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EFFECTS OF FERTILITY CONTROL ON POPULATIONS OF UNGULATES: GENERAL, STAGE-STRUCTURED MODELS

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Abstract: Regulating the abundance of ungulate populations using hunting can prevent populations from reaching levels that cause harm to natural and human dominated systems. However, there are an increasing number of cases where hunting is infeasible, and in such cases, fertility control has been widely advocated as an alternative means for controlling populations. Here, we develop simple analytical models offering general insight into the feasibility of using fertility control to regulate the abundance of ungulates. The models are structured in stages to represent variation in the duration of effect of fertility control agents. Analysis of these models offers several predictions, amenable to testing in field studies. (1) More than 50% of fertile females will need to be maintained infertile to achieve meaningful reductions in ungulate numbers even when fertility rates are low. (2) The relationship between the proportion of females maintained infertile and the steady state density is highly nonlinear. This means that small errors in estimating levels of infertility can lead to large errors in achieved density. It also means that managers should expect to see little change in steady-state density across a broad range of delivery rates. (3) The efficacy of fertility control as a management technique depends strongly on the persistence of the effect of the fertility control agent and the ability of managers to recognize previously treated animals. (4) Fertility control using long-lived agents can be more efficient than culling in regulating ungulate numbers. (5) Treating small populations with irreversible agents magnifies the likelihood of population extinction relative to treatment by culling. As with all techniques, managing population fertility must extend from a sound understanding of the influence of management actions on the state and dynamics of the population.

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Controlling the abundance of ungulates by regulating harvest is one of the prevailing activities of wildlife managers throughout North America. Regulated harvest offers a variety of benefits to society, including recreation for citizens and revenue for agencies and private enterprise. An additional benefit is a reduction in animal numbers, which if left unchecked, can cause significant harm to natural and human-dominated systems (Jewell and Holt 1981, Diamond 1992, Garrott et al. 1993, McCullough et al. 1997).

Although harvest can be an effective method for manipulating ungulate numbers, there are a growing number of situations where it is not feasible. The rapid expansion of urban and suburban areas during the last decade has brought populations of ungulates in close proximity to high densities of people (Adams 1994, McCullough et al. 1997). This proximity restricts the use of hunting as a management tool because it poses unacceptable risks to human safe-

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ty (Decker and Connelly 1989, Brush and Ehrenfeld 1991, McAnnich 1993, Wright 1993), and because urban residents often oppose hunting animals on ethical grounds (Decker and Connelly 1989, McCullough et al. 1997). In addition, there are many conservation areas, national parks prominent among them, where hunting is proscribed by law and policy (Leopold et al. 1963, Wright 1993, Porter et al. 1994, Wagner et al. 1995, Frost et al. 1997, Porter and Underwood 1999). In all of these situations, culling animals to regulate populations has significant liabilities, and as a result, wildlife managers are seeking innovative alternatives to lethal methods of population control (Kirkpatrick and Turner 1985, Bomford 1990, Brush and Ehrenfeld 1991, Diamond 1992, Garrott et al. 1993, McCullough et al. 1997).

Fertility control has been widely advocated as an alternative to culling (reviewed by Kirkpatrick and Turner 1985, Bomford 1990, Tuyttens and Macdonald 1998). Field and laboratory studies have evaluated efficacy of delivery of contraceptives to ungulates (Jacobsen et al. 1995, McCallum 1996, DeNicola et al. 1997, Fayrer Hosken et al. 1997, Kirkpatrick et al. 1997, Nielsen et al. 1997, Turner et al. 1997), and models have been developed to represent effects of fertility control on the population dynamics of individual species (Garrott and Siniff 1992, McAninch 1993, Swihart and DeNicola 1993, Boone and Wiegert 1994, Seagle and Close 1996). However, despite these efforts, a broad understanding of effects of fertility control on ungulate populations has failed to emerge. The absence of such understanding retards efforts to apply fertility control as a method to control ungulate numbers.

Here, we develop a general theory of regulation of ungulate populations using fertility control agents. Specifically, our objective was to develop simple analytical and simulation models offering general insight into the following questions. (1) What proportion of females in a population must be infertile to maintain the population at target densities? (2) How does fertility control compare with culling as a means to regulate ungulate populations? (3) How does duration of efficacy of contraceptives influence the number of animals that must be treated to achieve target population levels? (4) To what extent does timing of delivery (pre- or post-breeding) influence the ability of contraceptives to regulate ungulate numbers? (5) Do assumptions on the operation of density dependence modify the insights gained above? (6) Is fertility control a "safe" method for regulating small populations vulnerable to stochastic, local extinction? We provide a general mathematical basis for screening the feasibility of fertility control before developing detailed, species-specific models or conducting extensive field applications.

METHODS

Base Model

Because our purpose was to develop general insights, we used a model that was as simple as possible (Levins 1966, Starfield et al. 1990, Starfield and Bleloch 1991, Starfield 1997). The model needed to be sufficiently detailed to mimic the behavior of ungulate populations in a reasonable way, but it also needed to avoid detail peripheral to the questions posed above. To meet this challenge, we began with a simple formulation and incrementally added detail needed to meet our objectives.

Our starting point was a stage-structured model representing species where adult survivorship is high and where density dependence operates primarily on recruitment, characteristics that are particularly faithful to the life histories of many species of ungulates (McCullough 1979, Houston 1982, Skogland 1990, Bartmann et al. 1992, Gaillard et al. 1993, Putman et al. 1996). The model was formulated as

$$F_{t+1} = F_t(S + m - \beta(F_t - H_t)) - H_t, \quad (1)$$

where F_t is the density of females at time t, S is the annual per-capita survival rate of females, m is the maximum number of breeding females annually recruited to the population per breeding female, β is the linear slope of the relationship between recruitment of females to breeding age and female density, and H_t is the number of females culled at time t. The model predicts the preharvest population density of females using 3 parameters (S, m, β) and a single decision variable, H_t. Although the model represents a single stage explicitly (adult females), it implicitly represents sexually immature juveniles via the recruitment term. The explicit juvenile stage drops out of equation (1) via model reduction (Caswell 1989:100).

The model is based on several simplifying assumptions. First, we assume that the probability of survival of adults does not change with age. Thus, we treat longevity as the outcome of a

series of independent Bernoulli trials rather than being fixed at a specific age (Gurney and Nisbet 1998:80). Culling is assumed to occur after breeding, but before recruits are born. We assume that density dependence is linear and operates entirely on recruitment. That is, the number of new breeding females recruited to the population is directly proportionate to the postharvest population density of females. Effects of nonlinear density dependence will be included as part of the analysis of the model.

It is possible to formulate the model using an explicit parameter for the density at which recruitment = 0 (i.e., $K_r = m/\beta$). Alternatively, we can represent effects of density dependence using a slope (β) and intercept (m) for the relationship between density and recruitment. We used this approach because we believe these parameters offer useful biological interpretations; the parameter β represents the "strength" of density dependence and the parameter m represents the maximum possible recruitment rate. Both of these representations prove useful when interpreting model analysis.

Although this model is extremely simple, it can represent the dynamics of ungulate populations with reasonable fidelity (Fig. 1). A more detailed model would achieve higher levels of realism, but only at the expense of reduced clarity and generality.

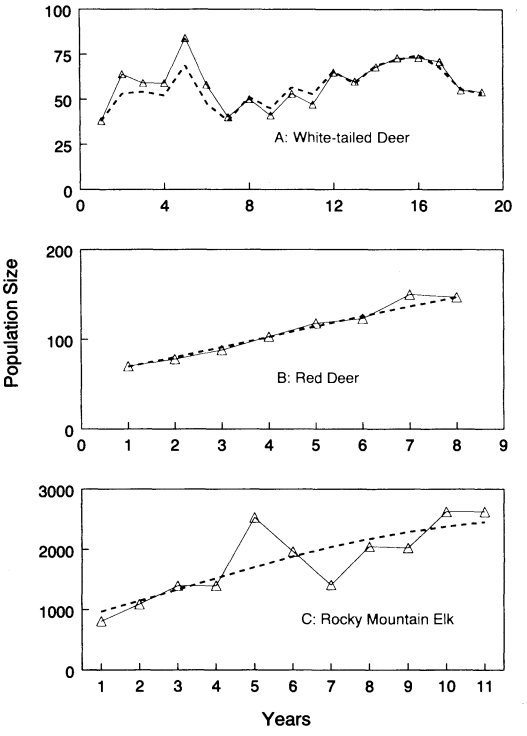

Fig. 1. Comparison of model projections (dashed line) with observed trajectories of ungulate populations (triangles). Model parameters were estimated by minimizing the squared difference between observations and predictions. A close fit between observations and predictions allows the conclusion that the model contains sufficient detail to reasonably represent trajectories of ungulate populations if its parameters are properly estimated. Data are from [A]—McCullough (1979: Table 3.2), [B]—Clutton-Brock et al. (1982), [C]—Houston 1982.

Model Development

We modified equation (1) to represent effects of fertility control as an alternative to culling. Node diagrams and the matrix form of the model are shown in Fig. 2. Here, we summarize our modifications using finite difference equations.

We defined the number of animals harvested annually as

$$H = cF_t. \quad (2)$$

Thus, c is the per capita rate of culling, the number of breeding females culled annually per breeding female in the population. It follows that the effects of culling can be represented in the model as

$$F_{t+1} = F_t(1 - c)(S + m - \beta(1 - c)F_t). \quad (3)$$

We will refer to equation 3 as the culling model.

To allow comparison with fertility control, we redefined c as the per capita rate of delivery of contraceptives. There are now 2 stages that we wish to track in the population; females that are fertile (i.e., F_t) and those that are infertile (i.e., I_t). We define fertile animals as those that are capable of breeding and carrying a fetus to term. We define infertile animals as those that cannot successfully breed. When the effect of the contraceptive persists for a single breeding season, then the dynamics of the fertile (F_t) and infertile stages (I_t) can be represented as

$$F_{t+1} = F_t(1 - c)(S + m - \beta N_t) + (1 - c)I_t, \text{ and} \quad (4)$$

$$I_{t+1} = F_t c(S + m - \beta N_t) + I_t Sc, \quad (5)$$

where

$$N_t = F_t + I_t. \quad (6)$$

However, if the effects of the contraceptive persist for more than 1 breeding season, then we must account for the time since the animal was treated. This accounting can be accomplished

by adding stages to the model. If the duration of the contraceptive is finite, then the dynamics of the fertile and infertile stages can be represented as

$$F_{t+1} = F_t(1-c)(S + m - \beta N_t) + (1-c)I_{\tau_t} \quad (7)$$

$$I_{1_{t+1}} = F_t c(S + m - \beta N_t) + I_{\tau_t} Sc \quad (8)$$

$$I_{2_{t+1}} = I_{1_t} S, \quad (9)$$

$$I_{3_{t+1}} = I_{2_t} S, \quad (10)$$

$$I_{4_{t+1}} = I_{3_t} S, \quad (11)$$

$$I_{\tau_{t+1}} = I_{\tau-1_t} S, \quad \text{and} \quad (12)$$

$$N_t = F_t + \sum_{i=1}^{\tau} I_{i_t}, \quad (13)$$

where τ = the duration of the contraceptive in number of breeding seasons. Thus, the number of stages in the model is $\tau + 1$, 1 stage for the breeding females and 1 stage for each year that animals are infertile following treatment. The total population of females is the number of animals summed across stages (equation 13). We will refer to equations 7–13 as the fixed duration model.

It is possible to develop agents that will render animals infertile for life (reviewed by Muller et al. 1997, Tuyttens and Macdonald 1998). To represent effects of such agents, only 2 stages need to be tracked (fertile and infertile animals), and the dynamics of these stages are connected only by the rate of addition of animals to the infertile pool and the effect of total density on recruitment. Thus, the dynamics of the population are given by

$$F_{t+1} = F_t(1 - c)(S + m - \beta N_t), \quad (14)$$

$$I_{t+1} = F_t c(S + m - \beta N_t), \quad \text{and} \quad (15)$$

$$N_t = F_t + I_t. \quad (16)$$

We will refer to equations 14–16 as the lifetime duration model.

The fixed and lifetime duration models depend on the same set of assumptions described for the culling model. However, these models require the additional assumptions that treatment is applied immediately before breeding occurs and that managers can differentiate between animals that have been treated and those that have not been treated. We will examine the consequences of these assumptions as part of model analysis. Time of census in the model is

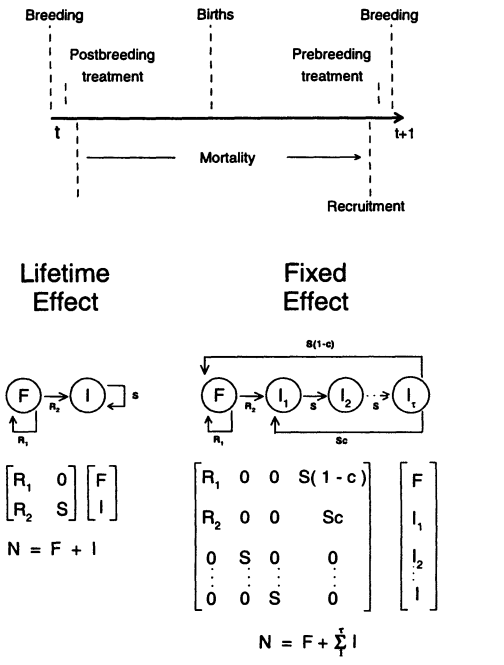

Fig. 2. Schematic of model structure and assumptions. The upper panel depicts assumptions on timing of events represented in the model. The central panel shows the structure of the model in node and matrix formats. The lower panel shows representations of assumptions about timing of delivery and the operation of density dependence.

assumed to occur annually, immediately after breeding (Fig. 2), and all fertile animals are assumed to be bred at the time of census. We chose breeding rather than births as the point of reference for census because the state of the animal that influences model behavior is its fertility rather than its age.

Model Analysis

Deterministic Analysis.—We used standard matrix techniques and algebraic manipulations (see Appendix I for details) to derive several quantities of interest. These included: K_e, the equilibrium density of animals in the absence of fertility control or culling; K_c, the equilibrium

density of animals as a function of delivery or culling rate; c^*, the delivery rate required to maintain a target population density; P, the proportion of the population that must be infertile to maintain a target population density; and n^*, the number of animals that must be treated or culled annually to maintain a target population density. To examine the effects of timing of delivery on model predictions, we changed model formulations to represent delivery after breeding (Fig. 2). In this case, delivery of contraceptives did not prevent birth by treated animals during the current time step (because treated animals were assumed to be pregnant at the time of delivery) but did prevent births during the subsequent time step (Fig. 2). To examine effects of nonlinearity in density dependence, we reformulated the model (Fig. 2) so that recruitment remained relatively constant at low population density and then declined as threshold densities were reached (Bartmann et al. 1992). To examine consequences of relaxing assumptions on known fertility status of animals, we developed an estimator to show how many animals must be treated to achieve a treatment target, given unknown fertility status. This estimator deals with the multinomial case of sampling with replacement, and estimates necessary treatment rates, assuming that animals could be treated more than once.

Stochastic Analysis.—We performed Monte Carlo simulations to examine the relative ability of culling and fertility control to regulate animal numbers near a target density. These simulations were organized in 4 model scenarios that included 2 types of management and 2 sources of stochastic variation. Management types include static and adaptive. Static management applied the same per capita rate of delivery annually, a rate that would achieve a target population density if applied accurately. Adaptive management changed the delivery or culling rate in response to differences between realized density and an acceptable range of target densities. The acceptable range was defined as the target ±25%. If the population exceeded the target by ≥25%, then the culling or delivery rate was increased by 10% until the population returned to acceptable levels. If the population dropped below the target by ≥25%, then culling or fertility control was discontinued until the population recovered to a density within the acceptable range.

In addition to 2 types of management, model scenarios included 2 sources of stochastic variation; process and observer. Process variation was included to mimic density independent effects of the environment on population dynamics. To represent this variation, adult survival varied according to a log-normal distribution with mean 0.9 and variance 0.1. We varied the effect of density on recruitment (i.e., β) using a normal distribution with a mean of 0.009 and variance 0.01. To assure that stochastic effects on mortality and recruitment were correlated, we derived them from the same normally distributed variate at each time step. Observer variation was included to mimic the error in estimates of population size that are typical in managing ungulate populations. To represent observer variation, delivery and culling rates were based on an estimate of the current population density derived by sampling from a normal distribution with a mean equal to the model density at time t (F_t) and a standard deviation equal to 15% of the mean. Initial conditions for population density were set at the target density. Initial conditions for population composition (i.e., fertile–infertile stages) were set at levels equivalent to the deterministic steady-state at that target density. We conducted 1,000 model runs of 50 years each for 2 target densities, set as percentages (25 and 75%) of the steady state that would occur in the deterministic model when culling or fertility control was not applied (i.e., K_e). We applied 4 methods of population control: culling, and fertility control with the duration of treatment equal to 1 year, 3 years, and lifetime. For each set of model runs, we recorded the total number of animals treated or culled, the overall variance in population density, and the number of years that the population was outside the acceptable ranges. We also recorded the frequency of extinction and the persistence time of the population.

Procedures for Analysis and Plotting.—Analytical results were obtained using Maple V (Waterloo Maple, Waterloo, Ontario, Canada). All analytical results were checked for accuracy using simulation of difference equations with Microsoft Excel (Microsoft Corporation, Redmond, Washington, USA). Monte Carlo simulation experiments were conducted using Visual Basic for Applications running under Excel. For the purpose of illustrating analytical results in plots, we chose parameter values that are reasonable for white-tailed deer (*Odocoileus virginianus*; $m = 0.6$, $S = 0.9$, $\beta = 0.005$) and that

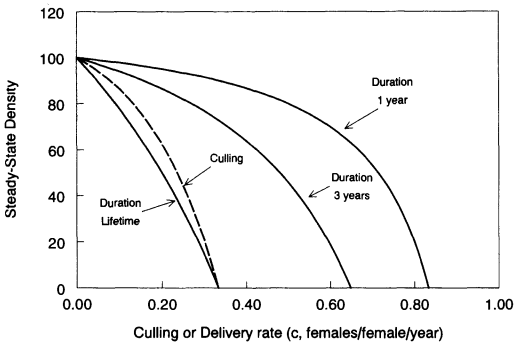

Fig. 3. Model predictions (equation 18) of steady-state density as a function of culling or delivery rate using parameter values $m = 0.6$, $S = 0.9$, $\beta = 0.005$. Different lines show differences due to variation in duration of efficacy of contraceptives.

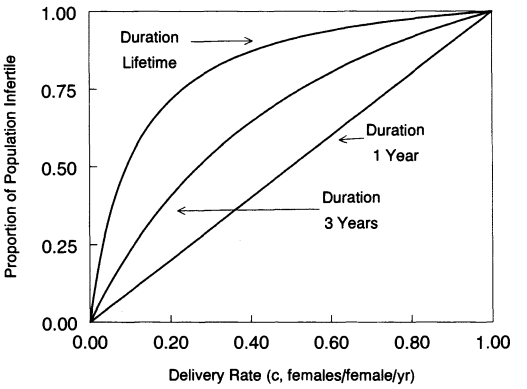

Fig. 4. Model predictions (equation 19) of effects of delivery rate on the proportion of the female population that is infertile. Different lines show differences due to variation in duration of efficacy of contraceptives. Parameter values are $m = 0.6$, $S = 0.9$, $\beta = 0.005$.

produced an equilibrium value of population density = 100 in the absence of any treatment or culling. Because density can scale to any unit area, our plots can be viewed in absolute or relative terms. For example, $N = 90$ can be accurately interpreted as an absolute density (e.g., 90 animals per unit area) or as a percentage of steady state population density that would be achieved in the absence of treatment (e.g., 90% of K_e).

RESULTS

Equilibrium Population Density and Composition

In the absence of treatment or culling (i.e., $c = 0$), all models predicted that the population would reach a steady state density, K_e,

$$K_e = \frac{S + m - 1}{\beta}. \quad (17)$$

As the rate of treatment with contraceptives increases, models predicted that steady state density declines in a nonlinear fashion (Fig. 3) according to

$$K_c = \frac{S + m + S^{\tau}c - Sc - cm - 1}{(1 - c)\beta}. \quad (18)$$

In the lifetime case, the S^{τ}_c term drops out of equation (18).

Fixed-effect models predicted a nonlinear, threshold relationship between equilibrium density and treatment rates. We found a broad range of treatment rates that exert relatively weak effects on steady state density, particularly for short-duration models (Fig. 3). Increasing duration of fertility control tended to make this relationship more linear (Fig. 3).

Fixed-effect models illustrated that when the duration of the contraceptive is 1 breeding season, the proportion of the population that is infertile (P) is equal to the treatment rate (i.e., $P = c$). However, when effects persist beyond a single season, infertile animals accumulate in the population treated annually, and as a result, $P > c$ (Fig. 4). Analysis of the model (Appendix A) suggests that the relationship between P and c depends on the duration of the effect of the contraceptive and the adult survival rate, i.e.,

$$P = \frac{(1 - S^{\tau})c}{1 + Sc - S - S^{\tau}c}, \quad (19)$$

but does not depend on birth rate (m) or the strength of density dependence (β). In the lifetime case, this simplifies to

$$P = \frac{c}{1 - S + Sc}. \quad (20)$$

The relationship between target density (N^*) and the proportion of the population that is infertile (P) is identical for all fertility control models (Fig. 5),

$$N^* = \frac{S + m - Pm - 1}{\beta(1 - P)}. \quad (21)$$

Equation 21 shows that increasing dependence of recruitment on density amplifies the effect of fertility control on the steady state population size. That is, achieving a given target density will require a smaller proportion of infertility in populations where density-dependent effects

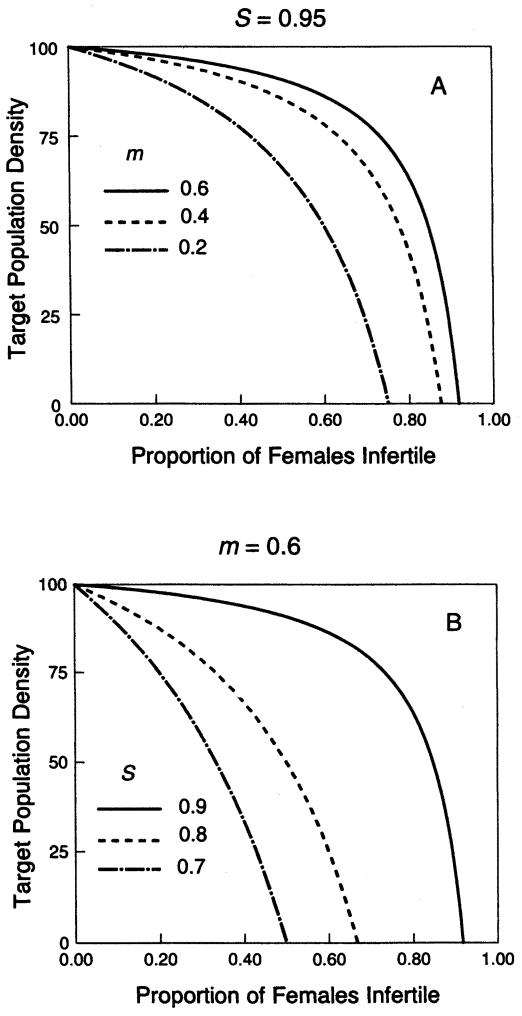

Fig. 5. Model predictions (equation 21) of the proportion of the females that must be infertile to achieve a target population density assuming a constant ecological carrying capacity of K_e = 100. Panel A shows this proportion with adult female survival rate held constant (S = 0.95) and maximum recruitment rate varying. Panel B shows this proportion with maximum recruitment rate held constant (m = 0.6) and survival rate varying.

are stronger than in populations where such effects are weak.

Population managers need a relatively easy way to estimate the proportion of the population that would need to be maintained infertile to achieve a given target density. This can be obtained as follows. By rearranging equation 21, we find P as a function of the target density:

$$P = 1 - \frac{1 - S}{m - \beta N^*}. \quad (22)$$

This result simply says that for any steady-state population density, N^*, the proportion of the population that is fertile (i.e., $1 - P$) must equal the adult per capita mortality rate divided by the per capita recruitment rate (i.e., females per female per year). When this is true, the number of animals recruited will equal the number that die.

Note that the duration of the contraceptive determines the rate of delivery that must be applied each year to achieve a given level of population infertility, (equation 19), but the duration does not affect the level of infertility required to achieve a given steady-state density (equation 22). Equation 22 also illustrates that the necessary proportion infertile will increase with increasing adult survival and maximum recruitment. It will decline with increasing "strength" of density dependence.

Equation 22 requires an estimate of β, which can be difficult to obtain. However, it is possible to derive an expression for P that does not depend on estimating β. Defining Δ as a proportion of the steady-state density (i.e., $N^* = \Delta K_e$) and substituting the right hand side of

$$\Delta K_e = \Delta\left(\frac{S + m - 1}{\beta}\right) \quad (23)$$

for N^* in equation 22, we obtain an expression for the proportion of animals that must be infertile to achieve a target density specified as a proportion of the untreated steady state, i.e.,

$$P = 1 - \frac{1 - S}{m + \Delta(1 - S - m)}. \quad (24)$$

Thus, when Δ = 1, there is no reduction in density; when Δ = 0, the population goes extinct. Equation 24 is an important result because it provides a general rule of thumb for estimating the effort required to regulate a population using fertility control without knowing the strength of density dependence. This rule requires estimates of only 2 parameters (adult female survival rate and maximum recruitment rate of females) and a decision on the targeted reduction, Δ.

Vital Rates and Population Composition at Equilibrium

Equation 24 also reveals that a relatively large proportion of the population must be infertile in order to achieve meaningful reductions in density (Fig. 5). When adult survival is high (>95%),

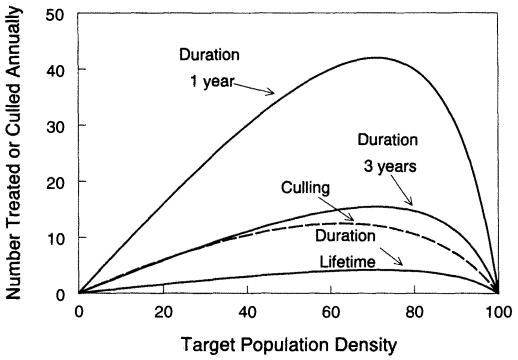

Fig. 6. Model predictions (equations 25–28) of the number of animals that would need to be treated or culled to maintain a population at the specified target density. Parameter values are $m = 0.6$, $S = 0.9$, $\beta = 0.005$. Different lines show differences due to variation in duration of efficacy of contraceptives.

it is reasonable to assume that >60% of the breeding females will need to be infertile to achieve meaningful reductions in population density, even for species with very low intrinsic rates of increase (e.g., $m = 0.2$, Fig. 5). The shape of the relationship between the proportion of the population that is infertile and steady-state density is more sensitive to differences in adult survival than to differences in maximum rates of recruitment. This relationship remains strongly nonlinear as long as adult survival is high. However, as adult survival declines, the relationship approaches linearity (Fig. 5).

Number of Animals Treated or Culled at Equilibrium

When effects of fertility control agents persist for only 1 year, then the proportion of the population that is infertile equals the delivery rate. Moreover, in this case, the number of animals that must be treated the first year to achieve a given target is the same as the effort required in subsequent years. However, when regulating populations with fertility control agents that persist for >1 breeding season, there are 2 types of effort required: the effort required to achieve a specified level of infertility in the population, and the effort required to maintain that level of infertility. Here, we examine this maintenance effort and compare it with culling.

In populations regulated with culling, the number of animals harvested annually (n) to maintain a given target density, N^*, at steady state is simply

$$n = c^*N^*, \quad (25)$$

where c^* is the culling rate needed to maintain a population at the target density. For the culling model, c^* is given by

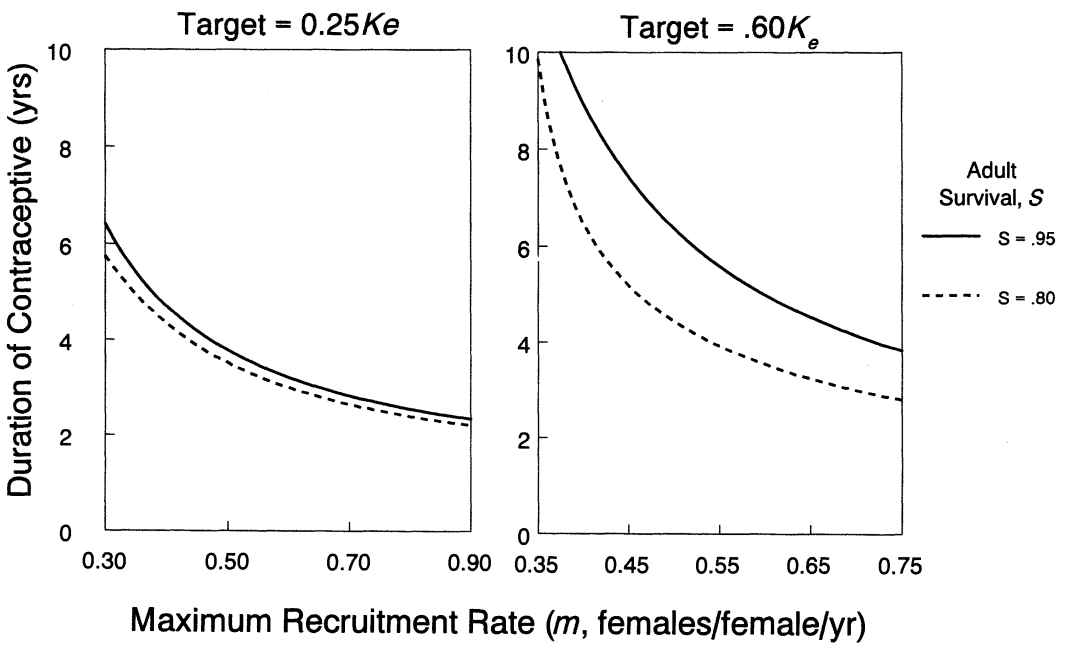

Fig. 7. Model predictions of the duration of contraceptive delivered to an animal needed to cause the same effect on population growth as culling the animal (equations 25 and 28).

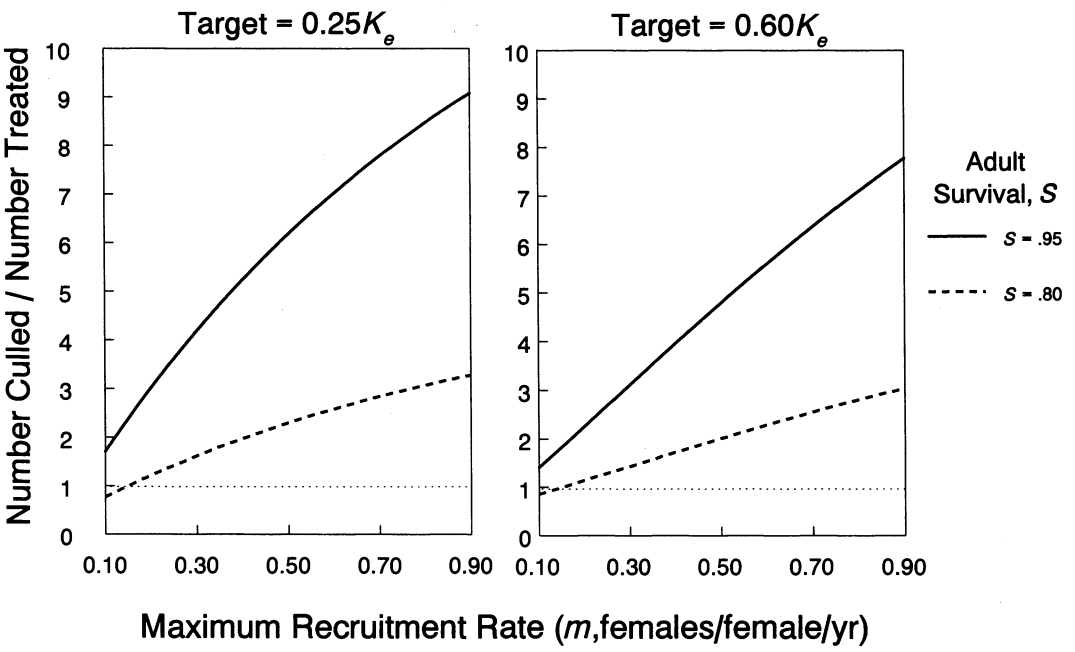

Fig. 8. Model predictions of the ratio of the number of animals that would need to be culled relative to the number treated with contraceptives to maintain steady states at target densities. The horizontal dotted line shows where the number culled equals the number treated (equations 25–28).

$$c^* = (2N^*\beta - S - m + \sqrt{S^2 + 2Sm + m^2 - 4N^*\beta})/(2N^*\beta). \quad (26)$$

In populations regulated with fertility control, there are 3 cases relevant to the number of animals treated annually (n) to maintain a given target density. In the case of fixed duration agents lasting a single year, the delivery rate at equilibrium (c^*) is the same as the proportion of the population that is infertile, so using equation 22, we calculate the number treated as

$$n = c^*N^* = PN^* = \left(1 - \frac{1 - S}{m - \beta N^*}\right)N^*. \quad (27)$$

In the case of agents with duration >1 year and greater than a lifetime, the number that must be treated annually is

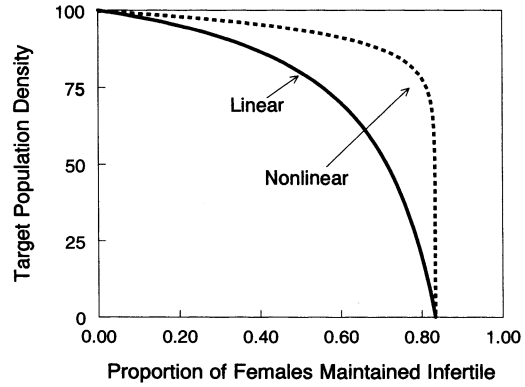

Fig. 9. Comparison of model predictions of the relationship between the target density and the proportion of the population that is infertile, assuming linear (solid line) and nonlinear (dashed line) representations of the effects of density dependence on recruitment.

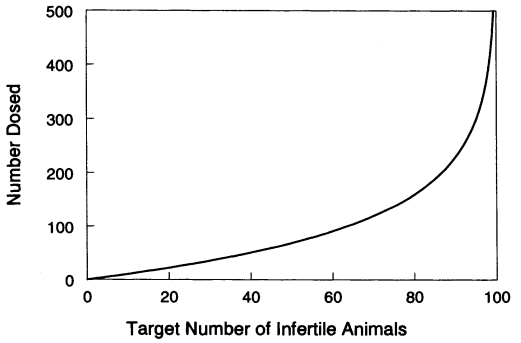

Fig. 10. Predictions of the number of animals that would need to be dosed to achieve a given level of infertility in a population, assuming animals are not marked and are subject to treatment more than once. The axes can be interpreted as a number of individuals in a population of 100, or as percentages.

Table 1. Results of 1,000 model runs of 50-year duration with stochastic variation. Static management applied a constant, per capita culling or delivery rate needed to achieve the target density. Adaptive management adjusted the culling or delivery rate to keep the population within ±25% of the target density. Process variation mimicked stochastic environmental effects on adult survival and juvenile recruitment. Observer variation mimicked uncertainty in estimating population size. Control methods included culling and 4 contraceptives differing in duration, compared with the no management case (i.e., "Natural"). Results include average population size (*N*) and its coefficient of variation. We also report percentage of runs for which population density dropped below 1 (% extinct) and mean time to extinction averaged across model runs (persistence time in yr).

Management type	Variation type	Control method (duration of contraceptive)	Target density							
			25				75			
			N	CV	% extinct	Persistence time	*N*	CV	% extinct	Persistence time
Static	Process	Culling	22.1	51.3	0.5	49.9	69.1	31.7	0.2	49.9
		1 yr	23.2	46.3	0.2	49.6	71.3	28.4	0.1	50.0
		3 yr	23.3	49.3	2.6	49.5	71.8	28.8	0.1	50.0
		Lifetime	19.7	61.3	37.6	41.7	69.2	33.4	3.2	49.3
		Natural	105.5	25.2	0.0	50.0	108.0	23.0	0.0	50.0
Static	Process and observer	Culling	21.6	65.4	1.3	49.4	68.7	32.2	0.0	50.0
		1 yr	29.9	56.2	0.5	49.8	71.2	31.0	0.0	50.0
		3 yr	23.3	85.1	12.3	45.0	72.3	41.8	1.3	48.7
		Lifetime	17.6	87.1	40.1	34.2	69.2	34.7	3.8	49.2
		Natural	105.4	25.7	0.0	50.0	108.1	23.0	0.0	50.0
Adaptive	Process	Culling	27.3	37.2	0.0	50.0	75.8	50.6	0.2	49.9
		1 yr	28.9	29.4	0.5	49.9	79.0	29.2	0.0	50.0
		3 yr	31.0	35.8	0.6	49.8	79.0	43.4	0.0	49.9
		Lifetime	22.7	76.7	10.1	37.7	74.2	37.5	1.0	49.6
		Natural	105.5	25.7	0.0	50.0	108.1	29.4	0.0	50.0
Adaptive	Process and observer	Culling	27.3	36.6	2.5	49.5	75.7	31.3	0.2	49.9
		1 yr	31.8	37.0	1.0	49.8	80.5	29.2	0.0	50.0
		3 yr	34.9	56.44	9.5	47.8	84.5	43.4	5.9	48.7
		Lifetime	27.0	62.26	29.4	44.8	75.2	37.6	1.4	49.7
		Natural	103.4	34.23	0.1	50.0	108.9	29.4	0.0	50.0

Table 2. Modeling estimates of proportion of the target sex that must be infertile to achieve target population objectives. Assumed annual growth rate in absence of treatment = 1.49.

Species	Population objective	% infertile	Reference
White-tailed deer	Negative growth rate[1]	90%	(Swihart and DeNicola 1993)
Feral horses	Reduce population from 600 to range of 300–600	68–82% depending on treatment regime	(Garroutt et al. 1992)
White-tailed deer	Maintain at ½ carrying capacity	80%	(Boone and Wiegert 1994)
Feral horses	Reduce growth rate (i.e., lambda) to 1	90–95%	(Garrott and Siniff 1992)
White-tailed deer	Reduce population size	>50%	(Seagle and Close 1996)
White-tailed deer	Maintain at ½ carrying capacity	96%	(Nielsen et al. 1997)

$$n = c^*(1 - P)N^*(S + m - \beta N^*) + c^*\rho N^*, \quad (28)$$

where c^* is the treatment rate required to achieve a given target density, N^*P is the portion of the population that is infertile at time t as a result of applying c^*, and ρ is the proportion of the population that becomes fertile during $t \rightarrow t + 1$ given a treatment rate = c^*. The components of equation 28 are as follows. The variable P is given by equation 22. The equilibrium treatment rate is

$$c^* = \frac{S + m - 1 - \beta N^*}{S + m - S^\tau - \beta N^*}. \quad (29)$$

The proportion of the population that will become fertile during a time step is

$$\rho = \frac{S^{\tau-1}(1 - S)c^*}{1 + Sc - S - S^\tau}. \quad (30)$$

In the lifetime duration case, the S terms with exponents drop out of the expression for c^* and $\rho = 0$.

Equations 25 and 28 allow comparison of the effort required to control populations using culling or fertility control. Because these equations predict the number of animals that must be treated or culled to maintain populations below K_e (Fig. 6), they are analogous to classical production functions, also known as yield curves (Roughgarden 1997).

Effect of Duration of Contraception

We asked the question, "On average, how long must the effect of a contraceptive persist in an animal to exert the same effect on population-level production as culling that animal?" To address this question, we chose a broad range of parameter values to represent diverse life-history characteristics of ungulates, and applied those values to equations 25 and 28. Models suggested that for a broad range of assumptions about survival and maximum recruitment, delivery of a contraceptive to an individual animal would have the same effect as culling that animal if the effects of the contraceptive persisted for 2–5 years (Fig. 7). Briefer duration is needed when adult survival rates are low. Increasing strength of density dependence reduces the required duration; weakening it increases the required duration.

When contraception persists for the lifetime of the animal, models predicted that in most cases, the effort required to regulate a population at a specified density using fertility control will be less than the effort required by culling (Fig. 8). Exceptions to this result occur when adult survival is low and recruitment is low, that is, when populations grow slowly (Fig. 8). For species with high rates of adult survival and high recruitment rates, the number of animals culled could be almost an order of magnitude greater than those treated with contraceptives at the same target density (Fig. 8). Thus, if we define efficiency of control in terms of the number of animals that must be culled or treated annually to maintain a population at a specified target, then culling will be more efficient than lifetime duration contraception only for slowly growing populations. In all other cases, models suggest that treatment with lifetime effect contraceptives is likely to be more efficient than culling. However, it must be understood that these estimates of effort specify the number of animals that must be treated annually to maintain a steady state population with a proportion infertile, P (equation 22). These are not estimates of the effort required to achieve P in the pretreatment population.

Consequences of Changing Model Assumptions

Assumed Pre-breeding Delivery.—In all results above, we assumed that contraceptives were delivered to females before breeding occurs. It is possible to change the model to represent delivery after breeding by assuming that treated animals give birth during the current time step but are infertile during the subsequent one (Fig. 2). An important result emerged from this analysis. We found that when contraceptives are delivered postbreeding, there is a fundamental limit to the magnitude of the reduction in population density that can occur using fertility control,

$$N^* \geq \frac{m - 1}{\beta}, \quad (31)$$

or, solving in terms of a proportionate reduction,

$$\Delta \leq \frac{m - 1}{S + m - 1}. \quad (32)$$

The right-hand-side of equation 32 gives the steady-state density of females that occurs when the delivery rate = 100%. At this density, each fertile female produces exactly 1 female offspring surviving to reproductive age and the proportion of fertile females in the population equals the adult mortality rate.

Equations 31 and 32 are important because they reveal that it may not be possible to achieve meaningful regulation of populations where contraceptives are delivered after animals have been bred, even when the entire population of fertile females is treated. If animals can breed at 1 year of age, then a cohort of animals always escapes the postbreeding treatment, and this escape may allow populations to grow to levels that are close to K_e, if recruitment rates are sufficiently high. Equations 31 and 32 raise serious doubt about the efficacy of fertility control as a means of regulating populations whenever yearlings contribute significantly to breeding and contraceptives are delivered after they are bred.

Assumed Linear Density Dependence.—Changing the form of the relationship representing density dependence from linear to nonlinear did not change the qualitative results obtained above (Fig. 2). The composition of the population at equilibrium (i.e., equations 19 and 20) does not depend on the form of density-dependent relationships. However, the relationship between equilibrium population size and the proportion of the population infertile changed when we changed the formulation for density dependence. In the case of nonlinear density dependence,

$$N^* = \frac{\ln\left(\frac{S + m - Pm - 1}{1 - S}\right) + \alpha}{\theta}, \quad (33)$$

where α and θ are parameters controlling the shape of the nonlinear relationship between recruitment and density.

Note that this expression does not depend on the duration of the effect of the contraceptive, as we saw earlier for the linear case. Plotting equation 33 shows that nonlinearity in density dependence can sharpen the threshold relationship between N^* and P (Fig. 9). In the neighborhood of this threshold, small errors in estimating the proportion of the population that is infertile can lead to very large errors in equilibrium population size. Nonlinear density dependence amplifies the consequences of such errors. Nonlinearity in density dependence also expands the range of levels of infertility in the population (i.e., P) over which we expect virtually no change in equilibrium density (Fig. 9).

Assumed Knowledge of Fertility Status.—One of the assumptions of our base model is that managers can differentiate between fertile and infertile animals when delivering fertility control treatments. There are cases where this assumption is reasonable, for example, when treated animals can be visually marked, or when it is possible to achieve the desired delivery rates by treating young of the year. However, there will be many cases where the fertility status of animals is unknown. This means that some animals are likely to be treated more than once, thereby increasing the number of "doses" of fertility control agents that must be delivered to achieve a target delivery rate.

This is essentially a problem in multinomial sampling with replacement. That is, after an animal is given a dose of contraceptives, it is released into the population unmarked, and is subject to "resampling" a later time. Because it is unmarked, its fertility status is unknown, and it will be redosed if it is resampled. We are interested in estimating the number of doses (d) that must be delivered in order to treat a target number of animals (η) in a population of size

N. We first consider the case where the duration of contraceptives is a single breeding season and all infertile animals must be dosed annually to maintain infertility. In this case, it can be shown (Appendix B) that the number of animals (d) that must be dosed to expect η animals to be infertile at the end of the treatment campaign can be estimated as

$$d = \frac{\ln\left(\frac{N-\eta}{N}\right)}{\ln\left(\frac{N-1}{N}\right)}. \quad (34)$$

Alternatively, when the duration of contraceptives persists for more than a year, there will be some animals in the population that will remain infertile and do not need to be retreated. Define I as the number of animals in the population that are infertile at the start of a treatment campaign and that remain infertile during the subsequent year. In this case the number of animals that must be dosed to expect that η additional animals are infertile is given by

$$d = \frac{\ln\left(\frac{N-I-\eta}{N-I}\right)}{\ln\left(\frac{N-1}{N}\right)}. \quad (35)$$

Equations 34 and 35 portray the large inefficiencies associated with treating a significant proportion of a population whenever treated animals are not easily distinguished from untreated ones (Fig. 10). For example, a population of 100 fertile animals would require dosing 160 animals to reasonably expect 80 infertile animals in the population after treatment (Fig. 10). A population of 100 animals, 80 of which are infertile at the time of delivery, would require dosing 69 animals to expect that only 10 additional animals would be infertile during the subsequent year. This result illustrates that the feasibility of treating populations with fertility control agents depends in a fundamental way on knowing the fertility status of individual animals at the time of treatment. In the absence of such knowledge, achieving meaningful reductions in steady state density could require delivering doses in excess of the total number of females in the population.

Stochastic Models

We compared the ability of different control regimes to maintain populations at target densities in the face of environmental stochasticity and error in estimation of population size. For all model runs, simulations of fixed-effect fertility control with single year duration showed the lowest coefficients of variation around target population densities, lowest likelihood of extinction, and longest persistence times (Table 1). Results from the culling model resembled those of single-year duration fertility control, but tended to show slightly higher variability and slightly greater probability of extinction. Lifetime effect fertility control models predicted the highest levels of variability and greatest chance of extinction. In the extreme case, extinctions occurred in >40% of all model runs for lifetime effect representing static application of fertility control with process and observer variation to populations at 25% of K_e (Table 1). Population variability and indicators of extinction risk for models of fertility control with 3-year duration showed effects intermediate to the culling model and the lifetime model. For all models, simulations of adaptive management scenarios increased variability in population density (as measured by coefficients of variation), but reduced probability of extinction and increased persistence time.

DISCUSSION

Relationships to Earlier Models

The models we offer differ in 4 fundamental ways from previous modeling efforts directed at understanding effects of fertility control on vertebrates (Garrott 1991, Garrott and Siniff 1992, Swihart and DeNicola 1993, Boone and Wiegert 1994, Seagle and Close 1996, Pech et al. 1997, White et al. 1997). First, our models are structured in stages where each stage represents the time since a female was treated. Earlier models have been structured by animal age, irrespective of time since delivery. As a result, previous efforts failed to consider effects of duration of contraceptives or the interaction of duration with other model parameters. Stage structure allows us to offer the first analysis of the consequences of variation in the duration of fertility control agents. As a result, we are able to show that animals with long lifetimes (i.e., high adult survival rates) can be effectively controlled with long duration contraceptives, even

when they have relatively low fertility rates. This contrasts with earlier efforts that argued that r-selected species with short lives and high fertility were most appropriate targets for regulation by controlling fertility (e.g., Hone 1992).

Second, we offer closed-form solutions for relationships among delivery rate, proportion of the population infertile, number of animals treated, and target population densities. In the past, estimating these relationships demanded laborious trial and error using simulations (Garrott et al. 1992, Boone and Wiegert 1994, Seagle and Close 1996). Unlike previous results, our models enjoy analytical solutions that provide a rapid and general way to approximate the effort required to stabilize a population using fertility control, without developing detailed simulation models.

Third, our models are the first to consider effects of different fertility control regimes on risks of extinction of small populations. We demonstrate that small populations regulated with long-lived contraceptives will be vulnerable to stochastic extinction.

And finally, we are the first to explicitly consider the inefficiencies associated with resampling unmarked animals when delivering fertility control agents. We show that failing to know the fertility status of individuals can create large inefficiencies in achieving desired levels of infertility in real-world populations.

Although our models differ in important ways from earlier ones, we also reinforce previous findings. Similar to other modeling results, we found that more than half of the breeding females must be infertile to achieve meaningful reductions in population density (Fig. 5). It is true that a smaller infertile fraction may be required for species with low adult survival and low recruitment rates (Fig. 5), but these are not usually the species that cause problems of overabundance. These general results are consistent with several, more detailed studies of effects of fertility control on ungulates (Table 2), as well as, field tests in small mammals (McCallum 1996). It follows from these results that there may be situations where it is logistically impossible to treat a sufficient portion of the population to achieve population objectives. This may explain, in part, why field studies have failed to demonstrate that fertility control can maintain populations at lower densities (McCullough et al. 1997). We should not expect any change in steady state when the proportion of the population treated is small (McCullough et al. 1997).

Our models reveal that this problem is exacerbated by the nonlinear relationship between the proportion of the population treated and the steady-state density (Fig. 4). That is, there is a broad range of levels of infertility producing essentially no change in population equilibrium. This relationship could make it very difficult to assess the effect of different levels of population infertility in field studies because widely differing levels of infertility could result in very similar steady-state densities. Similar nonlinear relationships have been identified in other modeling efforts (Boone and Wiegert 1994, McCallum 1996, Seagle and Close 1996). This problem is magnified in populations where the fertility status of animals is not known (e.g., equations 34 and 35, Fig. 10). In this case, the number of doses that would need to be delivered to achieve a meaningful reduction in population density could easily exceed the total number of animals in the population.

Efficiency of Fertility Control Relative to Culling

There is broad disagreement about the relative efficiency of lethal and nonlethal means of controlling animal abundance. It has been argued that fertility control is more efficient than culling because infertile animals remain in the population, maintaining density-dependent feedback to recruitment and survival (Knipling 1959, Sturtevant 1970, Knipling and McGuire 1972). In contrast, others contend that there are virtually no circumstances in which fertility control will be more efficient than lethal methods (Bomford 1990, Garrott 1995, Nielsen et al. 1997, Swinton et al. 1997). Some workers even assert it is not possible to reduce population numbers with nonlethal control (Seal 1991).

The models we offer can resolve these differences of opinion. There are 2 types of efficiency in population regulation. The first type assesses the amount of time required to bring an overabundant population to an acceptable lower density. There is no question that culling is more efficient than fertility control when efficiency is defined in these terms. Using lethal methods, it is theoretically possible to bring a population to a desired lower density in 1 season of effort. In contrast, the maximum rate of reduction of a population regulated by fertility control is simply $1 - S$, the natural adult mor-

tality rate. Assuming that all fertile females are treated ($c = 1$), our models show that the annual growth rate of the population (λ) is S. Because $S < 1$, the population will decline when $c = 1$, but it may decline very slowly if the adult survival rate is high.

In contrast, it is also possible to define efficiency in terms of the number of animals that must be treated annually to maintain a population at a specified target density. By defining efficiency as the number of animals that must be treated, we deliberately ignore the time required to treat them or the monetary expense of treatment. These costs are highly variable and situation specific. Thus to achieve generality, we will restrict our discussion to the number of animals that must be treated or culled and will allow the reader to apply cost estimates to weight efficiency in a case-specific fashion.

When efficiency is defined in terms of the number that must be treated annually, our models reveal that long-duration fertility control agents can be more efficient than lethal methods if the fertility status of treated animals is known. Although this result may be surprising at first glance, a small thought experiment reveals the intuitive basis for our result. Assume that you have 100 breeding animals with a survival rate of 0.9. On average, each of these animals produces a single female offspring every year. Nine of these offspring must enter the population to offset the losses of adults who die annually. It follows that 91 recruits must be culled to maintain the population at 100 animals. Now, presume instead that the population is stabilized by controlling fertility. Ninety animals are sterilized, leaving 10 fertile. One of the fertile animals dies each year after reproducing. Thus, there are 10 fertile offspring, replacing 9 infertile deaths and 1 fertile death. Only 9 of the recruits must be sterilized to maintain a level of infertility in the population = 90%. However, this result does not hold for short-duration agents. In this case, the 90 fertile adults and 90 recruits must be treated to maintain 90% infertility in the population.

Obviously, this is a grand oversimplification of population dynamics, but it illustrates the fundamental logic underpinning the efficiency of fertility control relative to culling. The number of females that must be treated or culled to maintain a given density depends on the difference between the total number of births and the total number of natural deaths. When a large proportion of long-lived species is sterile, this difference will be likely smaller than in fertile populations that are culled because very few animals will be reproducing. Reducing the duration of the fertility control agent increases the number that must be treated annually until this number eventually exceeds the number culled.

MANAGEMENT IMPLICATIONS

We can provide 4 general insights for implementing fertility control relative to culling. First, the efficacy of fertility control as a means to regulate populations pivots on the ability to develop agents with persistent effects. Second, we also point out that the expense of fertility control will never compete favorably with the revenue that can be provided by licensed hunting, regardless of the duration of contraceptives. Fertility control will only be cost-effective for situations where recreational hunting isn't feasible and where population control can only be achieved by professional treatment, lethal or nonlethal. Third, the feasibility of achieving target levels of infertility in the population will require knowledge of fertility status of individuals. In cases where this status is not known, the inefficiencies associated with redosing animals can increase the number of animals that must be treated by several fold. Finally, the greatest efficiency can be obtained by combining culling with contraception such that an initial reduction in animal numbers is maintained by annual treatment of marked animals with long-acting fertility control agents.

The reason that lifetime duration fertility control can be an efficient method for maintaining a population at target densities also explains why the approach is difficult to implement initially and why it poses grave risks to the persistence of small, closed populations. Our models suggest that it is relatively easy to maintain a population near a given steady state, once a sufficient number of animals have been sterilized. Achieving this initial level of infertility in the population will often be a formidable task, however, and accomplishing that task means that the breeding population will be small. Such breeding populations are subject to stochastic risks of loss of genetic variability and even extinction, as our models illustrated. It follows that while fertility control may not affect survival of individuals, it can easily be lethal to populations.

This creates a genuine dilemma for manag-

ers. Small, closed populations are the most likely candidates for regulation by fertility control. This is simply because delivery of fertility control agents to a sufficient number of animals is most likely to be logistically feasible when populations are small and bounded in space. However, these populations are also the most prone to extinction if managers make mistakes in applying fertility control agents. The nonlinearity in many of the relationships we observed magnifies the likelihood of such errors. It follows that although fertility control can be an efficient means of regulating animal numbers, that efficiency creates risk for the population manager. As with all other techniques, managing population fertility must be grounded in a fundamental understanding of the state of the population and the role of management in influencing its dynamics. This simple fact reinforces the conclusions of Garrott (1995) that careful field studies applying control techniques over long time periods are needed to thoroughly understand effects of fertility control.

We offer a suite of predictions that can be usefully tested in field studies. Our models provide a sketch of the dynamics of populations regulated by fertility control. Offering a sketch rather than a blueprint requires that we avoid many details. Understanding those details will no doubt prove necessary to minimize unpleasant surprises in managing fertility of ungulate populations (Caughley et al. 1992, Nettles 1997). However, by offering general rather than detailed models, we provide broad insights useful as a starting point for future studies.

We identify 2 urgent needs for future work on fertility control agents. Development of techniques that render animals infertile with a single dose offer a promising alternative to culling in situations where it is feasible to mark animals to know their fertility status. Thus, work at the level of the individual animal should emphasize increasing the duration of effect of fertility control agents. Studies at the level of the population must demonstrate that fertility control can achieve meaningful reductions in rates of increase in the field. Although models like ours can suggest situations where fertility control could be efficacious in controlling abundance of ungulates, such efficacy remains in doubt until field studies demonstrate such control in real-world populations.

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APPENDIX A

Example Derivation of Deterministic Results

We used a standard series of steps to analyze all models. Here, we illustrate those steps using fixed-effect model, assuming prebreeding delivery of contraceptives. We begin with a state vector $[F\ I_1\ I_2\ I_3]$ for the case where duration of fertility control persists for 3 years. The corresponding projection matrix is

$$\begin{bmatrix} (1-c)(S+m-\beta N) & 0 & 0 & S(1-c) \\ c(S+m-\beta N) & 0 & 0 & Sc \\ 0 & S & 0 & 0 \\ 0 & 0 & S & 0 \end{bmatrix} \quad (36)$$

We obtain the characteristic polynomial of the matrix:

$$0 = \lambda^4 - \lambda S^3 c - S\lambda^3 - m\lambda^3 + \beta N\lambda^3 + Sc\lambda^3 + cm\lambda^3 - c\beta N\lambda^3. \quad (37)$$

By iterating over several values of τ, we observe a general series in characteristic polynomials for all $(\tau + 1 \times \tau + 1)$ matrices:

$$0 = \lambda^{\tau+1} - \lambda S^\tau c - S\lambda^\tau - m\lambda^\tau + \beta N\lambda^\tau + Sc\lambda^\tau + cm\lambda^\tau - c\beta N\lambda^\tau. \quad (38)$$

When the population is at equilibrium, $\lambda = 1$. Therefore, to find a general expression for the equilibrium density as a function of delivery rate we set $\lambda = 1$ and solve equation 38 for N, obtaining:

$$N^* = \frac{S + m + S^\tau c - 1 - Sc - cm}{\beta(1-c)}. \quad (39)$$

Solving for c, we find the delivery rate (c^*) needed to achieve a specified target density (N^*),

$$c^* = \frac{S + m - N^*\beta - 1}{S + m - N^*\beta - S^\tau}. \quad (40)$$

To find the composition of the population (i.e., the proportion of each stage of infertility in the population) we obtain the eigenvectors of several projection matrices corresponding to different values of τ. For each matrix, we extract the first element ($v[1]$) of the normalized vector (corresponding to an eigenvalue of 1), which gives the proportion of the population that is fertile,

$$v[1] = \frac{1-c}{(1-c)/c + 1 + S + S^2 + S^3 \ldots + S^\tau}. \quad (41)$$

To find the infertile proportion (P), we subtract $v[1]$ from 1 and simplify,

$$P = \frac{(1 - S^\tau)c}{1 + Sc - S - S^\tau c}. \quad (42)$$

In a similar way, we observe the last normalized element of the eigenvector for several values of τ and find that the proportion of the population that is in the final stage of infertility (ρ) is given by a series which simplifies to

$$\rho = v[\tau + 1] = \frac{(1-S)S^{\tau-1}c}{1 + Sc - S - S^\tau c}. \quad (43)$$

We then derive a general expression for the proportion of the population (P) that must be infertile to achieve a target density N^*. We substitute the right hand side of equation for c in equation 42, and after some algebra, obtain

$$P = 1 - \frac{1-S}{m - N^*\beta}. \quad (44)$$

To remove β from the denominator, we observe from equation 39 that the equilibrium density in the absence of fertility control (i.e., $c = 0$) is

$$K = \frac{S + m - 1}{\beta}. \quad (45)$$

Defining Δ as the proportional reduction in population density below K (i.e., $N^* = \Delta K$), it follows that

$$N^* = \Delta K = \frac{\Delta(S + m - 1)}{\beta}. \quad (46)$$

Substituting the right hand side of equation 46 for N^* in equation 44,

$$P = 1 - \frac{1 - S}{m + \Delta(1 - S - m)}. \quad (47)$$

To derive an expression for the number of animals treated (n) to maintain a target density, we observe from the projection matrix and the state vector that the number of animals treated at equilibrium is

$$n = c^*F_t(S + m - \beta N^*) + c^*I_{t_\tau}, \quad (48)$$

which is simply the treatment rate multiplied by the steady state number of fertile females at time t plus the number of females that are recruited plus the number that regain fertility during $t \rightarrow t + 1$. Substituting $(1 - P)N^*$ for F_t and ρN^* for I_{t_τ} we have

$$n = c^* (1 - P)N^* (S + m - \beta N^*) + c^* \rho N^*, \quad (49)$$

Substituting the right-hand-side of equation 40 for c^*, rhs equation 44 for P and rhs equation 43 for ρ provides the needed expression for n in terms of the decision variable N^* and the parameters m, S, and β.

APPENDIX B

Derivation of an Unbiased Estimator for the Number of Doses that must be Delivered to a Population of Unknown Fertility to Achieve a Target Level of Infertility

We assume that a simple random sample of d animals is selected with replacement from a population of N animals. All of the animals in the sample are dosed. At the beginning of the sampling effort, the population contains I infertile animals. In order to establish the results shown in equations 34 and 35 we note that equation 35 is a special case of equation 36 where $I = 0$. We define t_i as an indicator random variable that equals 0 if the *ith* fertile animal is still not dosed after d selected animals are treated and equals 1 if it has been dosed at least once for $i = 1, 2 \ldots, N - I$. We define a as the number of additional infertile animals that are added to the population after d doses are delivered,

$$a = \sum_{i=1}^{N-I} t_i. \quad (50)$$

The expected number of additional infertile animals is given as

$$\eta = E(a) = \sum_{i=1}^{N-I} E(t_i) = (N - I)E(t_i). \quad (51)$$

Define $P(t_I = 1)$ as the probability that $t_i = 1$; then,

$$E(t_i) = P(t_i = 1) = 1 - P(t_i = 0) = 1 - \left(\frac{N - 1}{N}\right)^d. \quad (52)$$

Thus,

$$\eta = (N - I)\left(1 - \left(\frac{N - 1}{N}\right)^d\right). \quad (53)$$

Solving equation 53 for d gives equation 35. The variance of a can also be easily determined.