Analysis and Management of Animal Populations

Modeling, Estimation, and Decision Making

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- The population can be maintained below that level by removing the growth increment.

For a logistic population, the MSY is achieved by maintaining population size at $K/2$, the point at which population growth is maximum. This result is of course specific to the logistic model, and other models can be expected to exhibit different equilibrium values.

We note that an analogous treatment is possible for the continuous model

$$dN/dt = rN(t)[1 - N(t)/K] - H(t),$$

with $H(t)$ now representing instantaneous harvest. Equilibrium for this model is defined by $dN/dt = 0$, which requires that

$$H(t) = rN(t)[1 - N(t)/K]$$

or

$$h(t) = r[1 - N(t)/K].$$

But this is the same equilibrium condition as for the discrete model, with the same formulas (properly interpreted in terms of instantaneous rather than discrete rates) for maximum sustainable yield and equilibrium population size.

11.1.3. Compensatory and Additive Mortality

The concept of compensatory mortality was introduced previously in Chapters 3 and 8. Recall that under the compensatory mortality hypothesis (CMH), increasing harvest rates are compensated by density-dependent changes in nonharvest mortality factors. Thus, when harvest rates are reduced, densities are higher than they otherwise would have been, and natural mortality rates consequently increase. Conversely, as harvest rates increase, densities are lowered and natural mortality operates at a reduced rate. For complete compensation, there is no change in the annual mortality rate with changes in harvest rate, up to a threshold harvest rate, the maximum value of which is the mortality rate in the absence of hunting (Anderson and Burnham, 1976).

Opposing the CMH is the additive mortality hypothesis (AMH), which presumably operates in the absence of density-dependent mechanisms that would affect nonharvest mortality. Thus, the AMH implies that as harvest mortality increases, total annual mortality increases proportionately, thereby producing a linear relationship between harvest rate and annual mortality.

Recall that these relationships were described in Chapter 8 in terms of the relationship between survival and harvest rates. The additive mortality hypothesis was defined there by an approximately linear relationship between survival and harvest, with strict additivity producing an equivalent decrease in survival rate when harvest rate is increased. On the other hand, compensatory mortality was defined by changes in nonharvest sources of mortality that compensate for changes in harvest mortality, so that within limits, survival rate remains unchanged as harvest rates increase.

Here we describe the compensatory and additive mortality hypotheses in terms of the relationship of harvest to mortality (rather than survival). Assume that harvest mortality occurs at the beginning of the year and is followed by nonhunting mortality. Then a simple linear model relating nonharvest mortality $m(t)$ in the presence of harvest to mortality from harvest $h(t)$ is given by

$$m(t) = m_0 + \beta h(t), \tag{11.9}$$

where m_0 is natural mortality in the absence of harvest, with $-1 \leq \beta \leq -\alpha m_0$ and α close to 1 (e.g., $\alpha = 0.95$) (Anderson and Burnham, 1976). The value $\beta = -1$ represents complete compensation, with $\beta = -\alpha m_0$ representing strict additivity. Thus, we have

$$m(t) = m_0 - h(t) \tag{11.10}$$

under the CMH, which essentially says that within limits the sum of hunting and nonhunting mortality remains constant:

$$m(t) + h(t) = m_0.$$

Note that Eq. (11.10) describes a linear relationship between nonhunting mortality and hunting mortality, with a slope of -1. Because the CMH operates through compensatory decreases in nonharvest mortality $m(t)$, the amount of compensation cannot exceed m_0, and this value provides an upper bound for the threshold C, beyond which additional harvest mortality becomes additive.

On the other hand, Eq. (11.9) with $\alpha = 1$ yields

$$\begin{aligned} m(t) &= m_0 - m_0 h(t) \\ &= m_0[1 - h(t)] \end{aligned} \tag{11.11}$$

under the AMH. This expression is intuitively reasonable, because an animal must survive harvest mortality {with probability $[1 - h(t)]$} in order to have a chance of dying from nonharvest mortality. As with Eq. (11.10), this equation describes a linear relationship between hunting and nonhunting mortality. However, the slope of the relationship in Eq. (11.11) is $-m_0$, which is greater than -1. Thus, the reduction in nonhunting mortality attendant to increases in hunting mortality is less than that occurring beyond the threshold C under the CMH.

The relationship between $m(t)$ and $h(t)$ is summarized under both the CMH and the AMH in Fig. 11.2a. Note that $m(t)$ declines with increasing $h(t)$ under both hypotheses, as a result of competition between risks associated with harvest mortality and nonharvest mortality (animals killed by harvest cannot be lost to other sources). However, this decline does not represent compensation, which is engendered by density-dependent mortality mechanisms (Anderson and Burnham, 1976; Nichols *et al.*, 1984d). The region between the two curves represents the range in potential compensation, from completely compensatory (lower curve) to completely additive (upper curve).

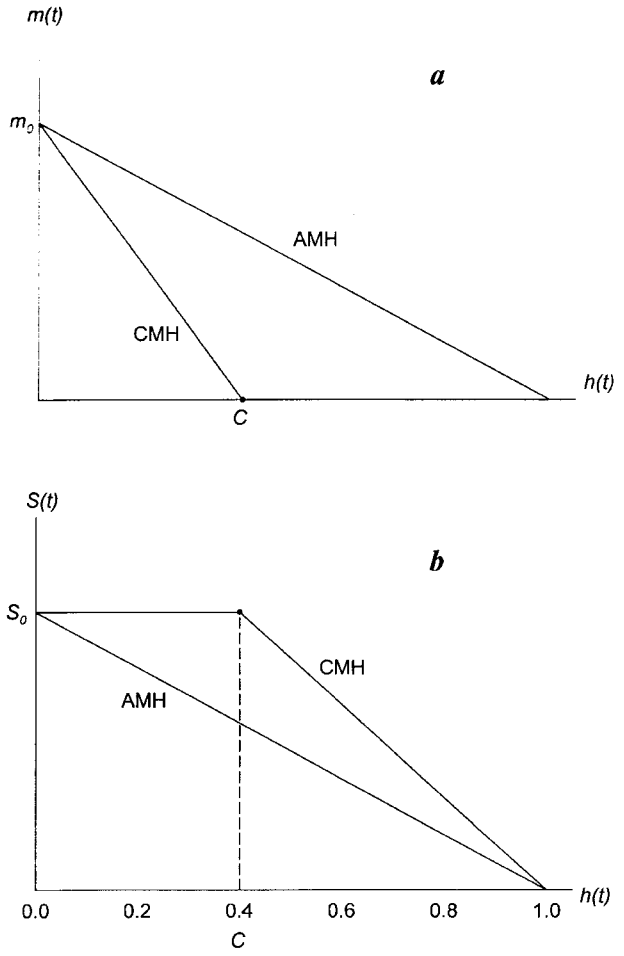

FIGURE 11.2 Hypotheses of compensatory (CMH) and additive (AMH) mortality. (a) Relationship between natural mortality m and harvest rate h. Under both hypotheses, m declines from m_0 (natural mortality in absence of harvest, here taken as 0.5) as h increases, because of competition between these sources of mortality. Under the CMH, the decline is steeper and is sufficient to balance h. (b) Relationship between annual survival S and harvest mortality h. Under the AMH, each increment in h is additive to overall mortality, resulting in a linear decline in annual survival. Under the CMH, there is complete compensation up to the threshold C—in this example $C = 0.4$; the maximum potential for compensation, $C_{max} = m_0$, is 0.5 in this example.

By definition, total annual mortality is the sum of mortality from both harvest and nonharvest sources; that is,

$$1 - S(t) = h(t) + m(t),$$

so that

$$\begin{aligned} S(t) &= 1 - h(t) - m(t) \\ &= 1 - h(t) - [m_0 + \beta h(t)]. \end{aligned}$$

Substitution of $m(t)$ from Eq. (11.10) yields

$$S(t) = 1 - m_0$$

under the CMH [for $h(t) < C$], whereas substitution of $m(t)$ from Eq. (11.11) yields

$$S(t) = S_0[1 - h(t)] \tag{11.12}$$

under the AMH. Thus, the effect of the additive hypothesis is essentially to add a harvest component to nominal mortality m_0, thereby decreasing the survival rate as in Eq. (11.12) (Fig. 11.2b). On the other hand, the CMH leaves unchanged the survival rate over a range of values for harvest rate up to the compensation limit, with declines thereafter as harvest rate increases:

$$S(t) = \begin{cases} \left[\dfrac{1 - h(t)}{1 - C}\right] S_0 & h(t) > C \\ S_0 & h(t) \leq C. \end{cases} \tag{11.13}$$

Thus, compensatory harvest has no effect on population dynamics if the harvest rate is sufficiently small, but reduces survival if the harvest rate is in excess of C.

The relationships in Fig. 11.2a represent the results of a mechanism (density-dependent, compensatory mortality) by which a relationship between changes in harvest rates and survival rates (Fig. 11.2b) arises. The contrast between the phenomenological and mechanistic modeling of compensation can be clarified by a slight recasting of the definition of annual survival:

$$S(t) = \theta_t[1 - h(t)]$$

for $0 \leq h(t) \leq 1$, where θ_t is survival from nonharvest sources, and may vary according to density or abundance. If this compensating variation in nonharvest survival is thought to occur immediately after the harvest period (e.g., in the wintering period for waterfowl), then a reasonable model for θ_t may be

$$\theta_t = \frac{e^{a+bN(t)[1 - h(t)]}}{1 + e^{a+bN(t)[1 - h(t)]}},$$

where $N(t)[1 - h(t)]$ represents the number of animals surviving the harvest period and thus influencing nonharvest mortality. From this model one can deduce compensatory relationships that are very similar to that portrayed in Eq. (11.13) and Fig. 11.2b, except now the strength of the compensatory relationship varies according to the initial (preharvest) population size. In the special case for which nonharvest mortality is independent of density, the coefficient b in the expression is zero, and the expression simplifies to

$$S(t) = \theta[1 - h(t)]$$

for $0 \leq h(t) \leq 1$, which is equivalent to the additive model in Eq. (11.11). Values of the coefficient $\beta < 0$ indicate density dependence and reflect a degree of compensation.

11.1.3.1. Partial Compensation Model with Variable Thresholds

The CMH and AMH hypotheses represent extremes, in that the density-dependent mechanism for the CMH is thought to compensate perfectly for changes in harvest rates up to the theoretical threshold $C = 1 - S_0$. On the other hand, it is assumed under the AHM that nonharvest mortality is independent of density, so that harvest is not compensated by changes in nonharvest mortality. These hypotheses provide logical extremes of a gradient of possible responses to harvest. However, it is perhaps more reasonable to expect compensation, if it occurs, to be less than complete, with the strength of compensation depending on both life history attributes and environmental conditions.

Under a partial compensation hypothesis (PCH), it is assumed that below a threshold C, compensation occurs, but at a level so as not to compensate completely for harvest mortality. Thus, annual survival declines with increasing harvest, but not as much as if there were no compensation (Fig. 11.3). This hypothesis is a special case of Eq. (11.9) with $-1 < \beta < -m_0$. We illustrate the PCH for a case where $\beta = -0.75$, intermediate between complete compensation ($\beta = -1$) and complete additivity ($\beta = -m_0 = -0.50$). PCH implies that there is a density-dependent relationship between nonharvest mortality and abundance, but that this relationship is not as strong as under complete compensation, and therefore is inadequate to balance completely changes in harvest mortality (Fig. 11.3b). Both the slope of the compensatory relationship and the threshold beyond which harvest mortality must be additive may depend on the life history of the animal (Patterson, 1979; Conroy and Krementz, 1990). For example, long-lived animals have a low natural mortality rate in the absence of harvest, so that the total amount

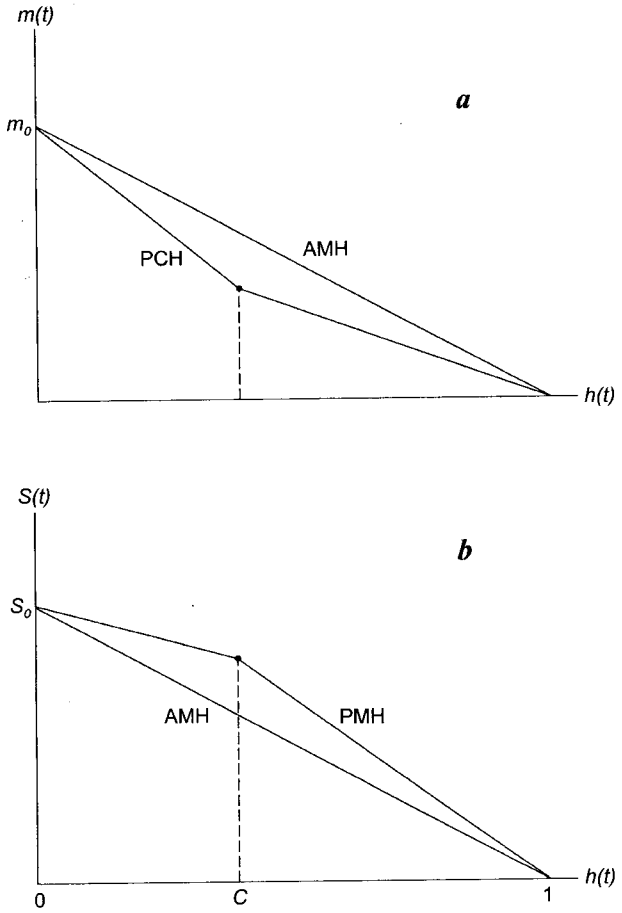

FIGURE 11.3 Hypotheses of partially compensatory (PCH) and additive (AMH) mortality. (a) Relationship between natural mortality m and harvest rate h. Under both hypotheses, m declines from m_0 (natural mortality in absence of harvest, here taken as 0.5) as h increases, because of competition between these sources of mortality. Under the PCH, the decline is steeper, but not sufficient to balance h completely. (b) Relationship between annual survival S and harvest mortality h. Under the AMH, each increment in h is additive to overall mortality, resulting in a linear decline in annual survival. Under the PCH, there is partial compensation up to the threshold C.

of possible compensation is lower than for shorter lived animals, where m_0 is higher (recall that m_0 provides an upper bound for C; i.e., $C \leq m_0$). However, so-called K-strategists, which have a lower threshold with respect to harvest mortality, may also have stronger density-dependent mortality responses (and thus stronger compensation) below that threshold than r-strategists (Conroy and Krementz, 1990). In addition, even for species in which compensation might be theoretically strong, temporally varying environmental conditions can override density dependence in some years, effectively creating variable thresholds to harvest mortality (Conroy and Krementz, 1990).

It is important to remember that the AMH, CMH, and PCH relate only to the relationship between harvest and mortality rates. Because population growth is determined by reproductive rates as well, it is possible for increases in harvest rates to be balanced (i.e., compensated for) by increasing reproductive rates, even if mortality operates according to AMH (i.e., there is no mortality compensation). Indeed, many arguments for compensation in large herbivores focus on the reproduction side of life histories, often suggesting that increased population levels (e.g., in populations subjected to no or low harvest pressure) result in depression of birth rates because of nutritional stress or other factors (Fowler, 1987; Gaillard *et al.*, 1998, 2000). Of course, similar arguments can be made for mortality rates, e.g., increased population levels result in increased starvation during wintering periods. Although much of the discussion regarding compensation in waterfowl has focused on mortality (Anderson and Burnham, 1976; Nichols *et al.*, 1984d; Conroy and Krementz, 1990), temporary decreases in survival with increasing harvest rates (under additive mortality assumptions) still might not result in decreased population growth, because of density-dependent increases in reproduction rates (e.g., Nichols *et al.*, 1984d).

Finally, it is important to keep in mind that nearly all arguments regarding compensation refer to the relationship between per capita rates of mortality from harvest and other causes, and corresponding rates of population growth. Knowledge of the absolute numbers of animals harvested or dying from other causes is inadequate to make inferences about the overall population impacts of harvest, unless (1) these statistics can be used to compute per capita rates, and (2) the relationship between rates of harvest, rates of other mortality, and overall rates of population growth can be established.

11.1.4. Methods for Determining the Impacts of Harvest

Much of harvest management is based on an underlying conceptual or mathematical model of the effect of harvest on population vital rates. While we advocate the use of models as a provisional basis for management, the literature is replete with examples in which models have been used in the absence of adequate empirical justification, with potentially serious impacts on resources (e.g., Conroy, 1993; Heppell and Crowder, 1996). Here we briefly examine some methods for evaluating the impacts of harvest on populations and thus for selecting an appropriate harvest model and estimating its parameters. This discussion is general, and a full development must await the estimation methods developed in Part III and the optimal decision methods in Part IV, where the estimation and assessment of harvest impacts are treated in some detail.

11.1.4.1. Observational Studies

Most of the studies dealing with the impact of harvest investigate empirical relationships between harvest rates and population growth rates, birth rates, and death rates based on time series of historical data. We term these *observational* studies (Section 6.8.2), to distinguish them from experiments in which a deliberate attempt is made to manipulate harvest rates or population densities under design conditions (see Chapter 6). For example, band-recovery methods have been used extensively to investigate the effects of hunting on survival of waterfowl (e.g., Anderson and Burnham, 1976; Nichols and Hines, 1983; Burnham *et al.*, 1984) [see reviews in Nichols *et al.* (1984d), U.S. Department of the Interior (1988), and Nichols (1991b)]. Though these studies have provided good evidence for compensatory mortality in some species, the evidence for other species is incomplete. Even for the heavily investigated mallard (*Anas platyrhynchos*), inferences suffer from an overreliance on statistical tests based on attributes of additivity vs. compensation, rather than the testing of mechanistic hypotheses of density dependence (Nichols *et al.*, 1984d; Conroy and Krementz, 1990).

11.1.4.2. Experimental Studies

Because of the absence of direct manipulation and experimental controls (see Chapter 6), purely observational studies suffer from an inability to ascribe causation. There have been few studies in which harvest rates or population densities have been directly manipulated in order to observe the population response (e.g., Bartmann *et al.*, 1992). Obviously, a manipulative study is feasible only under special conditions, which are especially difficult to meet with large mobile populations. Nonetheless, we believe that experimental approaches, perhaps under constrained designs, are needed to answer questions about the impacts of harvest (Anderson *et al.*, 1987).

11.1.4.3. Adaptive Resource Management

We advocate a particularly powerful method for scientific management, *adaptive resource management* (ARM) (Walters, 1986). In ARM, the emphasis is placed on decision-making (e.g., the setting of harvest regulations) to reach a long-term resource goal (e.g., maxi-