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The Chitty Effect: A Consequence of Dynamic Energy Allocation in a Fluctuating Environment

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An important biological feature of cyclic populations of voles and lemmings is phase-related changes in average body mass, with adults in high-density phases being 20–30% heavier than those in low-density phases of a cycle. This observation, called the ''Chitty effect,'' is considered to be a ubiquitous feature of cyclic populations. It has been argued that understanding the Chitty effect is fundamental to unraveling the enigma of population cycles. However, there exists no agreement among biologists regarding the causes of the Chitty effect. Here, I propose a simple hypothesis to explain the Chitty effect, based on phase-related, dynamic allocation of energy between reproductive and somatic effort. The essence of the hypothesis is that: (1) reproduction is suppressed in animals born or raised in the later part of the increase phase by environmental factors, including social influences; (2) suppression of reproduction limits the amount of energy that is diverted for reproductive effort, and forces a disproportionately greater amount of surplus power (the energy left after the energetic costs of standard and active metabolism are met) to be allocated for somatic effort; (3) the surplus energy, above and beyond what is required for routine biological activities, will allow continuous growth and deposition of additional body mass, which causes an increase in body mass; and (4) animals grow to a larger size as a population enters the peak density phase, causing an increase in the average body mass. The Chitty effect is predicted to be most pronounced at the late increase or peak phase of a population cycle. Possible causes of reproductive suppression include direct or indirect influences of the environmental factors. The Chitty effect may be a consequence, not a cause, of population cycles in small mammals.

Key Words: **age at maturity; arvicoline rodents; Chitty effect; DEB model; dynamic energy allocation; lemmings; population cycles; reproductive effort; reproductive suppression; somatic effort, voles.**

INTRODUCTION

Cyclic fluctuations in the abundance of some small mammal populations have been the subject of much research over the past seven decades (e.g., Batzli, 1992; Krebs, 1996). An important feature of cyclic populations of voles and lemmings is phase-related changes in the average body mass, with adults in high-density phase being 20–30% heavier than adults in the low-density phase of a cycle (Boonstra and Krebs 1979). This phenomenon, known as the "Chitty effect" (Boonstra and Krebs, 1979), is considered to be a ubiquitous feature of cyclic populations (see Boonstra and Krebs, 1979; Lidicker and Ostfeld, 1991, for reviews). Several authors have argued that understanding the Chitty effect is fundamental to explaining population cycles (e.g., Krebs *et al.*, 1978; Chitty, 1987; 1996; Krebs, 1996). The occurrence of larger animals at high-density phases of population cycles is particularly puzzling because the high-density phase is generally characterized by an

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adverse social environment, and lowest levels of resources peril capita (Chitty, 1987). Although some authors have reported no correlation between the body size and phase of a cycle (Ferns, 1979, Myllymäki, 1977), an overwhelming body of empirical evidence shows phase-related changes in body mass for most cyclic populations (e.g., Boonstra and Krebs, 1979; Chitty, 1996; Krebs, 1996).

The discovery of the Chitty effect gave rise to an important hypothesis of population cycles, the Chitty–Krebs model (*sensu* Lidicker and Ostfeld, 1991). This model considers large body size as a characteristic of a genotype that is favored by natural selection at high-density phases (Krebs *et al*., 1978; Lidicker and Ostfeld, 1991). However, recent findings that body mass is not significantly heritable and that larger body size confers no obvious fitness advantage have raised questions about the validity of the Chitty–Krebs model as an explanation of the Chitty effect (Boonstra and Boag, 1987; Boonstra and Hochachka, 1997; Lidicker and Ostfeld, 1991). Lidicker and Ostfeld (1991) found that large animals neither occupied the best habitat nor possessed any obvious fitness advantage at high-density conditions, two features predicted by the Chitty–Krebs model. These findings led Lidicker and Ostfeld (1991) to conclude that the Chitty effect may be a consequence of prolonged periods of favorable environmental conditions. Oksanen and Lundberg (1995) suggested that changes in foraging time and reproductive effort in response to predation risk could cause phase-specific shifts in body mass, but did not provide a mechanism. Here, I propose a simple hypothesis to explain the Chitty effect, based on the dynamic allocation of energy between reproductive and somatic effort during different phases of a population cycle.

REPRODUCTIVE SUPPRESSION AT HIGH-DENSITY PHASES

Abundant empirical evidence indicates that reproduction is inhibited at the high-density phase of cyclic populations. Among various reproductive parameters that are adversely affected, suppression of sexual maturity of juveniles is particularly remarkable. Gustafsson *et al.* (1983) demonstrated that high population density suppresses sexual maturation in *Clethrionomys glareolus.* Several other studies have shown that reproduction, particularly in young animals, is suppressed at high-density phases (e.g., *Clethrionomys* spp.: Bondrup-Nielsen and Ims, 1986; Bujalska, 1985; Gilbert and Krebs, 1991; Gilbert *et al*., 1986; Tkadlec and Zejda, 1998a,b; Lofgren, 1989; Nakata, 1989; Saitoh, 1981; *Microtus* spp.: Boonstra, 1989; Boyce and Boyce, 1988; Myllymäki, 1977; Ostfeld *et al*., 1993; Rodd and Boonstra, 1988; *Lemmus* spp.: Pitelka, 1973; Krebs, 1964). These studies suggest that animals born in a high-density phase may not start reproduction until the following year, whereas those born during a low-density phase may breed soon after they are weaned.

THE PRINCIPLE OF DYNAMIC ENERGY ALLOCATION

Animals can ingest only a limited amount of food, and only some of the ingested energy is assimilated. The portion of the assimilated energy left after the energetic costs of standard and active metabolism are met is "surplus power" (Stearns, 1992). Surplus power is partitioned into two important biological processes (Hirshfield and Tinkle, 1975; Perrin and Sibly, 1993; Stearns, 1992): somatic effort (e.g., growth, development) and reproductive effort (e.g., maturation, offspring production, reproductive behaviors). Until sexual maturity, energy is liberally allocated to somatic effort, which allows rapid growth and development. Once maturity is achieved, however, the surplus power must be optimally allocated to each of the two processes. Because somatic effort and reproductive effort directly compete for the surplus power, an increase in energy allocated for somatic effort must necessarily be accompanied by a corresponding decrease in energy that can be allocated for reproductive effort, and vice versa. This type of energy allocation conflict is thought to result in trade-offs among life history traits (Stearns, 1992).

Reproduction is energetically expensive in small mammals (McNab, 1986; Millar, 1988) and is apportioned differently by males and females because of sex-specific differences in reproductive strategies. In males, energetic costs of reproduction are often primarily associated with reproductive behaviors. In females, pregnancy and especially lactation are energetically the most expensive reproductive processes (Millar, 1988).

When resources are abundant, and the levels of somatic and reproductive effort do not change drastically, the proportion of energy allocated to each of the two processes should remain fairly constant over time. However, if energy demands for somatic or reproductive effort change periodically, the allocation of energy must be dynamic to meet changing energy demands of the two competing biological processes. Such is the case in cyclic populations. At high density, reproduction is suppressed,

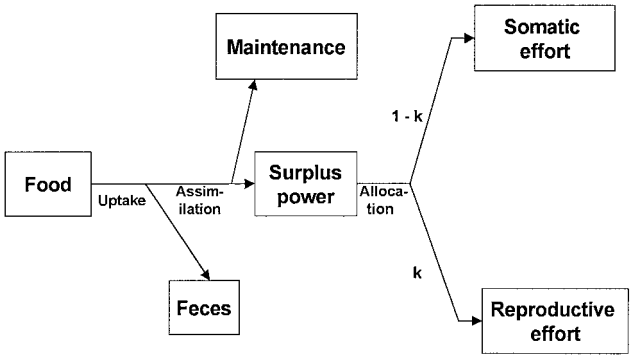

FIG. 1. Schematic representation of a dynamic energy budget (DEB) model. See text for details.

and this limits the amount of energy that can be allocated to reproductive effort and forces a disproportionately greater amount of surplus power to be allocated to somatic effort. As population density declines and environmental conditions become favorable, reproductive suppression is relaxed, and the surplus power is again optimally allocated to both biological processes.

Such dynamic allocation of energy can be modeled with dynamic energy budget models (Kooijman, 1993; Nisbet *et al.*, 1996; Perrin and Sibly, 1993; Ross and Nisbet, 1990). When assimilated energy A is greater than the energetic cost of maintenance M, surplus power ($P = A - M$) is available for somatic and reproductive efforts. If a fraction k of the surplus power is allocated to reproductive effort, then the remaining portion $(1-k)$ will be available for somatic effort (growth and deposition of additional body mass; Fig. 1). The somatic (S) and reproductive (R) processes can be described by differential equations

$$\frac{dR}{dt} = kP$$
$$\frac{dS}{dt} = (1-k)P. \qquad (1)$$

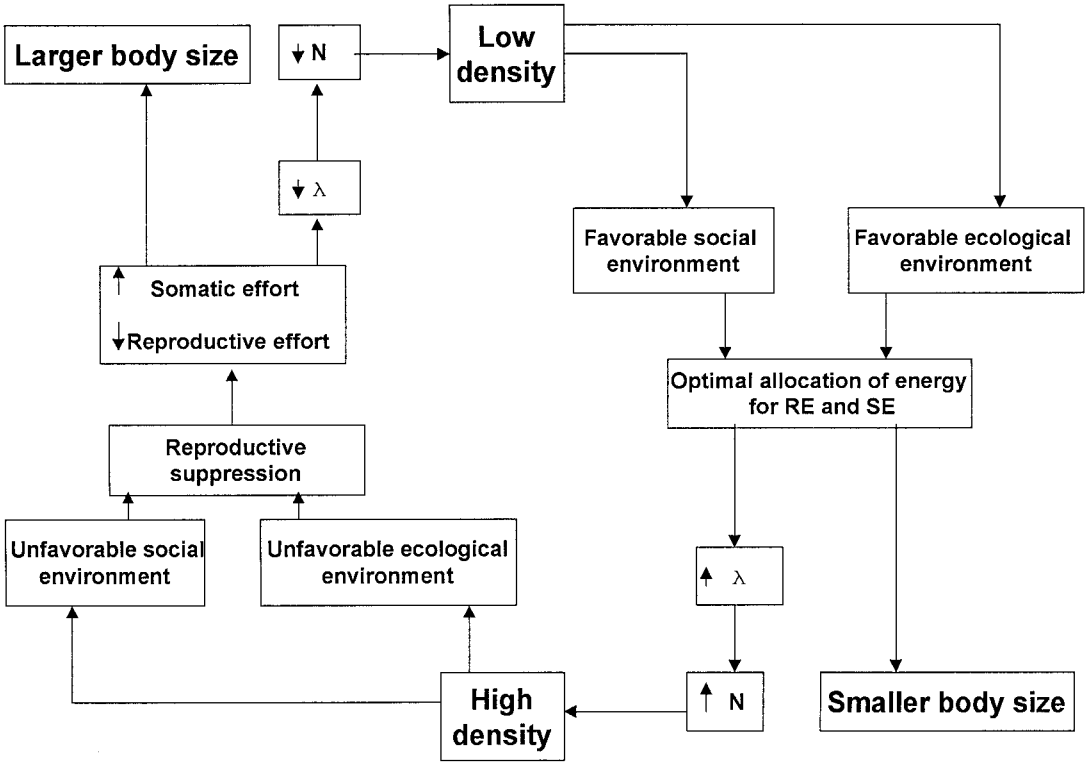

FIG. 2. The hypothesized model of the Chitty effect. When population density is low, surplus power is assumed to be optimally allocated for reproductive effort and somatic effort such that the population growth rate (λ) is maximized. Because maximum possible energy is devoted for reproductive effort (RE), somatic effort (SE) would be primarily limited to somatic maintenance. Because only a limited amount of energy is available for growth, growth is arrested and body size is smaller. As the population size increases, however, reproduction is suppressed, which forces a disproportionately larger amount of surplus power for somatic effort. The energy available for somatic effort, above and beyond what is required for somatic maintenance, is then used for structural growth and for deposition of additional body mass. This causes the average size of animals to be larger than those in low-density phases. Because of low reproductive effort, population size declines, and the cycle is repeated.

Equation (1) implies that changes in energy allocated for one of the two processes will determine energy that can be allocated for the other process.

When population density is low, natural selection may act to optimize energy allocation to maximize population growth rate; this is reflected in the rapid increase of cyclic populations following the "low" phase. As population density increases, the environment becomes less favorable and reproductive suppression begins to be manifested. Toward the end of the increase phase reproductive suppression becomes severe and reduced reproduction contributes to the cessation of growth, and ultimately decline, of the population. Suppression of reproduction in animals born at the later part of the increase phase limits the amount of energy that would otherwise be allocated for reproductive effort. Restriction in the amount of energy allocated for reproductive effort shunts a disproportionately greater amount of surplus power to somatic effort. A balance between somatic and reproductive effort at low-density phase and an increase in somatic effort as density builds up would cause phase-specific changes in somatic effort.

A disproportionately greater amount of energy allocated for somatic effort could influence body size distribution at the population level. Adult animals could use the excess energy to put on additional body mass and, to a lesser extent, for structural growth. However, surplus energy available for somatic effort would have a more dramatic effect on juveniles than on adults. Arvicoline rodents generally grow rapidly for the first 4 weeks of their life, or until sexual maturity is attained (Campbell and Dobson, 1992). When reproduction is suppressed and additional energy is available for somatic effort, growth may continue at a relatively fast rate until sexual maturity is attained. Thus, spring- or summer-born animals that may not achieve sexual maturity until next spring at the high-density phase (e.g., Bondrup-Nielsen and Ims, 1986; Gilbert and Krebs, 1991; Gilbert *et al.*, 1986; Lofgren, 1989; Saitoh, 1981) would grow for a much longer period compared to those born at the low or early increase phase (which attain sexual maturity soon after they are weaned.) A longer period of growth would inevitably lead to a larger body size, in terms of both structural size and body mass (Fig. 2).

When a population proceeds from the valley to the peak phase, the distribution of body size in the population shifts to a larger size. Individuals that fall on the right tail of the body size distribution (Fig. 3) are the "extra large" animals, considered by the Chitty–Krebs model to be a genotype favored by natural selection at high densities (Boonstra and Krebs, 1979; Chitty, 1960).

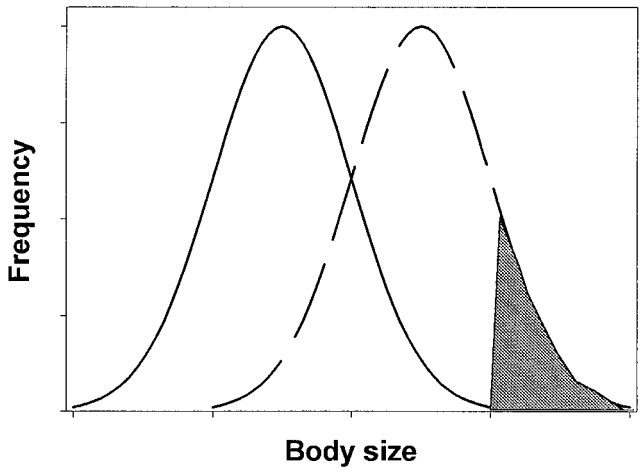

FIG. 3. The hypothesized shift in body mass distribution in cyclic populations. The body size is predicted to be smaller in a low-density phase (solid line). As the population moves to a high-density phase, body size distribution shifts to a larger size (broken line). The shaded region represents the "extra large" animals observed in the high-density phase of cyclic populations. See text and Fig. 2 for details.

ASSUMPTIONS AND PREDICTIONS

Inherent in my hypothesis is the assumption that reproduction, particularly of juveniles, is suppressed at high-density phases, and this assumption seems to hold for most cyclic populations (e.g., Boonstra, 1989; Gilbert and Krebs, 1991; Lofgren, 1989; Nakata, 1989; Tkadlec and Zejda, 1998a,b). A second assumption is that food resources are not critically limiting at hight-density phases. This assumption is supported by the observation that food supplementation studies have failed to stop population cycles (e.g., Desy and Batzli, 1989; Ford and Pitelka, 1984; Schweiger and Boutin, 1995). Finally, I assume that, when reproduction is suppressed, the portion of surplus power that would be allocated for reproductive effort had reproduction not been suppressed is allocated for growth and for the deposition of additional body mass. The idea of dynamic energy allocation has been empirically supported in several animal taxa (e.g., Baldwin and Sainz, 1995; Kooijman, 1993; McManus and Travis, 1998; Nisbet *et al.*, 1996; Ross and Nisbet, 1990), and has also been discussed in the context of small mammal population cycles (e.g., Krebs, 1993; Lidicker and Ostfeld, 1991; Stenseth and Ims, 1993; Ugland and Stenseth, 1985). However, the validity of this assumption remains to be tested in cyclic populations of arvicoline rodents. If these assumptions hold, the Chitty effect is predictable.

Reproductive suppression, particularly of young animals, should begin to be manifested as density builds

up at the later part of the increase phase and the environment becomes unfavorable; it should be most severe in animals born or raised toward the end of the increase phase. Such individuals should grow to a larger average size as a population enters the peak phase. Although animals born or raised at the peak phase may also be reproductively suppressed and are capable of growing to a larger size, they do not survive long enough to grow large because life expectancy of animals following the peak phase is short (e.g., *M. agrestis*: Myllymäki, 1977; *M. pennsylvanicus*: Boonstra and Rodd, 1983; *M. ochrogaster*: Getz *et al*., 1997). Thus, the Chitty effect is predicted to be most pronounced around the peak density phase, and not in the decline phase of a population cycle. These predictions are consistent with empirical data (Boonstra and Krebs, 1979; Chitty, 1996).

CAUSES AND CONSEQUENCES OF REPRODUCTIVE SUPPRESSION

Thus far, I have argued that phase-related changes in body mass could result from dynamic allocation of energy during various phases of a population cycle, but possible mechanisms for reproductive suppression have not been discussed. Several factors can inhibit sexual maturation in cyclic populations of small mammals (Oli and Dobson, 1999). Nutrition affects growth as well as maturation processes, and malnutrition can cause delayed maturity (Andreassen and Ims, 1990). Naturally occurring phenolic plant compounds (Berger *et al*., 1977) and protease inhibitors produced by plants as a defence against herbivory (Seldal *et al*., 1994) can adversely influence growth as well as maturation processes in arvicoline rodents. Although underlying mechanisms are not clearly understood, numerous studies have found that the maturation rate is inversely related to population density in cyclic populations (e.g., Boonstra and Rodd, 1983; Boyce and Boyce, 1988; Gilbert and Krebs, 1991; Gustafsson *et al*., 1983; Rodd and Boonstra, 1988; Tkadlec and Zejda, 1998a,b; Wiger, 1979)

Suppression of reproduction by factors in the social environment has been demonstrated by numerous studies (e.g., Bujalska, 1985; Gilbert *et al*., 1986; Kruczek and Marchlewska-Koj, 1986; Kruczek *et al*., 1989; Saitoh, 1981). The most common mechanism of social suppression of reproduction is shown to be puberty-delaying pheromones secreted by littermates or adult females at high population density (e.g., Batzli *et al*., 1977; Getz *et al*., 1983; Lepri and Vandenbergh, 1986; Rissman and Bronson, 1987; Rissman *et al*., 1984; Vandenbergh, 1987, 1994). Adult females might release puberty-delaying pheromones if the environment is perceived to be unfavorable, and the young animals respond to these chemosignals physiologically (Lepri and Vandenbergh, 1986; Rissman *et al*., 1984). Dominant individuals of both sexes may also suppress reproduction in other individuals by behavioral means (Krebs *et al*., 1978).

Another possible mechanism for density-dependent suppression of reproduction is a response to pre- or postnatal stress (Boonstra *et al*., 1998; Christian, 1980; Hansson, 1989; Mihok and Boonstra, 1992). When the environment is unfavorable, environmental factors may act as non-specific stressors, and trigger a stress response (Christian, 1980). The primary stress response is increased hypothalamus–pituitary–adrenal activity, which is inversely related to hypothalamus–pituitary–gonadal function (Christian, 1980, Handa *et al*., 1994). Suppression of hypothalamus–pituitary–gonadal activity then causes reproductive suppression. The experimental evidence that predation risk, a non-specific stressor (Boonstra *et al*., 1998), significantly inhibits reproduction in *Clethrionomys* voles (Heikkilä *et al*., 1993; Ylönen and Ronkainen, 1994) and snowshoe hares (Boonstra *et al*., 1998) suggests that the stress response can suppress reproduction in cyclic populations.

Although each of the above mechanisms can potentially cause reproductive suppression at high-density phases, it is possible that two or more factors may act synergistically. Also, there may be other factors as yet not well understood that can directly or indirectly influence age at maturity. In the absence of conclusive evidence, it would be unwise to discount any possible mechanism of reproductive suppression as implausible. I note, however, that the explanation of the Chitty effect presented here does not rely on a particular factor or a mechanism of reproductive suppression. As long as sexual maturation is suppressed in high-density phases, the Chitty effect should be manifested regardless of the factors or mechanisms involved.

Foregoing reproduction reduces current reproductive success, but reproducing under unfavorable environmental conditions can adversely influence lifetime reproductive success (Bronson, 1985). Refraining from reproduction at a time when environmental conditions are less than adequate for successful reproduction might allow suppressed animals to put on additional body mass and gain experience which could enhance survival and future reproductive success (Drickamer and Mikesic, 1990; Lepri and Vandenbergh, 1986; Rissman *et al*., 1984; Viitala, 1987; Ylönen and Ronkainen, 1994) Also, reproductive suppression of young animals will result in a

lower density, and thus less competition for resources, in subsequent generations. Thus, reproductive suppression under unfavorable environmental conditions that prevail in a high-density phase of a population cycle could be adaptive, because such a strategy would allow a population to respond to the deteriorating environmental quality (Drickamer and Mikesic, 1990; Lepri and Vandenbergh, 1986; Negus *et al*., 1992; Oksanen and Lundberg, 1995; Ylönen and Ronkainen, 1994). Such a density-dependent reproductive strategy has also been suggested to be evolutionarily stable in a strongly seasonal environment where most cyclic species evolved (Kaitala *et al*., 1997).

RELEVANCE TO POPULATION CYCLES

The dynamics of a population are determined by the population growth rates, which, in turn, are functions of demographic variables. Fluctuations in population size such as those observed in cyclic populations cannot, therefore, be explained without considering demographic causes of such fluctuations. Recently, Oli and Dobson (1999) addressed this issue and suggested that phase-specific changes in age at maturity are the primary demographic cause of population cycles. Using a demographic model, they showed that changes in fertility or survival rates within the range observed in natural populations cannot cause large-scale fluctuations in population size unless such changes are accompanied by realistic changes in age at maturity. Oli and Dobson (1999) suggested that at a low-density phase environmental conditions are favorable, and animals are capable of breeding soon after they are weaned. Early maturity then causes generation time and reproductive life span to decrease, and recruitment to reproductive age class to increase. Consequently, the population grows rapidly. As the population increases, however, the quality of the environment begins to deteriorate. Consequently, physiological and behavioral responses (e.g., puberty-delaying pheromones, stress response, aggression) to unfavorable environmental conditions begin to be manifested. These responses then suppress reproduction, particularly in young animals. Delayed maturity will cause an increase in generation time, and a decrease in reproductive life span and recruitment to the reproductive age class. These demographic changes will arrest, and ultimately reverse, the population growth, leading the population to a low-density phase.

Because the Chitty effect is considered to be a ubiquitous feature of cyclic populations (Boonstra and Krebs, 1979; Chitty, 1996; Krebs, 1996), an explanation of the Chitty effect must necessarily be consistent with a mechanism of population cycles. The ideas presented here are logically consistent with the demographic mechanism of population cycles recently suggested by Oli and Dobson (1999). Both of these ideas consider phase-related, density-dependent changes in the age at which reproduction begins to be at the core of the demographic machinery that produces population cycles as well as the Chitty effect. I have argued in this paper that the Chitty effect is a consequence of phase-related, dynamic allocation of energy for somatic and reproductive efforts, which in turn is caused by reproductive suppression of young animals at high-density phases. Thus, the Chitty effect likely is an epiphenomenon, a by-product of demographic processes that produce cyclic fluctuations in abundance. This conclusion is consistent with an earlier suggestion that the Chitty effect is a consequence, not a cause, of population cycles (Lidicker and Ostfeld, 1991).

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