given Assumption 1, learning and accumulative population stress can be ignored. Thus, net fecundity is inversely related to parental level of aggression on an individual basis.

Assumption 3 was generalized so that aggressives were always more successful in breeding competition, regardless of density.

It was now possible to rigidly specify the assumptions so that they could be modeled. The parameters of aggressive level (i.e., success in breeding competition) and fecundity were specific to the genotype and constant over density changes.

The model

The final form of the assumptions is:

1) Level of aggression is solely genetically determined by simple Mendelian selection of two alleles at one locus.

2) Recruitment is inversely related to female parental level of aggression (female genotype).