OPTIMAL REPRODUCTION UNDER PREDATION RISK: CONSEQUENCES FOR LIFE HISTORY AND POPULATION DYNAMICS IN MICROTINE RODENTS

by

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THESIS SUBMITTED IN PARTIAL FULFILLMENT OF

THE REQUIREMENTS FOR THE DEGREE OF

DOCTOR OF PHILOSOPHY

in the Department

of

Biological Sciences

SIMON FRASER UNIVERSITY

September 1994

APPROVAL

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DEGREE: DOCTOR OF PHILOSOPHY

TITLE OF THESIS:

OPTIMAL REPRODUCTION UNDER PREDATION RISK: CONSEQUENCES FOR LIFE HISTORY AND POPULATION DYNAMICS IN MICROTINE RODENTS

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Title of Thesis/Project/Extended Essay

Optimal reproduction under predation risk; Consequences for life history and population dynamics in microtine rodents

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ABSTRACT

Because predation is a predominant cause of mortality in many populations of small mammals, it is likely to have played a major role in the evolution of their behaviour. I show theoretically that risk of predation associated with reproduction reduces an individual's optimal reproductive effort, test that hypothesis under field conditions, and analyze the implications for population dynamics.

A model of optimal reproductive effort within a life history context demonstrates that predation risk by itself is sufficient to produce fitness optima in reproductive effort. Changes in predation risk result in changes in optimal effort in the opposite direction, and a reproductive strategy which facultatively responds to changes in risk maximizes fitness in circumstances in which predation risk changes.

This prediction is experimentally tested using meadow voles, Microtus pennsylvanicus, in field enclosures. Voles' perception of predation risk was controlled by adjusting the available cover, and their differing perceptions of risk in the high- and low-cover treatments was verified by measuring giving up density (GUD). Actual predation rate did not differ between treatments, however; nor did vole density, or the quantity or quality of food. During the experiments I measured indices of reproductive effort such as vole activity (using electronic detectors), foraging (by taking fecal plate samples), and number of young (by live trapping). Voles in the low risk treatments were more active, foraged more and reproduced more, than voles which perceived themselves to be at high predation risk.

Predation risk affects both the equilibrium population size and the stability of the population through its effect on litter size decisions. Analysis of a simple population model incorporating facultative adjustments in litter size demonstrates that individual reproductive decisions made within a life history context are tightly linked to the population's dynamics, not only because individual decisions in aggregate influence population dynamics, but also because the dynamics affect individual decisions by influencing the value of an individual's reproduction. Because the resulting population dynamics feed back into litter size decisions, a system of this sort can exhibit either point-stable, cycling, or chaotic population dynamics.

DEDICATION

My family nourished and protected my curiosity from the earliest time I can remember, and both my parents sacrificed to provide their children with opportunities to grow. In addition, good fortune decreed that my two sets of grandparents would participate in my life into adulthood, providing me with wonderful memories and a sense of place. I owe a great deal to this combination of adults in my early life; some of our good fortune we recognize immediately, some of the deeper truths are slower to take form.

It is with great pleasure that I dedicate this thesis to my family, and particularly to my father, George Henry, who first told me about the apes.

M.D.
August, 1994

ACKNOWLEDGMENTS

Although it is not possible to mention everyone who contributed significantly to the development of the research described in the pages to follow, nor to thank friends sufficiently for their ongoing support, the enthusiasm, friendship, support, and labour of many friends and acquaintances has meant a great deal to me, and has played a key role in making this work possible. To all who helped in this effort, my thanks.

Many wide-ranging discussions with fellow graduate students, especially John Reynolds and Clive Welham helped me to refine a perspective that emphasized the operation of selection on individual strategies. My interaction with John in particular made me realize the importance of proper accounting techniques.

The ideas developed here had their origins for me in many discussions with Ron Ydenberg, whose fruitful ideas have been important to the development of this research and who has continued to be vitally interested in the development of the work throughout. Ron also encouraged me to conduct my field research in Creston and made it easier to get started with the field work there.

Larry Dill, my senior supervisor, has also played a key role, and I particularly wish to thank Larry for his thoughtful contributions during the development of the experimental design, for help and observations in the field, and for providing a perspective on many research issues which

has broadened my own. While you have been supportive throughout my work at SFU, Larry, I have particularly valued your encouragement and cooperation during the last months of writing.

I also wish to thank the Creston Valley Wildlife Management Area and its staff for providing me with an ideal environment to conduct the field research, for friendly advice, and for the generous loan of material and equipment. In particular I wish to acknowledge the contribution of Brian Stushnoff for his interest in the work, for his friendship, and for his assistance in obtaining financial support.

From my first days in Creston, I have enjoyed the friendship and encouragement of Dave Klassen, who I first met when he was the area manager for Ducks Unlimited. Dave has enriched my thought with his perspective on conservation, contributed his own material and labour to the field research, and most importantly, always been ready with a rock-solid friendship that I treasure deeply.

The field research could not have been conducted without the diligent efforts of field assistants, and I particularly wish to mention the contributions of Dorothy Hill, who cheerfully did much more electronics than either of us had bargained for and who helped get the first field season rolling in Creston, and Bob Setter whose considerable skill, good humor, and sense of adventure lightened the back-breaking tasks associated with the initiation of the full-scale experiments. Dean Mulligan volunteered his time and enthusiasm at critical times and

provided assistance which I greatly needed. Thanks to Laura Hooker for her hard work and challenging insight. Michal Strauss brought dedication, intellectual curiosity, and insight to the work which helped to keep alive my own wonder at nature.

My thanks, too, to Alex Fraser, who provided an important link to SFU when I was in the field, helped to get needed field equipment to Creston quickly, and who volunteered a considerable amount of his time to grind my vegetation samples and arrange for their analysis. Alex's knowledge and help made my experience much more enjoyable and productive.

Financial support for this work from the Science Council of BC GREAT program to myself and from NSERC through an operating grant to Larry Dill is gratefully acknowledged.

Most importantly, I wish to thank Anne de Jager. Anne supported my efforts with ideas, encouragement and enthusiasm. At times her financial commitment was critical in providing me with the freedom from worry I needed to develop my thoughts. In the field, Anne gave freely of her organizational skills during several forgone vacations and labored as my most dedicated and hard working field assistant under extremely trying circumstances. She shared in the sweat and tears, in the field and at home, and in doing so received more than her share of both. Without your encouragement, enthusiasm, and hard work I would not have completed this work, Anne. Thank you.

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PROLOGUE

Adaptive flexibility in the behaviour of animals is well established, but the idea that individuals might exhibit plasticity in life history traits is much less well developed. Nevertheless, the notion that selection acts to optimize reproductive effort over an individual's lifespan, perhaps the basic tenet of life history theory, almost seems to demand the ability to facultatively adjust reproductive investment, for the key parameters which determine the optimal allocation of reproductive effort at each point in time (survival probability and population growth rate), both may fluctuate dramatically. Strategies which incorporate facultative adjustment in reproductive effort to meet the changing optima that result should be strongly selected for. Thus, just as investment in reproduction has been shown to change through an individual's lifespan in an evolved obligatory response to factors such as parity and senescence, it should also be capable of adjusting in response to factors that are predictable only in the shorter run, such as predation risk.

In fact, it was a consideration of reproduction under predation risk which initially led me into the avenues of thought sketched above. Predation risk associated with reproductive activity can impact the optimal level of reproduction just as any other cost of reproduction, although risk is unique in that it is probabilistic. An individual that survives a reproductive bout may not show any physiological effect of the cost of predation risk, but the effects of risk will be reflected in the reproductive decisions taken by the individual during the course of the reproductive event. Unlike other costs of reproduction, however,

predation risk varies through time for many species; thus, the appropriate investment in reproduction may well change in response.

However, if the reproductive output of the individuals in a population changes, the dynamics of the population itself will be affected. Thus, population level phenomena are linked to reproductive decisions taken at the individual level. What has been less clear is that the population's dynamics also impact the optimal decisions of individuals. It is adaptive for individuals in an expanding population to shift resources into current reproduction, and, conversely, in a declining population to withold reproductive investment until a future time. Thus both levels of phenomena (individual and population) should influence each other, and the resulting behaviours and dynamics will be closely linked.

I address each of the major points raised above in the following chapters. Chapter 1 develops an approach to understanding optimal reproduction under predation risk, and demonstrates that if individuals do facultatively adjust their reproduction, increases in risk associated with reproduction will result in lower reproduction (in this case litter size). Chapter 2 presents a field test of the effect of predation risk on reproduction of meadow voles, Microtus pennsylvanicus, which demonstrates that reproduction does respond in the predicted manner to the level of predation risk as perceived by the voles. Chapter 3 considers the links between optimal reproductive behaviour of individuals and the dynamics of their population.

CHAPTER 1

OPTIMAL REPRODUCTIVE BEHAVIOUR UNDER PREDATION RISK

INTRODUCTION

While predation's direct and indirect effects have often resulted in the evolution of fixed life history patterns (e.g. Lynch 1980), a growing body of evidence indicates that facultative responses in life history strategies have also evolved in response to predation risk (e.g. Dodson & Havel 1988). A flexible life history should be adaptive for species that face a trade-off between a varying predation risk and other components of fitness such as growth (Skelly & Werner 1990, Werner & Hall 1988), mating activity (Sih et al. 1990), brood size (Nur 1984a,b), or the avoidance of other mortality factors (Holomuzki 1986a,b). In general, adaptive responses to predation risk can powerfully shape individual behaviours (reviewed by Lima & Dill 1990) and we should expect to find them incorporated into life history strategies.

Predation risk plays the role of a cost of reproduction when risk increases with reproductive effort, because future reproduction must be traded off against current reproduction. However, predation risk assumes a somewhat different role than physiological costs (such as decreased future fecundity or weight loss) because its effect is probabilistic, unlike physiological costs.

Predation risk is sometimes associated with one or more of the physiological costs of reproduction (eg. weight loss, Nur 1984a, Reid 1987) which might predispose the organism to falling victim to a predator. However, predation risk can function quite independently of other costs. In fact, Lima (1987) has argued that even if no other costs were associated with avian reproduction, predation risk would produce

an optimal clutch size in the same manner as physiological costs. An animal capable of reproducing without physiological diminution of its ability may still experience increased predation risk while producing and raising its young. Although the individual may be capable of producing offspring repeatedly, predation risk lowers the probability that it will do so.

In reviewing the effect of reproduction on parental survival, Roff (1992:170) concludes that the literature in general supports the concept of a survival cost to reproduction. Just as the search for mates may expose adults to greater predation, the search for sufficient resources to rear young to independence may expose parents to increased predation by requiring them to be more active, to leave hiding places more frequently, or to spend more time occupying relatively unsafe feeding habitat. Indeed, since female mammals spend more time and energy rearing young than attracting mates or supporting embryogenesis, increased probability of mortality resulting from raising young may represent a relatively high cost of reproduction.

In particular, small mammals typically experience heavy predation and one would expect that the risk of predation has been a powerful force in the evolution of their reproductive strategies. Ydenberg (1987) has suggested that individuals may reproduce less when faced with high predation risk and that changes in their behaviour may influence small mammal population cycles. In this paper I develop a theoretical approach to optimal reproduction under predation risk by modelling optimal litter size when predation risk is the only cost of reproduction. I then extend the model to include life history considerations. While the theoretical considerations are not restricted to small mammals, certain

aspects of the model, such as litter size, are oriented toward an understanding of their reproductive strategy, a consideration which originally motivated this work.

REPRODUCTION AND PREDATION RISK

In constructing these models I first assume that reproductive success is maximized in a single reproductive bout and then introduce life history considerations by assuming that reproductive success is maximized over the individual's lifetime. In both cases, simple models can predict optimal litter size for a female reproducing in a risky environment.

I assume that a female's chance of surviving decreases as she attempts to raise a larger litter, either because she is more vulnerable (larger or slower, for example) or because she spends more time exposed to predators while foraging (see below). Thus, the female faces an optimization problem--if her reproductive strategy places too much emphasis on her own survival she will raise too few young, but if she attempts to raise too many young she increases the chances of her own death and the subsequent death of any dependent offspring. The problem facing the female is to select the appropriate litter size for the predation risks she faces. However, predation risk may change over time, in which case the optimal litter size may also change. Females which adjust their litter sizes should be more fit than those which do not.

There is evidence that microtine females do adjust reproductive effort in response to changing optima both pre- and postpartum. Taitt et al. (1981) found that Microtus townsendii populations which were protected from predation risk with additional cover bred earlier in the season and exhibited quadruple the number of females pregnant. Other prenatal mechanisms of adjustment include suppressed reproduction in

Clethrionomys glareolus in the presence of predators (Ylönen 1989), and increased incidence of pregnancy failure at high density in M. pennsylvanicus (Mallory & Clullow 1977). Post-natal mechanisms of litter size reduction have not yet been tested for their relationship to predation risk, but suggestive findings include poorer parental care and maternal infanticide in M. pennsylvanicus and Neotoma floridana (McShea & Madison 1987, McClure 1980, respectively), differential defense of young against conspecifics depending on neonate age (Mallory & Brooks 1978), litter size response to food for M. californicus (Krohne 1980) coupled with evidence that M. ochrogaster eat less when exposed to predation risk (Desy & Batzli 1989), and postpartum litter size reduction in M. pennsylvanicus at high density (McShea & Madison 1989).

Therefore, I assume that females can adaptively adjust the number of young they raise, either through physiological mechanisms before parturition or through behavioural mechanisms after birth. Such variation in litter size represents a conditional life history strategy which may be employed by a single individual, and thus the need to assume a genetic polymorphism (Chitty 1958, 1960) is obviated.

Below I present models of optimal reproduction in which predation risk associated with reproductive effort serves as the cost of reproduction, and litter size is adjusted facultatively to maximize fitness. Animals are assumed to have a lifespan of indefinite length without senescence, restricted only by the predation risk they incur as a result of foraging for the energetic demands of maintenance and reproduction. First, a model which considers only one reproductive bout is presented,

followed by a model which places facultative adjustment of litter size within a life history context.

COST OF REPRODUCTION

It is clear that small mammals support the increased energetic demands of pregnancy and lactation primarily through current food intake (Millar 1975, Stebbins 1977, Genoud & Vogel 1990, Kenagy et al. 1990). The additional exposure to predation due to the increased foraging activity required is likely to decrease survivorship.

Therefore, I assume that the probability an adult female will survive the current reproductive bout is inversely related to litter size and denote it S(L). The derivative of S(L) with respect to litter size, S'(L), must be negative since survival is assumed to decline with increasing litter size. In order to emphasize the role that predation risk might play, I assume in the model that the entire cost of reproduction lies in the increased likelihood of predation as foraging effort increases to support larger litter sizes.

FITNESS BENEFIT OF INCURRING RISK

The expected contribution of the current reproductive bout to the female's fitness is the expected number of surviving offspring (times the relatedness coefficient, 0.5) plus the likelihood she survives the bout. Assuming the offspring cannot survive without the mother, the expected number of surviving offspring is given by $S(L)\cdot f(N)\cdot L$, where f(N) is the probability of juvenile survival. The expected total fitness contribution of the reproductive bout is then given by:

$$RS = S(L) + (.5)\cdot S(L)\cdot f(N)\cdot L \qquad [1.1]$$

The juvenile survival probability, f(N), is assumed to be a declining function of density. Decreased survivorship of juveniles at higher densities may result from a number of mechanisms including infanticide (Mallory & Brooks 1980, Caley & Boutin 1985), food limitation coupled with lower competitive ability of young animals (Desy & Batzli 1989), and increased dispersal into less favorable habitats.

OPTIMAL LITTER SIZE

The litter size which maximizes reproductive success, L*, is found by setting the derivative of RS with respect to L to zero and solving for L. This results in:

$$L^* = -S(L)/S'(L) - (1/.5)\cdot(1/f(N)) \qquad [1.2]$$

Although the precise specification of adult survival, S(L), is not critical to the model, some reasonable assumptions lead to simple results. Assume that adult females risk randomly occurring predation at a constant rate R per unit of food energy obtained, and that the energetic requirement of a female is equal to her maintenance requirement, m, plus an additional cost, c, per offspring. Then the probability that the female survives the reproductive period is an exponentially decreasing function of litter size:

$$S(L) = \exp\{-R\cdot(m+c\cdot L)\} \qquad [1.3]$$

Note that by a simple manipulation, Eqn [1.3] can also be expressed:

$$S(L) = \exp\{-R \cdot c(m/c+L)\} \qquad [1.4]$$

where the product R·c in the exponent represents predation risk per offspring, rather than per calorie. The general shape of this family of survival curves is shown in Figure 1.1 for three values of predation risk.

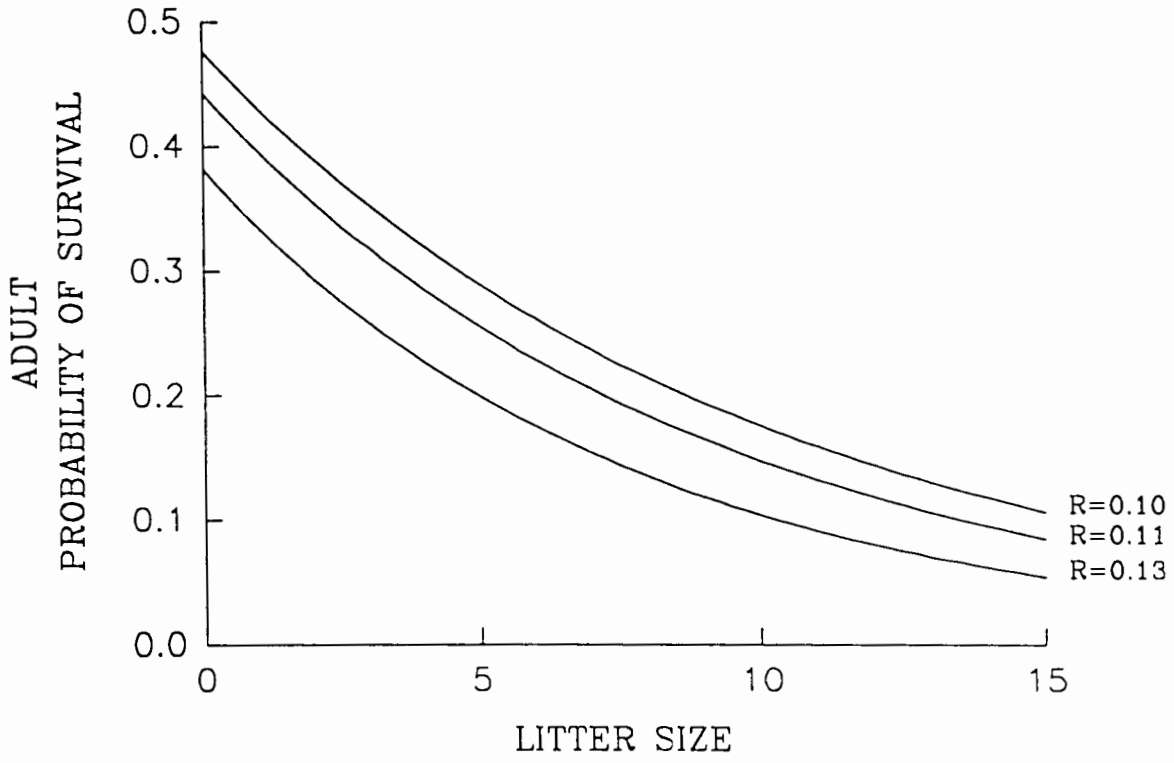

Fig. 1.1. Adult survival function. The family of curves specified by Eqn [1.3]. Curves are shown for R=0.10, 0.11, 0.13 and m=763 Kcal, c=103 Kcal (typical of M. pennsylvanicus, Innes & Millar 1981).

Substituting Eqn [1.3] into Eqn [1.2] gives a simple solution for optimal litter size:

$$L^* = 1/(R \cdot c) - (1/.5) \cdot 1/f(N_t) \qquad [1.5]$$

Specification of the juvenile survival function, $f(N_t)$, completes the one-period model. I have assumed the generalized reverse sigmoidal curve shown in Figure 1.2 and specified by:

$$f(N_t) = 1/(1+e^{b(N_t - N_{.5})}) \qquad [1.6]$$

Substituting Eqn [1.6] into Eqn [1.5] results in the general solution for L^* shown in Figure 1.3. The precise shape of the juvenile survival curve used is not critical to the resulting solution for the one period model, whose important features include a decline in litter size with increasing predation risk, and with increasing energetic cost of producing young, c. Interestingly, the maintenance cost for females, m, drops out of the solution (see Eqn [1.5]).

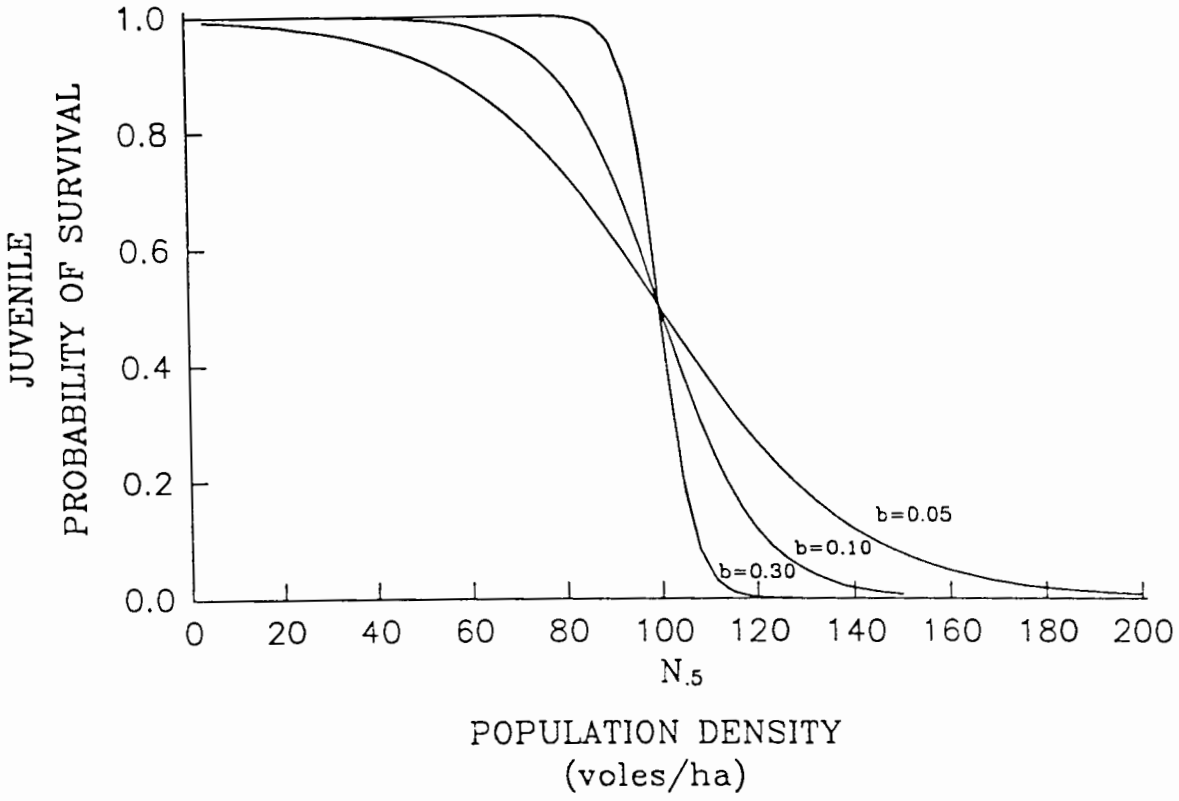

Fig. 1.2. Density dependent juvenile survival function. The family of curves specified by Eqn [1.6]. Two parameters determine the function: $N_{.5}$, which is the population density at which each offspring has a 50% chance of survival; and the slope parameter, b, which determines the steepness of the function as it passes through its mid-range. Curves are shown with $N_{.5}$=100, b=0.30, 0.10, 0.05.

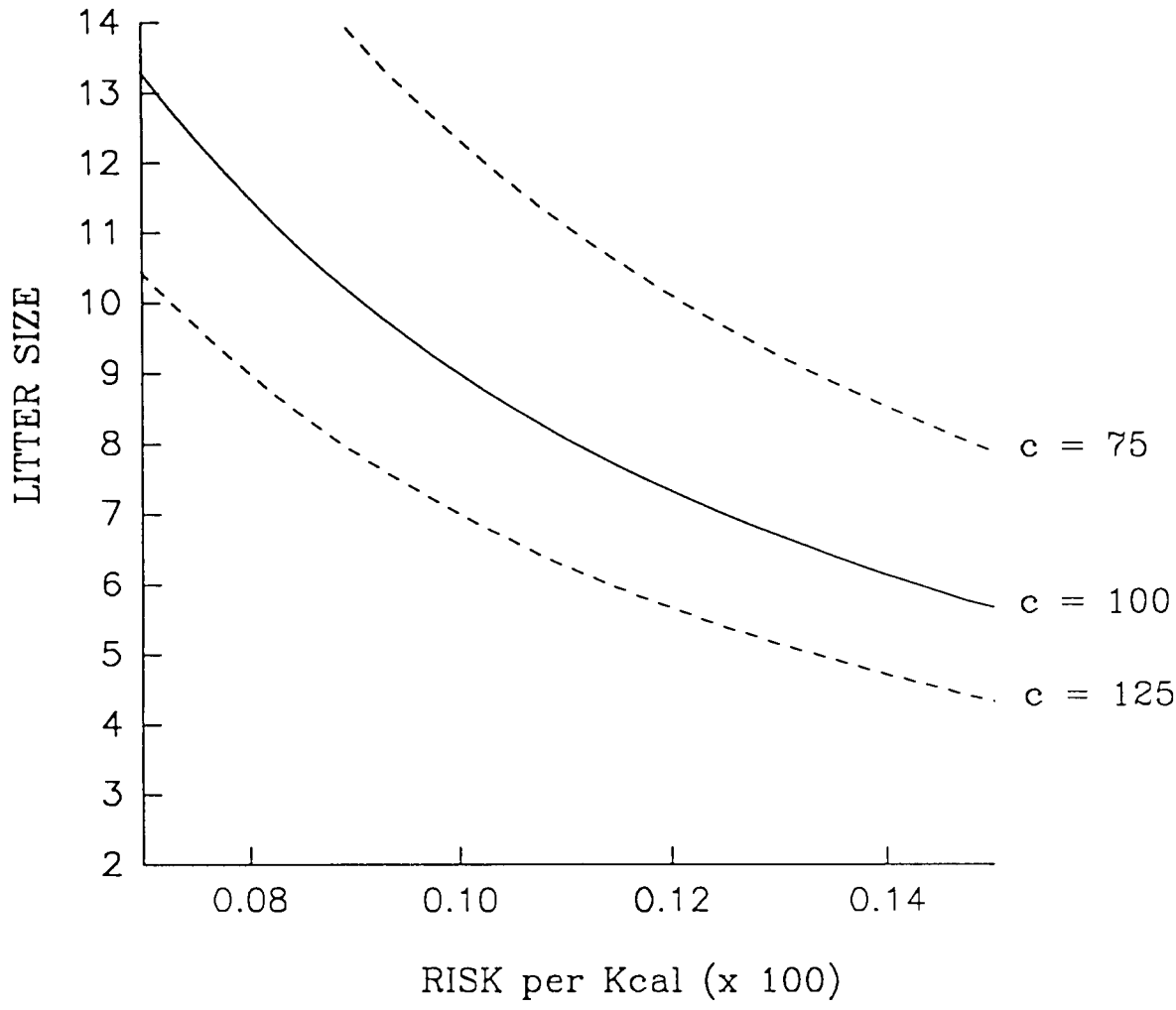

Fig. 1.3. Solution to the one period fitness-maximizing model. The effect of risk on litter size in the one period model. Shown for typical c=100 Kcal/offspring ± 25%, with b=0.30, N_5=100, and N=5 (low population density). Litter size has been graphed against risk per Kcal ($\bar{R}$) in order to show the effect of offspring cost (c) on litter size, but subsequent graphs show risk per offspring (=R·c) on the x-axis.

LIFE HISTORY CONSIDERATIONS

Eqn [1.2] maximizes reproductive success for the current bout, but natural selection favors traits which maximize the reproductive value of the individual calculated over its lifetime (Fisher 1930). Reproductive value differs from lifetime reproductive success because, in addition to fecundity, it takes into account both the likelihood that the individual is still alive to reproduce at each point in the future and the growth rate of the population, which serves to discount the value of future reproduction. The reproductive value of an individual at the beginning of a reproductive bout is given by:

$$RV_1 = S(L_1)f(N_1)L_1 + 1/\lambda_1 S(L_1)S(L_2) + \ldots \qquad [1.7]$$

where: L_t = litter size at beginning of reproductive period t, and

$\lambda(L_t) = N_{t+1}/N_t$ = population growth rate in period t, which naturally varies with the litter sizes of females in the population.

Eqn [1.7] may be rewritten:

$$RV_1 = S(L_1)\cdot f(N_1)\cdot L_1 + 1/\lambda_1 \cdot S(L_1)\cdot RRV_2 \qquad [1.8]$$

where: RRV_2= residual reproductive value, that is reproductive value calculated from the beginning of period 2 rather than period 1.

Reproductive value in Eqn [1.8] cannot be maximized using the standard procedure employed for [1.1] above, because [1.8] represents a frequency-dependent situation: the reproductive value of any given litter size decision, L_1, to the individual employing it depends on the population growth rate λ_1, which in turn depends on the litter sizes chosen by all females in the population. Thus, the optimal litter size in

this model will be an evolutionarily stable strategy (ESS), denoted L_{ESS1}. It is found by writing RV in [1.8] as a function of both the population's average litter size for time period 1, L_1 and the litter size of an individual which deviates from the population strategy, $\hat{L}_1$:

$$RV_1 = RV(L_1,\hat{L}_1) = S(\hat{L}_1)f(N_1)\hat{L}_1 + 1/\lambda_1(L_1)S(\hat{L}_1)RRV_2 \qquad [1.9]$$

Differentiating with respect to L_1 and solving for L_{ESS1}:

$$\partial RV_1/\partial\hat{L}_1\Big|_{\hat{L}_1=L_1} = \{S'(\hat{L}_1)f(N_1)\hat{L}_1 + S(L_1)f(N_1) + 1/\lambda_1 S'(\hat{L}_1)RRV_2\}\Big|_{\hat{L}_1=L_1} = 0 \qquad [1.10]$$

$$L_{ESS1} = \hat{L}_1 = -S(L_1)/S'(L_1) - 1/f(N_1)\cdot 1/\lambda(L_1)\cdot RRV_2 \qquad [1.11]$$

Note that consideration of optimal reproduction within a life history framework changes the resulting optimal litter size (compare Eqns [1.2] and [1.11]). The extent to which ESS litter size differs from the single period fitness maximizing litter size depends on the ratio of expected future success to current population growth (RRV_2/λ_1). It will sometimes be adaptive for females to play it a bit safer (i.e., smaller L_{ESS1}) in order to be more certain of obtaining the benefit of future reproduction. On the other hand, larger litter sizes will be more adaptive when the population is growing rapidly (λ_1 large) and when there is little likelihood of future reproductive success (RRV_2 small).

Although Eqn [1.11] cannot be solved explicitly for L_{ESS1} (because λ incorporates the adult survival function in a manner which does not

permit explicit solution), an iterative solution can generally be attained when all the parameters are specified (see Chapter 3, Appendix 3B). The resulting solution for L_{ESS} can be compared with L^* at any population density (see Figure 1.4). The ESS litter size is less than L^* for all values of predation risk, but the difference is greater at higher density because λ_1 is smaller, causing the ESS solution to put more emphasis on future reproduction (see Eqn [1.11]). Most importantly, increased risk results in reduced litter size for both L_{ESS} and L^*.

When predation risk changes, animals may perceive the change either as one which holds over the long term or as a short term aberration. To this point I have assumed that a change in risk indicates a long term change. In terms of this model the difference between the two scenarios is that in the first instance, $S(L_t)$ in Eqn [1.7] has the same value for risk, R, in all periods. However, if animals perceive a change in risk as short term, R will not be the same for all periods, and the adult survival function in Eqn [1.7] might be better expressed as $S(L_t,R_t)$. This difference would, of course, carry through to the solution of the model.

It can also be shown, however, that if risk is allowed to vary through time, the derivative of ESS litter size with respect to current predation risk is negative. Thus, an increase in risk results in an effective postponement of some reproductive effort to future bouts, whether the increase in risk holds for only one period or for all time.

It is also worthwhile to note that residual reproductive value, RRV_2 in Eqn [1.11], is itself a product of the solution, since its value depends

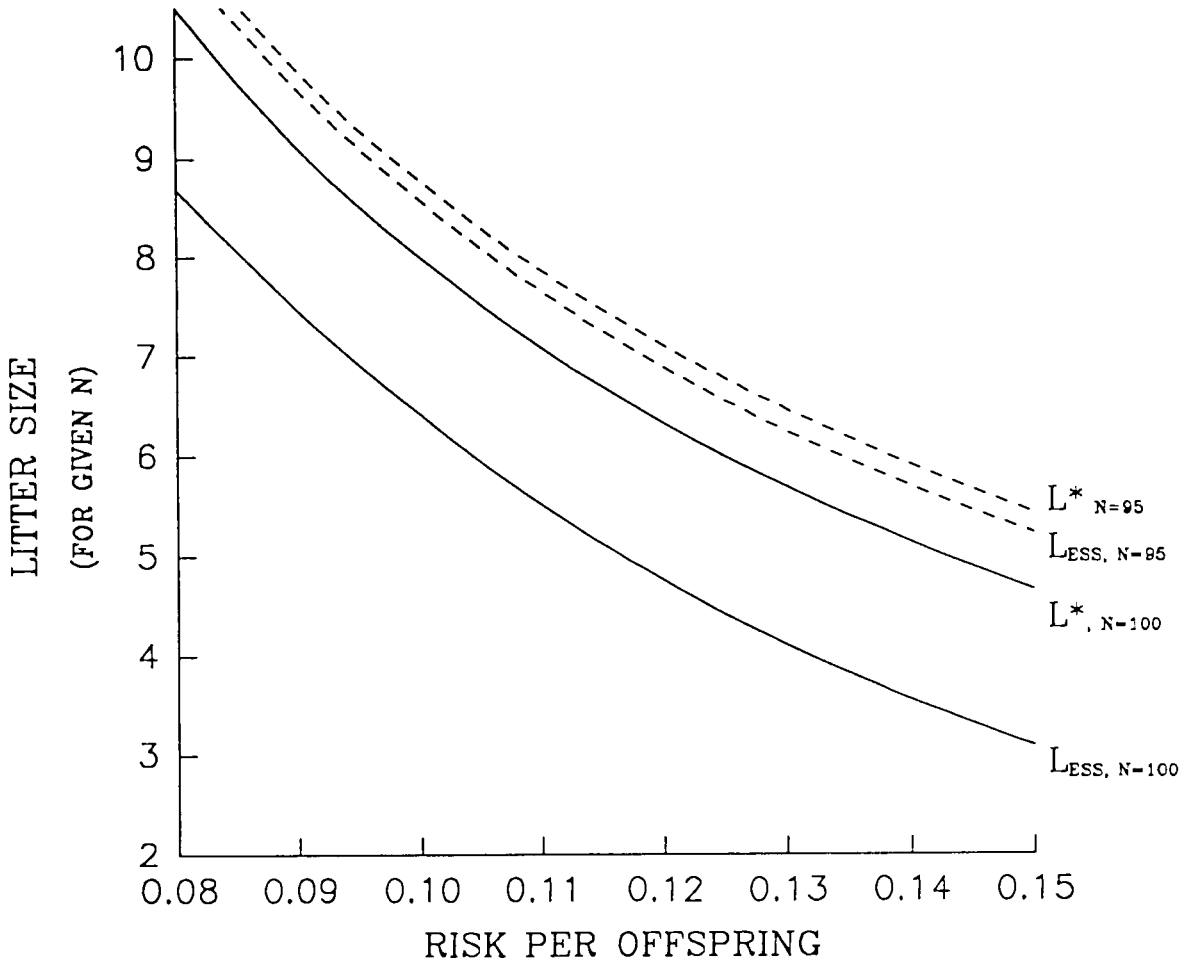

Fig. 1.4. Comparison of litter size strategies. L_{ESS} and single period fitness maximizing litter size L^* for $N=.95N_{.5}$ and $N=N_{.5}$. Each point along the L_{ESS} curves represents the result of an iterative solution to Eqn [1.11]. Curves calculated with b=0.30, $N_{.5}$=100, m=763 Kcal, c=103 Kcal

on the optimal litter size at each future bout (see Eqns [1.7] and [1.8]). Thus, ESS litter size and residual reproductive value are determined simultaneously, a point to which I return in the discussion, and in Chapter 3.

DISCUSSION

The life history model demonstrates that predation risk can function as a cost of reproduction independently of any physiological trade-off against future reproduction. This is true even though predation risk functions in a probabilistic manner, i.e., even though no individual can predict whether predation will end its life during a given reproductive bout, its expected reproduction is well-defined once its litter size is determined, as is the value of its potential future reproduction.

The model presented here relies on a fundamental tenet of life history theory: individuals choose strategies which maximize reproductive value at each reproductive bout. However, reproductive value is difficult to measure and is often replaced by a simple summation of the number of offspring successfully produced, termed lifetime reproductive success. The two measures of fitness are equivalent only when the population is stable, because in a changing population offspring produced in the future have a different fitness value to the parent than offspring produced currently. The more rapidly a population changes the more the two measures of fitness will diverge. In a population which may change over time, it is necessary to take the rate of population change (λ) explicitly into account, in order to allow proper discounting of future reproduction.

Explicit consideration of the role of population change helps to clarify two points. First, optimal behaviours of individuals and population dynamics are firmly linked. Individual reproductive decisions influence the density and rate of change of the population, while at the

same time these population parameters influence the optimal allocation of individual reproductive effort between the present and the future. I return to a consideration of the links between individual and population level phenomena in Chapter 3.

Secondly, optimization of reproductive effort is frequency-dependent and requires an ESS solution. An individual's optimal investment in current reproduction depends on the reproductive decisions of others because the fitness value of future reproduction is affected by the rate of population change, which itself is determined by the reproductive decisions made by all the individuals in the population.

The assumption of optimal behaviour on the part of individuals can have important implications for other areas of ecology. For example, we have traditionally tended to think of individual life history decisions as deriving from fecundity and mortality schedules set out in a standard life table, which determine an individual's residual reproductive value. But the direction of causality may well be reversed. Although no mortality or fecundity schedule is built into the model presented here, and no senescence is assumed, individuals in effect generate their own schedules by pursuing particular litter size strategies. In effect they choose the rate at which to die through their reproductive decisions.

The aggregate result of these decisions over the lifetime of individuals in a population produces what we commonly interpret as mortality and fecundity schedules. However, these schedules represent the results of decisions taken in response to particular combinations of circumstances (in this model, levels of predation risk and population density). Seen within this context, the life table loses much of its

predictive value because it becomes a reflection of individuals' decisions rather than a determinant of those decisions.

Although this conclusion is particularly clear for the model presented, it has general applicability because when facultative adjustment of reproductive effort occurs, individuals also select the level of costs to incur and thus determine, at least in part, their own fecundity and mortality schedules.

The model I have presented specifically addresses only optimal litter size, but other questions, such as the optimal age at dispersal or at first reproduction, could be addressed within a similar framework by, for example, expanding the approach to allow for age-specific effects. I have excluded other effects from this analysis in order to clarify the possible role of predation risk as a cost of reproduction, to stress the importance of frequency-dependence, and to emphasize the role that facultative reproductive decisions may play not only in determining current reproduction, but also in establishing residual reproductive value.

In the next chapter I present a field test of the model's central prediction, that increased predation risk will result in decreased current reproductive effort.

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CHAPTER 2

THE EFFECT OF PREDATION RISK ON REPRODUCTION IN MEADOW VOLES (Microtus pennsylvanicus)

INTRODUCTION

Animals may adjust their reproductive investment in response to predation risk for two reasons. If reproduction carries an associated increase in predation risk, parents may have a better chance of successfully raising their current young by attempting to raise fewer young. They may also increase the likelihood of surviving to reproduce in the future, when the risk may be lower. Elsewhere (Chapter 1), I have shown theoretically that the ability to adjust reproductive investment is adaptive when predation risk is variable. My emphasis in conducting the current study has been to test the effect of predation risk on reproduction.

Microtine rodents appear to be an ideal group on which to test these ideas. Many species experience fluctuating predation pressure while producing and caring for large and variable numbers of offspring (reviewed by Innes 1978). Although a growing body of literature indicates that predation plays an important role in determining the behaviour and population dynamics of small mammals, the effects of predation risk are often not distinguished from those of predation itself, nor has it been generally recognized that these individual responses potentially have a greater impact on both life history and population parameters than predation's direct effects. Although there is evidence that predators of microtines play a role in determining the latter's activity level and reproductive rates (e.g. Taitt et al. 1981, Taitt and Krebs 1981, Kotler et al. 1988, Adler and Wilson 1989), and a significant body of thought that the nature of small mammal population dynamics is

determined by predators (Birney et al. 1976, Hansson 1984, Henttonen et al. 1987, Hanski et al. 1991), it is unclear to what extent the observed phenomena are actually due to individual prey responses to predation risk rather than to the direct effects of predation itself.

Evidence already exists that small mammals alter their foraging activity and habitat use in response to predation risk (Kotler et al. 1991, Longland 1991), and that predation risk is increased by activity (Kaufman 1974). In this paper I present the results of field experiments designed to examine the effect of predation risk on the primary reproductive decision faced by microtine females - how many young to produce and attempt to raise in a given reproductive period.

I first develop and test a method of altering meadow vole (Microtus pennsylvanicus) assessment of predation risk without changing other factors which might affect life history decisions, such as vole density, mortality rate, or food availability (Cole & Batzli 1979, Taitt & Krebs 1981) using both field and laboratory experiments. Then, I examine the effect of predation risk on vole activity, foraging and reproduction in field experiments, testing the hypotheses that higher predation risk will result in less activity and foraging, and decreased reproduction, quite independently of the direct effects of predation per se.

MANIPULATION OF PERCEIVED PREDATION RISK

METHODS

Field experiments to determine the effect of predation risk on reproductive output and activity level of voles were conducted in 1990 and 1991 on the Creston Valley Wildlife Management Area, a large (7000 ha) conservation area approximately 700 km east of Vancouver, BC.

I selected two sites separated by approximately 1 km for the experiments. The two sites were chosen to be as similar as possible; both had flat terrain with a dominant vegetative cover of reed canary grass (Phalaris arundinacea), and both sites were located in areas known from initial trapping to be inhabited by M. pennsylvanicus.

At each site I constructed four square enclosures, each 15 m on a side. The enclosures were placed adjacent to one another so that the overall shape of the fenced area at each site was that of a large (30 x 30 m) square subdivided into four quadrants.

The design and operation of the enclosures was intended to meet two objectives: to prevent voles from entering or leaving, and to prevent actual predation from occurring while still exposing enclosed voles to an environment which they perceived to be a risky one, particularly with regard to avian predators. The walls of the enclosures were constructed of galvanized steel roofing material, approximately 91 cm wide, buried half-way in the ground. To ensure that baseline cover available was as similar as possible among enclosures, dead vegetation remaining from previous years was removed before vegetative growth started each spring.

35 nestboxes measuring 15 cm square x 10 cm high were placed in a regular grid pattern within each enclosure.

Within each enclosure a narrow border of approximately 25 cm was kept clear of vegetation with a weedeater to discourage voles from attempting to tunnel under the wall. To make it more difficult for a vole or small predator to climb in or out of the enclosures, posts used to support the wall were made of metal and placed so that the wall extended about 8 cm above the top of each post. Where possible posts were placed on the outside of the enclosure wall. Outside the enclosures the vegetation in a wider border of approximately 5 m was kept mowed to a height of 10 cm to discourage small predators. A three-wire electric fence was erected above the perimeter wall of each site to deter coyotes (Canis latrans) and elk (Cervus elaphus) from entering or damaging the enclosures. Experience has demonstrated that large herbivores are capable of inflicting significant havoc incidental to their grazing activities. To discourage avian predators from actually taking enclosed voles, vertical wooden stakes, extending to a height of 1.2 m, were positioned on a 2 m grid pattern within each enclosure.

Numerous natural predators were observed near the experimental sites, including kestrels (Falco sparverius), great horned owls (Bubo virginianus), marsh hawks (Circus cyaneus), red-tailed hawks (Buteo jamaicensis), weasels (Mustela erminea), and coyotes. In order to manipulate predation risk without altering food availability, I added cover to low-risk treatment enclosures. Because M. pennsylvanicus do not eat clean wheat straw (unpubl. data) it was used as the added cover. Each low-risk enclosure was given an even covering of straw, applied at a level of 0.2 kg/m^2, after the natural vegetation had reached a height of

approximately 60 cm. Within a few days the straw had worked its way down into the vegetation and appeared to provide a natural cover for voles. Grass cover and straw have been previously shown to reduce predation rate for voles (Taitt et al. 1981, Adler & Wilson 1989).

Two of the four enclosures at each site were treated with straw (low-risk) and two were used for high predation risk treatments. Within each site, diagonally opposite enclosures were given the same predation risk treatment so that adjacent enclosures received opposite treatments. Because each enclosure received the opposite risk treatment from its two neighbors the effect of any undetected differences in microhabitat due to unidentified variables such as soil quality should have been minimized.

Meadow voles used to stock the enclosures were trapped in the vicinity of the enclosures (within 0.5 km) in nearly identical habitat to that in the enclosures, and were matched by sex and by weight (within 0.5 g) across the enclosures. All animals used in the experiment were selected for maturity (scrotal testes in males, and developed nipples or perforate vaginas in females). Voles selected were individually marked by toe clipping and released into the enclosures (10 females, 2 males per enclosure) within 4-h of capture. Site 1 was stocked on June 26, 1990, but Site 2 was flooded at that date because of an extremely wet spring and was not stocked until August 1.

EFFECT OF RISK TREATMENT ON FOOD ABUNDANCE AND QUALITY: VEGETATION SAMPLING

Because the cover treatments for predation risk might have also affected the quantity or quality of vegetation available for food, I tested for such effects in separate pens, from which all vertebrate grazers were excluded. These exclosures were constructed in 1991 at Site 1, within

30 m of the large enclosures, in identical terrain and vegetation, and were separated from each other by approximately 10 m. Each of the four exclosures received the same preparation as the main enclosures. Then each exclosure was divided in two, with one half given a straw covering identical to that used in the low risk treatments in the large enclosures (0.2 kg/m^2) and the other half left alone (like the high risk treatments).

Vegetation in the exclosures was sampled three times: once immediately after the cover treatment (July 8), and twice during the growing season (July 24, Aug. 22). All four exclosures were sampled on the first date to provide baseline data, but on each of the next dates only two of the exclosures were sampled. This strategy was chosen to minimize the possibility that the sampling procedure itself would affect vegetative growth by opening gaps and allowing in extra sunlight.

Each time I sampled the vegetation in an exclosure, I clipped 5 30-cm square quadrats of vegetation from the high risk treatment and 5 quadrats from the low risk treatment in that exclosure. To minimize trampling in the relatively small exclosures, the quadrats were selected before vegetative growth began in such a manner that the first sampling involved clipping the outermost quadrats in each exclosure, allowing room for the worker to perform subsequent sampling without damaging vegetation, and so that the initial sampling did not expose quadrats used in subsequent sampling to additional light. The same sampling pattern was used for each small exclosure.

As each quadrat was clipped, any straw was removed and the remaining vegetation placed in individually marked brown paper bags and air dried at approximately 40°C until completely dehydrated. The dry weight of each sample was recorded for later analysis.

Because the reproduction experiment described below depends on equal food availability across treatments, I hoped that no difference could be detected. To provide as strong a case as possible, data were analyzed using parametric statistics to maximize power and minimize the possibility of a type-2 error. Sample weights from each sampling date were tested for homogeneity of variance across treatments and enclosures using Bartlett's test and then analyzed for differences between strawed and non-strawed treatments using ANOVA.

Since the cover treatment might also have affected the quality of the vegetation, I analyzed the vegetation samples taken on the final sampling date for both nitrogen and carbon content. After each of the twenty available samples was powdered in a grinder and thoroughly homogenized, two subsamples of about 2 g drawn from each of the samples were each chemically analyzed twice using the combustion analysis method (Carlo-Erba Elemental Analyzer Model 1106). The results from the chemical analyses were assessed using ANOVA following Bartlett's test.

DEPLETION EXPERIMENT

I required a field method of comparing a vole's assessment of risk under different treatments in order to ensure that the experimental treatments had the desired effect. One such method is to measure the "giving up density" (GUD) for food search using pans containing a mixture of food and non-food items (Brown 1988; Brown et al. 1988; Kotler 1984). As food is depleted in a pan, each subsequent food item takes longer to find and thus exposes the forager to greater risk of predation. An animal should quit foraging when the predation risk that

would be incurred in finding another food item exceeds the value of the food item. The density of food remaining in the pans provides a measure of the giving up density and thus a means of comparing animals' perceptions of risk across experimental treatments.

Because the GUD technique relies critically on the assumption that search time increases as food is depleted in the pans, I conducted laboratory experiments to test for this depletion effect to ensure that my adaptation of the method was valid. In separate runs I presented voles with food pans stocked with three known food densities and measured their search times in order to determine whether longer searches were necessary to find individual food items at lower food densities compared to higher ones.

Ten voles (5 male and 5 female) from the same wild population used in the field experiments were set up for observation in opposite sex pairs in large (30 x 50 x 25 cm) aquaria placed so that they received natural light during the day. An observer location was established in a darker area about 2 m from the aquaria and separated from the aquaria by a reflective pane of glass which made the observer less visible from the aquaria side. Aquaria were separated from each other by translucent shields so that activity in one would not influence the others.

Voles were placed in their aquarium at least 3 days before any data were collected, provided with sufficient water and bedding material and given carrots and reed canary grass. In addition, a pan measuring 19 x 9 x 6 cm was filled with a well-mixed combination of cubed carrots and coarse sawdust (25g:115g) and settled into the bedding material at one end of each aquarium. This mixture was replaced daily during the familiarization period.

Six hours prior to an experimental run any food remaining in the aquarium was removed, and at the start of each run a fresh pan containing the appropriate carrot/sawdust mixture was placed in the aquarium. Cubes of carrots were cut as uniformly as possible (average weight 0.85 g) with a vegetable dicing machine. I presented three mixtures of food and filler: 25 g cubed carrots to 115 g sawdust (high food density), 15:115 (medium), and 5:115 (low) in separate runs and recorded the voles' activities until either 5 g of carrots (6 pieces) had been removed or 10 min had elapsed. During each run, activities of individual voles were timed using a personal computer programmed to function as an event recorder. Each vole's activity was timed and recorded in one of four categories: successful search for food, unsuccessful search, eating, and other activity.

Search time for each carrot cube was calculated as the sum of the search times occurring since the last piece was found; thus, the length of unsuccessful searches as well as the eventual successful search contributed to measured search time.

Data were collected for at least six runs on each animal (2 runs at each of the three initial food densities). Data were analyzed using the Kruskal-Wallis test across the three categories of food density, and using pair-wise Mann-Whitney U tests between pairs of categories.

VOLES' ASSESSMENT OF PREDATION RISK: GUD EXPERIMENT

I measured the effectiveness of the cover treatment in altering the voles' perceived predation risk by measuring giving up density (GUD) of food in the experimental enclosures themselves. Pans were filled with a mixture of carrots and sawdust (25:115 g) and placed in the enclosures

near mid-day for a period of 2-h, after which the pans were removed. Pans placed within the low-risk enclosures were covered with a thin layer of straw approximating the cover within the enclosure. After an initial week to allow the animals to locate the pans, data collection began. After each run, carrots and sawdust remaining in each pan were separated and weighed (to within 0.2 g). The ratio of carrot/sawdust weight remaining was used as the index of giving up density.

Runs during the first week of this experiment (mid-July 1990) consisted of two pans in each enclosure at Site 1 (for a total of eight pans per run). During the next two weeks of the experiment four pans per enclosure were used at both Site 1 and Site 2 (16 pans per run at each site). The data were analyzed separately for each site using the Mann-Whitney U test.

RESULTS

VEGETATION ABUNDANCE AND QUALITY

Examination of vegetation sample weights (Table 2.1) suggested little difference across treatments on any sampling date. I confirmed homogeneity of variance for each date (Bartlett's test: P = 0.79, 0.21, 0.59, respectively), and then employed nested ANOVA, which did not indicate a treatment effect (Table 2.1). Power analysis (Zar 1984) performed on the data to determine the minimum detectable difference between pooled treatment means for α=0.05 resulted in values of 4.3g, 8.5g, and 8.4g for July 8, July 24, and August 22, respectively, indicating that any substantial difference in vegetative production would have been detected.

Table 2.1. Results of vegetation sampling. Dried weight (g/30cm^2) of vegetation samples by treatment for each sample date

	HIGH RISK TREATMENT (NO STRAW)			LOW RISK TREATMENT (STRAW)			
EXCLOSURE	MEAN	SE	N	MEAN	SE	N	ANOVA P
			July 8				
a	82.4	(9.0)	5	75.2	(4.6)	5	
b	98.3	(7.3)	5	103.8	(7.4)	5	
c	98.0	(7.7)	5	104.4	(5.4)	5	
d	100.8	(10.7)	5	83.9	(5.6)	5	
pooled	94.9	(4.4)	20	91.8	(4.0)	20	0.561
			July 24				
a	97.8	(5.4)	5	101.8	(8.8)	5	
b	115.4	(7.9)	5	128.0	(15.2)	5	
pooled	106.6	(5.7)	10	114.9	(9.4)	10	0.436
			Aug 22				
c	136.2	(7.3)	5	131.2	(5.3)	5	
d	134.8	(8.8)	5	149.3	(14.7)	5	
pooled	135.5	(5.4)	10	140.3	(8.0)	10	0.630

ANOVA analysis of nitrogen and carbon content of the vegetation samples, following a Bartlett's test for homogeneity of group variances, also showed no treatment effect ($P = 0.834$ and 0.687 for carbon and nitrogen, respectively). Thus, it would appear that the straw cover treatment at the level applied here had no effect on either the quantity or quality of vegetation available to the voles.

DEPLETION EXPERIMENT

The duration of 389 searches for food in the GUD pans was measured in the depletion experiment. Searches tended to be longer at low food density (Table 2.2) with a significant difference in search times across the three categories of food density (Kruskal-Wallis $P < 0.001$). Comparison of search times between pairs of density categories using Mann-Whitney tests indicated significant differences between search times at high density food compared with medium and low density, but not between medium and low density.

GUD EXPERIMENT

Giving up densities at both sites (Figure 2.1) were significantly lower in enclosures treated with straw cover. That is, in enclosures with straw cover voles foraged in the food pans until food was further depleted and search times were longer than where they had no straw cover. This justifies the assumption that the voles perceived the enclosures with cover to be less risky than those without.

Table 2.2. Results of the depletion experiment. Search time (s) for food at three food densities (g). (Kruskal-Wallis P < 0.001)

FOOD DENSITY	N	SEARCH TIME MEAN*	(SE)	Median
0-5	158	13.3 a	(1.1)	9.1
10-15	64	11.9 a	(1.5)	7.6
20-25	167	6.0 b	(0.5)	3.6

*Means followed by the same letter are not significantly different (i.e., pairwise Mann-Whitney U probability > 0.05).

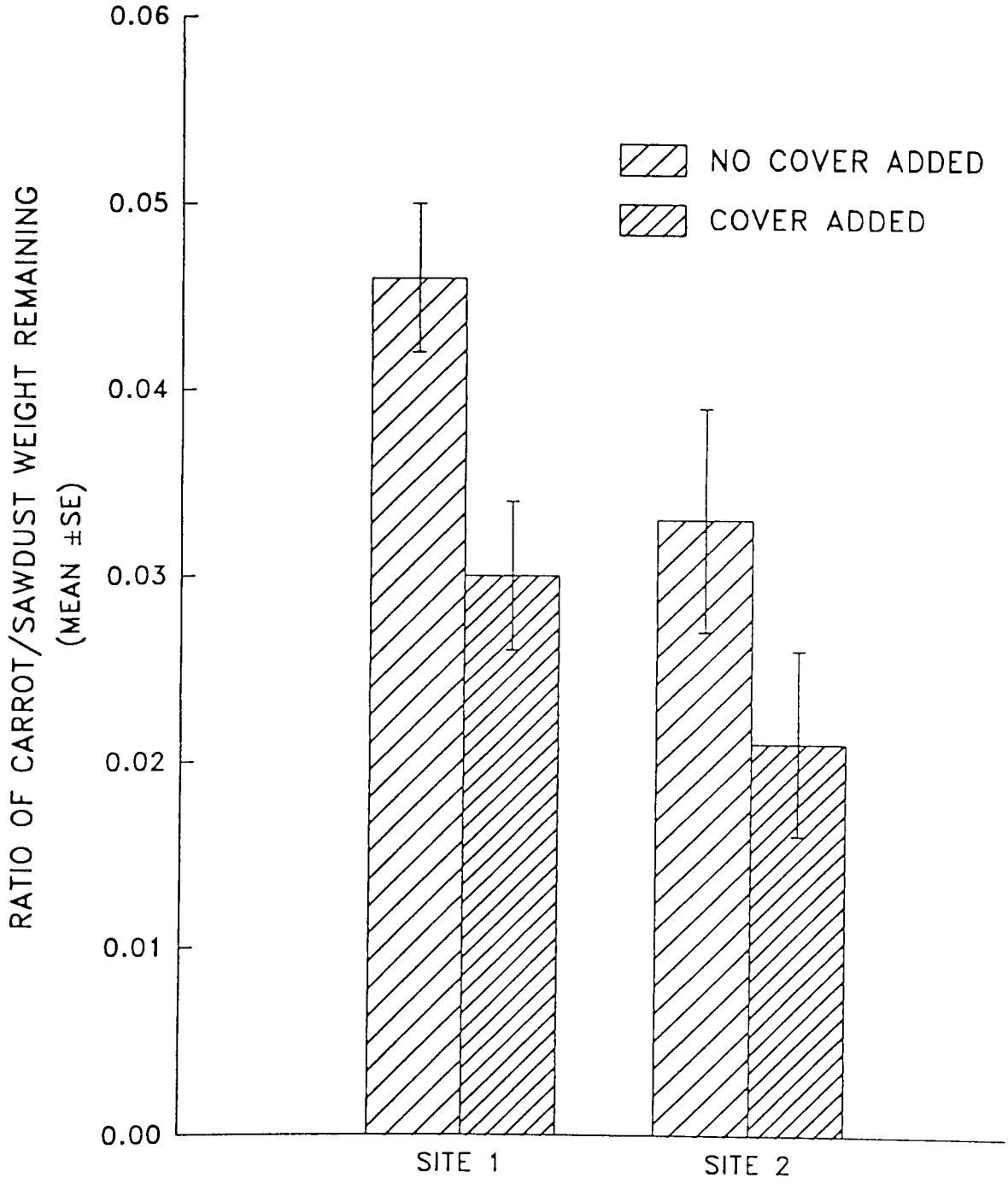

Fig. 2.1. Giving up density (GUD) in food pans in high and low risk treatments. Site 1: $N_1=N_2=100$; Site 2: $N_1=N_2=55$, Mann-Whitney P=0.003, 0.019, respectively.

VOLE MORTALITY

I attempted to manipulate vole assessment of predation risk while discouraging actual predation from occurring by means of the predator-exclusion fencing and vertical stakes. To determine if, in spite of these efforts, actual predation might have been higher in the 'high-risk' treatment, I calculated the mortality rate of voles in both treatments at Site 1 and at Site 2 (Table 2.3). There was no significant difference in mortality rates across risk treatments at either site, confirming that the actual predation risk for voles was unaltered by the risk treatments even though their perceptions of risk were altered, as the GUD experiment demonstrated.

Table 2.3. The effect of risk treatment on mortality of adult females. Mean (SE) 2-week mortality rate and ANOVA probability at each site by risk treatment.

		TREATMENT		
LOCATION	N	High Risk (No straw)	Low Risk (Straw)	P
Site 1	14	.04 (.02)	.11 (.03)	.96 (NS)
Site 2	6	.20 (.14)	.14 (.06)	.35 (NS)

ANOVA of mortality rate on risk treatment with enclosures nested within risk treatment at each site. 1-Tail probability with H_a: high-risk mortality rate > low-risk mortality rate.

EFFECT OF PERCEIVED PREDATION RISK

METHODS

REPRODUCTION

Live-trapping was conducted within the enclosures in 1990 on a bi-weekly basis, for three purposes: firstly, to provide an on-going check on the density of adult voles in each enclosure; secondly, to provide a basis for establishing the minimum number of adult females alive for calculating reproductive output at the end of the experiment, and finally, to provide data on the number and weight of juveniles reaching trappable age and allow for their release into the wild. These data allowed me to test for the effect of perceived predation risk on reproduction by comparing the production of trappable juveniles per female in the high and low predation risk treatments while insuring that vole density did not vary between treatments.

Traps were set out in a 2 m grid pattern (49 traps per enclosure) at the beginning of the season and left locked open. Traps were pre-baited with a few slices of fresh carrot the evening before each bi-weekly trap session. These carrots were almost invariably gone at dawn the next morning when the traps were baited again and armed. Throughout the trapping day the traps were checked approximately every hour and captured voles were removed to a nearby processing station where they were individually identified, weighed, and examined for reproductive condition. Captured adults were held individually in covered buckets supplied with grass and carrots until trapping was complete (so that they would not be recaptured the same day), then each adult was released at

its capture location. Voles born in the enclosures (juveniles) were immediately released to the wild after processing.

When trapping was complete, I tallied the number of adults accounted for in each enclosure, and added adult replacements when necessary to maintain the initial density and sex ratio. In general, a fresh caught replacement was added when one of the experimental adults was not trapped on two successive trapping sessions.

When all trapping sessions for the field season were completed, I produced a final estimate of the number of adult females alive for each trap date by summing the number of females trapped on a given date plus the number of females not trapped but known to be alive because they were trapped subsequently. Females were only included in the count up to the last date on which they were trapped. Thus, for the purposes of subsequent analysis, the number of females is arbitrarily assumed to change only in a step-wise fashion on a trap date.

The birth date of each juvenile was estimated by back-dating from its trap date using its weight when trapped, and an assumed birth weight and daily growth rate (3.0 g and 0.85 g/day, respectively; Morrison et al. 1977). I used these putative birth dates to estimate the number of juveniles born in each period which subsequently reached trappable age (approx 2-3 weeks). Although the method of back-dating should be reasonably accurate, the precise birth date is not critical because the estimate is used only to place the birth within the appropriate two-week period between trapping sessions, not to identify the specific day of birth.

Dividing the number of juveniles born in each 2-week period by the number of females alive during that period produced the index used as

the measure of reproduction, the number of successful juveniles born per adult female for each trapping period. The reproductive data thus calculated were compared across treatments for each site.

ACTIVITY AND FORAGING

I used three different methods to measure activity, one aimed at measuring movement per se (electronic detectors) and two intended to be more specific measures of food consumption (fecal plates and marked grass stems).

These experiments were conducted in 1991 at Site 1. Because the vegetation in the enclosures had been somewhat degraded by experiments in previous years and by over-winter foraging by voles, I made efforts to return the enclosures to their original state of homogeneous grass cover before re-introducing voles.

In early April, before vegetative growth started, all voles remaining in the enclosures from previous experiments were live-trapped and released to the wild. At that time I began preparation for the experiments by the burning dead vegetation and old straw in situ. Subsequently I removed thistles (Cirsium spp.) manually and then applied a common 2,4-D based lawn herbicide (Later's Weed-B-Gon, TM) on May 10. Reed canary grass was re-planted (May 15) where it was slow in developing (approximately equal amounts of seed being required in each enclosure), and all four enclosures were treated with a light application of 16-20-0 fertilizer (3 kg per enclosure) on May 30. These efforts resulted in an even growth of grass throughout all the enclosures, resembling that of the previous year.

Vegetation inside the enclosures was maintained at a height of 60 cm with a weedeater during the entire field season in order to make it easier to perform the experiments without altering the cover or breaking vegetation. Aside from these manipulations of the vegetation, the enclosures were prepared in the same manner as for the experiments on reproduction (same predator risk treatment, nestboxes, predator stakes, electric fencing, etc.)

Only wild-caught female voles were used to stock the enclosures. I took this decision because if the voles had been allowed to reproduce, I would have expected more offspring in the low-risk treatments (based on the 1990 experiments). The presence of these additional offspring might have produced the appearance of increased activity, and thus biased the results toward the hypothesis. Ten adult females were stocked in each enclosure on June 29, matched by weight across enclosures. During the activity experiments vole density in the enclosures was monitored by live-trapping (Aug 14, Aug 25).

To measure vole movement, I designed an electronic detector/counter which sensed whenever an object blocked the 30 cm path of its infra-red beam. Thus, a vole was counted whenever it crossed the beam path of one of the detectors. The detector was designed to trigger only when its beam was blocked for at least 0.2 s, to reduce the likelihood of it triggering when large insects or wind-blown vegetation crossed its path, and once triggered to remain insensitive for about 0.3 s to avoid multiple counts if a vole happened to move down the length of its beam. The beam was positioned 2.0 cm above the ground. Because voles were the only vertebrates in the enclosures, and because the detectors were designed to ignore extraneous environmental noise, the

detector counts are an accurate measure of the number of times voles crossed the detector beam in each observation period. I built 32 of these units for use in the field.

The detectors were placed in the field in two different configurations in successive phases of the experiment. Initially (Aug 22-Aug 27) a 4-m grid pattern was employed using a total of 32 detectors, 8 per enclosure. In the second phase of the experiment (Aug 27-Sept 1) detectors were placed at the 7 locations in each enclosure identified as the most active vole sites by analysis of pellet numbers on the fecal plates (see below). In order to make the detector readings as independent as possible, no detector was placed within four meters of another. Data collected from these two detector configurations were analyzed separately.

During both phases of the experiment, detector counts were recorded manually at seven times each day (0530, 0630, 0845, 1300, 1700, 1830, 2030 h). Counts were converted to an average rate per hour in each enclosure for analysis (two enclosures high risk and two enclosures low risk). Grid pattern data was collected over 12 observation periods while most active location data was collected over 32 observation periods. Log-transformed rates were compared across treatments using ANOVA (nested by enclosure) on the most active location data and the simpler non-parametric Friedman's rank test on the grid data, where there was clearly no difference between the two treatments.

To obtain a measure of activity focused specifically on food intake, I set out small wooden plates (7.5 cm square) in the enclosures which could be used to obtain counts of both fecal pellets and grass blades dropped by the voles. I placed 49 fecal plates in a 7x7 grid pattern (2 m

spacing) in each of the enclosures and recorded data twice daily (0700, 1900 hr) from Aug 16 to Sept 1. I counted the numbers of fecal pellets and blades of green, freshly cut grass on each plate, scraped it clean and replaced it in position.

Data was collected over 24 observation periods in two enclosures for each of the risk treatments. For each observation period, I calculated: the proportion of plates with fecal pellets; the average number of fecal pellets per plate; the proportion of plates with fresh blades of grass; and the average number of blades per plate in each enclosure. I compared each of these variables across treatments using nested ANOVA.

In another approach to measuring foraging intensity I marked grass stems in each enclosure so that the proportion of marked stems which were later grazed could be compared across treatments. One healthy stem within the vicinity of each fecal plate was marked with a small piece of flagging tape about 45 cm above the ground. Thus, 49 stems were marked in each of the four enclosures on Aug 16. Stems which had been cut by foraging voles were noted as the fecal plates were read, and a final census of ungrazed and grazed stems was carried out on Sept 1.

RESULTS

REPRODUCTION

In total, more juveniles were captured in the low-risk treatments than in the high-risk treatments at each site (Figure 2.2). More

importantly, the reproductive rate (juveniles per female per trap period) in the low risk treatments statistically exceeded that in the high risk treatments when the data for both sites were pooled (Figure 2.3) . When the data for each site are analyzed separately, only Site 1 shows a highly significant difference (Figure 2.4). Examination of the data for each trap period shows that the number of juveniles produced per female was higher in the low-risk treatments in 8 of the 10 trapping periods (Table 2.4)

Had the probability of vole re-capture differed between treatments, the results presented here might have been biased. To check for this possibility, at the end of the field season I calculated the probability of re-capture as the number of captures divided by the number of voles known to be alive for each treatment. The probability of capture in the low and high risk treatments was nearly identical (95.0% and 98.3%, respectively).

ACTIVITY AND FORAGING

The period during which data on activity were obtained by any of the three techniques extended from Aug 16 to Sept 1. Although the density of voles declined somewhat during this period (Table 2.5) the average density was the same for each treatment. I judged the differences to be minor and made no adjustments to the data.

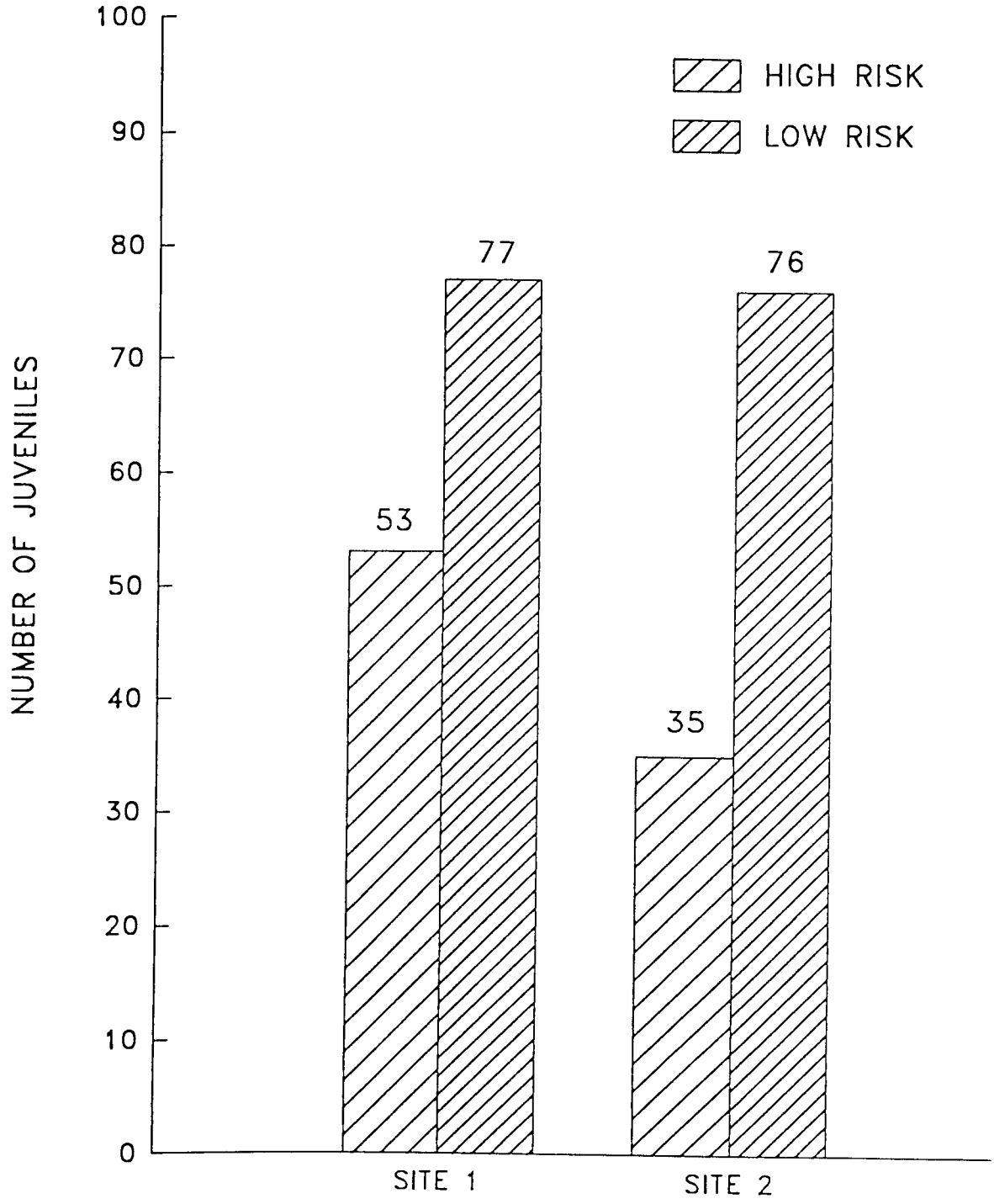

Fig. 2.2. Total number of juveniles captured in the high and low risk treatments at each site.

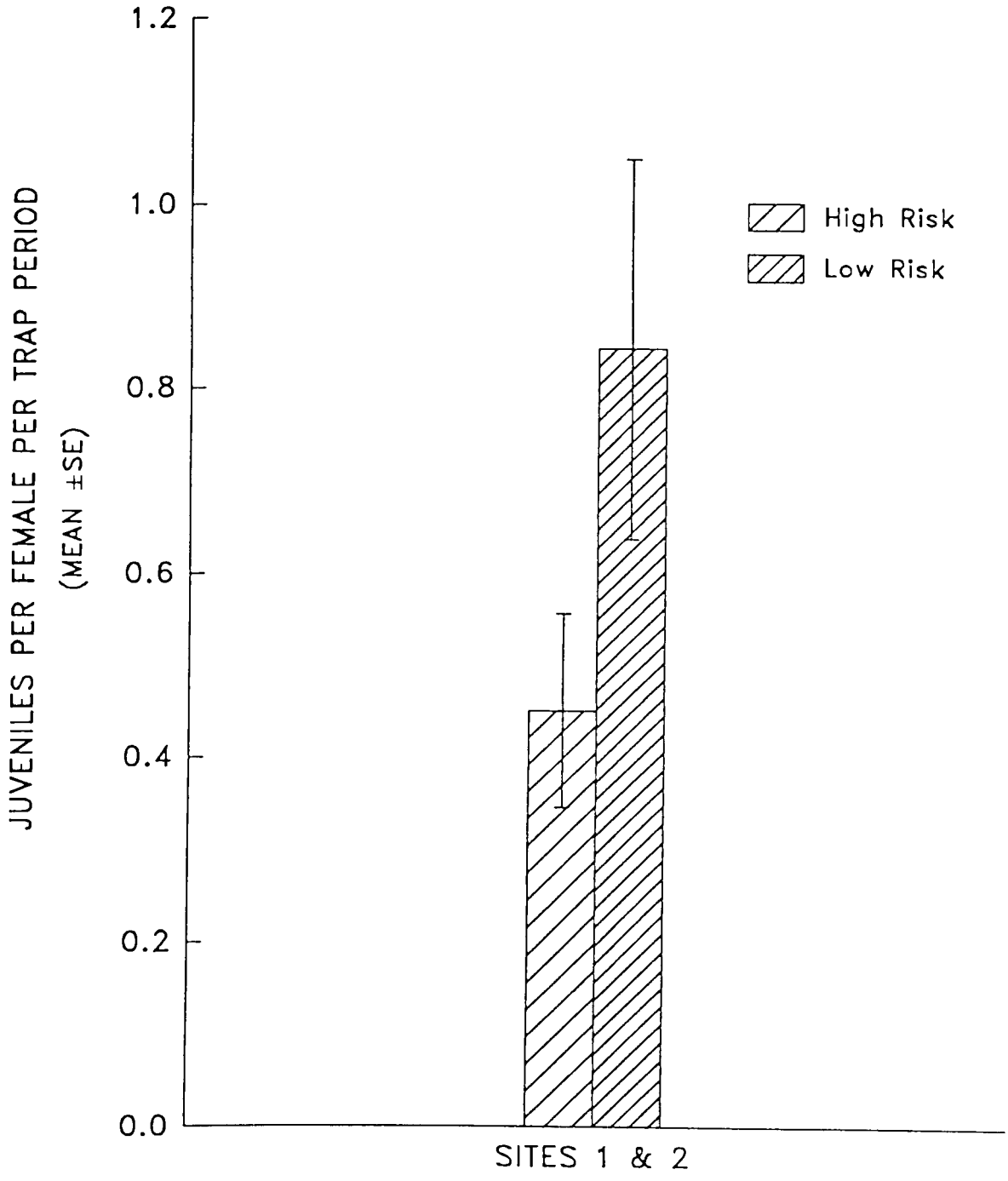

Fig. 2.3. Reproductive rate at high and low predation risk. Number of juveniles per female averaged across trapping periods (± one standard error). Site 1 and Site 2 pooled, 10 trapping periods in total. Repeated measures anova P=0.018 (1-tailed).

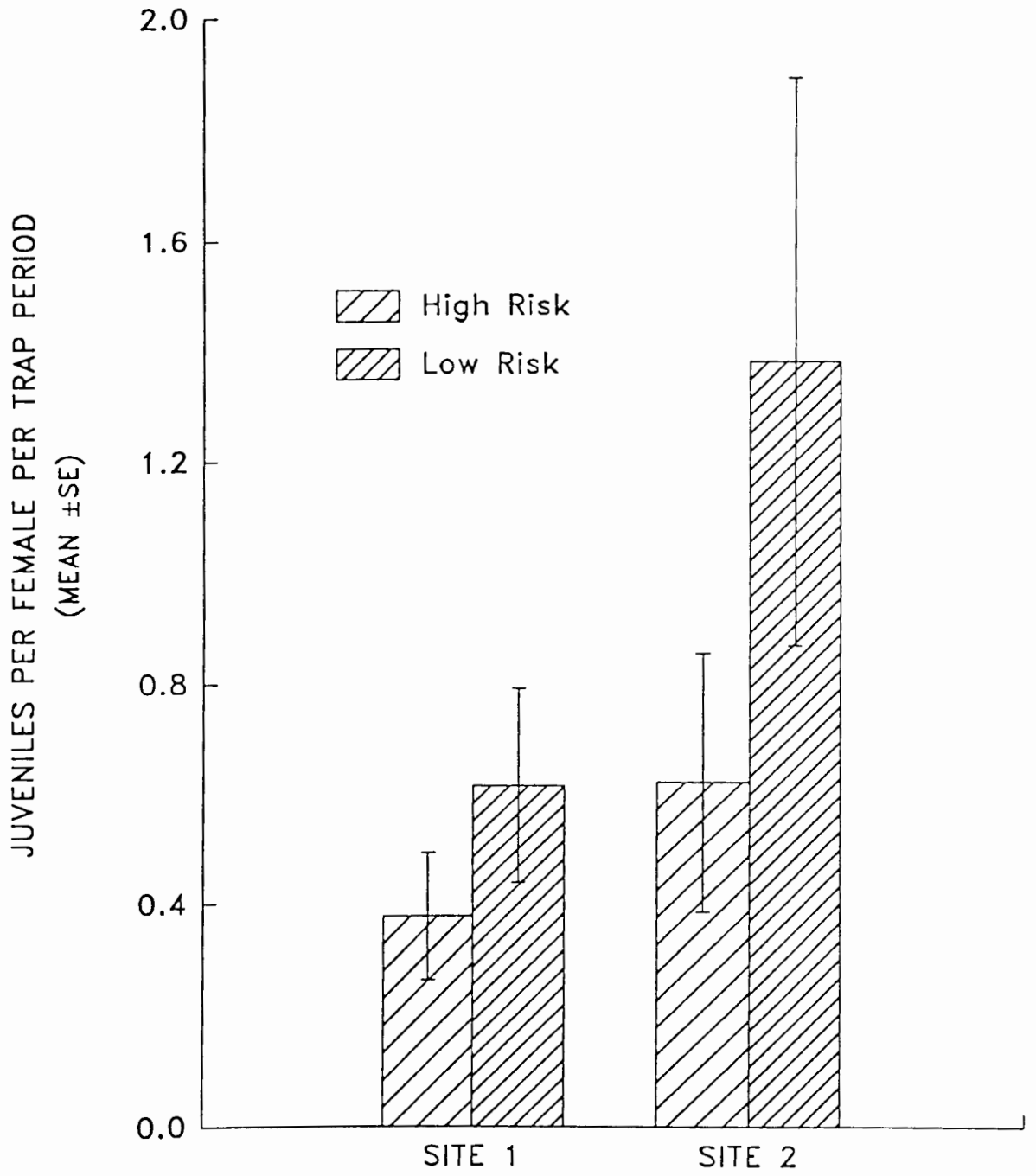

Fig. 2.4. Reproductive rate at high and low predation risk for the two sites separately. Number of juveniles per female averaged across trapping periods (± one standard error). Site 1 and Site 2, 7 and 3 trapping periods respectively. Repeated measures anova P=0.017, 0.125 (1-tailed) respectively.

Table 2.4. Average number (SE) of juveniles born per female in each trapping period which survived to trappable age. Two replicates per treatment at each site.

	SITE 1		SITE 2	
Trapping Period	High Risk	Low Risk	High Risk	Low Risk
1				
2	0.2 (0.1) <	0.4 (0.1)	---	---
3	1.2 (0.4) <	1.8 (0.4)	---	---
4	0.3 (0.3) <	0.4 (0.3)	---	---
5	0.5 (0.0) <	0.9 (0.5)	---	---
6	0.3 (0.0) <	0.6 (0.3)	1.1 (0.5) <	2.8 (0.6)
7	0.1 (0.1) =	0.1 (0.1)	0.4 (0.3) <	0.9 (0.4)
8	0.0 -- <	0.1 (0.1)	0.4 (0.4) >	0.3 (0.3)
Mean	0.4	0.6	0.6	1.3

Table 2.5. Vole density during activity experiments at Site 1. Number of voles known to be alive in each enclosure.

	June 29	Aug 14	Aug 25
High risk			
Enclosure 1	10	9	9
Enclosure 4	10	8	7
Low risk			
Enclosure 2	10	9	8
Enclosure 3	10	8	8

The average rate of electronic detector counts on the grid pattern (Table 2.6) differed only slightly and non-significantly between treatments. However, detectors placed at the most active locations do indicate a significant difference between treatments ($P < 0.001$): voles in the low-risk treatments triggered the detectors more than twice as often (Table 2.6).

The proportions of plates containing fecal pellets and fresh grass blades were both significantly higher in the low risk treatments, suggesting that voles in that treatment moved about more extensively. In addition, both the number of fecal pellets per plate and the number of blades of grass per plate were significantly higher in the low-risk enclosures, supporting the idea that voles foraged more where they felt safer. However, although the proportion of marked stems grazed in the low-risk treatment was higher the difference was not significant (Table 2.6).

Table 2.6. Activity experiments. Mean (S.E.) of various activity indices in the high and low risk enclosures, and the statistical analyses.

ACTIVITY MEASURE	N	TREATMENT: HIGH RISK	TREATMENT: LOW RISK	P	METHOD
Detector Data					
Grid Pattern					
Average rate of counts	24	3.0 (0.4)	3.2 (0.4)	NS	Friedman's
Most Active Locations					
Average rates of counts	64	1.4 (0.2)	3.1 (0.3)	<.001	Nstd ANOVA
Fecal Plates					
Fecal Pellets					
Proportion of plates with fecal pellets	48	0.24 (0.02)	0.30 (0.01)	<.01	Nstd ANOVA**
No. fecal pellets/plate	48	0.74 (0.07)	1.08 (0.10)	<.01	Nstd ANOVA*
Blades of Green Grass					
Proportion of plates with green blades	48	0.12 (0.01)	0.17 (0.01)	<.01	Nstd ANOVA
No. blades/plate	48	0.68 (0.09)	1.36 (0.14)	<.001	Nstd ANOVA*
Marked Stems					
Proportion of stems grazed	98	0.18	0.26	NS	

*ANOVA data square root transformed for normality.

**ANOVA data arcsin transformed for normality.

DISCUSSION

This work has shown that it is possible to alter the predation risk which voles perceive (GUD experiment) without altering the quality or quantity of their food supply (vegetation sampling) or changing the actual level of predation (mortality data). Therefore, differences in reproduction, activity level, and foraging between the high and low risk treatments could not have been due to variation in either food availability, vole density, or direct predation, but must have resulted from facultative adjustments in behaviour made by the voles in response to differences in predation risk which they perceived.

Manipulation of vole assessment of predation risk is, of course, central to the arguments presented here. I have used giving up density (GUD) to provide a comparison of the perceived risk in the two risk treatments, but GUD may be sensitive not only to predation risk, but also to food availability in the enclosures. Any difference in the quantity or quality of vegetation between treatments might have reflected itself in a difference in the GUD measured in the food trays and been incorrectly interpreted as a difference in perceived predation risk. However, the results of the vegetation sampling clearly eliminate this possibility since neither vegetation quantity or quality differed between treatments.

There was also concern that the measures I took to prevent actual predation from occurring, i.e., excluding terrestrial predators and discouraging avian predators with vertical stakes, could have already created such a safe environment for the voles that additional cover would have no effect, but the results of the GUD experiments also eliminate this

possibility. The GUD experiment thus provides very strong evidence that the animals' assessment of predation risk differed across treatments.

The results of the reproduction experiment indicate that female voles which perceive their environment as relatively dangerous raise fewer offspring to independence (i.e., to trappable age). Because I was only able to count offspring after they were trapped, I cannot determine the mechanism which produced this result, except to argue that it is almost certainly a result of decisions made by the adult females. Thus, while the difference in offspring production may manifest itself either pre- or post-partum, this work indicates that it is intimately tied to the reproduction-mortality trade-off facing the adult female. It is not likely, for example, that the smaller number of juveniles trapped in the high risk treatments resulted from higher predation on juveniles there, since adults did not experience a higher predation rate in those enclosures.

Evidence exists that female bank voles (Clethrionomys glareolus) avoid copulating in the presence of mustelid odor and also reduce their acitivity level (Ylönen 1989, Ronkainen & Ylönen 1994). This change in reproductive behaviour has been interpreted as an antipredatory adaptation (Ylönen & Ronkainen 1994).

It is also possible that female voles make adjustments to litter size in utero. McShea and Madison (1989) found that there were a greater number of live embryos just before parturition than there were live births and that the differences were correlated with factors affecting the likelihood of offspring survival. However, their data were insufficient to distinguish between immediate pre- and post- partum mortality, and they argued that neonate mortality immediately following birth was most likely.

Females may reduce their litter post-partum in a number of ways, varying from reduced defense of the nest against conspecific adults, as Mallory & Brooks (1980) showed for collared lemmings (Dicrostonyx groenlandicus), to selective killing of the young as seen in meadow voles (McShea & Madison 1987). A third strategy has been observed in the laboratory; food-restricted female wood rats (Neotoma floridana) actively rejected their young male offspring, and physically removed them from the nest (McClure 1981).

Although the timing and nature of the events which led to reduction in offspring production in this study could not be determined, energetic considerations suggest that post-partum reduction may be an effective and flexible strategy. For voles, as with other mammals, lactation is considerably more expensive and less efficient than gestation (Glazier 1990). Female voles ingest significantly more food per day while lactating than while pregnant (Innes & Millar 1981) and presumably must endure a greater risk of predation mortality in order to obtain the extra food. Thus, parturition marks the time when a much greater commitment of reproductive effort is required, and it may also be an appropriate time for females to reduce litter size when predation risk is high.

Taken together, the results of the activity and fecal plate experiments are consistent with those of the reproduction experiments. Voles in the high-risk treatment moved about less, ate less, and carried less grass about. I would suggest that all three represent adjustments in behaviour to reduce the danger from predators, and that these results are closely linked, and probably causally related, to lowered reproductive output. Overall, these results provide experimental support for the

predicted facultative adjustment of reproductive investment under predation risk derived theoretically from life historical considerations in Chapter 1.

The present approach to examining the interaction of predation risk and reproduction appears to be unique. A more typical design involves controlling brood size and measuring its effect on some aspect of the parent's welfare, such as survival rate or body weight. A drawback of this approach is that a small change in mortality rate is difficult to measure with attainable sample sizes. It is important to note, however, that although a change in predation risk may have only a small effect on the mortality associated with reproduction, it may still be significant in terms of the life history strategy of the animal and may result in behavioural changes.

The present experiments have taken a different approach in that I controlled one aspect of the adults' environment, perceived predation risk, while measuring reproductive output. An advantage of this approach is that it is unnecessary to measure predation risk, only to establish that the animals' perception of risk differs across the treatments. This design allows the animals themselves to determine if a higher perceived predation risk is sufficient to alter their reproductive investment and provides an output variable which is relatively easy to measure.

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CHAPTER 3

THE POPULATION IMPLICATIONS OF OPTIMAL REPRODUCTIVE BEHAVIOUR

INTRODUCTION

While it has long been suspected that predation plays a key role in the fluctuating population dynamics of microtine rodents, no theoretical framework advanced to date has enjoyed a great measure of success. However, it is now recognized that the risk of predation, as opposed to predation per se, has powerful effects on the behaviours of animals in such diverse arenas as foraging, sociality, and reproductive strategies (reviewed by Lima and Dill 1990). In fact, predation risk may influence the litter size of small mammals if individuals are capable of facultatively adjusting their reproductive investment, as I demonstrated theoretically using a life history framework in Chapter 1. Field evidence indicates that such changes in reproductive effort occur quickly in response to differences in predation risk for the meadow vole, Microtus pennsylvanicus (Chapter 2).

It seems likely that adaptively flexible life history strategies would impact population dynamics, and that there must be an intimate link between population level phenomena and individual behaviour, with causal links running in both directions. The fitness contribution of a given reproductive output depends on population density and growth rate while these population variables are determined in large part by individual reproductive behaviours. Thus, it seems worthwhile to explore the implications of this life history model developed in Chapter 1 for the population dynamics of small mammals.

While several life history approaches have attempted to provide a theoretical perspective for explaining microtine population cycles

(Schaffer and Tamarin 1973, Stenseth 1978, Morris 1984), none has explicitly taken the risk of predation into account, nor have any of the existing models been shown to produce continuing fluctuations in population density in the absence of periodic environmental variation. As Morris (1984) recognized, life history models which generate cyclic population densities without relying on external perturbations to the system are likely to be more powerful in explaining microtine population dynamics than those which require external "kicks". Nevertheless, a life history approach remains appealing because such an attack on the problem might ultimately be used to derive the population consequences which result when individuals maximize lifetime reproductive success by optimizing such traits as age at first reproduction, offspring number and size, length of breeding season, winter breeding activity, or age at dispersal. In this chapter I extend the model of optimal reproduction developed in Chapter 1 to investigate the inter-relationship between optimal reproductive effort and population dynamics by concentrating on one aspect of life history, the optimal number of offspring for a female to bear and raise during each reproductive period.

THE MODEL

The population level consequences of optimal litter size decisions can be determined by modelling a population in which individuals produce offspring according to the optimality equation for ESS litter size developed in Chapter 1:

$$L_t = -S(L_t)/S'(L_t) - 1/f(N_t) \cdot 1/\lambda(L_t) \cdot RRV_{t+1} \qquad [3.1]$$

where:

L_t = litter size in reproductive period t,

$S(L_t)$ = adult survival probability, i.e., the probability of survival until the next time period for females with litter size L. The specific functional form used is given in Chapter 1,

$S'(L_t)$ = derivative of $S(L_t)$ with respect to L_t,

$f(N)$ = juvenile survival probability, i.e., the probability a juvenile will survive until the next time period. Note density dependence. The specific functional form used is given in Chapter 1

$\lambda(L_t)$ = N_{t+1}/N_t = population growth rate in period t,

RRV_{t+1} = residual reproductive value at the beginning of reproductive period t+1, as defined in Chapter 1.

The density of reproducing females at the beginning of the next reproductive period, N_{t+1}, is then given by the sum of the expected survivors and their surviving offspring, who are reproductive individuals in period t+1. In calculating the expected number of surviving offspring, I assume that the survival of the mother is essential to juvenile survival:

$$N_{t+1} = S(L_t) \cdot N_t + L_t \cdot N_t \cdot S(L_t) \cdot f(N_t) \qquad [3.2]$$

Eqns [3.1] and [3.2] neglect some factors which may affect the population dynamics of any particular species. For example, age-specific

effects are taken into account only insofar as adults are distinguished from neonates. Secondly, according to Eqn [3.2] neonates never reproduce, and all surviving adults reproduce identically, regardless of age. Since the age of first reproduction is known to vary within many species of microtines (Krebs and Myers 1974), it would be more realistic to allow at least two adult reproductive tactics (representing decisions to reproduce and to forego reproduction) to co-occur in the model. However, concern that the essential aspects of the ideas be clearly presented has led me to employ the simplification represented in the equations given.

EQUILIBRIUM POPULATION DENSITY AND DYNAMIC STABILITY

Note that Eqn [3.2] may be reformulated as:

$$N_{t+1} = S(L_t)\cdot\{1 + L_t\cdot f(N_t)\}\cdot N_t = \lambda_t\cdot N_t \qquad [3.3]$$

where $\lambda_t = S(L_t)\cdot\{1 + L_t\cdot f(N_t)\}$ [3.4]

At the equilibrium population density $N_{t+1} = N_t = N_{eq}$ and $\lambda = 1$ by definition. Therefore, from Eqn [3.4]:

$$f(N_{eq}) = \{1-S(L_{eq})\} / \{S(L_{eq})L_{eq}\} \quad [3.5]$$

or

$$N_{eq} = f^{-1} [\{1-S(L_{eq})\} / \{S(L_{eq})L_{eq}\}] \quad [3.6]$$

Note: f^{-1} indicates an inverse function (i.e., if $y=f(x)$ then $x=f^{-1}(y)$).

Eqn [3.6] shows that equilibrium population density is determined by litter size at the equilibrium point, given the juvenile and adult survival schedules (f and S, respectively), but it does not provide an explicit solution for N_{eq}. One might have hoped that substituting the expression for litter size (Eqn [3.1]) into Eqn [3.6] would cause L_t to drop out of the equation and result in an expression for N_{eq} in terms of the underlying parameters which determine the specific shapes of S and f. However, no such explicit solution is possible when litter size follows an ESS strategy such as Eqn [3.1]. N_{eq} and L_{eq} are jointly determined, a fact which underscores the fundamental link between individual and population level phenomena. An iterative procedure similar to one described below is required to find L_{eq} and N_{eq}.

N_{eq} does not necessarily represent a point equilibrium--that is, density may not stabilize at N_{eq}, but may oscillate around N_{eq} in stable

limit cycles (May 1973). In fact a wide range of population trajectories may result from Eqn [3.2], ranging from dynamically stable through complex limit cycles and chaotic behaviour.

The conditions necessary for point stability of the equilibrium population density in difference equation models have been discussed by others (May 1974, Clark 1976). Point stability requires that:

[3.7]

A geometric interpretation of this condition can be based on the fact that in the neighborhood of a stable equilibrium point the population growth rate, λ must be inversely related to density, for otherwise slight perturbations from the equilibrium density will result in run-away conditions. However, if the inverse relationship is too strong, overcompensation (Clark 1976) results and any small perturbation from equilibrium density will cause the system to overshoot the equilibrium in the opposite direction and generate a continuously fluctuating population density even in the absence of further environmental fluctuations. Thus, the slope of N_{t+1} on N_t must fall within the range specified by Eqn [3.7] in order for density to stabilize at an equilibrium point.

The stability condition can be made specific to the present model by substituting [3.2] and [3.6] into Eqn [3.7]. This results in:

$$-1 < N_{eq} \cdot S(L_{eq}) \cdot L_{eq} \cdot f'(N_{eq}) + 1 < 1 \qquad [3.8]$$

The central role played by reproductive effort in determining the nature of the population's dynamics can be seen by noting that each of

the variables in this expression as well as that for equilibrium population density (Eqn [3.6]) is itself a function of equilibrium litter size.

RESIDUAL REPRODUCTIVE VALUE IN THE MODEL

While the discussion above illustrates the central role facultatively adjustable reproductive strategies play in determining population dynamics, further elaboration of the performance of a population composed of such individuals requires a closer examination of the expression for optimal litter size, Eqn [3.1].

A conceptual problem arises when the meaning of the residual reproductive value term (RRV_{t+1}) in Eqn [3.1] is considered. The problem is that the expression for residual reproductive value includes the individual's fecundity at each reproductive period in its future, L_{t+1}, L_{t+2}, ..., but Eqn [3.1] is intended to determine optimal fecundity at each point in time; that is, the very values the equation is to determine are necessary for its solution. The individual cannot decide how much to invest in the present without knowing the value of its potential future reproduction, but each of those values in turn depends on a future beyond, and so on.

The broader issue is the question as to what meaning might be attached to the concept of residual reproductive value when fecundity and mortality are to a large extent under the control of the individual. This problem is central to life history theory and it can best be understood when contrasted with the situation arising when both fecundity and mortality schedules are assumed to be fixed.

If fecundity and mortality are fixed, residual reproductive value, calculated as the discounted sum of expected reproductive success over

the remaining lifespan of the individual, allows a straightforward allocation of reproductive effort between the present and the future. However, if adult survival and fecundity are not fixed but are in part the result of optimal decisions taken by individuals, as the current model assumes, then RRV depends on reproductive decisions as yet unmade.

The key to solving this dilemma lies in the recognition that reproductive effort in all relevant time periods must be simultaneously determined. It is incorrect to assume that an optimization process should place primacy on either current reproduction or potential future reproduction, because neither is necessarily more valuable than the other.

The introduction of facultative rather than obligatory reproductive strategies requires that residual reproductive value be viewed not as a fixed potential inherent in the biology of the animal, which serves as a determinant of reproductive allocation, but as a result of the decisions of the individual. Residual reproductive value must be determined in the process of allocation, not used as a determinant of that allocation. I provide a practical approach to actually solving the mathematical problem in the following section.

SOLUTION TO THE MODEL

Given these considerations for residual reproductive value, the model is mathematically complete. Eqn [3.1] for L_t and Eqn [3.2] for N_{t+1} along with the survival functions, $S(L_t)$ and $f(N_t)$ (specified in Chapter 1) are sufficient to determine the population trajectory through time (N_{t+1}, N_{t+2}, N_{t+3}, ...) and the succession of ESS litter sizes through time (L_t, L_{t+1}, L_{t+2}, ...). Although it is not possible to solve the equations explicitly for L_t

and N_t, I have developed an iterative approach which generally provides ESS litter size to any degree of accuracy desired.

The method of solving the model is described in detail in Appendix 3.A; it essentially involves truncating the expression for RRV_{t+1} in Eqn [3.1] at t+T, defining an initial set of guesses for litter size in each period from L_t to L_{t+T}, estimating the error involved in the original set of guesses, and correcting the initial guess until some accuracy criteria is reached.

The specific trajectories for N_t and L_t which result from this solution procedure vary with predation risk, R, and with the parameters which determine the slope and shape of the adult survival curve (m and c) and the juvenile survival curve (b and $N_{.5}$). In the case of chaotic dynamics the trajectories also vary with the initial population density, N_t.

RESULTS OF THE MODEL

As well as the course of population density through time, the iterative process described in Appendix 3.A can be used to compute the phase plot of N_{t+1} vs. N_t. These calculations are presented for three levels of predation risk in Figure 3.1. The striking effect of the differences between these curves is illustrated in Figure 3.2, where the trajectories of population density and litter size through time corresponding to the phase curves are shown. The same parameter values representing physiological costs (m and c) were used in each of these plots, and are representative of the meadow vole,

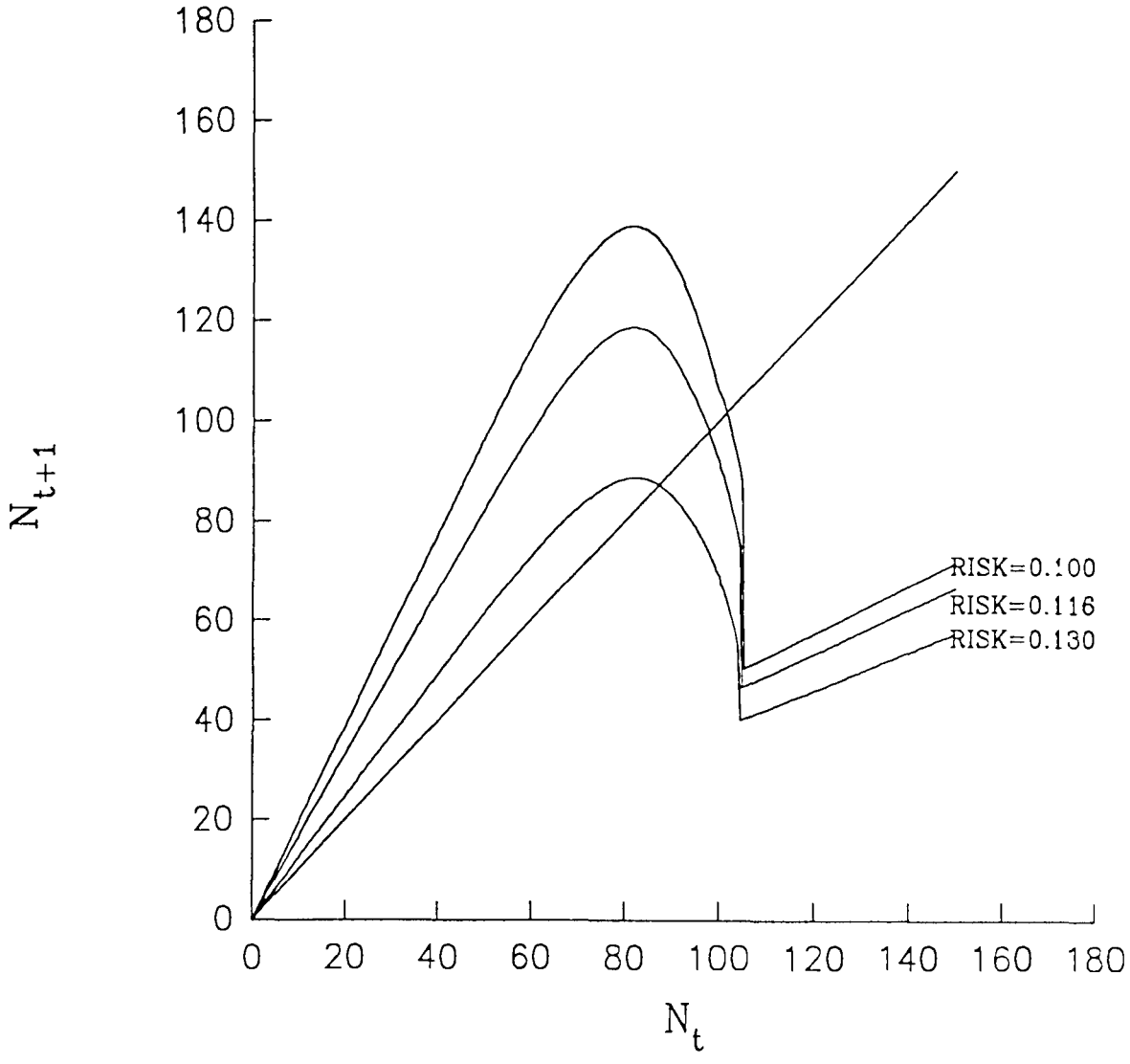

Fig. 3.1. Phase plot of N_{t+1} vs. N_t for predation risk per offspring (R·c) values of 0.100, 0.116, 0.130. At higher risk, both the maximum population density attainable and the equilibrium density (point where $N_{t+1} = N_t$) are lower. Higher risk stabilizes the population because the slope $\partial N_{t+1}/\partial N_t$ evaluated at equilibrium density, N_{eq}, is lower. Parameter values: b=.010, $N_{.5}$=100, m=763 Kcal, c=103 $K\chi\alpha\lambda$.

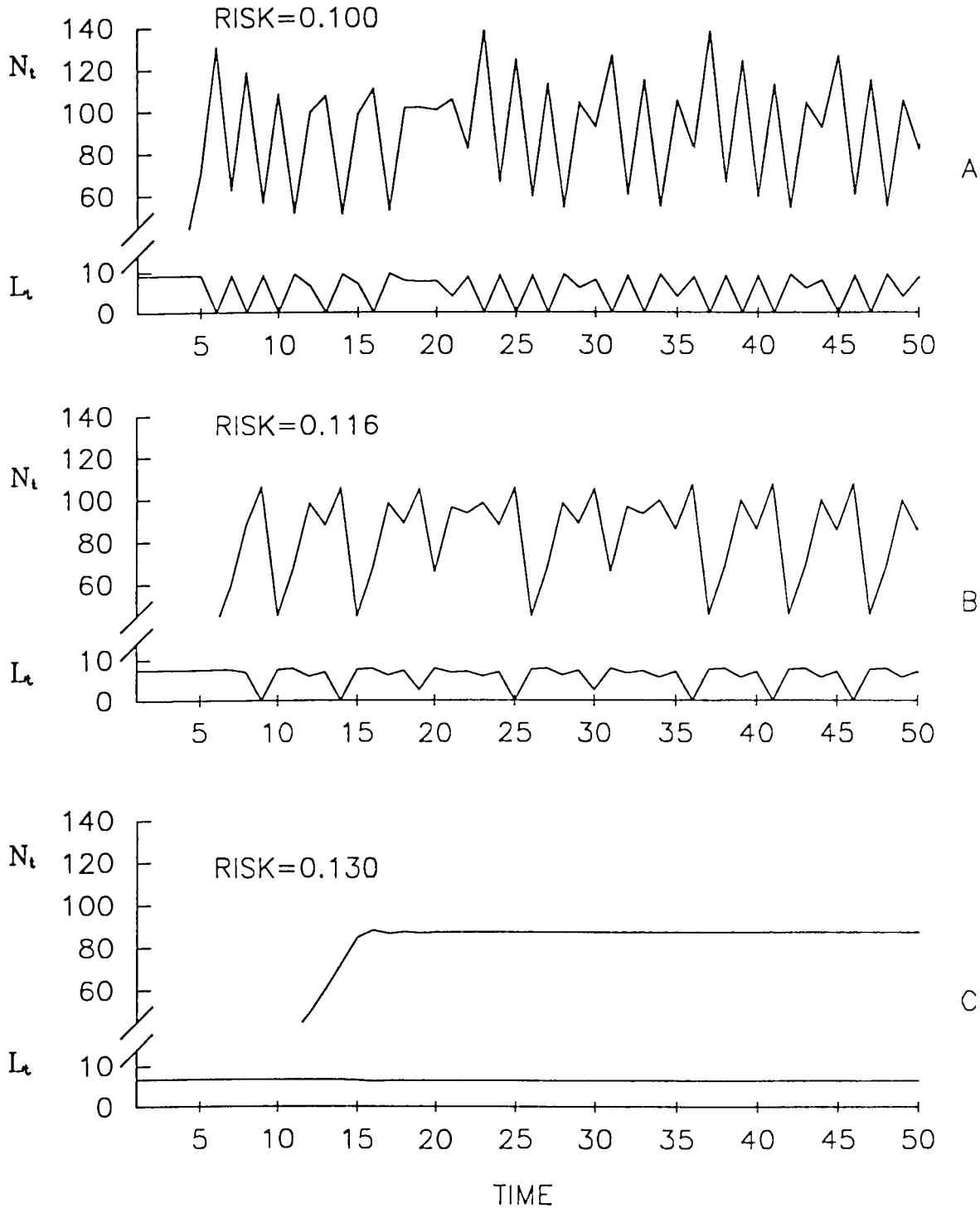

Fig. 3.2. Trajectories of N_t and ESS litter size through time for the phase curves in Fig. 3.1. a) Risk = 0.010, b) Risk = 0.116, c) Risk = 0.130. Other parameters as in Fig. 3.1.

Microtus pennsylvanicus (Innes and Millar, 1981). The same organism pursuing an optimal reproductive strategy can exhibit population dynamics which range from chaotic (Figure 3.2a) through limit cycles (b), and stable-point equilibria (c), as a result of litter size decisions made in response to the level of predation risk.

While the time trajectories help to visualize the population, the phase plot is of particular interest. The equilibrium population density, N_{eq}, where each phase curve crosses the 45° line is important for two reasons: firstly, the dynamic stability of the population is determined by the slope of the phase curve at N_{eq} (see Eqn [3.7]), and secondly, this equilibrium population density provides a point of reference for comparing the performance of the model under different assumptions.

For example, the theoretical relationship between litter size at N_{eq} and predation risk is shown for two vole species in Figure 3.3. The parameter values used are typical of M. pennsylvanicus and Clethrionomys gapperi, with the former having a higher maintenance requirement. My point here is not to compare the two vole species but to show the consequences for the performance of the model using different parameter values representative of microtines. Note that for both species equilibrium litter size tends to vary inversely with risk. Secondly, equilibrium litter size differs for the two species.

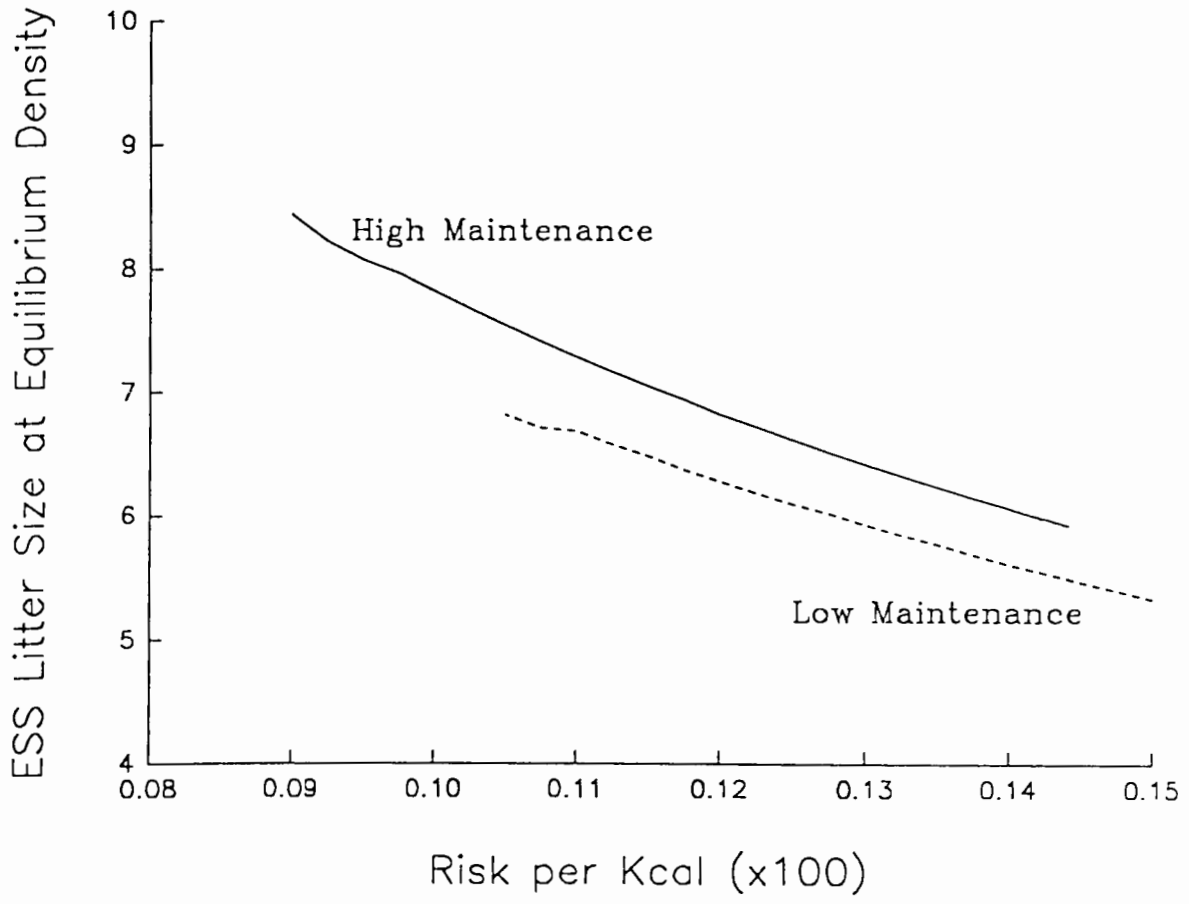

Fig. 3.3. The effect of predation risk on ESS litter size at equilibrium population density. Two species with differing maintenance requirements: M. pennsylvanicus (m = 763 Kcal, c = 103 Kcal) and C. gapperi (m = 589.8 Kcal, c = 109.8 Kcal) (Innes and Millar, 1981). Other parameters: b=.010, $N_{.5}$=100.

Interestingly, the predicted difference in litter size arises not because of a difference in the cost of each offspring, which is approximately the same (100 Kcal) for both species, but because the maintenance requirement for adults differs. The direction of the difference in litter size at first appears counterintuitive. One might expect that the species with the lower maintenance requirement would produce a larger litter because it could do so without incurring as great a risk. However, the predicted difference stems from the fact that the additional maintenance requirement of M. pennsylvanicus and its consequently greater "baseline" risk decreases its residual reproductive value and results in a greater emphasis on current reproduction (see Eqn [3.1]).

The litter size at N_{eq} for a given predation risk, as determined from Figure 3.3, can be used to examine other aspects of the model's performance. In Figure 3.4, equilibrium population density is graphed against litter size at N_{eq} for the two species. For any given litter size at N_{eq}, C. gapperi has a higher equilibrium population density because of its lower maintenance requirement and associated lower predation risk.

Litter size at N_{eq} also establishes the stability of the population (Figure 3.5). Whenever litter size at equilibrium is such that $\partial N_{t+1}/\partial N_t$ is greater than -1 (i.e., above the X-axis in Figure 3.5) the population is point-stable. When $\partial N_{t+1}/\partial N_t$ falls below the X-axis the dynamics first exhibit limit

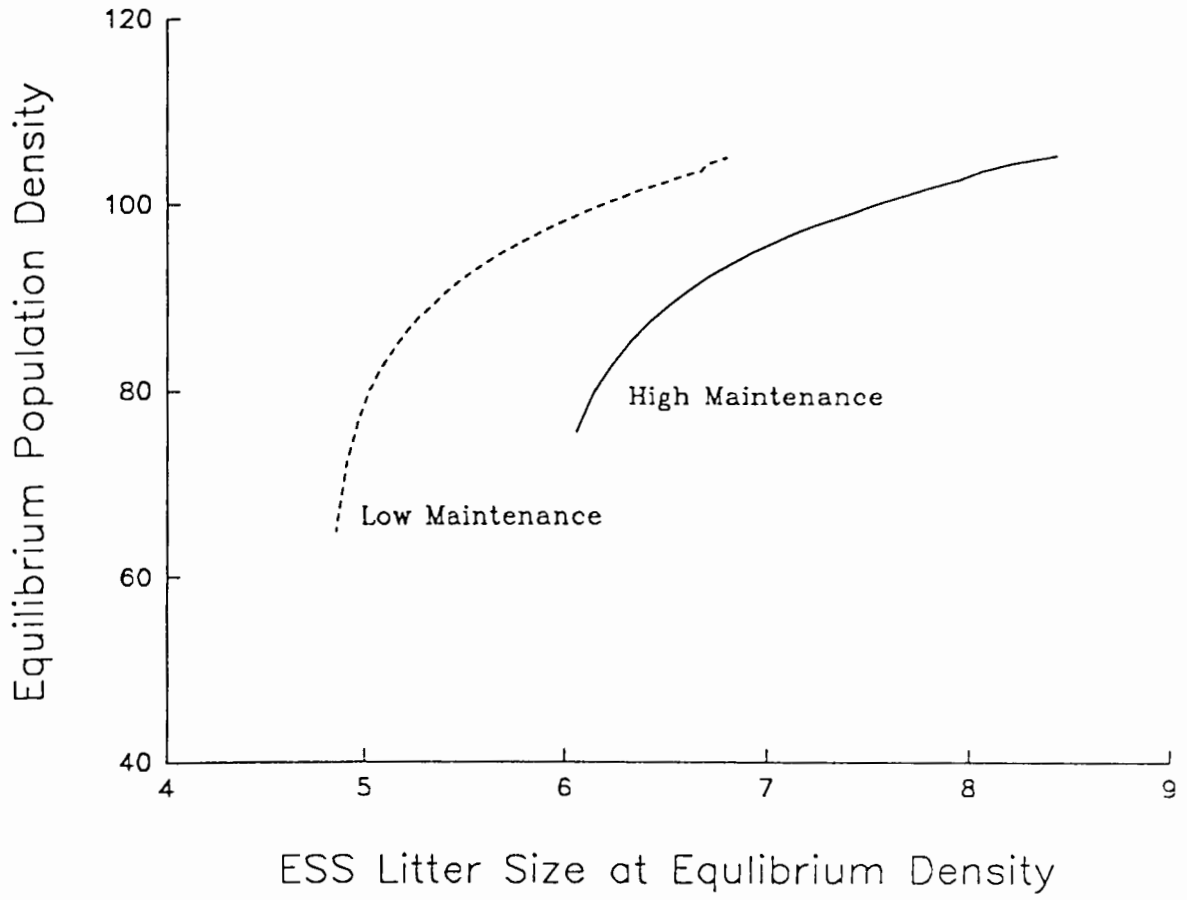

Fig. 3.4. Relationship between equilibrium population density and litter size at equilibrium. For two species with different maintenance requirements (same parameters as in Fig. 3.3).

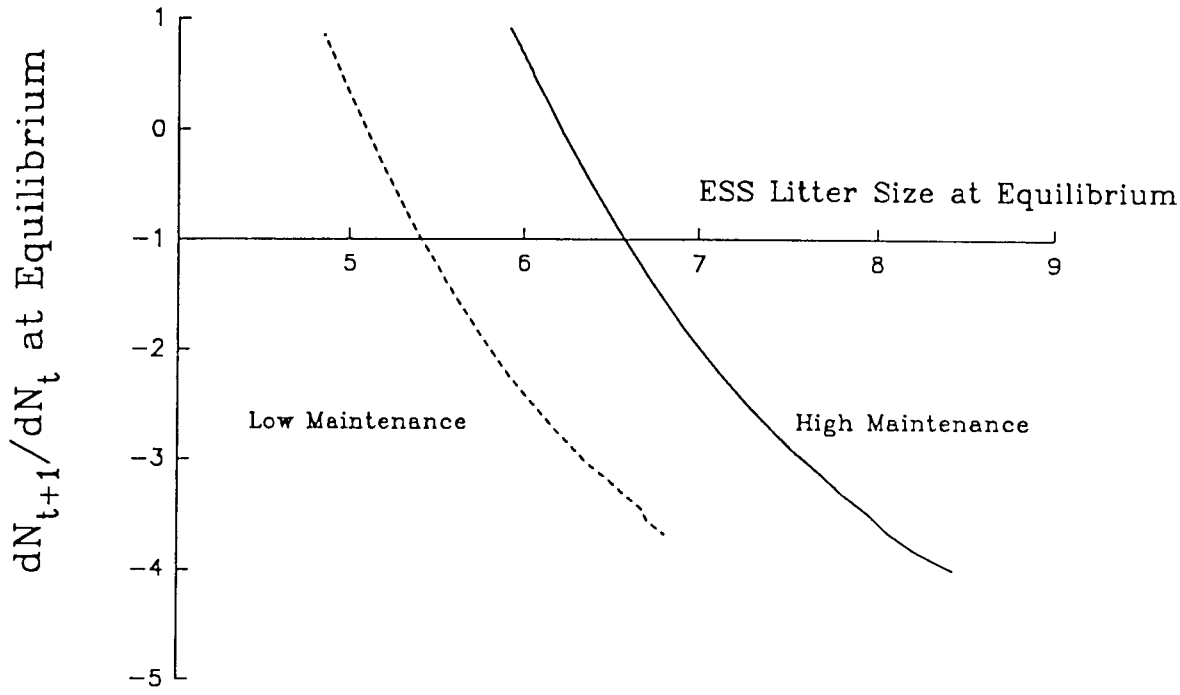

Fig. 3.5. Population stability. Slope of the phase curve at N_{eq} vs. ESS litter size at N_{eq}. Values above the x-axis result in stable-point equilibria; values below the axis result in limit cycles or chaotic population dynamics (as per Eqn [3.7]).

cycle and then chaotic trajectories as litter size increases. The mathematics for determining the critical point at which cycling dynamics give way to chaotic behaviour have been elucidated (May and Oster 1976) but are beyond the scope of this chapter. Returning to the specific example, Figure 3.5 indicates that for the same litter size at N_{eq}, C. gapperi would tend to have a less stable population, and for some values of litter size would demonstrate cycling or chaotic population dynamics while M. pennsylvanicus would exhibit a stable-point equilibrium.

DISCUSSION

The fitness maximizing litter size (L_t in Eqn [3.1]) serves as a decision variable which integrates nutrient requirements for maintenance and reproduction, predation risk, and population parameters (density and growth rate) in producing a behavioural tactic. In turn, its value has important consequences for population level phenomena such as dynamic stability and population density.

Considering the simplicity of the model,the behaviour of ESS litter size is interesting. Because neither a fixed life span nor senescence are assumed in the model, a vole enters each subsequent reproductive period in the same state as it entered the first. Yet although the individual's capability of reproduction, physiological costs, and its risk of doing so remain constant, voles in the model vary their reproductive effort across reproductive bouts. This is because both the value of current reproduction and the individual's residual reproductive value vary depending on population density and growth rate. Fitness maximization will lead the individual to vary its litter size as these population variables change and as expectations of the immediate future change.

The consequences of such changes in reproductive investment can be seen in Figure 3.2, where significant changes in population density follow changes in litter size by one period. This effect is probably seen most clearly in Figure 3.2b, where each major decline in density is preceded by a decline in litter size, a pattern which has been observed in at least one microtine species, M. montanus (Pinter 1986).

It is worth noting here that while populations might also fluctuate in response to changing predation risk, each population in Figure 3.2 is subject to constant risk. Thus, risk is not driving these model systems, but rather establishing the conditions under which litter size decisions are made. A constant level of risk will result in cycling whenever optimal litter size at N_{eq} lies in the unstable region of Figure 3.5. Changes in risk over the course of a population cycle may accentuate or retard the effect, but are not necessary for cycling to occur.

Different risks incurred during reproduction might explain why some microtine species fluctuate dramatically and others do not (Taitt and Krebs 1985), and why species known to cycle do not exhibit cycles in all locations studied (Hansson and Henttonen 1985). A connection between increased cover and increased cycling has been reported for Microtus spp. (Birney et al. 1976). Variations in risk and the subsequent effect on ESS litter size might also explain the conclusion of Sandell et al. (1991) who found a continuum of populations and species exhibiting dynamics from nearly stable to chaotic.

In attempting to make the point that optimal behaviour of individuals can generate surprising population dynamics, I have kept the model as simple and straightforward as possible, but some additions might add to the model's realism. Examples include age-specific reproductive strategies and frequency dependence in the survival functions.

The major area remaining to be addressed is the discreet nature of the population model. In general, analyses of microtine population dynamics have assumed continuous population growth without providing explicit justification (see Schaffer and Tamarin 1973, Stenseth

1978). Such models employ differential equations rather than the difference equation of [3.2]. Simple differential equations without time lags exhibit rather straightforward behaviour around population equilibria, whereas even very simple difference equations can exhibit complex cycling around equilibrium points (May 1974).

There is reason to believe that reproduction in microtines exhibits some synchrony, in which case the difference equation approach employed here is appropriate. For example, microtine reproduction may be synchronized repeatedly on an annual basis by factors specific to each species. Lemmings and voles, which often cease reproduction in winter, appear to be stimulated by fresh spring vegetation to begin reproducing (Negus and Berger 1977). Such a cessation and re-initiation of reproduction should result in females reproducing more or less synchronously in the spring, although synchrony may be lost as the summer progresses. In warmer and drier climates the same synchronizing role may be played by dry summer periods during which reproduction ceases. Of course it is also possible for models which assume continuous rather than synchronized reproduction to produce cyclic behaviour, but in general such models tend to be more complicated.

I have demonstrated that when individuals pursue optimal life history strategies in risky environments, population level effects including cycling may result. In doing so I have expanded the potential role which behavioural decisions, particularly response to predation risk, may play in determining population dynamics.

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APPENDIX 3.A - ITERATIVE SOLUTION FOR ESS LITTER SIZE AND POPULATION TRAJECTORY

The approach described below can be used to solve Eqn [3.1] for ESS litter size, L_t, and to find the next period's population density, N_{t+1}, given the initial population density, N_t and predation risk, R. Other required values are the defining parameters for the adult survival function (m, c) and for the juvenile survival function (b, $N_{.5}$)

Of course once the method is implemented to find N_{t+1} it can be repeated to find L_{t+1} and N_{t+2} and so on to produce a trajectory of litter size and population density through time. The same method can be used to produce data for the phase plot (N_{t+1} vs. N_t) by completing the procedure repeatedly for values of N_t along the X-axis of the phase plot.

The first step toward the solution involves recognizing that although individuals in this model have no fixed life span, potential contributions to reproductive success sufficiently far into the future have little effect on RRV_{t+1} in Eqn [3.1] because the probability that the individual will still be alive becomes diminishingly small. Therefore events beyond some time horizon need not enter into the computations for the current period, and the computation of RRV_{t+1} can be truncated at some point T periods into the future. Of course, the time horizon must be chosen sufficiently far into the future, but the assumption that reproductive events beyond the horizon contribute little to the decision can be verified after each set of iterations.

As described in the main body of the text, it is necessary to simultaneously estimate all of the litter size variables, present and future. A column vector of potential ESS litter size values, **L**, is defined

which contains one element for each reproductive period from the current period (t) to the time horizon (t+T). The elements in the **L** vector serve as initial guesses for the ESS litter size in each time period, but the initial values serve only as starting points and make little difference to the process; it is sufficient to start with an **L** vector filled with zeroes. A corresponding vector of population density, **N**, is then calculated using Eqn [3.2] and the appropriate element in the **L** vector .

Now note that Eqn [3.1] is of the form:

$$\mathbf{L} = f(\mathbf{L}, \mathbf{N}, \bullet) \qquad [3.A.1]$$

where "•" indicates variables independent of **L** and **N**.

If the initial guess for each element in the **L** vector had been correct, Eqn [3.8] would hold exactly, but obviously the initial guess is not usually correct. Therefore define an error vector **e**:

$$\mathbf{e} = \mathbf{L} - f(\mathbf{L}, \mathbf{N}, \bullet) \qquad [3.A.2]$$

and correct the previous guess for **L** as follows:

$$\mathbf{L}_{new} = \mathbf{L}_{old} - \mathbf{e} \qquad [3.A.3]$$

The process is repeated by forming a new **N** vector and evaluating the second guess by recalculating **e**. The process usually converges toward a specific **L** vector with the elements of **e** becoming smaller and smaller. When all the elements in **e** are sufficiently small the iterations are halted and the first element in **L** is taken as the estimate of the ESS litter size:

$L_t = \mathbf{L}(1)$

This set of iterations results in a single value for the ESS litter size at time t and a corresponding value for population density in the next period, N_{t+1} (= $\mathbf{N}(2)$). The process is then repeated in its entirety for the second period, and so on until the ESS litter size and population density for as many time periods as required have been calculated.

Note that the number of time periods which can be calculated is not limited by the time horizon. The time horizon determines how far "down the road" the process looks in determining the current litter size decision, but once the consequences of that decision have been calculated, the process is repeated for the next time period using a time horizon which extends one time period further into the future.

CONCLUSIONS

I demonstrated in Chapter 1 that predation risk influences optimal reproductive effort whether viewed within the context of a single reproductive bout or within a life history context. Although predation risk has no direct physiological effect unless the predation event occurs, risk is nevertheless a cost of reproduction within a life history context because it imposes a present-future tradeoff on reproductive individuals. If reproduction entails an increase in predation risk, the expectation of future reproduction is lowered.

Within a life history perspective, optimization of reproductive effort is frequency dependent, requiring an ESS solution. Both components of the ESS reproductive strategy, present- and expected future-reproduction, are jointly determined as the individual chooses its strategy. Thus, residual reproductive value is determined by individual decisions rather than by the interaction of fixed fecundity and mortality schedules

If predation risk varies between habitats or through time, individuals capable of facultatively adjusting their reproductive effort appropriately should be favored.

In Chapter 2, I tested the prediction that reproduction varies inversely with predation risk. Using meadow voles, Microtus pennsylvanicus, I developed and tested a method of adjusting the predation risk perceived by voles without altering their mortality or the food available to them. Using the giving up density (GUD) technique I demonstrated that voles perceived a higher level of predation risk in the high risk treatment than in the low risk treatment. The GUD approach was justified in a laboratory experiment showing that voles searching for food in a mixture of food and filler experience longer search times at

lower food densities (depletion effect). Survival of the voles in the two treatments did not differ, and vegetation sampling and analysis showed that neither the quantity nor quality of food differed between risk treatments.

However, the reproductive rate for voles in the high risk treatment was significantly lower than in the low risk treatment. Activity measured with electronic detectors was also significantly lower in the high risk treatment, as was the level of foraging measured by counts of fecal deposits and of grass clippings. The results suggest that in habitats in which they perceive risk to be high, voles restrict their foraging rates and thus their reproductive output.

Such individual optimization provides a link between individual decisions and population level phenomena such as density and population growth rate, because each depends on the other. In Chapter 3 a population model incorporating facultative adjustments in litter size demonstrated that individual reproductive decisions are tightly linked to the population's dynamics. Risk of predation, through its influence on optimal litter size, can determine both population stability and equilibrium density. Thus, the adaptive plasticity in life history patterns proposed in Chapter 1 and field tested in Chapter 2 carries consequences not only for our understanding of individual behaviour but also for the dynamics of populations.