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John Citta
The University of Montana

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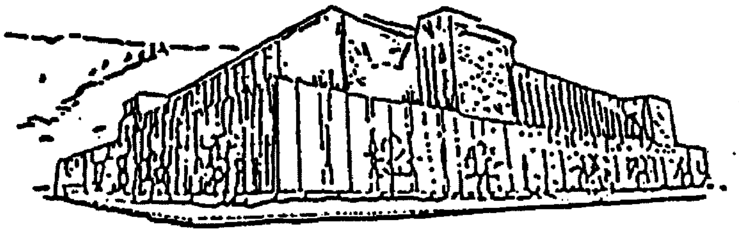

HOW TO CHANGE THE GROWTH OF WILDLIFE POPULATIONS: APPLICATIONS AND EXPERIMENTAL TESTS OF DEMOGRAPHIC SENSITIVITY ANALYSIS

by

John Citta

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Approved by:

Dr. L. Scott Mills, Chairperson

Dean, Graduate School

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Wildlife biologists must often decide how best to manage species, despite incomplete knowledge, scarce funding, and the complexity of natural systems. This thesis addresses one promising tool for wildlife biologists: matrix-based demographic sensitivity analysis. By sensitivity analysis, I am referring to methods which measure the "sensitivity" of population growth rate to perturbations in specific vital rates (i.e. birth and death rates) associated with an organism's life history.

I address sensitivity analyses in two chapters. The first chapter is an example of how such methods are used to provide insight into the management of brown-headed cowbirds (*Molothrus ater*). Collectively, the analyses indicate that natural variation of egg survival likely determines population growth when mean values of egg survival are low (yet plausible) or when high variation exists around mean rates. When the natural range of egg survival does not encompass low rates, yearling survival increases in importance. Due to uncertainty in vital rates, it is currently impossible to ascertain the true sensitivity of these two vital rates. Management actions that decrease only adult survival on breeding ranges are not expected to regulate population growth. In contrast, trapping on wintering ranges are expected to be more effective as these techniques reduce both adult and yearling survival. However, the impacts of winter trapping may be swamped by high egg survival.

In the second chapter of this thesis, I experimentally test predictions of four commonly used matrix-based methods of sensitivity analysis for common deer mice (*Peromyscus maniculatus*). Deer mice provided us with a study system in which I could easily monitor and manipulate entire populations.

All analyses indicated that population growth rate should be sensitive to perturbations in adult female survival and relatively insensitive to perturbations in juvenile female survival. I then tested these predictions using three treatments: (1) a 50% reduction of adult female survival; (2) a 50% reduction of juvenile female survival; and (3) a control. Counter to the predictions, treatments were equally capable of reducing population growth rate over a short time span and had few effects over longer periods of time. Using my field data, I identify non-stable age distributions, vital rate compensation, and demographic stochasticity as factors that may have lead to my lack of treatment effects. Both non-stable age distributions and compensation of vital rates can drastically alter the predicted sensitivity of vital rates. Using methods which allow the practitioner to incorporate actual age distributions can avoid the problem of non-stable age distributions and post-treatment monitoring can be used to incorporate the effects of compensation. If monitoring programs are designed to estimate vital rates, compensation can be identified and used to re-evaluate predictions of sensitivity, thereby improving management.

PREFACE

Wildlife biologists must often provide management insights, despite incomplete knowledge, scarce funding, and the complexity of natural systems. Clearly, objective and cost-effective tools that provide a reliable assessment of management options are needed. Ideally, such tools can be used to provide insight into the efficacy of management options before management is implemented, thereby reducing negative consequences and cost associated with inappropriate management.

Two of the most commonly used modeling tools are habitat-based models and demographic-based models. Habitat-based models generally relate the presence or density of a species with habitat characters. The goal of such models is usually to assess wildlife habitat and predict population responses to habitat alterations. The most well known examples are Habitat Suitability Index (HSI) models (Schamberger et al. 1982;Van Horne and Wiens 1991) and Habitat Capability (HC) models (see Wisdom et al. 1986; Morrison et al. 1992). Habitat models generally relate the presence and/or density of species to specific habitat characters (Van Horne and Wiens 1991). While such modeling is relatively inexpensive, factors which influence populations may be unrelated to habitat (Schamberger and O'Neil 1986; Kellner et al. 1992) and it is questionable if density is correlated with individual fitness (Van Horne 1983, Vickery et al. 1992). As a possible solution, many suggest that habitat models be tested with demographic data (e.g. Van Horne and Wiens 1991) or that "habitat suitability" be defined in demographic terms (e.g. Kellner et al. 1992).

In contrast to habitat-based models, demographic-based models focus directly upon the underlying birth and death rates of a population. While demographic data is more expensive to collect, resulting models do not assume that habitat is correlated with underlying demography. Most demographic based models do not consider how habitat attributes relate to demography, or if habitat is considered, these models often focus on the role of habitat placement for species persistence (e.g. metapopulation models).

This thesis addresses one promising demographic-based tool for wildlife biologists: matrix-based sensitivity analysis. These analyses are described as matrix-based because they combine estimates of reproduction and survival in Leslie or Leftkovitch matrices (i.e. rows and columns of demographic data) which account for known population structuring (See Caswell 1989; Burgman et al. 1993). By sensitivity analysis, I am referring to methods which use these matricies to measure the "sensitivity" of population growth rate to perturbations in specific vital rates (i.e. birth and death rates) associated with an organism's life history (e.g. Crouse et al. 1987; Brault and Caswell 1993; Crowder et al. 1994; Crooks et al. 1998). These analyses are typically used to identify which vital rates are most important for altering the population growth of a species. Once identified, important vital rates can be targeted for management or guide management decisions. While it is unclear what role sensitivity analyses play in final management plans, such analyses are regularly used to make recommendations for management for many species of plants and animals (see table 1 on page v).

I address sensitivity analyses in two chapters. The first chapter is an example of how such methods are used to provide insight into the management of species. The second is an experimental test used to determining if such analyses can provide useful management insight.

Table 1. Selected examples of when sensitivity analysis is used to make management recommendations.

Species	Author(s)
Desert tortoises (*Gopherus agassizii*)	Doak et al. 1994
Yellow mud turtles (*Kinosternon flavescens*)	Heppel et al. 1996
Kemp's ridley sea turtle (*Lepidochelys kempi*)	Heppel et al. 1996
Loggerhead sea turtles (*Caretta caretta*)	Crouse et al. 1987; Crouse et al 1994
Palm species (*Thrinax radiata* and *Coccothrinax readii*)	Olmsted and Alvarez-Buylla 1995
American ginseng (*Panaz Quinquefolium*)	Carron and Gagnon 1991
Madagascar palm (*Neodypsis decaryi*)	Ratsirarson et al. 1996
Astragalus scapoides	Lessica 1995
Florida manatees (*Trichechus manatus latirostris*	Marmontel et al. 1997
Wild rabbits (*Oryctolagus cuniculus*)	Smith and Trout 1994
California gnatcatchers (*Polioptia c. Californica*)	Akcakaya et al. 1997
Greater prairie-chickens (*Tympanuchus cupido pinnatus*)	Wisdom and Mills 1997
Northern pintails (*Anas acuta*)	Flint et al. 1998
Lesser snow geese (*Anser caerulescens caerulescens*)	Rockwell et al. 1997
Greater snow geese (Anser caerulescens atlantica)	Gauthier and Brault 1998

For the first chapter, the brown-headed cowbird (*Molothrus ater*) is used as an example. Brown-headed cowbirds are obligate nest parasites: they do not incubate or feed their own young, but rather lay their eggs in the nest of other species, called hosts, which incubate and feed the cowbird young. If the host is unable to reject the cowbird egg, the host is then left with abandoning the nest, building a new floor in the nest (killing any eggs present), or incubating the foreign egg(s). Hosts that cannot reject cowbird eggs often experience decreased nest success (e.g. Hofslund 1957, McGeen 1972, Mayfield 1977, Elliott 1978, Brittingham and Temple 1983, Weatherhead 1989). Because cowbirds are associated with the declines of numerous host species (e.g. Kirtland's warbler - *Dendroica kirtlandii,* least Bell's vireo - *Vireo bellii pusillus*, and the black-capped vireo - *Vireo atricapilla*) they are of intense management interest (See Kelly and Decapita 1982).

Using published vital rates for brown-headed cowbirds, I conducted three commonly used methods of sensitivity analyses. The objectives were to: (1) determine the relative importance of various demographic components on brown-headed cowbird population growth rates; (2) determine the robustness of model predictions when vital rate estimates vary due to measurement error and/or environmental variation; and (3) discuss the implications of this analysis for management and research.

While working on this project, I identified a number of problems that could result in incorrect management. First, compensation of vital rates may lead to incorrect predictions. For example, if managers increase reproduction of a specific age class, survival may decrease, due to higher population densities. The structure of such compensation is rarely known. Unknown compensatory structure is especially problematic for sensitivity analysis, because the goal is to make an *a priori* assessment of management alternatives.

Second, most matrix-based analyses are deterministic and therefore rely upon the assumption that populations are at stable age distribution (SAD) or that fluctuating age distributions are unimportant (but see Tuljapurkar 1997). SAD is the proportion of individuals in any age or stage class over time, given a constant matrix. It is unlikely that populations in fluctuating environments exist at SAD for long periods of time. Currently, it is unknown how deviations from SAD in a fluctuating environment affect sensitivity analyses.

Third, matrix-based sensitivity analysis may be difficult to translate into well defined management actions. Although it is typically assumed that management actions should focus upon the vital rates with the highest impact upon population growth rate, this may be misleading, because the sensitivity of population growth is dependent on both the inherent sensitivity of a vital rate and the amount that a given vital rate varies (Mills et. al. In Press). Vital rates predicted by models to be of high importance may actually vary little in nature (Pfister 1998, Gaillard et al. 1998) or may be impossible to manipulate effectively. Interpretation is further complicated by the fact that limiting factors (Silvertown et al. 1996) and/or stochastic events may regulate long-term population growth independent of management actions.

Clearly, it is necessary to have a better understanding of how well sensitivity analyses predict population growth in actual management scenarios. In the second chapter of this thesis, I experimentally test predictions of four commonly used matrix-based methods of sensitivity analysis for common deer mice (*Peromyscus maniculatus*).

Deer mice provided us with a study system in which I could easily monitor and manipulate entire populations. I use the results to address the effects of fluctuating age distributions, compensation of vital rates, and how such analyses should be interpreted for the management of species. Furthermore, I identify how such techniques can be improved for future use.

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Chapter 2

Chapter 1: What do demographic sensitivity analyses tell us about controlling Brown-headed Cowbirds?

John J. Citta

Wildlife Biology Program, School of Forestry, University of Montana, Missoula, MT 59812.

ABSTRACT

While Brown-headed Cowbird (*Molothrus ater*) control efforts are fairly common, the effects of control programs on cowbird populations are unknown. I apply analytical-based and simulation-based demographic sensitivity analysis to the problem of cowbird management. Collectively, the analyses indicate that natural variation of egg survival likely determines population growth when mean values of egg survival are low (yet plausible) or when high variation exists around mean rates. When the natural range of egg survival does not encompass low rates, yearling survival increases in importance. Due to uncertainty in vital rates, it is currently impossible to ascertain the true sensitivity of these two vital rates. Management actions that decrease only adult survival on breeding ranges are not expected to regulate population growth. In contrast, trapping on wintering ranges are expected to be more effective as these techniques reduce both adult and yearling survival. However, the impacts of winter trapping may be swamped by high egg survival. When this analysis is combined with life history and logistical realities, I believe that widespread trapping efforts will be largely ineffectual for controlling cowbird

populations on either breeding or wintering ranges. I suggest that cowbird vital rates be specifically examined with respect to host communities, vegetation type, and land use in order to rank management priorities.

INTRODUCTION

Land managers have long realized that Brown-headed Cowbirds (*Molothrus ater*) may decrease nesting success of passerine hosts (e.g. Hofslund 1957, McGeen 1972, Mayfield 1977, Elliott 1978, Brittingham and Temple 1983, Weatherhead 1989). Due to the negative effect cowbirds have on some host species, land managers have attempted to control cowbird populations since the early 1970s. For example, control programs in Michigan typically remove 3,000 or more female cowbirds and cowbird eggs yearly (Kelly and DeCapita 1982; DeCapita, pers. comm.) and trapping efforts on the Ft. Hood military reservation in Texas remove upwards of 3,000 to 5,000 female cowbirds per year (J. D. Cornelius, pers. comm.). These control programs usually target cowbirds to protect federally listed endangered species and commonly involve the removal of adults from feeding areas (Rothstein et al. 1987), the removal of adults and yearlings from communal wintering areas (J. D. Cornelius, pers. comm.), and to a much lesser extent, the removal of eggs from host nests. While cowbird control efforts are fairly common and such efforts are capable of decreasing parasitism rates, the effect of such efforts on cowbird population growth remain unknown.

Better knowledge of cowbird population dynamics is necessary to assess the efficacy of current management strategies and to aid the design of more efficient management strategies. Here I apply traditional techniques and new matrix-based techniques of sensitivity analysis to investigate how different management options may influence cowbird population dynamics. Specifically, I use sensitivity analysis to determine how managers can most effectively decrease the growth rate of cowbird populations. The objectives are three-fold: (1) to determine the relative importance of various demographic components on Brown-headed Cowbird annual population growth rates (λ); (2) to determine the robustness of model predictions when vital rate estimates vary due to measurement error and/or environmental variation; and (3) to discuss the implications of this analysis for management and research.

METHODS

I examine the sensitivity of annual population growth rate (λ) to perturbations in specific Brown-headed Cowbird vital rates with traditional analytical-based and new simulation-based techniques.

ANALYTICAL-BASED TECHNIQUES

Traditional sensitivity analyses (Caswell 1989) are analytical techniques used to evaluate expected response of population growth rates to perturbations in single vital rates (i.e. birth or death rates) one-at-a-time and by equal amounts. Sensitivity, as defined by Caswell (1989), is the absolute infinitesimal change in population growth rates given an absolute infinitesimal change in a vital rate, while all other vital rates are held constant. If a is a matrix of transition probabilities, v and w are the vectors of reproductive values and stable age distributions (SAD) associated with matrix a, respectively, and $\langle \mathrm{v\ w} \rangle$ is the scalar product of the two vectors, the sensitivity of matrix element a_{ij} (row i, column j) is equal to:

$$\textit{Sensitivity } (s_{ij}) \text{ of } a_{ij} = \left(\frac{v_i\, w_j}{\langle v\, w\rangle}\right) \tag{1}$$

Elasticities are similar, but are calculated on a proportional scale, where λ is the geometric population growth rate at SAD:

$$\textit{Elasticity } (e_{ij}) \text{ of } a_{ij} = \left(\frac{v_i\, w_j}{\langle v\, w\rangle}\right)\left(\frac{a_{ij}}{\lambda}\right) \tag{2}$$

Intuitively, elasticity is the sensitivity of a_{ij} weighted by its proportional change with λ. The change in vital rates and λ is assumed to be infinitesimal and linear.

When matrix elements are composed of more than one vital rate, component sensitivities and elasticities can be calculated for each vital rate that appears in one or more matrix elements. Chain rule differentiation is required for each a_{ij} that contains a particular vital rate x. For n elements that contain vital rate x, the sensitivity and elasticity of x are:

$$\textit{Component sensitivity of vital rate } x = \sum^{n}\left[\left(s_{ij}\right)\left(\textit{product of non-}x\textit{ components}\right)\right] \tag{3}$$

$$\textit{Component elasticity of vital rate } x = \left(\textit{Component sensitivity of vital rate } x\right)\left(\frac{x}{\lambda}\right) \tag{4}$$

What do analytical techniques of sensitivity analysis imply biologically? Because sensitivity and elasticity are partial derivatives, they represent the slope of the relationship between a small change in a vital rate to the corresponding change in λ. Traditionally, researchers and managers have assumed that vital rates with high sensitivities or elasticities should be the focus of management actions, as perturbation of these vital rates produce the greatest change in λ. This assumption is not always correct (Mills et al. in press).

SIMULATION-BASED TECHNIQUES

Traditional sensitivities and elasticities may mislead managers, because inevitable variation imposed by nature, by management action, or by measurement error will not be infinitesimal or equal across all vital rates (Gaillard et al. 1998, Mills et al. in press). To account for vital rate variation on scales that are neither absolutely or proportionally equal across vital rates, I also use the sensitivity technique used by Wisdom and Mills (1997). Upper and lower limits of vital rates, determined from literature review, are incorporated into high and low matrices and a computer program constructs 1,000 matrices with each vital rate of each matrix randomly chosen from a distribution bounded by the high and low values. A population growth rate (λ) is then calculated for each matrix. The relative importance of a stage specific vital rate is assessed by regressing λ for each replicate against the value of that rate for all replicates to derive coefficients of determination (R^2). In terms of traditional definitions of sensitivity, R^2 for any component vital rate is analogous to the squared sensitivity weighted by the relative variance of a vital rate (H. Caswell, pers. comm.).

The regression method is appealing, because it allows variation in particular vital rates to alter according to the scale perceived to occur in the field. With the regression technique, variation in vital rates can be incorporated to represent natural amounts of variation, levels of variation imposed by management, or measurement error. Furthermore, vital rates can be selected from distributions that mimic natural distributions. For the selection of vital rates, a uniform distribution was favored over other distributions. Without knowing how likely different vital rates are, all vital rates should have equal likelihood of selection and this distribution evaluates the scenario where extreme changes in rates under management have the same likelihood as small changes from the current mean.

Because all possible λs are plotted, the regression technique also has the advantage of being able to detect non-linearities that traditional methods may not. This is similar to the covariance technique used by Brault and Caswell (1993), but is computationally and intuitively easier to manage. If non-linearities do not exist in the data, then varying vital rates on absolute and proportional scales should produce similar results as traditional sensitivities and elasticities (Mills et al. in press).

Model Structure and input for cowbird analysis

I use two-stage Leftkovitch matrices (see APPENDIX A) to model cowbird populations. Stage specific demographic data form the matrix and the model projection interval is 1 year. Eigenanalysis of the matrix, or projection of the matrix over time, provides annual population growth rates (λ). Consequently, all techniques of sensitivity analysis assume populations are at stable age distribution (SAD). It is an all female model, a reasonable approach given the excess of adult males in natural populations (Darley 1971, Arnold 1983). Fecundities are divided in half to account for female eggs only and are multiplied by annual cohort survival to account for a post-breeding census.

Model input, in terms of estimated stage-specific ranges of vital rates, is taken from the literature (Table 1). The top row of the matrix (F_{11} and F_{12}; see APPENDIX A) contains reproductive information based on both survival of females to breed and number of eggs laid (fecundity). Unfortunately, cowbird fecundity has been difficult to determine and estimates of the number of eggs laid per female varied widely. Much of this variation is removed when daily laying rates are considered. When multiplied by the length of the breeding season, daily laying rates are likely to be the most accurate estimator of annual female fecundity (Rothstein et al. 1986). Consequently, only daily laying rates are considered and, to avoid non-constant laying rates over the breeding season, assume a 40 d breeding period within which laying rates are constant (Table 1). To determine the possible importance of low fecundity, I include Holford and Roby's (1993) fecundity estimates for calcium deprived individuals in captivity. From this data I estimate the suppressed daily egg laying rate to be approximately 0.37 eggs per day. While this figure is significantly lower than the lowest estimate of daily egg laying rate measured under natural conditions (0.51 eggs per day), inclusion in the model illustrates the consequences of extremely low fecundity on λ. The final assumption is that adults and yearlings have the same maximum and minimum daily laying rates. Although Jackson and Roby (1992) indicate that yearlings have lower fecundity rates than adults, the lowest measured daily laying rate for yearlings is not as low as the rate for calcium deprived individuals. This implies that the lower daily laying rate used in the model (that for calcium deprived individuals) represents a worst case scenario for both adults and yearlings.

Matrix element G_{21} is the mean survival from stage 1 to stage 2, and represents a composite of egg, nestling, and yearling survival. Egg survival is defined as the probability that an egg survives to hatch. This life stage is assumed to be 15 d. While the average incubation period is approximately 10 - 13 d (Briskie and Sealy 1990), these estimates do not include time before incubation is initiated. In other words, because eggs are likely to remain within the nest some number of days before incubation is initiated (see Nice 1954), a 15 d pre-hatching period is realistic. Nestling survival is defined as the probability that a nestling survives to fledge, given that it hatched. This period is assumed to be 10 d (Norris 1947, Hann 1937). Yearling survival is defined as the probability that a juvenile survives to breed, given that it fledges. This period is assumed to be the remaining 340 d of a cowbird's first year. Estimating yearling survival rates are problematic, because only one study (Woodward and Woodward 1979) quantified cowbird fledgling survival rates (only until independence at approximately 30 d). For an upper bound, I assume yearlings attain adult survival rates immediately after independence and combine the Woodward and Woodward (1979) yearling rate for the first month after fledging (0.48) with the highest estimate of adult survival for the remaining 310 d before breeding. This yields an upper bound for yearling survival of 0.32. The lower bound for yearling survival (0.15) is assumed equal to known lower bounds for Great Tits (*Parus major*) (Dhondt 1979). While using data from other species is not ideal, great tits are one of the only passerine species with known yearling survival rates, thereby providing insight into a lower bound of cowbird survival.

Matrix element P_{22} is the mean survival to remain within stage 2. This is simply an adult female survival rate between annual birth events, and is estimated via return rates or recoveries (Table 1).

RESULTS

If variation is artificially constrained to be small and equal around all vital rates, the simulation-based technique should rank the importance of all vital rates similarly to the traditional analytical-based sensitivity analysis (Mills et al. in press). Although traditional sensitivities and R^2 sensitivities are not directly comparable, because they are different statistics, with small and equal absolute change of ± 0.10 for each rate, the rankings of vital rate effects on λ are identical for both approaches (Fig. 1a). Likewise, incorporating proportional changes in the regression technique (± 10 %) produces similar rankings as traditional elasticities (Fig. 1b). The small deviations between the sensitivities or elasticities and the simulation-based measure are likely due to the effect of non-linearities on sensitivities or elasticities (Mills et al. in press). The vital rates with the highest sensitivities on an absolute scale of variation are yearling survival and nestling survival. The vital rates with the highest sensitivities on a proportional scale are egg, nestling, and yearling survival.

Of course, neither of these vital rate ranges, determined by fixed and equal absolute or proportional change, are likely biologically realistic. Therefore, I used the regression technique to determine R^2 sensitivities for the entire range of cowbird variation, letting different rates vary by different amounts according to the upper and lower bounds presented in Table 1; I refer to this as the empirical range of variation (Fig. 2 and 3). Egg survival alone appears to account for over 60% of the variation in population growth rates. The vital rate accounting for the next largest amount of variation in λ is yearling survival ($R^2 = 0.14$).

While the regression technique is likely to be more realistic than traditional methods because it selects vital rates from biologically realistic upper and lower bounds, it is possible that vital rate ranges that are too large or too small may artificially increase or decrease the R^2 value of a vital rate (Wisdom and Mills 1997). Unfortunately, the sparse data available for most species makes determination of vital rate ranges difficult. This is especially true for threatened or endangered species that are modeled the most, but is also true for common species such as cowbirds. A critical question is thus: what are the consequences of under- or over-estimating the range of variation in demographic parameters? If altering the range of an uncertain vital rate has little effect upon R^2, then accurate range estimation is unimportant. However, if R^2 is sensitive to small changes in the range of vital rates, then correct range estimation is critical. To assess this with the data, I altered the range of each vital rate one-at-a-time while holding the other vital rates at the empirical range width (Table 1). Vital rate ranges were decreased by 25 and 50% and increased by 25% (50% increases were not possible because some survival rates would exceed 1).

Generally, increasing or decreasing range widths results in a monotonic increase or decrease in R^2 values (Fig 4), as expected from the fact that R^2 for any component vital rate is weighted by the variance in that rate. Although I do find the statistically expected change in absolute R^2 values with changes in vital rate ranges, the biologically important result is that the relative rankings do not change for the vital rates that account for most of the variation in λ.

Because egg survival was identified as the vital rate most affecting population growth when vital rates vary between empirically determined bounds, and because decreasing the range of egg survival can decrease the R^2 value of egg survival. It is relevant to ask how much of a decrease in the range of egg survival is necessary before another rate replaces egg survival as the rate most affecting λ. In the most extreme case of a 50% decline in range width, most of the change in R^2 (40%) was partitioned as increased R^2 for yearling survival and that the total R^2 for yearling survival approached that of egg survival (compare Fig. 5 to Fig. 3).

In addition to range width, mean vital rates must also play a role in determining the effect of a change in any rate on λ. The variance around the regression line for egg survival is non-constant (Fig. 2a), indicating that R^2 values will change as the mean values of vital rates change. To investigate this further, I determined how sensitivities were affected by altering the mean egg survival rate, while holding the range of variation constant. To keep the total range of variation within the biologically plausible range of variation, the range of egg survival was restricted (50% of the empirical range), and the mean vital rate was decreased by 25% and increased by 25%. Even with a small range of egg survival, if the mean egg survival rate is low, then the egg stage has the highest R^2 (Fig. 6a). Alternatively, if the mean egg survival rate is high, yearling survival has the highest R^2 (Fig. 6b).

DISCUSSION

HOW VITAL RATES AFFECT POPULATION GROWTH

The egg survival stage is likely to be the vital rate that most affects population growth rate whenever the range of variation in egg survival is high or in situations where the mean egg survival rate is low. The only studies that examine cowbird egg survival across the entire community of hosts within an area yield mean rates of 0.08 (Elliot 1978) and 0.43 (Norris 1947), indicating that the sensitivities in any of the simulations are plausible. Unfortunately, it is not known how often low rates of egg survival occur and, more importantly, how much egg survival varies within and between sites over time. In situations with high mean egg survival rates and low levels of variation around those mean rates, the yearling stage may play the biggest role in impacting λ.

An obvious question is: with what degree of certainty are vital rate ranges estimated? This question is most critical with regard to egg survival. Range estimation for egg survival is problematic, because most studies observe only one host. Furthermore, cowbird researchers tend to study highly parasitized hosts that accept cowbird eggs and are parasitized enough to be analyzed statistically. Unfortunately, cowbirds do not exclusively parasitize one species, but typically parasitize a number of hosts within the breeding area. Host communities likely yield rates of egg survival that differ from the rates observed in any single species. Furthermore, egg survival rates are sure to be systematically overestimated, because egg ejections or eggs laid in inactive nests are unlikely to be detected. The extent of this bias is unknown and likely dependent upon the host community and vegetation type. While many host species are known ejectors, very little is know about how often these species are parasitized as eggs may be almost immediately ejected (Rothstein 1975, Friedman et al. 1977). Although not well quantified, the rate at which cowbirds lay eggs in abandoned nests appears to highly variable. Berger (1951) reported a rate of 1.35% for Song Sparrows (*Melospiza melodia*) and Freeman et al. (1990) reported a rate of 21.5% for Red-winged Blackbirds (*Agelaius phoeniceus*). The high rates of inappropriate egg laying noted by Freeman et al. (1990) appear to be a function of available perch sites; they hypothesized that without perches, the cowbirds were not able to assess correctly whether a nest was abandoned.

In summary, traditional analytical techniques indicate that egg, yearling, and nestling survival are the most sensitive vital rates. Regression-based techniques indicate that egg and yearling stages are the most sensitive. Regression-based techniques also indicate that the relative importance of egg versus yearling stages depends upon the range of variation and the mean rate of egg survival. Whenever egg survival rates are low or if the range of egg survival encompasses low rates then egg survival will most affect population growth. Adult survival, adult fecundity, and yearling fecundity were not important factors in any of the modeled scenarios.

LIMITATIONS OF MODELLING TECHNIQUES

Although sensitivity analysis is capable of revealing non-intuitive relationships, several limitations must be kept in mind. First, neither the analytical- nor simulation-based technique accounts for density dependent relationships. While positive or negative correlations between vital rates could be included within either the analytical-based technique (van Tienderen 1995) or within the simulation-based technique (Wisdom et al., unpubl. data), these data are not available. Furthermore, density dependent correlations between vital rates may change as management perturbations are intensified and these changes may not be predictable under current conditions.

Second, and related to density dependence, the techniques do not account for compensatory effects. One possible compensatory effect is the replacement of breeding females and may occur as present non-breeding "floaters" occupy empty egg laying areas. If removed females are replaced, then adult survival is not functionally decreased as modeled and the predicted sensitivity is biased high. I predict that the sensitivity (traditional and regression-based) of adult survival is maximized when non-breeding floaters are not present. Conversely, if many floaters are present, adult removals will not be effectual until the number of adult females drops below the amount necessary to parasitize all available nests. If cowbirds exhibited extremely high replacement rates, then it is unlikely that trapping of adults near sensitive host species would be effective. Trapping records from the effort to protect the Kirtland's Warbler show that most female cowbirds are captured within the first few weeks of the breeding season (Kelly and DeCapita 1982), indicating that trapping efforts are capable of removing all females within a short time period and that floaters are not a concern to this analysis.

Third, neither technique accounts for spatial considerations. As modeled, Brown-headed Cowbirds are treated as one large population and assumed perturbations are population-wide. Management actions must consider the ratio of the size of the target population to the size of the total population, because managing only a subset of individuals dilutes population-wide effects. In other words, if only a part of a cowbird population is managed, there will be little effect on the population as a whole. Identifying exactly what effect any given management action will have on cowbird populations will require delineation of population boundaries and knowledge of movement rates between populations within and between breeding seasons. Trapping records from the effort to protect the Kirtland's Warbler (Kelly and DeCapita 1982) and the Black-capped Vireo (Barber and Martin 1997) show that trapping does not reduce the number of cowbirds in subsequent years; this indicates that there is either a large level of movement between populations or that the target population is much smaller than the total population. Unfortunately, there are little or no data identifying the spatial structure and dynamics of cowbird populations.

Finally, matrix-based calculations of λ also assume populations are at stable age distribution (SAD). SAD is the proportional number of individuals in any age or stage class over time, given a constant matrix. It is unlikely that populations in fluctuating environments exist at SAD for long periods of time. Currently, it is unknown how deviations from SAD in a fluctuating environment affect either traditional or the regression-based techniques.

IMPLICATIONS FOR COWBIRD MANAGEMENT

Although egg survival is likely the vital rate which most affects population growth rate in many situations, it is nearly impossible to manage with current techniques and logistical constraints. I identify four problems with egg removal programs. First, host nests are difficult and expensive to find (Martin and Geupel 1993). Second, removing cowbird eggs may increase nest predation rates or result in nest abandonment (Major 1990, Gotmark 1993), although correct protocols can reduce disturbance (Martin and Geupel 1993). Third, to be effective, egg removals must target a large proportion of the total cowbird population. Egg removals will likely have to exist at scales much larger than study sites, management areas, and wildlife refuges. The exact scale of management will depend upon the size of the cowbird population and movement rates between populations, which remain unknown. Fourth, eggs are expected to be numerous. At the time of breeding, over 90% of the population is in the egg stage at stable age distribution; consequently, management actions may remove many eggs, yet have little impact upon total egg survival rates.

To illustrate how a management imposed change in a vital rate is affected by the number in that age class, an example is provided. Using the mean matrix (APPENDIX A) and assuming a population size of 5,000 cowbirds, there are over 4,600 eggs resulting from approximately 400 adults at the time of breeding (APPENDIX B). Suppose that from this population I remove equal numbers of eggs, nestlings, fledglings, and adults, assuming that this mortality was additive. I find that by removing small numbers of eggs, I impact total population growth very little compared to other stages (Figure 7). For example, on an individual basis, removing approximately 100 adults or fledglings will have the same impact as removing over 475 eggs, because there are fewer adults or fledglings in the population. In short, there are so many cowbird eggs that even large egg removals may have little impact upon total egg survival rates. While environmental variation and the consequences of having different host communities determine the population-wide survival rate of eggs, and therefore affect population growth, the effects of management on egg survival and the resulting changes in population growth are likely minuscule. When the problems of finding nests, human induced impacts upon hosts, large management scales, and the preponderance of eggs are considered jointly, egg removal is probably not a viable management option.

Because reducing egg survival is not a wide-scale management option, I must consider what vital rates can be managed. Currently, the most common management options are trapping on the breeding grounds and trapping on the wintering grounds. Trapping on the breeding grounds typically involves the trapping of adults, while trapping on the wintering grounds involves the removal of adults and yearlings. Each of these options are considered in turn.

Cowbird population growth rates are generally less affected by fluctuations in adult survival than other vital rates and the effects of adult removals may be masked by variation in egg and/or yearling survival. During the breeding season, the replacement of breeding females (via floaters and immigrants) exacerbates this problem and makes population growth rates even less sensitive to adult removals. To illustrate the significance of this problem, consider that cowbird trapping programs in Michigan typically remove 3,000 cowbirds per year with no noticeable decline in cowbird populations between years, despite the fact that virtually all individuals are removed during the breeding season (Kelly and DeCapita 1982; M. DeCapita, pers. comm.). The lack of any effect of trapping on cowbird populations may be due to either targeting only a small proportion of the total cowbird population or high rates of immigration. Either alternative leads to the same conclusion: that adult removal programs on breeding grounds are not likely to regulate populations unless they are conducted on a much larger scale. However, this does not invalidate trapping programs during the breeding season, as such programs are usually intended to protect sensitive host species at a local scale and can successfully do so (Kelly and DeCapita 1982, Barber and Martin 1997; M. DeCapita, pers. comm.; J. Cornelius, pers. comm.).

Trapping adult and yearling cowbirds on the wintering ranges is expected to be much more effective in controlling cowbird population growth. While population growth is not sensitive to perturbations in adult survival, adults are only a small proportion of the total population. Hence, adult survival may be greatly altered by removing only a small absolute number of adults (APPENDIX B). In contrast to adult survival, population growth is likely sensitive to perturbations in yearling survival; there are also relatively few yearlings in any given population. Therefore, the sensitivity analysis and the age distributions suggest that winter trapping programs are more likely to decrease cowbird population growth rates than by removing eggs or trapping on breeding grounds. In addition to these life history considerations, winter trapping has many logistical advantages because cowbirds concentrate on large communal wintering grounds.

Unfortunately, there are also serious limitations with using winter trapping to control cowbird populations. Removing cowbirds from all wintering areas may be logistically impossible, because wintering ranges extend from Texas into Mexico (Bray et al. 1974, Arnold 1983). Also, even massive control in a limited number of wintering areas may produce extremely diffuse effects on the breeding ranges (Rothstein and Robinson 1994), because individuals in one wintering range may breed throughout North America (Bray et al. 1974, Dolbeer 1982). Finally, the large effect of egg survival on population growth rate may make trap efforts on other age classes ineffectual. For example, note that for the lowest rates of yearling and adult survival, many of the matrices have positive growth rates (Fig. 2c). So, while winter removals of adults and yearlings are expected to be more effective than removing eggs or adults on the breeding ranges, they do not have a high likelihood of regulating population growth rates unless most or all wintering areas are targeted for management. Furthermore, the effects of winter removals may be swamped by natural variation in egg survival rates.

Given the formidable logistical difficulties in lowering the vital rates that most affect cowbird population growth, the most effective method of cowbird control is likely to be the management of land uses to disfavor cowbirds. Cowbird presence is often significantly correlated with the presence of livestock (Schulz and Leininger 1991, Knopf et. al. 1988, Mosconi and Hutto 1982; but also see Kantrud 1981), agriculture (Rothstein et al. 1984, Rothstein et al. 1987, Tewksbury et. al. in press), and forest fragmentation (Chasko and Gates 1982, Coker and Capen 1995, Tewksbury et al. 1998). By managing grazing patterns, availability of agricultural waste grain (often an important food source), and forest fragmentation, managers may be able to indirectly eliminate or at least control the presence of cowbirds before they parasitize host species.

IMPLICATIONS FOR COWBIRD RESEARCH

Much research has focused upon the effects of limiting cowbird fecundity or determining what limits cowbird fecundity. While this is a valid research topic for life history information, it is of little management interest unless fecundity can be decreased to rates near zero. I varied fecundity to rates lower than anything ever measured in nature (the calcium deprived rates) and then decreased that rate to assess the effect of larger variation in vital rate ranges. In all simulations but one (Fig. 6b) fecundity had the least effect on λ of any vital rate.

To date, most cowbird research has focused upon parasitism of specific host species, not upon parasitism of host communities within habitats or by land use practice. Because parasitism rates, predation rates, host communities, and the ability of hosts to fledge cowbirds vary across the landscape, it is unlikely that all vegetation types and host communities are equally productive for cowbirds. Furthermore, the presence of cowbirds may not reflect cowbird habitat quality. As long as adequate foraging habitat (feeding grounds) exist within flight range, cowbirds may parasitize host nests in habitats which barely provide positive growth rates or provide negative growth rates. By focusing research efforts upon cowbird vital rates in different vegetation types and host communities, researchers may be able to identify habitats and land use practices which are most important for (or possibly are responsible for) cowbird population growth. If the goal of management is to regulate populations of cowbirds, I suggest focusing management plans on regulating land uses which favor cowbirds in areas with positive cowbird growth rates. For example, livestock grazing in areas that have vegetation types and host communities that lead to negative cowbird population growth rates should be a lower management priority (assuming no endangered species are present) than livestock grazing in areas which lead to positive cowbird population growth rates. Currently there is no knowledge of how cowbird population growth rates may vary across combinations of vegetation types and host communities.

Last, more data are needed to understand cowbird population structure. The model assumes that cowbirds exist in one large population, because there are no data for constructing spatially-explicit models. Without more knowledge of population boundaries and how adult and juvenile cowbirds move between populations over time, managers will not be able to predict the true efficacy of management alternatives and may choose inappropriate scales for management.

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APPENDIX A: Brown-headed Cowbird stage-based model

COWBIRD LIFE CYCLE DIAGRAM AND MATRIX

The biologically relevant projection interval for cowbirds is 1 year, so elements within the matrix represent annual rates (Figure A1). However, in the first year of life, there are three relevant stages: egg, nestling, and yearling; thus I let the first year of life have egg, nestling, and yearling components. Only one adult stage is included (as opposed to annual age classes), because age specific adult survival rates are not available and management techniques target all adults concurrently.

Transition matrix:

$$\begin{bmatrix} F_{11} & F_{12} \\ G_{21} & P_{22} \end{bmatrix}$$

DEFINITIONS OF MATRIX ELEMENTS AND VALUES FOR THE MEAN MATRIX:

Mean survival from stage 1 to stage 2 [G(2,1)] = mean egg survival (0.38) x mean nestling survival (0.64) x mean yearling survival (0.24) = 0.06

Mean survival from stage 2 to stage 2 [P(2,2)] = mean annual adult survival = 0.47

Mean yearling fertility [F(1,1)] = mean daily laying rate (0.56) x laying period (40 d.) x proportion of female eggs (0.5) x mean first year survival (0.06) = 0.65

Mean adult fertility [F(1,2)] = mean daily laying rate (0.69) x laying period (40 d.) x proportion of female eggs (0.5) x mean adult survival (0.47) = 6.49

These transition probabilities are incorporated into a mean matrix and have the resulting stable stage distribution and deterministic population growth rate (λ):

$$\begin{bmatrix} 0.65 & 6.49 \\ 0.06 & 0.47 \end{bmatrix} \qquad \begin{bmatrix} 0.9244 \\ 0.0756 \end{bmatrix} \qquad \lambda = 1.184$$

STAGE SPECIFIC TIME INTERVALS

The projection interval is one year (365 d.):

1st year:

- egg survival stage: 15 d.
- nestling survival stage: 10 d.
- yearling survival stage: 340 d.

2nd year + (Adults): 1 yr.

APPENDIX B: Analysis of a how a given removal may affect Brown-headed Cowbird survival and population growth rate

This analysis is included to clarify how the sensitivity analysis must be interpreted. Specifically, I was concerned that the analysis would lead to time, effort, and money being prematurely applied towards egg removal programs. When environmental variance is included in the analysis, egg survival has the largest impact upon population growth rate. However, it is not clear how removing cowbird eggs actually alters egg survival rates. I investigate this link with the following crude analysis, where cowbird vital rates are decremented one-at-a-time by removing a specified number of individual eggs, nestlings, yearlings, or adults.

I start with the following assumptions:

1. There is a population of 5,000 cowbirds at the beginning of a breeding season.
2. This population has the vital rates of the mean matrix in APPENDIX A.
3. All mortality is additive (no compensatory effects) and immigration is nonexistent.
4. Populations are at stable age (stage) distribution.

To calculate the number of individuals in particular life stages within a population of 5,000, at time *t*, I must first determine the number of individuals at time *t-1*:

$$N_{t-1} \times \lambda = 5,000$$

$$N_{t-1} = 4222.97$$

By multiplying N_{t-1} by the stage distribution vector (APPENDIX A), the total number of eggs and adults at time *t-1* is:

$$4222.97 \text{ x } \begin{bmatrix} 0.9244 \\ 0.0756 \end{bmatrix} = \begin{bmatrix} 3903.72 \text{ eggs} \\ 319.26 \text{ adults} \end{bmatrix}$$

Then the matrix is multiplied by the stage distribution vector at time *t-1* to determine the initial number of individuals in each stage at time *t*:

Initial number of eggs: $(F_{11} \text{ x } V_1) + (F_{12} \text{ x } V_2) = 4621.81$

Initial number of adults: $(G_{21} \text{ x } V_1) + (P_{22} \text{ x } V_2) = 377.90$

Immediately after breeding, there is a population size of 377.90 adults and 4621.81 eggs. This is the total population of 5,000. To calculate the number of individuals in intermediate stages, the total number of eggs is decremented by egg, nestling, and yearling survival rates successively:

Initial number of nestlings: Initial number of eggs x egg survival rate = 1756.29

Initial number of yearlings: Initial number of nestlings x yearling survival rate = 1124.03

I simulate individual removals by decreasing the number of individuals in a life stage by increments of 40. This mortality is assumed to occur after the initial probabilistic mortality of that stage class (i.e. it is assumed to be additive). For example, if management removes 200 eggs, then the adjusted egg survival rate is calculated as follows:

$$\frac{(\text{Initial number of eggs} \times \text{original egg survival rate}) - 200 \text{ eggs}}{\text{Initial number of eggs}} = \text{New egg survival rate}$$

$$\frac{(4621.81 \times 0.38) - 200 \text{ eggs}}{4621.81} = 0.34$$

In this example, removing 200 eggs reduced egg survival rates by only 11%. The altered survival rates are then incorporated into the mean matrix to calculate the resulting population growth rate (Figure 7).

TABLE 1. VITAL RATES FOR THE COWBIRD TRANSITION MATRIX (SEE APPENDIX A)

Vital Rate	Average (N)	High	Low	Citations
Adult Fecundity[1]	0.69 (4)	0.8	0.51 (lab) 0.37 (calcium limited)	Scott and Ankney 1979 (California), 1980 (Ontario); Rothstein et al. 1986 (California); Holford and Roby 1993 (captive population)
Yearling Fecundity[1]	0.56 (1)	N/A	N/A	Jackson and Roby 1992
Egg survival	0.38 (pooled across 9 studies; N=1346 eggs)	0.68 (single species study)	0.08 (host community study)	Hann 1937; Norris 1947; Berger 1951; Hofslund 1957; McGeen 1972; Elliot 1978; Weatherhead 1989; Marvil and Cruz 1989; Smith and Arcese 1994
Nestling survival	0.64 (pooled across 6 studies; N=224 nestlings)	0.76	0.46	Hann 1937; Norris 1947; Berger 1951; Hofslund 1957; Marvil and Cruz 1989; Weatherhead 1989
Yearling survival	0.24	0.32	0.15	Dhondt 1979 for Great Tits; Woodward and Woodward 1979
Adult survival[2]	0.47 (4)	0.63	0.31	Darley 1971; Fankhauser 1971; Arnold 1983 (provides 2 estimates)

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[1] Total fecundity; this is divided by 2 to account for an all female model (See APPENDIX A).

[2] Female only rate

FIGURE 1. Comparison of component sensitivities and R^2 values for Brown-headed Cowbird vital rates with (a) range standardized on an absolute scale (plus or minus 0.10) and (b) range standardized on a proportional scale (plus or minus 10%). Only rankings are directly comparable between component sensitivities and R^2 values.

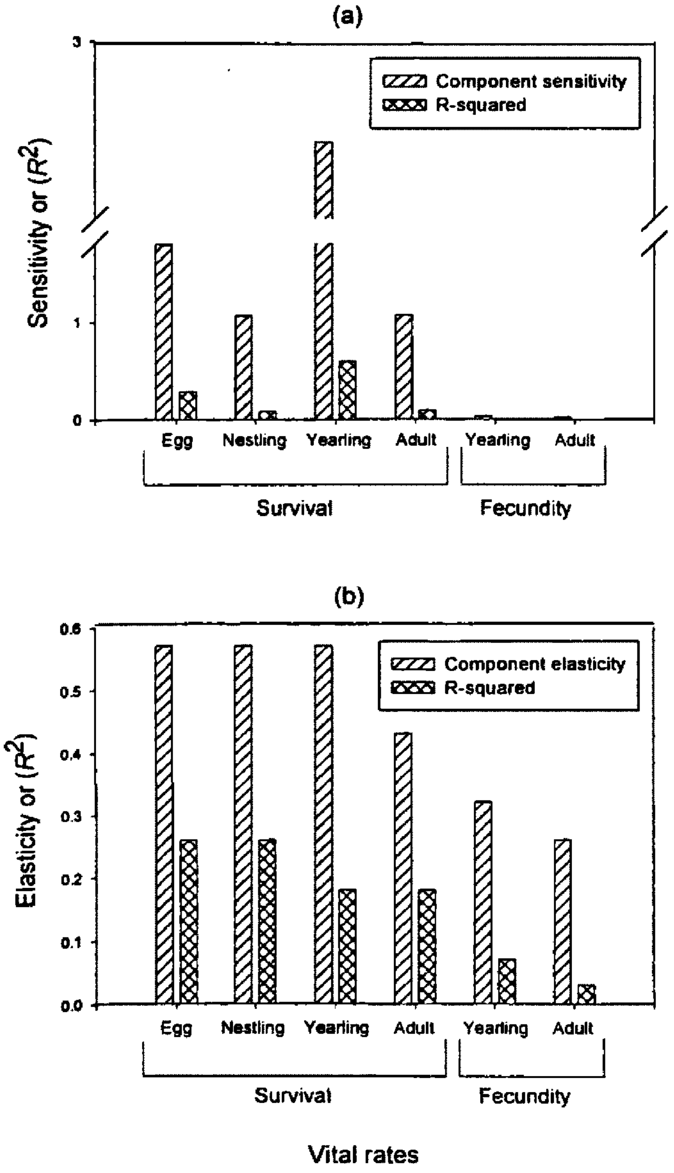


FIGURE 2. Amount of variation in Brown-headed Cowbird population growth rate (R^2) as accounted for by (a) egg survival rate, (b) nestling survival rate, (c) yearling survival rate, (d) adult survival rate, (e) yearling fecundity rate, and (f) adult fecundity rate in 1000 matricies with randomly selected vital rates. Coefficient of determination (R^2) and linear regression line presented.

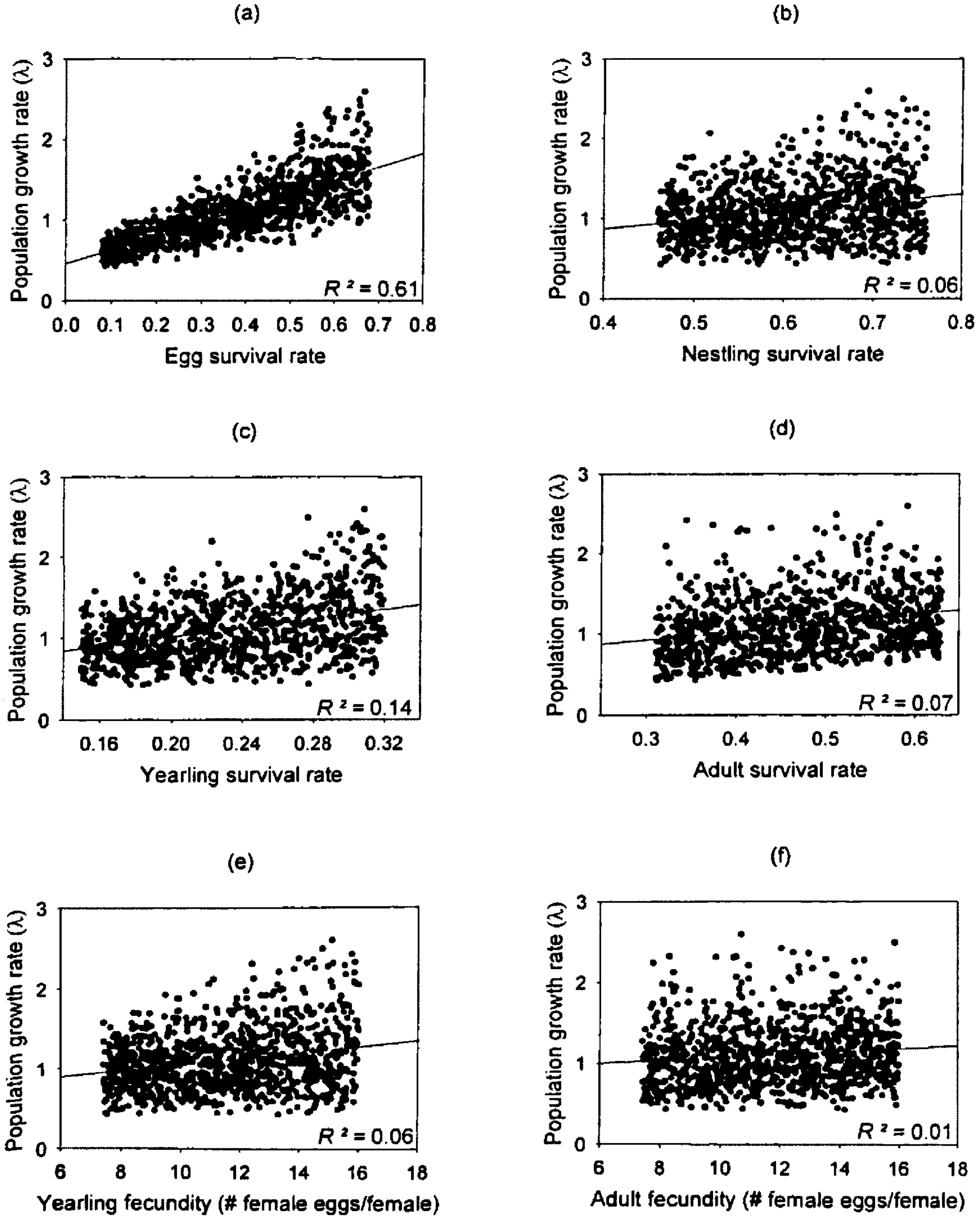


FIGURE 3. Sensitivities of Brown-headed Cowbird vital rates as indexed by the coefficient of determination (R^2) in 1000 matricies with randomly selected vital rates regressed against corresponding population growth rates.

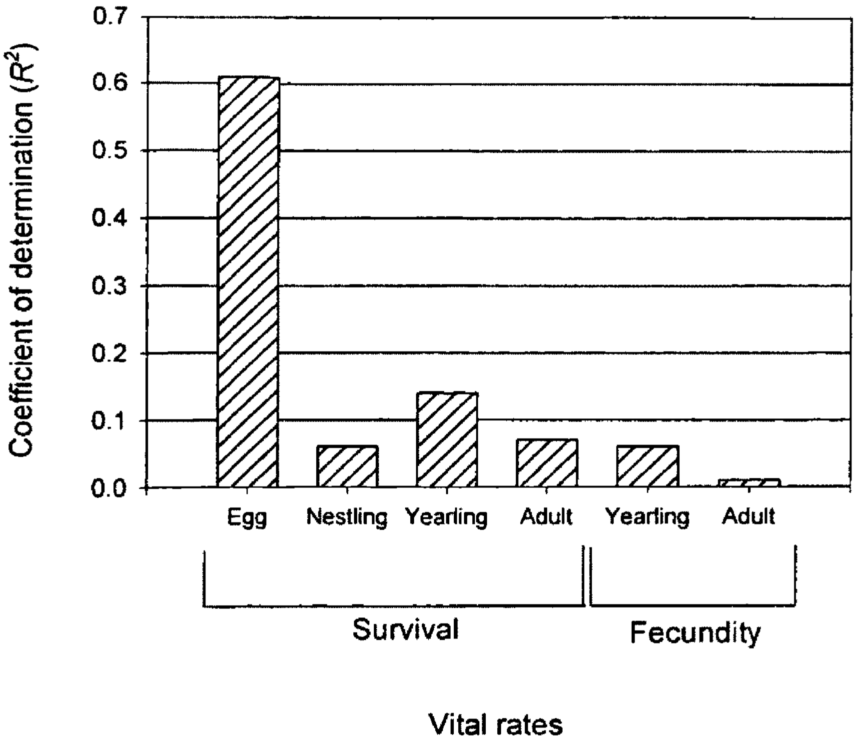


FIGURE 4. Changes R^2 for Brown-headed Cowbird life stages when vital rate ranges are altered. Each point equals R^2 for a vital rate range which is 50%, 75%, 100%, or 125% of the original empirical range, while all other vital rate ranges are held at the empirical range.

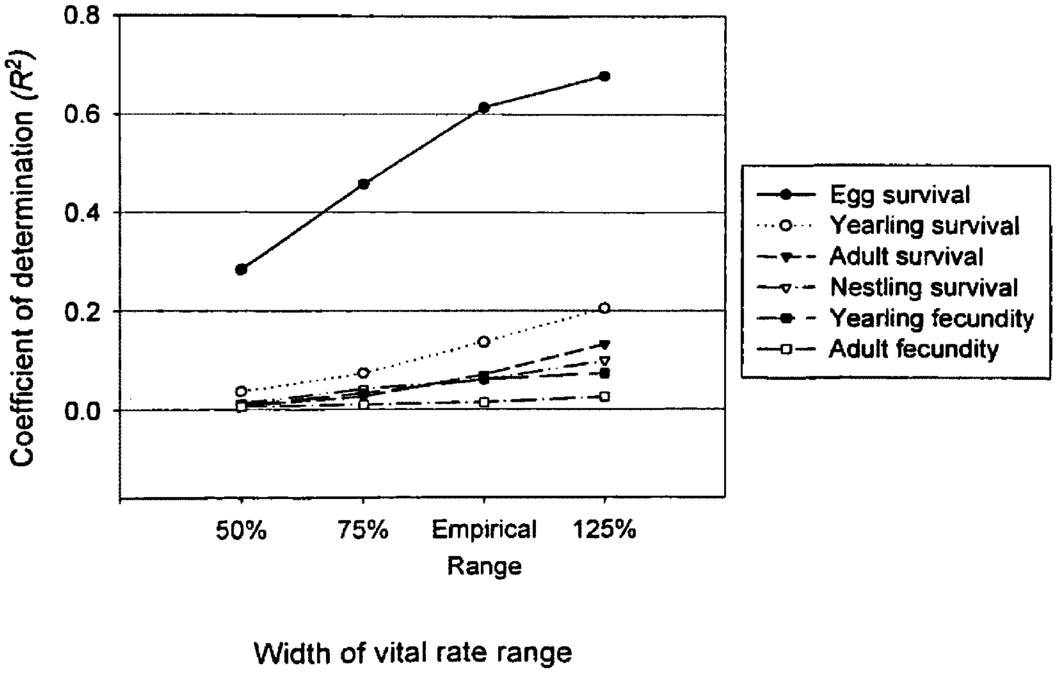


FIGURE 5. Sensitivities of Brown-headed Cowbird vital rates as indexed by the coefficient of determination (R^2) in 1000 matricies with randomly selected vital rates regressed against corresponding population growth rates when the range of egg survival is 50% of the empirical range. Compare to Fig. 3.

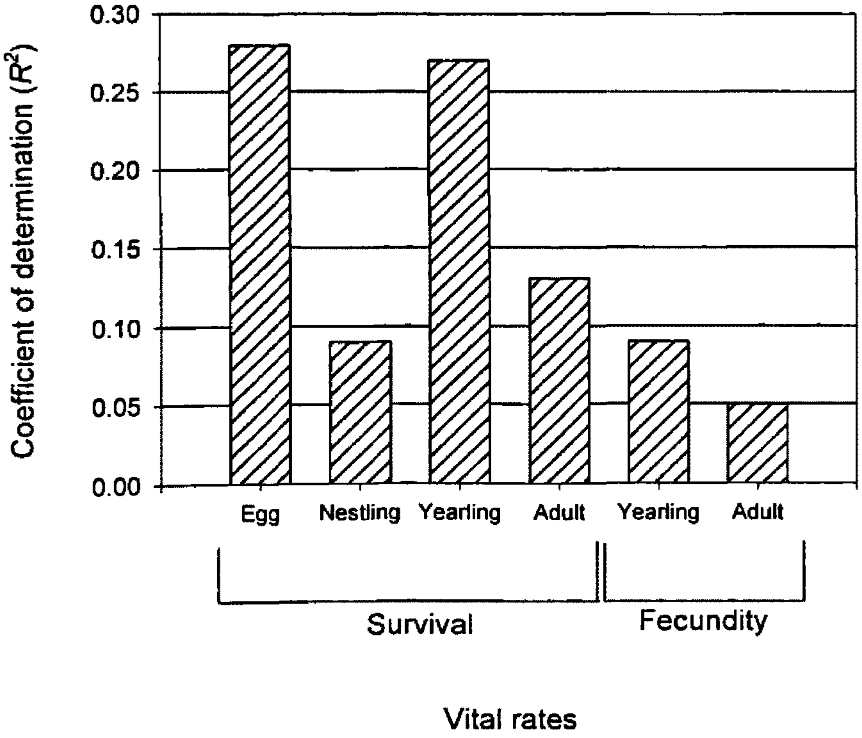


FIGURE 6. R^2 for Brown-headed Cowbird vital rates when the range of egg survival is 50% of the empirical range and the mean rate is (a) decreased 25% from the empirical mean (new mean = 0.29) and (b) increased 25% from the empirical mean (new mean = 0.48). All other vital rates have the empirical means and ranges.

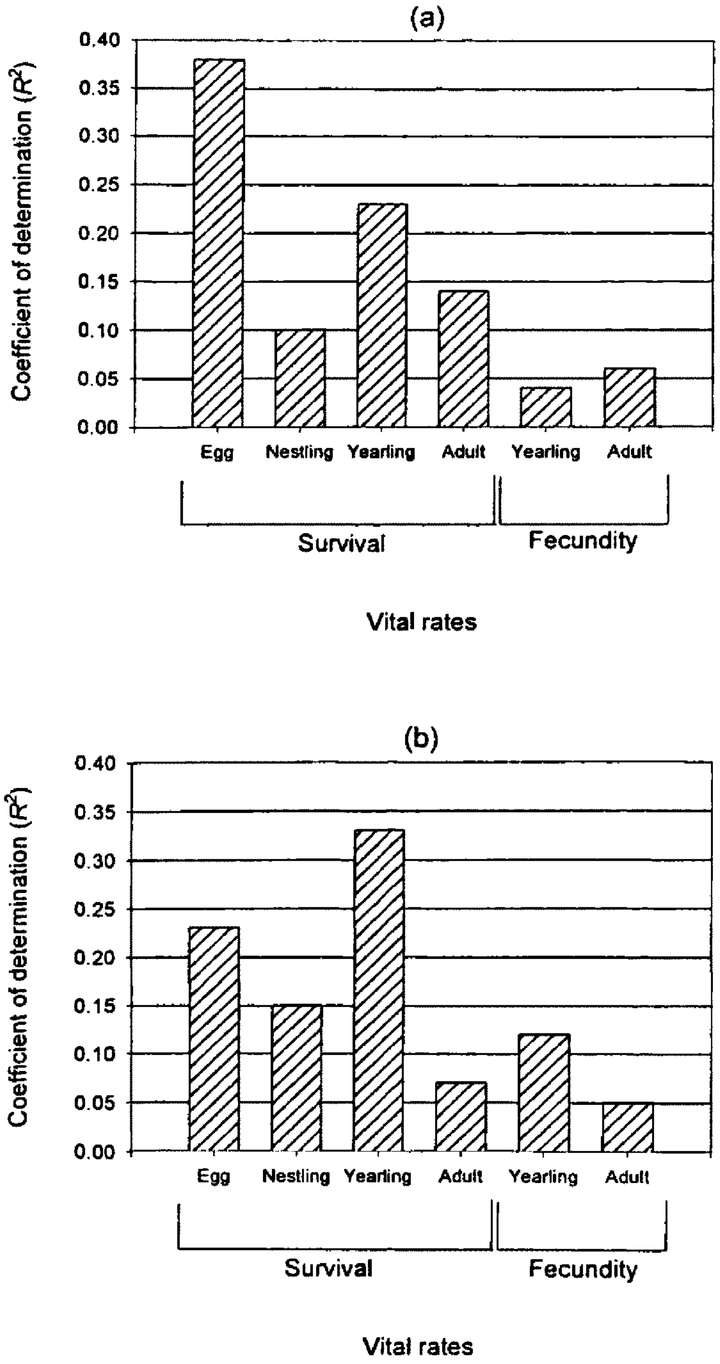


FIGURE 7. Number of individual eggs, nestlings, yearlings, and adults removed and the resulting population growth rates for a hypothetical population of 5,000 cowbirds. Removals are assumed to impose additive mortality. See APPENDIX B for details.

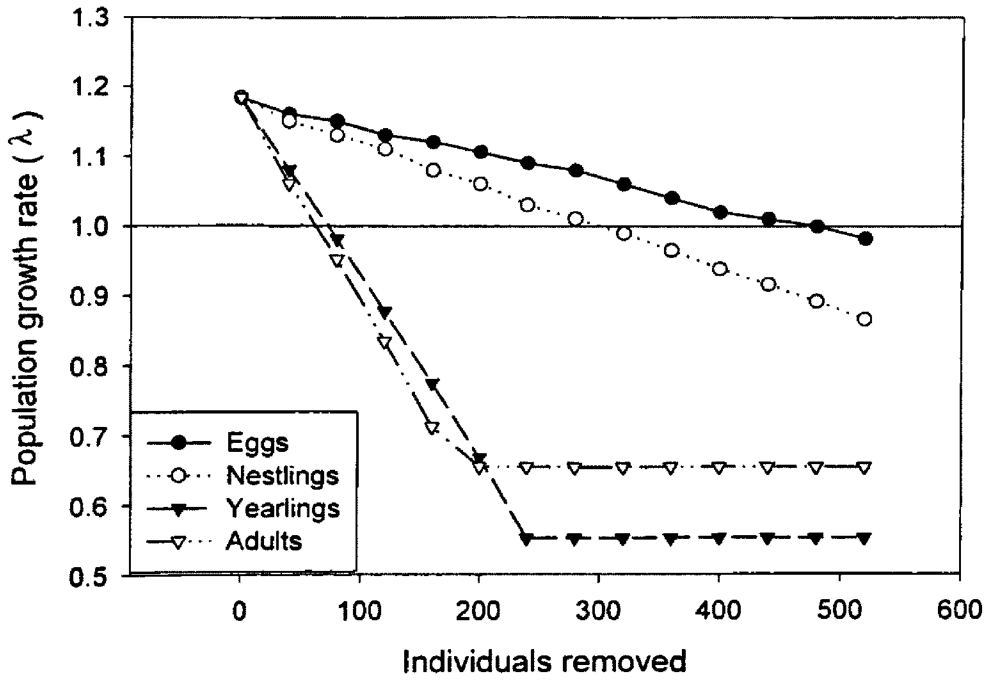
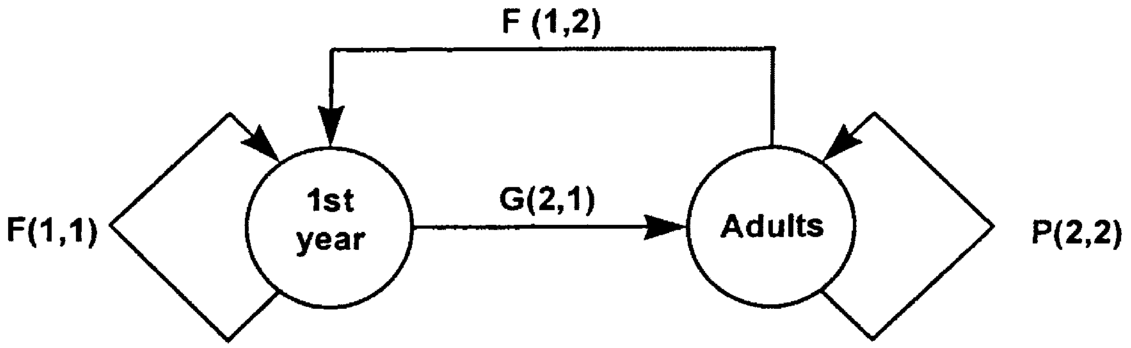


FIGURE APPENDIX 1. Life cycle diagram used for the cowbird Brown-headed Cowbird analysis.



Chapter 2: An experimental field test of sensitivity analysis models

John J. Citta

Wildlife Biology Program, School of Forestry, University of Montana, Missoula, MT 59812

ABSTRACT

While demographic sensitivity analysis is commonly used to guide conservation efforts, such analyses are virtually never field tested. I constructed a female-only, post-breeding matrix model for deer mice (*Peromyscus maniculatus*) and conducted four commonly used methods of sensitivity analysis: (1) elasticity; (2) Life Table Response Experiments (LTRE); (3) Life-stage Simulation Analysis (LSA); and (4) a simulation-based manual perturbation model. All analyses indicated that population growth rate should be sensitive to perturbations in adult female survival and relatively insensitive to perturbations in juvenile female survival. These predictions were tested using three treatments: (1) a 50% reduction of adult female survival; (2) a 50% reduction of juvenile female survival; and (3) a control. Counter to the predictions, treatments were equally capable of reducing population growth rate over a short time span and had few effects over longer periods of time. Using my field data, I identify non-stable age distributions, vital rate compensation, and demographic stochasticity as factors that may have lead to our lack of treatment effects. Because both non-stable age distributions and vital rate compensation can drastically alter the predicted sensitivity of demographic rates independent of population size, I recommend using methods of sensitivity analysis which

incorporate actual age distributions. By designing monitoring programs to estimate demographic rates, demographic compensation can be identified and used to re-evaluate predictions of sensitivity and improve management.

INTRODUCTION

The scarcity of funds and the inherent complexity of natural systems impose formidable barriers to managers striving to determine appropriate management of wild populations. Clearly, objective and cost-effective tools that provide a reliable assessment of management options are needed. Ideally, such tools can be used to provide insight into the efficacy of management options before implementation, thereby reducing negative consequences and cost associated with inappropriate management.

Recently, there has been much interest in matrix-based analyses. Such analyses combine estimates of reproduction and survival in a matrix-based format which accounts for known population structuring (Caswell 1989a; Burgman et al. 1993). One of the most popular uses for such matrix-based analyses are demographic sensitivity analyses (e.g. Crouse et al. 1987; Crowder et al. 1994; Marschall and Crowder 1996; Crooks et al. 1998), which measure the "sensitivity" of population growth rate to perturbations in specific vital rates (i.e. birth and death rates) associated with an organism's life history.

Matrix-based methods are appealing because they are relatively easy to use and focus directly on population growth rates. This is an improvement upon other assessment techniques, such as habitat modeling, which must assume a link between habitat factors, the presence or density of a species, and population growth (Van Horne 1983; Vickery et al. 1992). Similarly, key factor analyses (Morris 1959; Podoler and Rogers 1975) are based upon mortality and never address population growth rate (Royama 1996).

Despite the obvious advantages of examining population growth directly, matrix-based analyses have a number of problems. First, the analyses require data that are difficult to collect, and parameterizing matrix models is difficult even for well studied species. The compensatory structure (e.g. density dependent function) and spatial/temporal variation of vital rates are rarely known, even though techniques exists which can account for such complex vital rate structuring (see van Tienderen 1995). These problems are especially acute for endangered species, where *a priori* assessment of management alternatives is critical and experimentation may lead to species extinction. Consequently, most matrix-based analyses rely on a mean matrix of "best guess" vital rates. When variance in vital rates can be included in such analyses, it is usually assumed that vital rates do not covary and variance is rarely partitioned into real vital rate variance and sampling variance (as recommended by Burnham et al. 1987; Link and Nichols 1994).

Second, most matrix-based analyses are deterministic. In particular, metrics of population growth usually rely on asymptotic properties and assume a stable age distribution (SAD)(but see Tuljapurkar 1997).

Third, matrix-based sensitivity analysis may be difficult to translate into well-defined management actions. Although it is typically assumed that management actions should focus upon the vital rates with the highest impact upon population growth rate, this may be misleading, because the sensitivity of population growth is dependent on both the inherent sensitivity of a vital rate and the amount that a given vital rate varies (Mills et. al. In Press). Vital rates predicted by models to be of high importance may actually vary little in nature (Pfister 1998, Gaillard et al. 1998) or may be impossible to manipulate effectively (Citta and Mills In Press). Interpretation is further complicated by the fact that limiting factors (Silvertown et al. 1996) and/or stochastic events may regulate long-term population growth independent of management actions.

Clearly, it is necessary to have a better understanding of how well sensitivity analyses predict population growth in actual management scenarios. I conducted a matrix-based demographic sensitivity analysis for common deer mice (*Peromyscus maniculatus*) and then experimentally tested these predictions with different vital rate manipulations. Because the predicted changes in population growth did not occur, I used data from the experiment to examine biological processes that the sensitivity analyses did not account for.

STUDY SYSTEM

While sensitivity analysis is usually applied to the conservation of endangered or game species (e.g. Noon and Biles 1990; Doak et al. 1994; Wisdom and Mills 1997, Flint et al. 1998), field testing sensitivity analysis models with such species is difficult because the target species are scarce, have poorly understood demography, or are wide ranging and difficult to manipulate. I chose deer mice as a study species because they are abundant, have relatively well understood demography, small home range sizes, and high rates of population growth. Furthermore, sampling of small mammals is well-developed in theory and practice (e.g. Pollock et al. 1990) and deer mice populations are relatively easy to manipulate. This model system allowed me to collect a relatively large amount of data on population processes in a short period of time (< 2 breeding seasons).

Deer mouse model

Following the usual approach to sensitivity analysis, I first collected vital rates via literature review (Table 1), then created a stage-based, female only matrix model with a 30 d projection interval (Appendix A). A female-only model is justified, because females are usually considered to be the limiting sex. Females have smaller territories (Metzgar 1979; Van Horne 1981; Taitt 1981) with less home-range overlap than males (McCabe and Blanchard 1950; Blair 1940).

Density dependence of vital rates (the change of birth and death rates by animal density) was not included in the matrix model, because such relationships are largely unquantified – despite deer mice being relatively well studied. A number of studies provide evidence to show that adult female deer mice may regulate population densities in lab situations by either aggressively defending litters (Savidge 1974a,1974b; Ayer and Whitsett 1980) or by reproductively inhibiting juvenile female deer mice (Terman 1965, 1973, 1979, 1980). Field experiments also show an inverse correlation between the density of adult females and juvenile survival (Taitt 1981; Galindo and Krebs 1987). Adult male mice may also regulate population density by reducing juvenile survival (e.g. Healy 1967; Petticrew and Sadleir 1974) or by forcing non-breeding or subordinate male mice to disperse during the breeding season (Fairbairn 1977). Wolff (1985a) found that aggression was not detectable until densities exceeded 25-30 mice per hectare.

Density dependence was not included in the model, because the evidence is not sufficient to develop predictive functions that explicitly involve the rate of reproduction or survival as a function of population density. First, there are multiple mechanistic hypotheses which can explain the same compensatory phenomena. Second, while the laboratory-based studies (e.g. Terman 1965, 1973, 1979, 1980) are probably the most convincing, the densities at which females inhibit reproduction or express aggression are artificially high compared to wild deer mouse densities. Third, aggression rates are believed to interact with other factors, such as food availability (Taitt 1980; Gilbert and Krebs 1981; Teferi and Millar 1993), that make it difficult to parameterize a matrix model.

The matrix model also assumed that survival and reproductive capability are uncorrelated. Individuals may have correlated rates of survival and reproduction (i.e. individuals of poor condition likely survive and reproduce poorly). However, matrix models assume that all members of an age class have the same rates of survival and reproduction and, for deer mice, I could find no information to parameterize a more complex individual-based model.

METHODS OF SENSITIVITY ANALYSIS

I predict the sensitivity of population growth rate (λ) to perturbations in specific deer mouse vital rates with four techniques: (1) elasticity of a mean matrix; (2) Life Table Response Experiments (LTRE); (3) Life-stage Simulation Analysis (LSA); and (4) a simulation-based manual perturbation technique.

Elasticity of a mean matrix

Elasticities are defined as the proportional infinitesimal change in geometric population growth rate at stable age distribution (λ) given an proportional infinitesimal change in a vital rate, while all other vital rates are held constant (Caswell et al. 1984; de Kroon et al. 1986; Caswell 1989a). Specifically, for matrix element a_{ij} (row i, column j):

$$\textit{Elasticity } (e_{ij}) \text{ of } a_{ij} = \left(\frac{v_i w_j}{\langle v, w \rangle}\right)\left(\frac{a_{ij}}{\lambda}\right), \qquad (1)$$

where v and w are the vectors of reproductive values and stable age distributions (SAD) associated with matrix a, respectively, $\langle v, w \rangle$ is the scalar product of the two vectors, and λ is the dominant eigenvalue of the matrix. Because treatments were imposed on a proportional scale (see Experimental Methods section), the *a priori* predictions are based upon vital rate elasticity and not the analogous measure, sensitivity, that is more appropriate for infinitesimal absolute changes (Caswell 1989a, Horvitz et al. 1997).

When matrix elements are composed of more than one vital rate, component elasticities can be calculated for each vital rate that appears in one or more matrix elements. For n elements that contain vital rate x, the elasticity of x is:

$$\sum^{n}\left[\left(\frac{v_i w_i}{\langle v, w \rangle}\right)\left(\prod \text{non - x components}\right)\right]\left(\frac{a_{ij}}{\lambda}\right). \qquad (2)$$

Life Table Response Experiments (LTRE)

Life Table Response Experiments are closely related to traditional sensitivities and elasticities. To account for unequal variation in vital rates, LTRE weights traditional sensitivities by the variation in a particular vital rate (Caswell 1989b; Brault and Caswell 1993; Horvitz et al. 1997; Ehrlen and van Groenendael 1988). Because treatments were imposed on a proportional scale (see Experimental Methods section), elasticities were used to calculate LTRE effects rather than sensitivities. The weighted elasticity of vital rate a_{ij} is:

$$\sqrt{V_{ij}} \times e_{ij} \tag{3}$$

where $\sqrt{V_{ij}}$ denotes the standard deviation in matrix element a_{ij} and assumes no covariation in vital rates.

The weighted elasticities of the underlying vital rates can also be computed for n elements that contain vital rate x. The LTRE for component vital rate x is:

$$\sqrt{V_x} \times \sum^{n}\left[\left(\frac{v_i w_i}{\langle v, w\rangle}\right)\left(\prod \text{non - x components}\right)\right]\left(\frac{a_{ij}}{\lambda}\right). \tag{4}$$

While weighting elasticities by variance in vital rates improves predictions of population change, LTRE requires all other assumptions of elasticity analysis.

Life-stage Simulation Analysis (LSA)

The third method, Life-stage Simulation Analysis (LSA), accounts for large and/or simultaneous changes in vital rates. This is a simulation-based technique where replicate matricies are constructed with each vital rate of each matrix randomly chosen from a distribution bounded by empirically determined upper and lower limits (see Wisdom and Mills 1997). Population growth rate at SAD (λ) is then calculated for each matrix. The relative importance of a stage specific vital rate is assessed by regressing λ for each replicate against the value of that rate for all replicates to derive coefficients of determination (R^2). In terms of traditional definitions of sensitivity, R^2 for any component vital rate is analogous to the squared sensitivity weighted by the relative variance of a vital rate (H. Caswell, pers. comm.). Thus, this approach is similar to the analytical covariance technique used by Brault and Caswell (1993), but is simulation-based.

The regression method allows specification of the range and distribution of variation based on natural variation. Because all possible λ are plotted, the regression technique also has the advantage of being able to detect non-linearities that traditional methods may not (Wisdom et al. In Press).

Simulation-based manual perturbation technique

There are a number of sensitivity analyses that do not have a formal methodology, but that share one important attribute: effects of perturbations are evaluated by manually altering vital rates and comparing resulting population growth rates. While the modeler may examine any combination of perturbations believed to be important and these analyses can be very realistic, the modeler must also be able to choose representative perturbations from an infinite realm of perturbation combinations and levels. These techniques usually rank vital rate importance by how much a metric of population growth (usually λ) or likelihood of extinction changes when vital rates are perturbed (e.g. Noon and Biles 1990; Heppel et al. 1994; Akcakaya and Atwood 1997). For example, Heppel et al. (1994) alter each vital rate of red-cockaded woodpeckers (*Picoides borelais*) by 5, 10, and 25%; and rank the vital rates by how much λ alters due to each perturbation.

MODEL PREDICTIONS

Elasticities were derived for the mean matrix (Appendix A) using equation 2 (Figure1). Elasticities weighted by vital rate variance (LTRE) were generated using elasticities derived from the mean matrix and multiplying them by the variance in the total range of variation (equation 4) as determined via literature review (Table 1). For the third technique, Life-stage Simulation Analysis (LSA), I selected vital rates from a uniform distribution, bounded by the range of variation as determined via literature review (Table 1). I use a uniform distribution because there is no *a priori* knowledge to suggest another distribution, and because the experimental treatments should result in large changes from mean vital rates being as or more likely than small changes.

Last, I manually decreased the vital rates one-at-a-time in the mean matrix by 50% for the simulation-based manual perturbation analysis. This directly emulates the experimental treatments (see Experimental Methods section). The sensitivity of population growth is reported as the change in λ after vital rates are altered.

Interestingly, all four methods of sensitivity analysis indicate that monthly population growth is most sensitive to perturbations in adult survival (Figure 1). Thus, following traditional approaches in a management or research context, managers would focus the efforts upon increasing adult survival if deer mice were an endangered species or decreasing adult survival if deer mice were a pest species. I tested these *a priori* predictions in wild populations of deer mice.

EXPERIMENTAL METHODS

The three experimental treatments, conducted on wild populations of deer mice at the Lubrecht Experimental Forest in western Montana, were: (1) an adult removal treatment where 50% of all adult female mice were removed (thereby decreasing adult survival); (2) a juvenile removal treatment where 50% of all juvenile female mice were removed (thereby decreasing juvenile survival); and (3) a control treatment in which no mice were removed from the population. Removal treatments focused upon female mice, because females appear to be the limiting sex in deer mouse systems (Metzgar 1979;

Terman 1980; Van Horne 1981; Taitt 1981; Galindo and Krebs 1987). A removal of 50% of the target age and sex class should functionally decrease the survival rate of that age class by 50%. The hypothesis, based upon sensitivity analysis, was that control treatments would have the highest population growth rates, followed by lower population growth rates for juvenile removal treatments, followed by even lower population growth rates for adult removal treatments.

All experiments were carried out in replicate 0.81 ha pens (90 m X 90 m), to facilitate delineation of separate populations of deer mice and to eliminate the effects of dispersal. Pens had walls of high-density polyethylene plastic sheeting buried 1 foot below ground and extending 3 feet above ground; in both years of the study, there were low movement rates into and out of pens (1996 = 5.00%; 1997 = 1.25%). Movement rates between pens decreased in 1997, because trees near enclosure walls were encircled with sheet metal to prevent mice from moving through the forest canopy.

One trial with no replication (i.e. three pens with one treatment each) was conducted on naturally-occurring populations within the enclosures during the 1996 breeding season. In 1997, construction of the enclosures was completed and treatments were assigned according to a randomized block design with a total of three blocks. Each block had three pens with one treatment each. Because I could only trap and manipulate populations of mice within one block at a time and because each block of pens had slightly different levels of canopy closure, blocking controlled for time differences between treatments and slight differences in canopy closure. At the beginning of the 1997 breeding season, mouse densities were very low (< 1 mouse per hectare), presumably due to a record snowfall the previous winter. Consequently, I stocked the enclosures with mice. Each enclosure was stocked with a total of 12 mice – 3 juvenile males and females and 3 adult males and females – from the surrounding forest. I then let the mice adjust to their surroundings for 4 weeks prior to any trapping or manipulations.

Trapping followed Pollock's robust design (Pollock et al. 1990). Trap spacing was 12.8 meters, allowing for 49 sherman live traps on a 7 by 7 grid. At the beginning of the season, when daily capture probability (*p*) was high (0.75 - 1.0), all trap sessions were 3-4 d in length. As the season progressed and *p* decreased, trap sessions were lengthened to 6-8 d in length.

During the first capture session, I assessed population size and then removed 50% of the target age class. I then assessed population size and growth rate every two weeks after the initial date of manipulation.

Data analysis

The Lincoln-Petersen estimator (Seber 1980; Pollock et al. 1990) was used to calculate population size ($\hat{N}$) within each trap session (*t*) and calculate observed population growth rate rates over each interval of two weeks as:

$$\hat{\lambda}_{obs} = \frac{\hat{N}_{t+1}}{\hat{N}_t} \qquad (6)$$

Observed population growth rate (λ_{obs}) differs from the geometric population growth rate (λ) (the dominant eigenvalue of the matrix), because λ_{obs} does not require the assumption of SAD. After manipulating vital rates, λ_{obs} should converge on λ as age distributions stabilize.

Following Mood et al. (1974), we estimate variance of $\hat{\lambda}_{obs}$ as:

$$\hat{\mathrm{var}}\ \hat{\lambda}_{obs} = \mathrm{var}\left[\frac{N_{t+1}}{N_t}\right] \approx \left(\frac{N_{t+1}}{N_t}\right)^2 \left(\frac{\mathrm{var}(N_{t+1})}{N_{t+1}^2} + \frac{\mathrm{var}(N_t)}{N_t^2} - \frac{2\mathrm{cov}(N_{t+1}N_t)}{N_{t+1}N_t}\right) \qquad (7)$$

Because covariances cannot be calculated with the Lincoln-Petersen estimator, covariance is assumed to be 0.0. Point estimates of λ_{obs} had very low standard errors (Figures 2 and 3). I only statistically analyze the data from 1997, because λ_{obs} can only be calculated for a one month interval in 1996 (block 4; Fig. 3b) and all other estimates for λ_{obs} are for two week time intervals. However, the 1996 data has the same trend as all other data. The number of trap sessions differed between blocks, because of different starting dates and because a number of pens went extinct or nearly so. In 1997, there was an average of 8 mice per pen (after survival reductions) and I ceased monitoring a block when a pen went extinct.

RESULTS

Two weeks from the initial date of manipulation, it appeared that reducing survival had an effect on population growth rates. However, contrary to the predictions of sensitivity analyses, it did not matter what age class had reduced survival (Figure 4a). Actual population growth from two to four weeks (i.e. $\lambda_{obs} = N_{t=4\ \mathrm{weeks}} / N_{t=2\ \mathrm{weeks}}$) indicates that there was no longer any difference between any of the treatments (Figure 4b). In all cases, the precision of average λ_{obs} is very low. Low precision is due to true process variation and not sampling error, as the individual estimates of λ_{obs} all have small standard errors (Figures 2 and 3).

FACTORS INFLUENCING EXPERIMENTAL RESULTS

Sensitivity analysis predicted strong differences in population growth rate across the treatments, but no such effects were sustained. Four primary factors may have led to the lack of treatment effects: (1) experimental perturbations were insufficient to cause a large effect; (2) non-stable age distributions; (3) vital rate compensation; and (4) demographic stochasticity due to small population size. Because all of these could be relevant to the application of sensitivity analysis in wild populations, I consider each of these factors in turn. Where applicable, I re-evaluate sensitivity with data collected during the study (Table 2).

Insufficient perturbations

Sensitivity analyses usually assumes that vital rate perturbations change the mean values through time. However, instead of altering mean survival rates during the entire study, I lowered survival rates only once, as might occur under many management plans (scenario #2, Table 2). To investigate the effects of altering survival rates only once, I created a deterministic model which used the vital rates and matrix from the sensitivity analysis (Appendix A). Observed population growth rate was calculated as: $\lambda_{obs} = N_{t+1} / N_t$; hence the model did not let the population attain SAD before estimating population growth, but calculated population growth between each projection interval.

Using this projection model I assumed the starting population was at SAD and removed one half of the individuals in the target age class and then projected the model 5 projection intervals into the future. Population growth from one time step to the next changes only immediately after the perturbation (Figure 5). After 1 projection interval, λ_{obs} for each treatment varies little from the control λ_{obs}. However, treatment effects should still exist over short time intervals.

Non-stable age distributions

I next investigated the possible consequences of non-stable age distributions. All sensitivity analyses assumed populations were at stable age distribution (SAD). It is unknown how often populations in the wild are at SAD; however, because vital rates (and underlying age distributions) vary due environmental variation, predictions of sensitivity may also vary.

To evaluate the effect of non-stable age distributions, I first compared actual age distributions from the experiment (i.e. the empirical age distributions) with the stable distributions the model generated. Unfortunately, the exact age distributions could not be determined from field data, because the youngest of the three age classes (pre-weaning stage) is not trappable. Therefore, the proportion of adults (P_a) in the wild populations was used as an index to age distribution. Because the model indicates that at SAD there are equal proportions in all age classes (Appendix A), the observed proportion of adult females to juvenile females during the field study should equal 0.5. In the experimental pens, P_a was generally (in 9 of 12 cases) skewed low. With the exception of one outlier, all P_a values are between 0.2 and 0.6.

How much would a juvenile-biased sex ratio alter predictions of sensitivity? To simulate treatments, I assumed pre-weaning mice (which were invisible to sampling) were of the same proportion as juveniles and then projected λ_{obs} for various starting age ratios (P_a = 0.2-0.6), removed one half of the target age class, and calculated λ_{obs} (scenario #3, Table 2). As P_a decreases, the large predicted effect of decreasing adult survival decreases. Contrary to the *a priori* predictions, the impact of reducing juvenile survival exceeds that of reducing adult survival when P_a is biased below 0.3 (Figure 6).

Compensation of vital rates

The perturbations imposed on the populations of mice may have been counteracted by vital rate compensation. To investigate the effects of such compensation, empirical vital rates were estimated and then incorporated these vital rates into the projection model.

I estimated survival rate by treatment with actual field data using Pollock's robust design (Pollock et al. 1990, Kendall et al. 1997) and Program MARK (see Cooch and White 1998). Survival rate is estimated for the two blocks of pens which could be trapped for more than two consecutive primary capture occasions. Goodness-of-fit testing is poorly developed for complex models (Leberton et al. 1992) and model the fit of models could only be tested to the level of the population (see Appendix B for details). While the analyses did not detect differences in reproduction, there were higher post-treatment survival rates in pens where adult mice were removed (Figure 7).

I re-evaluated the predictions of sensitivity by incorporating the empirically derived survival rates into the projection model to investigate treatment specific compensation of survival rate across the empirical range of age ratios (scenario #4, Table 2). Vital rate compensation drastically alters the predicted sensitivity of λ_{obs}; after removing adult mice, the resulting higher post-treatment survival rates is predicted slightly increase population growth relative to the control treatment or have no effect (Figure 8). While λ_{obs} interacts with the proportion of adult females (P_a), this interaction has little effect (compare Figure 6 with Figure 8) and reducing juvenile survival consistently alters λ_{obs} more than reducing adult survival. However, neither treatment is expected to drastically alter λ_{obs}.

Demographic stochasticity

Demographic stochasticity, the variation inherent in a population because organisms reproduce and die as discrete units, may cause population growth rate to fluctuate greatly when population sizes are small (Burgman et al. 1993; Akcakaya et al. 1997). The maximum population sizes were as high as 30 mice, but during 1997 the average population size was 8 mice. Because of the low population sizes, demographic stochasticity could have overwhelmed treatment effects. I used a commercially available software package, RAMAS/age (Ferson and Akcakaya 1988), to model demographic stochasticity (Scenario #5, Table 2). RAMAS/age is a matrix-based simulation package which models demographic stochasticity by selecting vital rates from a binomial distribution. I input the mean matrix, starting age distributions, and compensatory vital rates into the program and then projected 500 replicate simulations one time step into the future; all simulations had a starting population size of 10 mice. I find that the demographic stochasticity could overwhelm treatment effects (Figure 9).

DISCUSSION

The best efforts to use sensitivity analysis to predict population change in deer mice were unsuccessful. Although the estimates of population growth were very precise, average growth rates across replicates had high variance. High variance is consistent with high levels of demographic stochasticity, but does not explain why sensitivity analysis incorrectly predicted point estimates of λ_{obs}.

Non-stable age distributions and vital rate compensation may have caused the unexpected point estimates of λ_{obs}. Observed age distributions were skewed away from SAD, towards juvenile mice, and the projection model indicated that this skewing could reduce or even reverse the predicted change in population growth resulting from the treatments. However, the simulations indicate that non-stable age distributions may be a minor problem compared to the compensation of vital rates. When predictions of sensitivity include empirical levels of compensation; that the change in observed growth rate (λ_{obs}) is much lower than originally predicted by the sensitivity models (compare Figure 6 with Figure 8).

The simulations indicate that non-stable age distributions and compensation may both be important, but what do the field data suggest? Immediately after the perturbations, λ_{obs} decreased for the survival reduction treatments (Figure 4a). This pattern is consistent with simulation predictions that account for non-stable age distributions, but not consistent with simulations that account for compensation of vital rates. From two to four weeks into the future (Figure 4b), observations are consistent with either predictions resulting from non-stable age distributions or vital rate compensation. Why was compensation not observed immediately after the experimental treatments? It is possible that vital rate compensation was not instantaneous (i.e. a time-lag existed) and that there was not enough data to identify such a time-lag.

Unfortunately, the effects of demographic stochasticity, variation in age ratios, and vital rate compensation cannot be separated. While deviations from SAD and vital rate compensation were observed and measured, the field data are not conclusive. I can only state that I did not observe the predicted effects and that all of these factors (deviations from SAD, vital rate compensation, and demographic stochasticity) may have played roles in obscuring treatment effects.

While populations of small mammals are clearly more variable than many populations of larger bodied vertebrates, the findings illuminate when sensitivity analysis is questionable and force consideration of how sensitivity predictions can be translated into management action. First, discrete treatments that do not permanently alter mean survival rates are not expected to change population growth rates for long periods of time. While this is not unexpected, commonly used models of sensitivity analysis do not explicitly explore the effects of discrete perturbations over short time periods. Specifically, models based on SADs are implicitly based on the assumption that vital rate perturbations are permanent.

Second, non-stable age distributions may play large roles in determining the true amount and direction of a vital rate perturbation. Environmental variation causes vital rates to fluctuate and results in fluctuating age distributions. Any perturbation that drastically altered age distributions in the recent past will alter predictions of sensitivity analysis in the near future regardless of current vital rates.

One special case of fluctuating age distributions is non-continuous breeding. As an example, deer mice only breed from the spring through the fall in the northern latitudes. In the spring, all individuals are adults and age distributions are drastically biased (i.e. the proportion of adults, $P_a \approx 1.0$). As new mice are born, age distributions become less biased towards adults and I would expect the sensitivity of λ_{obs} to perturbations in adult survival to be greatest in the early spring (when P_a is high) and lowest in the fall (when P_a is low). Interestingly, this change in sensitivity occurs regardless of the underlying vital rates. In other words, even if the vital rates in the matrix are invariant and representative of the population, the population will rarely be at SAD because breeding is not constant throughout the year. Hence, the true impact of perturbations depend upon when they are implemented.

Third, vital rate compensation may reduce or even reverse the predicted impacts of treatments. While this result is not unexpected, managers usually have little or no knowledge of what compensatory effects to expect. Fourth, the variance due to demographic stochasticity can be large and may limit how predictable models are when management perturbations are unreplicated.

CONCLUSIONS AND RECOMMENDATIONS

What does the experiment and subsequent modeling mean for practitioners of sensitivity analysis? First, age distributions can affect how well predictions match actual population growth. If age distributions are different from SAD or are likely to change, the preferred approach would be to use a simulation-based technique that calculates population growth from one projection interval to the next, without assuming SAD. Such models should also allow the practitioner to input actual age distributions.

Population Viability Analysis (PVA) may circumvent the assumption of SAD and provide a practical alternative to assessing vital rate sensitivity. Population viability models are usually matrix or individual-based and are traditionally used to estimate extinction risk. Usually, multiple stochastic simulations are projected in the future and extinction risk is indexed by the number of replicate simulations with population sizes below a threshold number at the end of the simulation period (See Burgman et al. 1993 and Beissinger and Westphal 1998 for detailed reviews). Population viability analysis models are stochastic and, therefore, vital rates and age distributions alter each time interval. As such, PVA models do not rely upon SAD; furthermore, many of the software packages can be parameterized with user defined age distributions at the beginning of each simulation (e.g. RAMAS and Vortex). Such a modelling framework can be used in conjunction with manual perturbations to rank the sensitivity of vital rates by summarizing how many replicate simulations have population sizes either increasing above or decreasing below a threshold population size. Akcakaya and Atwood (1997) and Marmontel et al. (1997) provide examples of how PVA models can be used to assess sensitivity of vital rates.

The problem of vital rate compensation is more difficult to overcome than non-stable age distributions. If the practitioner is not aware of compensatory mechanisms/functions, then management actions must be based upon models which temporarily ignore compensation. Without prior knowledge of compensatory structure, sensitivity analysis still provides the best indication of how to alter population growth rate. If management actions are performed in conjunction with monitoring programs that estimate vital rates, then management can be adapted to consider emerging compensatory patterns. In this study, I used field data to estimate survival rates and then used these empirical rates to re-evaluate predictions of sensitivity. Practitioners of sensitivity analysis could use this protocol to improve predictions of sensitivity and alter management perturbations as new knowledge arises.

My results also point to the importance of sustaining changes in vital rates. Unless mean vital rates are altered permanently, perturbation effects will be temporary and projection models will be necessary to determine how long (i.e. how many projection intervals) treatment effects will last.

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Table 1. Summary of deer mouse vital rates (female only).

Category	Mean rate	Range	Source	Number of estimates
Litter size	2.38	2.0 - 2.6	Blair 1940;	1
			Halfpenny 1980;	1
			Wolff 1985b;	1
			Millar et al. 1979;	2
Proportion Adults Breeding	0.61	0.39 - 0.84	Sullivan 1979;	6
			Sullivan and Sullivan 1981;	3
Time between litters	36 d	26.3 - 50 d	Millar and Innes 1983;	1
			McCabe and Blanchard 1950;	1
Pre-trappable survival	0.48 / 30 d	0.32-0.79[1]	Millar and Innes 1983;	4
			Sullivan 1979;	7
Time to ween	20 d	18.0 - 24.9	Millar 1982;	1
			Millar and Innes 1983;	1
			Millar et al. 1979;	1
			Halfpenny 1980;	1
			Millar 1985;	1
			King et al. 1963	1
Juvenile Survival	0.64 / 30 d	0.29 -0.86[1]	Sullivan 1979;	6
			Sullivan and Sullivan 1981[2] ;	3
			Van Horne 1981;	12
			Wolff 1985b	3
Adult Survival	0.61 / 30 d	0.26-0.83[1]	Sullivan 1979;	7
			Sullivan and Sullivan 1981;	3
			Van Horne 1981;	12
			Wolff 1985b	3

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[1] Vital rates of 0.00 or 1.00 are used to calculate mean rate, but not used in the range, as they cannot be typical of populations.

[2] Vital rates from Sullivan and Sullivan (1981) which are from "experimental" plots (treated with herbicides) are not used. I used only vital rates from the "control" or non-herbicide plots.

Table 2. Modeled scenarios, their data sources, and resulting predictions of vital rate sensitivity.

Scenario	Fecundity	Survival	Age distribution	Metric of population growth rate	Predictions of sensitivity
1. Matrix models are used to predict demographic sensitivity (Appendix A).	Literature review (Table 1).	Literature review (Table 1).	Stable age distribution (SAD)	Geometric population growth rate (λ).	Geometric population growth rate (λ) is much more sensitive to perturbations of adult female survival than juvenile female survival (Figure 1).
2. Actual perturbations and original matrix model used to examine observed population growth rate (λ_{obs}) instead of geometric population growth rate (λ).	Literature review (Table 1).	Literature review (Table 1).	Non-stable age distributions are calculated by the model from N_t to N_{t+1}.	Observed population growth rate $\lambda_{obs} = \frac{N_{t+1}}{N_t}$	λ_{obs} is more sensitive to perturbations in adult female survival than juvenile female survival, but effects last for only one projection interval (Figure 5).
3. The sensitivity analysis is improved with empirical age distributions.	Literature review (Table 1).	Literature review (Table 1).	Empirical age distributions.	$\lambda_{obs} = \frac{N_{t+1}}{N_t}$	Treatment effects are less for age distributions which are biased towards juvenile females. At high proportions of juvenile females, impacts of treatments are opposite of original predictions (Figure 6).
4. The sensitivity analysis is improved with empirical age distributions and empirical survival rates.	Literature review (Table 1) – data not sufficient to estimate reproduction	Empirical estimates (Figure 7).	Empirical age distributions.	$\lambda_{obs} = \frac{N_{t+1}}{N_t}$	λ_{obs} is more sensitive to reducing juvenile survival than for reducing adult survival (Figure 8).
5. Demographic stochasticity is incorporated into the sensitivity analysis to investigate the effects of small population size	Literature review	Empirical estimates	Empirical age distributions.	$\lambda_{obs} = \frac{N_{t+1}}{N_t}$	The error due to demographic stochasticity overwhelms treatment effects (Figure 9).

Figure 1. The sensitivity of population growth rate assuming stable age distribution (λ) to perturbations in specific vital rates for deer mice. All predictions are generated from the matrix model in Appendix A and with the methods described within the text. Because each technique (x-axis) yeilds a different metric of sensitivity, techniques are not directly comparable. However, qualitative rankings within techniques are comparable.

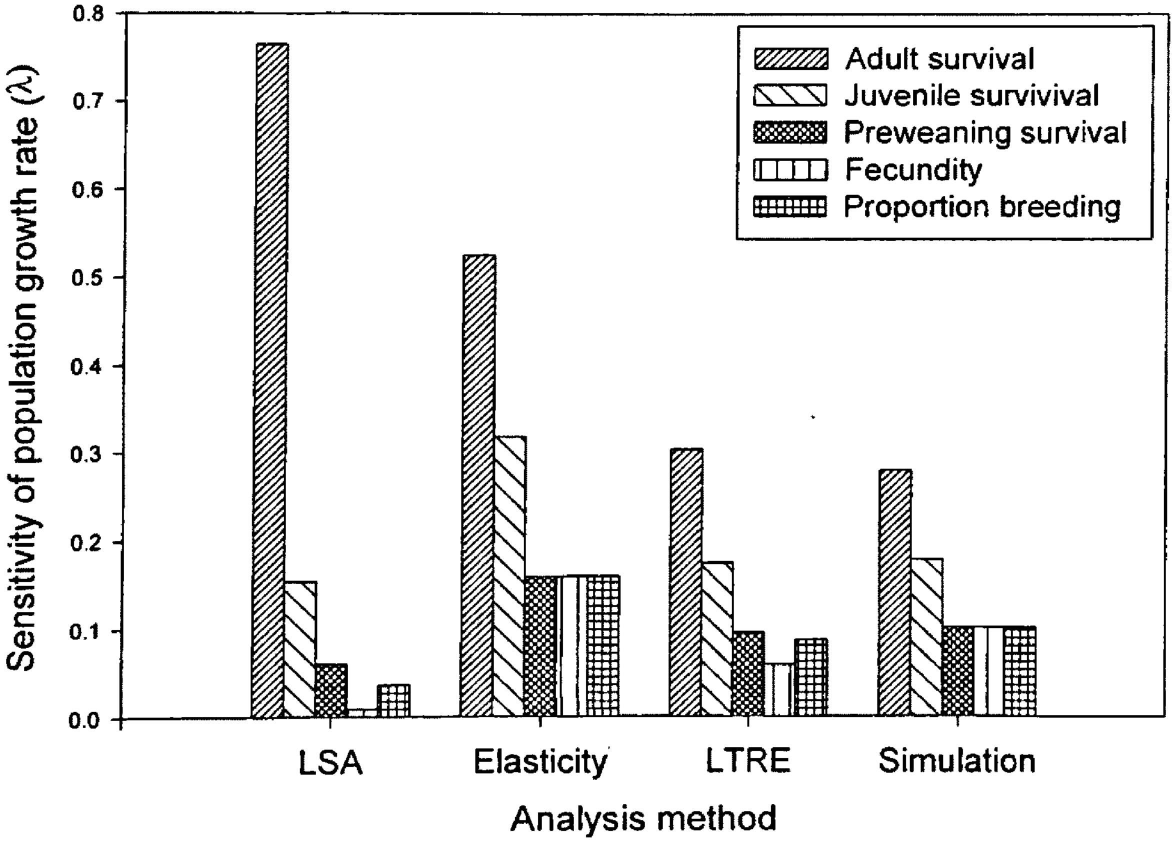


Figure 2. Estimates of observed population growth rate (λ_{obs}) and one standard error for (a) block 1 and (b) block 2 with the three survival reduction treatments. Each estimate of N was calculated with the Lincoln-Petersen estimator. Point estimates of λ_{obs} have low standard error due to high daily capture probability.

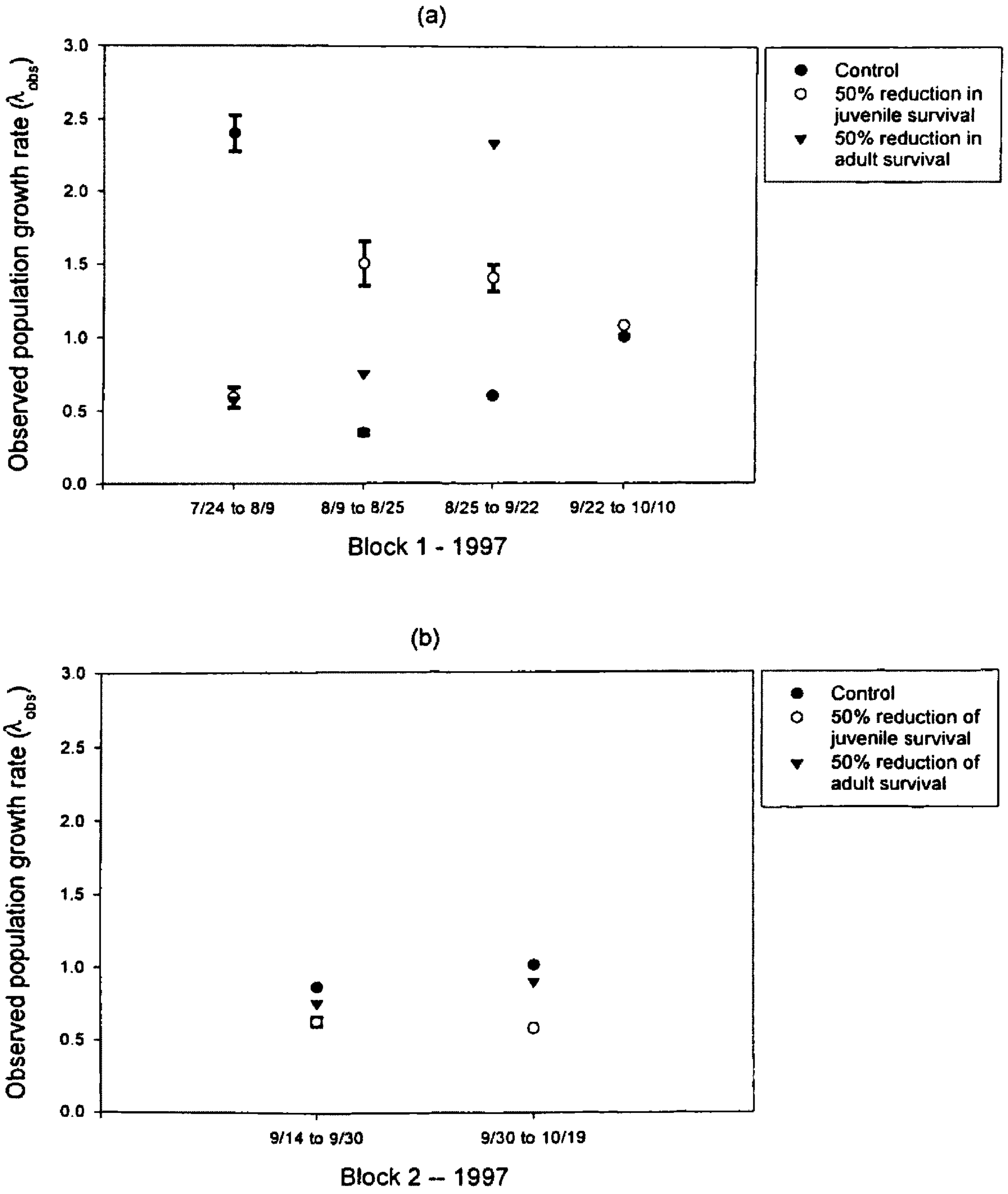


Figure 3. Estimates of observed population growth rate (λ_{obs}) and one standard error for (a) block 3 and (b) block 4 with the three survival reduction treatments. Each estimate of N was calculated with the Lincoln-Petersen estimator. Point estimates of λ_{obs} have low standard error due to high daily capture probability.

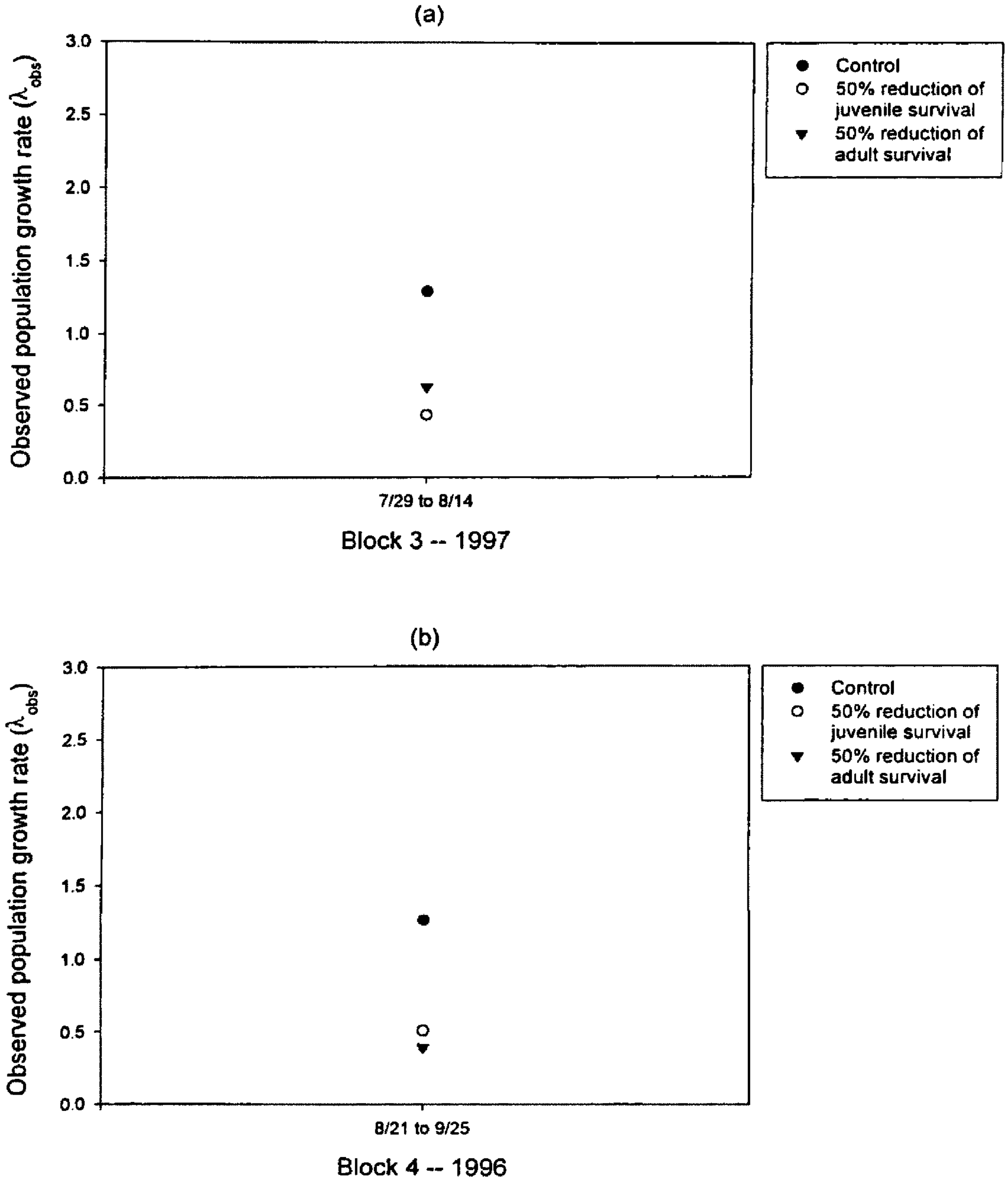
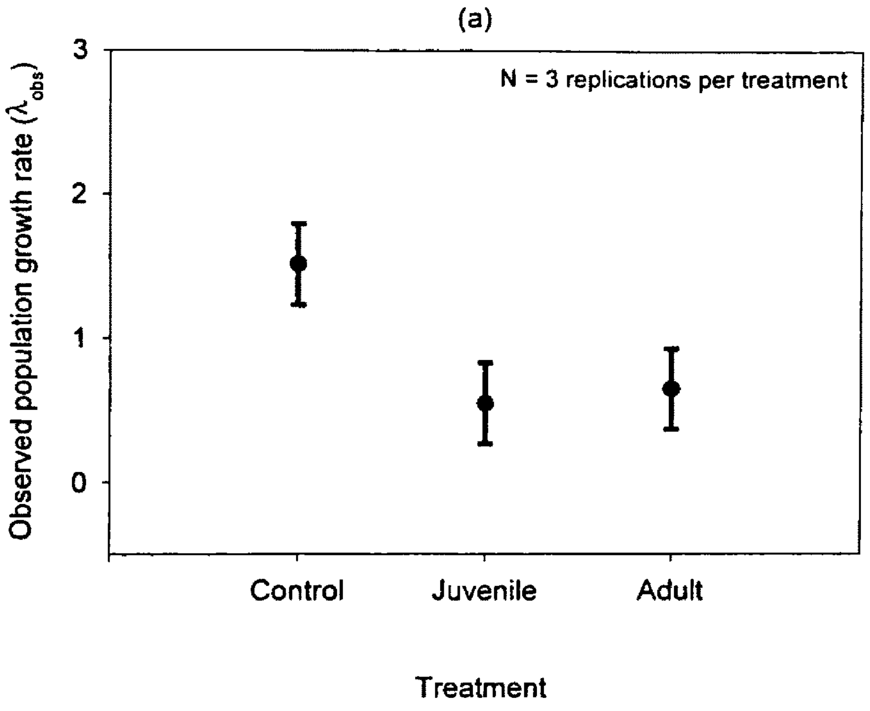


Figure 4. Change in observed population growth (λ_{obs} and 90%CI) following treatments of reducing survival by 50%. Presented are changes in λ_{obs} from (a) 0 to 2 weeks following treatment and (b) from 2 to 4 weeks following treatment.



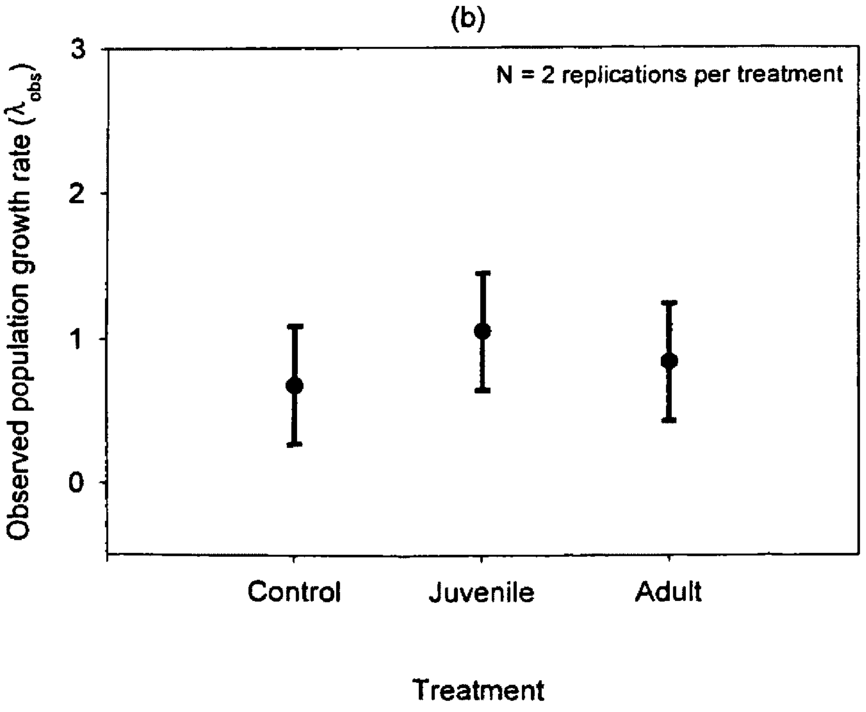


Figure 5. Modled impacts of treatments over 5 projection intervals. Perturbations were included in the model as they were performed in the experiment -- survival of the target age class was reduced by 50% prior to interval 1 and then populations are monitored over time. The change in observed population growth rate (λ_{obs}) is relative to the control treatment. After the initial perturbation λ_{obs} quickly recovers.

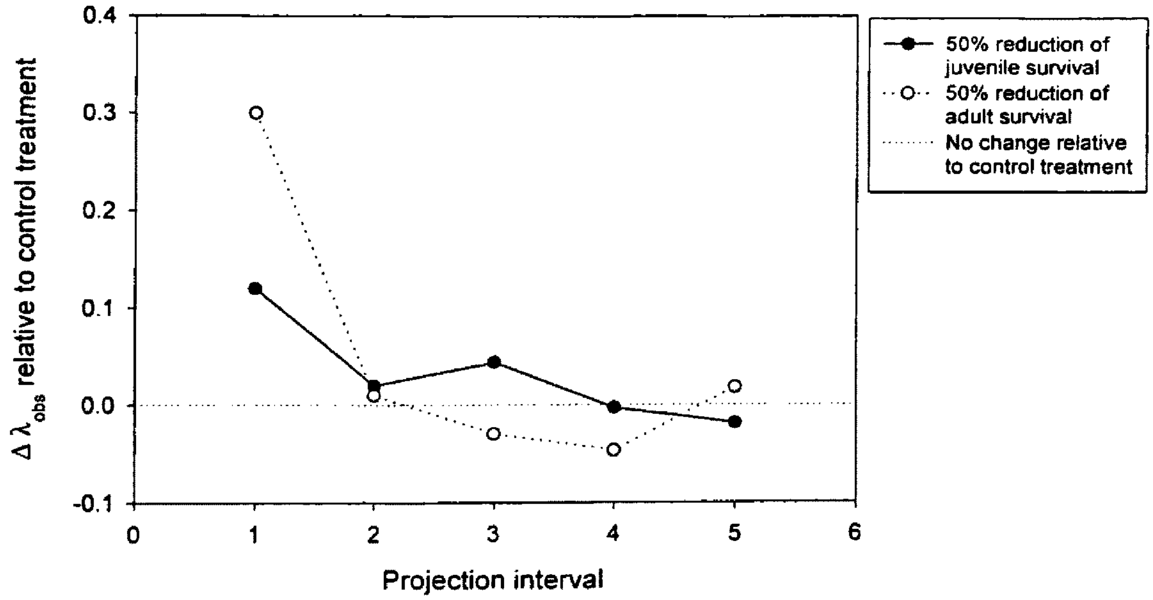


Figure 6. Modled impacts of biasing age distributions towards adult deer mice with five observed age distributions as indexed by the proportion of adults (P_a). Changes in observed population growth rate (λ_{obs}) are for one projection interval after the treatment perturbation. Perturbations were included in the model as they were performed in the experiment -- survival of the target age class was reduced by 50%. As P_a decreases to levels observed in the field experiment, reducing juvenile survival has equal or more impact than reducing adult survival.

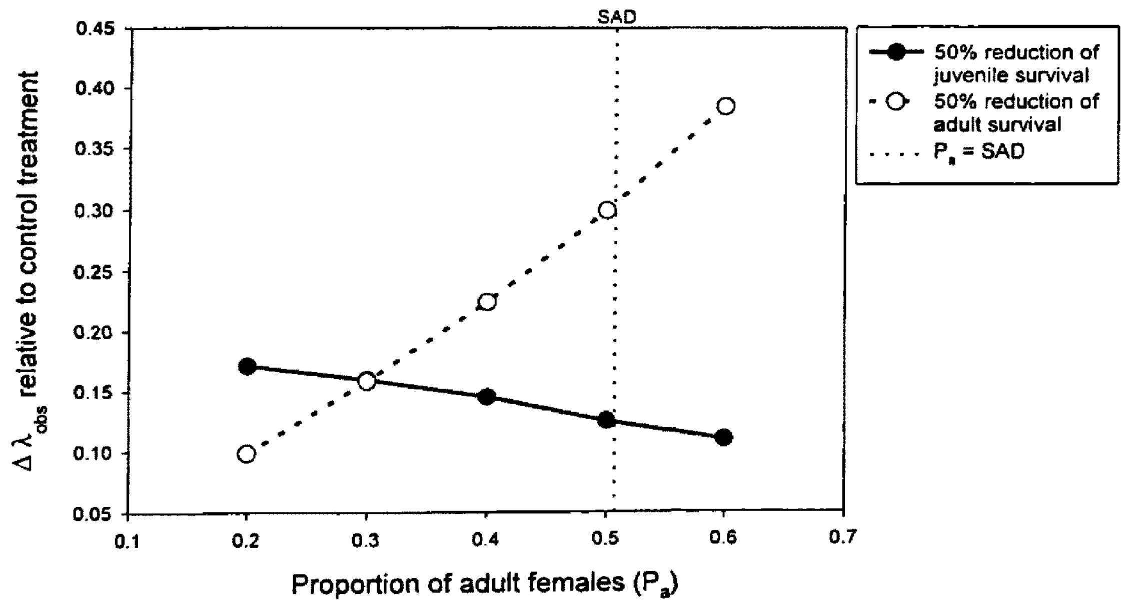


Figure 7. Survival rate and unconditional standard error specific to treatment. Treatments are: (1) control; (2) 50% reduction of juvenile female survival; (3) 50% reduction of adult female survival. Unconditional standard error indicates uncetainty in both the estimator and the information criterion used to select the models. Rates estimated with Program MARK (see Appendix B).

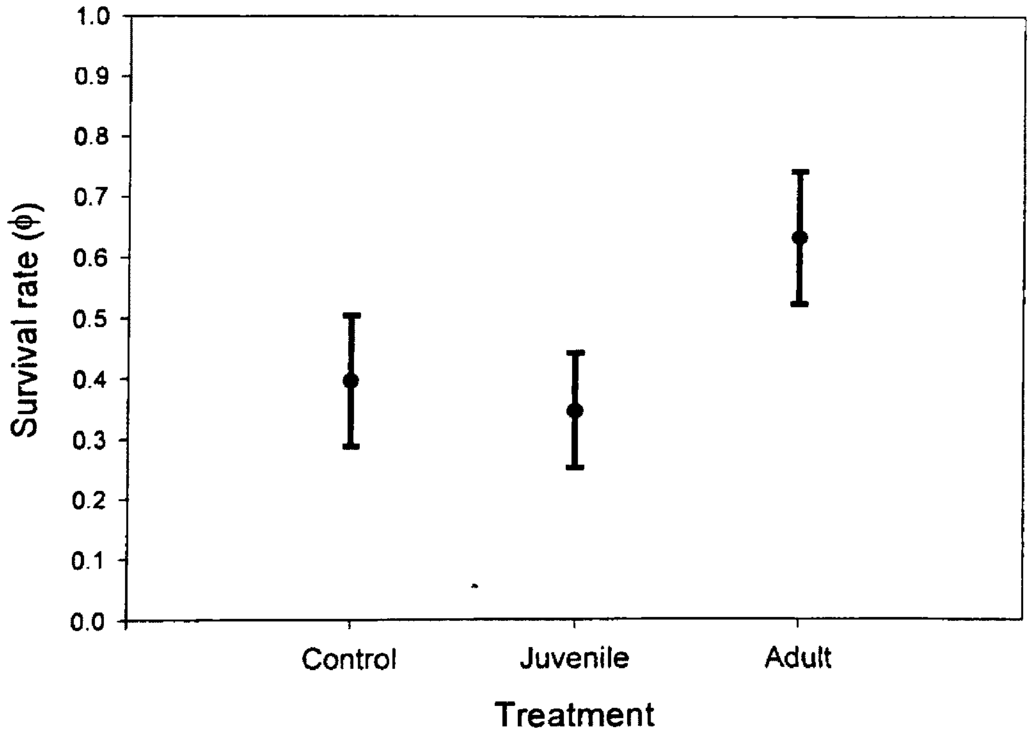


Figure 8. Modeled impacts of biasing age distributions towards adult deer mice across the empirical range of the proportion of adults (P_a) and while accounting for empirical treatment specific survival rates. Changes in observed population growth rate (λ_{obs}) are for one projection interval after the treatment perturbation. Perturbations were included in the model as they were performed in the experiment -- survival of the target age class was reduced by 50%. The y-axis ($\Delta\ \lambda_{obs}$) equals λ_{obs} of the control minus λ_{obs} of the survival reduction treatment. Compare with Figure 6; the empirical change in adult survival results in slightly higher λ_{obs} than control treatments.

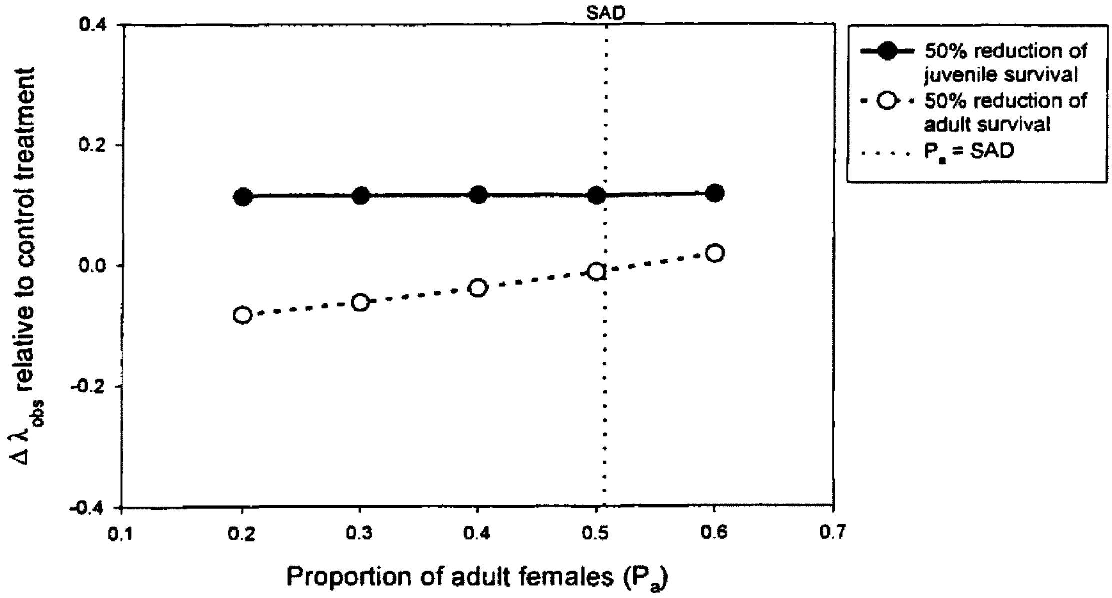
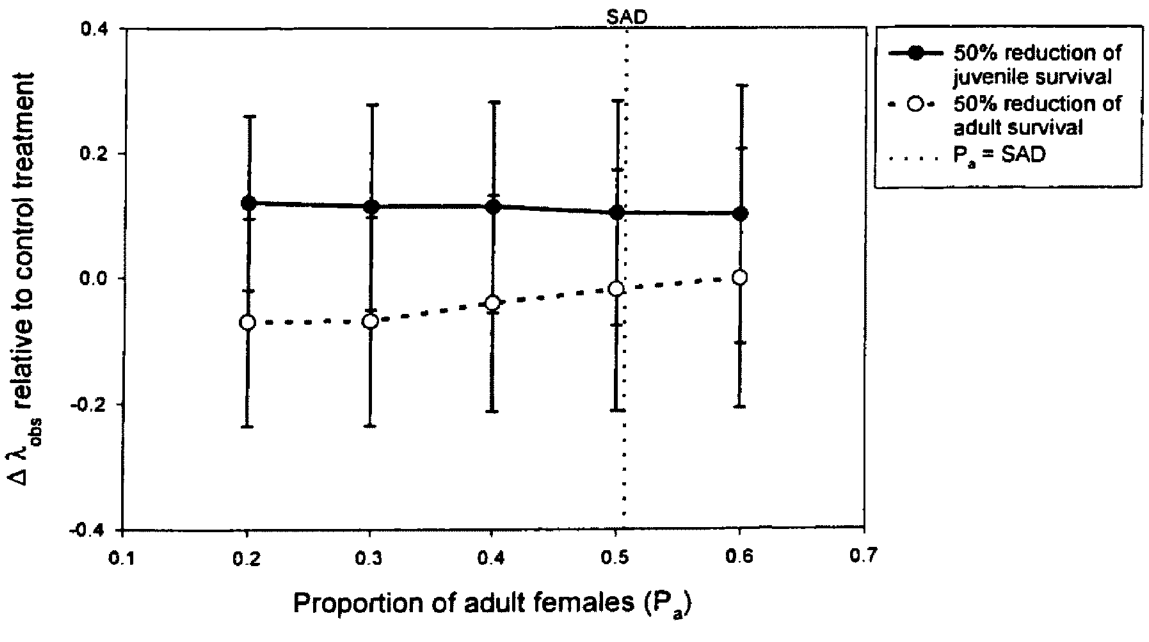


Figure 9. Modled impacts of vital rate compensation and demographic stochasticity across the empirical range of the proportion of adults (P_a). All simulations assumed a starting populaiton size of 10 mice. Error bars represent 1 standard deviation in population size across 500 replicate simulations. Changes in observed population growth rate (λ_{obs}) are for one projection interval after the treatment perturbation. Perturbations were included in the model as they were performed in the experiment -- survival of the target age class was reduced by 50%. The y-axis ($\Delta\ \lambda_{obs}$) equals λ_{obs} of the control minus λ_{obs} of the survival reduction treatment.

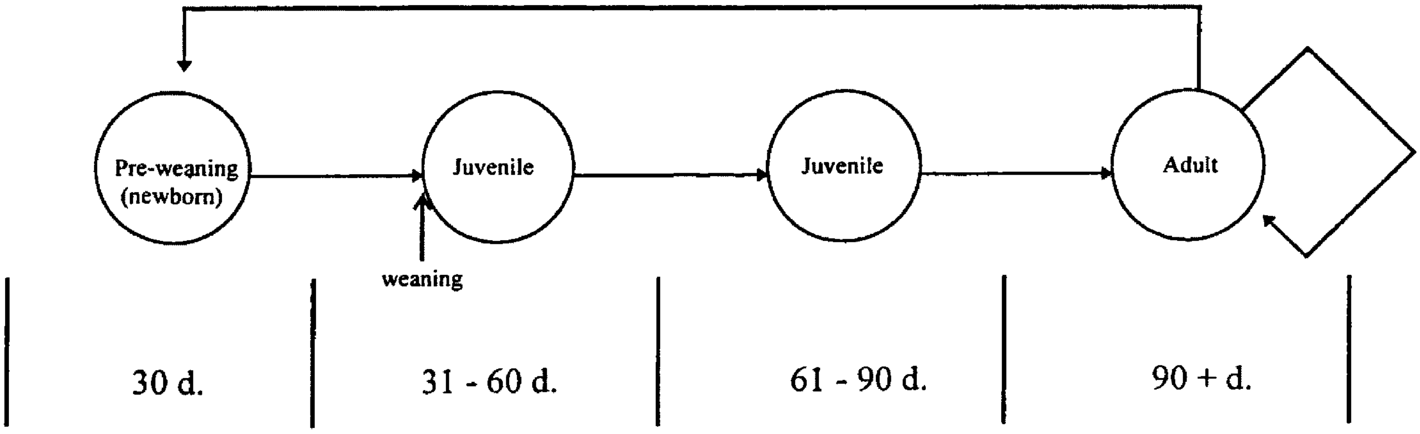


Appendix A. Deer mouse matrix model.

Projection interval: 30 d.

Stages:	Approximate ages (d.):	Duration of stage class (d.):
1 (pre-weaning)	<30	30
2 (1st juvenile)	31 - 60	30
3 (2nd juvenile)	61 - 90	30
4 (adult)	121 +	N/A

Deer mouse demography differs by season and I could not test the model in all seasons, the model was split into a breeding season and a non-breeding season matrix. Only the breeding season matrix is used for analysis; this matrix is diagrammed as:



Deer mouse matrix

Where:

P = survival to remain within a stage

G = survival to next stage

F = reproduction

H = proportion breeding

$$\begin{bmatrix} 0 & 0 & 0 & P_4F_4H_4 \\ G_1 & 0 & 0 & 0 \\ 0 & G_2 & 0 & 0 \\ 0 & 0 & G_3 & P_4 \end{bmatrix}$$

Mean and adjusted matricies

When mean vital rates for deer mice are combined in matrix format, geometric population growth rates (λ) are less than one. This is likely due to a systematic low bias in survival rates, which are a consequence of assuming all permanent emigrants from trap girds are mortalities. Trapping grids which cover small areas exacerbate this problem, because dispersers are less likely to be captured elsewhere on the grid and traps may only cover a small portion of an animal's home range. Because λ must be greater than 1.0 during the breeding season, I increased all survival rates by 20% from the mean rates from the literature. All adjusted vital rates are still within the reported range of variation. This "adjusted matrix" is used in all analyses which require a mean matrix (i.e. elasticity, LTRE, and simulation based analyses).

Adjusted matrix rate

SAD

Population growth at SAD

$$\begin{bmatrix} 0 & 0 & 0 & 0.94 \\ 0.58 & 0 & 0 & 0 \\ 0 & 0.77 & 0 & 0 \\ 0 & 0 & 0.77 & 0.73 \end{bmatrix} \qquad \begin{bmatrix} 0.34 \\ 0.19 \\ 0.14 \\ 0.34 \end{bmatrix} \qquad \lambda = 1.05$$

Appendix B. Survival rate estimation using program MARK.

Program MARK is a collection of open and closed estimators for population parameters. I used Pollock's robust design (Pollock et al 1990; Kendall et al. 1997) to estimate survival rates for the treatments. Modifying the notation of Kendall et al (1997):

N_{ikl} = Estimate of population size during primary session (*i*), in pen (*k*), with treatment (*l*), assuming closed populations (i.e. no birth, death, emmigration, or immigration).

ϕ_{ikl} = Survival rate of animals from primary session (*i*) to primary session (*i* + *1*) in pen (*k*), with treatment (*l*).

p_{ijkl} = The probability that an animal is captured in secondary sample (*j*) of primary sample (*i*), pen (*k*), and treatment (*l*), given that the animal is alive and in the sampled area during period (*i*).

c_{ijkl} = The probability that an animal is recaptured in secondary sample (*j*) of primary sample (*i*), pen (*k*), and treatment (*l*), given that the animal has been captured before, is alive, and is in the sampled area during period (*i*).

γ_{ijkl} = The probability that a previously captured animal is not in the sampling area at primary period (i), but will be recaptured during a later primary period. Kendall et al. (1997) separate g into two components: γ'' = the probability of emigrating from the sampling area, and γ' = the probability of remaining away from the sampling area one the animal has left. Because the populations are enclosed, I assume $\gamma'' = \gamma' = 0.00$.

Parameters which do not vary at specific levels are denoted by (.). For example, a model with $\{\phi_{ik.}\}$ assumes that survival rate is varies by primary period (*i*) and pen (*k*), but is invariant (equal) across treatments (.).

Goodness-of-fit (GOF) was tested for the full model with RD-SURVIVE (see Kendall et al. 1997). Because RD-SURVIVE cannot calculate GOF for highly complex models, I had to calculate goodness-of-fit for each pen separately and had to assume that ϕ was equal for all age and sex classes within a pen. I then summed the χ^2 approximations and adjusted the degrees of freedom to test the overall GOF of the most parameterized model (as done by Leberton et al. 1992) (Table Appendix B1).

I follow Burnham and Anderson (1992) and Lebreton et al. (1992) and use Akaike's Information Criterion (AIC) to determine which models are the most parsimonious. Where the log likelihood is determined by the maximum likelihood estimators in Program MARK and *k* is the number of parameters, AIC is calculated as:

$$\text{AIC} = (-2\log \text{Likelihood}) + (2k) \qquad \text{(AB 1)}$$

AIC penalizes the better fit (i.e. lower deviance) of more parameterized models by the number of parameters in the model. This tradeoff acknowledges the fact that as the number of parameters increases, the precision of those parameters decreases. The model with the lowest AIC is referred to as the most parsimonious model, in that it has the best tradeoff between model fit and the number of parameters.

Program MARK uses a modified version of AIC that corrects for small sample size bias; where *n* equals the number of observations, AICc is defined by Harvich and Tsai (1995) as:

$$\mathrm{AICc} = \mathrm{AIC} + \frac{2k\,(k+1)}{n - k - 1} \qquad \text{(AB 2)}$$

I defined 10 models for consideration *a priori*. Because there is no temporary emigration, γ is invariant and equal to 0.0. Furthermore, I expected the probability of capture (p) and recapture (c) to be equal because the populations were very "trap-happy" and daily capture rates were ≥ 0.75. If any variation existed in p or c, I expected this to be between primary sessions (i) and not between pens or treatments. Hence, most of the models were developed assuming p and c are equal and invariant or are equal and vary by primary session (i).

There is strong evidence suggesting that survival rate of adult removal treatments is different than those of juvenile removal or control treatments (Table AB2). Models with different survival rates by treatment (models 3 and 4) and models that assume survival rate is equal for controls and treatments which reduce juvenile survival, but differ for treatments that reduce adult survival (models 1 and 2), have the lowest AICc. Models that assume survival rates are equal for all treatments have relatively high AICc values and are less parsimonious (models 7 and 9).

Cooch and White (1998) recommend that all models within 2 AICc of the most parsimonious model be considered for vital rate estimation. Because there is uncertainty in both the estimation of a parameter (i.e. survival rate or population size) and uncertainty in model selection (i.e. none of the models are likely the true model), Buckland et al. (1997) recommend that models be averaged.

Because some models are more parsimonious than others, model averaging places greater weight on more likely models. Using AICc from equation AB2, the weight of model *K* equals (from Buckland et al. 1997):

$$w_k = \frac{\exp\left(\frac{-\mathrm{AICc}_k}{2}\right)}{\sum_{i=1}^{K}\exp\left(\frac{-\mathrm{AICc}_i}{2}\right)} \qquad k = 1,\ldots.,K. \qquad \text{(AB3)}$$

After calculating the weight of a model, the weighted average of parameter $\hat{\theta}$ across *K* models, denoted as $\hat{\theta}_a$, is calculated as:

$$\hat{\theta}_a = \sum_{i=1}^{K}\hat{w}_i\hat{\theta}_i \qquad \text{(AB4)}$$

Where $\hat{\mathrm{v}}\mathrm{ar}\left(\hat{\theta}_i \middle| M_i\right)$ is the estimated variance of parameter given model M_i, and $\left(\hat{\theta}_i - \hat{\theta}_a\right)^2$ is a term which quantifies the variation of $\hat{\theta}_i$ from the weighted average $\hat{\theta}_a$ across *K* models, the variance unconditional on any given model is:

$$\hat{\mathrm{v}}\mathrm{ar}\left(\hat{\theta}\right) = \left[\sum_{i=1}^{K}\hat{w}_i\sqrt{\hat{\mathrm{v}}\mathrm{ar}\left(\hat{\theta}_i \middle| M_i\right) + \left(\hat{\theta}_i - \hat{\theta}_a\right)^2}\right]^2 \qquad \text{(AB5)}$$

Hence the variance of parameter $\hat{\theta}$ is the product of the sampling variance in $\hat{\theta}$, given model *M* (i.e. $\hat{\mathrm{v}}\mathrm{ar}\left(\hat{\theta}_i \middle| M_i\right)$) and the variance in $\hat{\theta}$ across all models averaged (i.e. $\left(\hat{\theta}_i - \hat{\theta}_a\right)^2$). This product of variance for $\hat{\theta}$ is then weighted by w_i for each model M_i.

In other words, equation AB5 adds the sampling variance of $\hat{\theta}$ to the model selection variance in $\hat{\theta}$ and then weights this value by how likely the model is.

While models 1 through 4 are within approximately 2 AICc values of the most parsimonious model (Table AB2), I only used models 1, 2, and 4 for vital rate estimation, because I wanted average survival rates within treatments and model 4 examined each pen separately. The averaged point estimate and one unconditional standard error is shown for each treatment in Figure 9.

Table Appendix B1. Goodness-of-fit statistics from Program RD-SURVIVE with unpooled data.

Pen	Degrees of freedom	G statistic (unpooled)
1	25	2.017
2	25	17.742
3	25	7.392
4	25	10.511
5	25	8.192
6	25	6.479
Totals	150	52.333

Probability of a higher G statistic: $p \geq 0.999$

Table Appendix B2. Models used to estimate deer mouse survival rates.

Model	Parameters	AICc	Δ AICc	AICc Weight	Number of Parameters
	{N(ikl) ϕ(ikl) p(....) c(....) γ(....)} - p = c; ϕ of juvenile removal treatments equals ϕ of control treatments	208.151	0.000	0.3143	5
	{N(ikl) ϕ(ikl) p(i...) c(i...) γ(....)} - p = c; ϕ of juvenile removal treatments equals ϕ of control treatments	208.819	0.670	0.2251	7
	{N(ikl) ϕ(ikl) p(....) c(....) γ(....)} - p = c	209.430	1.280	0.1658	13
	{N(ikl) ϕ(i.l) p(i...) c(i...) γ(....)} - p = c	210.187	2.040	0.1136	9
	{N(ikl) ϕ(ikl) p(i...) c(i...) γ(....)} - p = c	210.360	2.210	0.1042	15
	{N(ikl) ϕ(ikl) p(....) c(....) γ(....)} - p does not equal c	211.601	3.450	0.0560	14
	{N(ikl) ϕ(...) p(....) c(....) γ(....)} - p = c; ϕ equal for all treatments	215.448	7.300	0.0082	3
	{N(ikl) ϕ(ikl) p(i...) c(i...) γ(....)} - p does not equal c	215.775	7.620	0.0070	18
	{N(ikl) ϕ(...) p(i...) c(i...) γ(....)} - p = c; ϕ equal for all treatments	216.091	7.940	0.0059	5