CHAPTER 8

Predation and wildlife populations

If there are any marks at all of special design in creation, one of the things most evidently designed is that a large proportion of all animals should pass their existence in tormenting and devouring other animals.

J.S. Mill (1874, from Taylor 1984:1)

The large, ferocious gray or buffalo wolf, the sneaking, snarling coyote, and a species apparently between the two, of a dark-brown or black color, were once exceedingly numerous in all portions of the Park, but the value of their hides and their easy slaughter with strychnine-poisoned carcasses of animals have nearly led to their extermination.

P. Norris (1881), Second Superintendent of Yellowstone National Park

INTRODUCTION

With its gore, its excitement, and its brutal finality, predation has always fascinated humans. Biologists have built on the core intrigue of predator–prey dynamics, co-opting the term **arms race** – widely used to refer to how human armies inevitably escalate technology to keep up with each other – to describe the evolutionary changes in both predators and prey (Dawkins & Krebs 1979). For wildlife prey we see speed, poisons, coloration, armor, alertness, and deception, matched on the field of battle by similar traits in the predator.

As wildlife population biologists, an understanding of predation is important because the public is vocal and curious about what happens to predators and prey, and because predation plays such an important role in population dynamics. Some of the most controversial issues in wildlife and conservation biology hinge on the extent to which predators affect prey numbers. As one recent example of a theme that has played out all over the world for centuries, on January 12, 2006 the *Idaho Statesman* newspaper reported that the Idaho Department of Fish and Game "plans to kill up to 75% of the wolves in the Lolo elk zone to bolster struggling elk herds there."

Do wolves and other predators control or adversely affect their prey, so that killing the predators will in fact bolster the abundance of their prey? Similarly, in the context of invasive species (Chapter 11), would a predator biocontrol agent successfully reduce the numbers of a pest or invasive species? Or might native species be driven inadvertently toward extinction by the introduced biocontrol predator?

To help shed light on these questions, this chapter focuses on the effect of predators on prey dynamics and, to a lesser extent, the effect of prey on predator numbers. I will emphasize concepts, avoiding a plunge into the sea of predator–prey models, including the famous Lotka–Volterra predator–prey equations. These have heuristic value to general ecology, but less practical value to applied population biology.

Before jumping in, we must define two key terms. First, let's look at the concept of predators controlling

Conservation of Wildlife Populations: Demography, Genetics, and Management, Second Edition. L. Scott Mills.

Box 8.1 Would invasive predator control be effective for two species of shearwaters?

For two species of New Zealand shearwaters, a management concern is whether control of exotic predators would be an efficient path to recovery. Predators include, most prominently, stoats (a type of weasel otherwise known as ermine), introduced to New Zealand in the 1880s, as well as other introduced mammalian predators such as rats and cats. The main factors that affect how the Hutton's and sooty shearwaters are affected by predators include the following:

- The location of colonies affects the suite of predators. Hutton's shearwaters nest above the snowline and stoats are their only substantial predator. By contrast, sooty shearwaters nest close to sea level and must contend with a suite of introduced predators including not only stoats but also cats and rats.
- The size of existing colonies affects the impact of predation. The two remaining colonies of Hutton's shearwaters contain about 110,000 and 10,000 breeding pairs. Because predator (stoat) numbers are limited by a lack of prey over the winter (when shearwaters and many other species are gone), the predation rate is fairly dilute. On the other hand, sooty shearwater colonies are much smaller, making the predation rate (number of kills/prey population size) much higher.

Therefore, predation on sooty shearwaters has led to low and highly variable breeding success and adult survival. To increase sooty shearwater abundance, the only real management solution is aggressive reduction of the whole suite of predator species, including not only stoats but also cats and rats. By contrast, the relatively low predation rates on Hutton's shearwater indicate that its population growth might be marginally affected by even complete stoat removal. Therefore, management strategies for Hutton's shearwaters should include minimizing destructive browsing by introduced mammals and establishing alternative breeding sites (Cuthbert et al. 2001, Cuthbert & Davis 2002, Jones 2002).

prey. Taylor (1984) has noted that the word **control** has been used in predator–prey discussion to mean almost anything and, therefore, nothing. When I refer to predators controlling prey I will specify particular outcomes, such as predation regulating prey numbers and affecting fluctuations around an equilibrium, or acting to limit prey at low numbers, including extinction.[1]

The second term to define is who might qualify as a predator or prey. Do herbivores prey on plants, do decomposers prey on dead animals, or granivores on seeds? Is a parasite or disease a predator? Is it predation if an animal kills one of its own species in a fight? For the purposes of this chapter I will focus primarily on animals killing and usually consuming animals, recognizing that even in this narrow definition there can be surprises: the main killer (and consumer) of preweaning snowshoe hares are not big-fanged carnivores, but rather red squirrels and ground squirrels (O'Donoghue 1994).

[1] Recall from Chapter 7 that **regulation** refers to maintaining numbers within an equilibrium range via negative density dependence, while limiting factors determine the actual equilibrium numbers and may be density-dependent or density-independent.

DOES PREDATION AFFECT PREY NUMBERS?

As you might expect, the best short answer to this question is sometimes or that it depends (Box 8.1). We do not have to look very far to see examples where predators limit prey population size – potentially all the way to extinction – or cause oscillations in prey abundance to be either exacerbated or dampened. Some of the most spectacular examples of control are with recently introduced predators, both when they arrive and after they are removed. Cats, rats, brown tree

snakes, and foxes have caused devastating extinctions around the world when they arrive in a new area. Indeed, 40% of the extinctions of birds on islands have been caused by predation by introduced animals (Estes et al. 2001).

One reason why native prey – particularly on islands – can be so badly affected by introduced predators is that the prey are a big step behind in the arms race, lacking the adaptations necessary to escape or even to fear the predators. The loss of anti-predator behaviors, leading to **ecological naiveté** of prey on islands, could arise either from the chance loss of key traits when an island is founded by a few individuals or from relaxed selection on anti-predator behaviors that are potentially expensive to maintain (Blumstein & Daniel 2005). As Quammen (1996:205–6) describes it:

> Loss of wariness is sometimes manifest as ingenuous nesting behavior: In the Galapagos, the blue-footed booby puts its eggs onto a bare patch of ground, unprotected, unconcealed, not even cushioned by a cradle of vegetation. Another form of ingenuous nesting involves building a nest in plain view on a tree limb, where it can easily be raided by a climbing predator. The Mariana crow practices that sort of reckless behavior on the island of Guam. A more cautious bird might at least conceal the nest, or place it beyond reach at the end of a thin branch, or suspend it in an elaborate woven pouch, as the tropical oropendolas do. But oropendolas are mainland species, surrounded by predators and obliged to be more cautious. Boobies can be boobies . . . These animals aren't imbecilic. Evolution has merely prepared them for life in a little world that is simpler and more innocent than the big world.

When they have evolved together, predators and prey interact on more equal footing, but prey density or fluctuations can still be affected by predation. The classic cycles of snowshoe hare in North America are driven at least in part by predation (Krebs et al. 1995), as are the regular, widespread cycles of northern small mammals (Korpimäki & Norrdahl 1998). More surprisingly, mammalian carnivores often kill other carnivores (**intraguild predation**), accounting for up to 89% of known mortalities in some species and at times limiting numbers (Palomares & Caro 1999, Donadio & Buskirk 2006).

A more subtle, but potentially pervasive line of evidence for effects of predators comes from changes more than one trophic level removed from a top predator. **Mesopredator release** (Soulé et al. 1988) occurs when mesopredators (mid-level predators) are regulated by top predators through either predation or competition. If the top predator is removed, a top-down **trophic cascade** can occur, whereby the mesopredators increase in number and in turn decrease abundance of their prey. A classic example was documented in southern California, where intensive urbanization has destroyed most of the native sage-scrub habitat (Crooks & Soulé 1999). With the decline or absence of coyotes from this system, both native mesopredators (striped skunk, raccoon, and grey fox) and exotic mesopredators (especially domestic cats) were released from predation and competition from coyotes (the cat response also occurred because without coyotes around owners tended to let their cats outside more often). The resulting high numbers of mesopredators cascaded into both higher overall prey mortality (cats around a single moderately sized canyon killed more than 500 birds, nearly 1000 rodents, and over 600 lizards per year) and reduced abundances of scrub-breeding birds. Trophic cascades initiated by vertebrate predators in terrestrial systems are fairly common in nature (Schmitz et al. 2000).

Despite the range of examples where predators do reduce numbers of their prey, we also see plenty of places in the wild where prey continue to persist and even flourish with predators in their midst. To foreshadow a theme of the chapter, prey are active participants in the life and death process, evolving and behaving to reduce their chances of being killed. Even predator-naïve animals can exhibit reactions of caution that can reduce vulnerability to novel predators. For example, the last population of the rufous hare-wallaby on the Australian mainland was destroyed by a fire and foxes in 1991, so that the species persisted only on two islands off the coast. However, captive-breeding trained hare-wallabies to avoid cat and fox predators that they would confront following reintroduction (McLean et al. 1996). Also, at a population level, the death of prey individuals, no matter how massive or macabre it may seem to us, does not necessarily result in a smaller prey population; consider that roughly one-third to one-half of all bird nests are destroyed by predators, but the decline of bird populations following such predation is certainly not inevitable (Côté & Sutherland 1997).

In short, predators and prey are entwined in a dance of evolution and population response. The best

generalization we can make on population response is that predation can certainly regulate and help limit numbers of prey, but is unlikely to drive prey populations to extinction unless introduced species are involved or the prey population is small and fragmented or otherwise affected by other recent perturbations (Macdonald et al. 1999). To extend this generalization, we will closely examine three main factors that determine whether a predator will limit or regulate prey in any particular case: the predation rate of the predator on the prey (in turn a function of predator and prey numbers, and the number of prey killed per predator), the degree to which the predation can be compensated for by the prey, and which individuals are killed. Considering these factors will help us answer the question of whether predators in a particular setting are likely to affect the dynamics of their prey.

FACTOR 1. DETERMINING HOW PREDATION AFFECTS PREY NUMBERS: PREDATION RATE

Prey face a world "red in tooth and claw" (as Lord Tennyson put it in his 1849 poem), populated by predators that can respond to an increasing number of prey by increasing their own numbers and by individually killing more prey. Therefore, the total number of prey killed will be a product of both the number of predators (the predator **numerical response**) and how many prey each individual predator kills (the predator **functional response**). So **predation rate**, or percentage of the prey population killed per unit time, is

$$\text{Predation rate per unit time} = \frac{\text{Number of prey killed}}{\text{Prey abundance}} \times 100 \quad (8.1)$$

Number of prey killed is the predator functional response times the predator numerical response. Next we will discuss the predator numerical and functional responses, separately, and then merge them to determine the number of prey killed and predation rates.

Numerical responses of predators

The **numerical response** reflects the change in number of predators as prey abundance changes; more precisely, it is the equilibrium numbers of predators present at a given prey density (there could be a time lag between current prey numbers and the eventual equilibrium predator number). Within a population, the numerical response will be a function of the predator's birth and death rates (which we know can be captured as λ or r), and how the available prey affect those birth and death rates.

In addition to the numerical response mounted from within the predator population, more rapid numerical increases in a predator's population can be driven by an **aggregative response**, whereby predators converge from elsewhere to consume prey. Aggregative responses are of special interest in the agricultural pest arena, because the numerical response of, say, an avian predator to an outbreaking insect pest would be much more rapid if accompanied by an aggregative response. To cite one such case, Carolina chickadees rapidly congregate in woodlands with greater densities of leaf-mining moths, aiding in suppression of the moth (Connor et al. 1999).

Predicting and interpreting numerical responses becomes more complicated with multiple species of predator or prey. With several predator species, reducing the abundance of one predator (say through predator control) could actually increase the numerical response of other predators due to trophic cascades or relaxed competition. This seems to be what happened in New Zealand when attempts to remove stoats to protect nesting birds (Box 8.1) increased introduced rat numbers (one prey of stoats), which in turn increased predation on sooty shearwaters (Lyver et al. 2000).

In the case of multiple prey species, if one or more prey species are better able to increase or sustain their numbers in the presence of predation, they may facilitate a numerical response in the predators that results in a decrease of other prey species. Thus, what seems like competition between alternate prey species may actually be enemy-mediated **apparent competition** (Chaneton & Bonsall 2000, DeCesare et al. 2010), where prey species affect each other's abundances through their effects on the numerical response of a shared predator. For example, woodland caribou in Canada are exposed to multiple native predators (especially wolves, cougars, and bears) that in turn are supported by multiple prey (especially moose and deer) that do quite well in the human-modified landscape. The incidental take of caribou by the abundant subsidized predators reduces caribou population growth via apparent competition (Wittmer et al. 2007).

Box 8.2 Introduced rabbits lead to hyperpredation by cats on native species

Rabbits have been introduced – usually intentionally – to hundreds of islands worldwide. They adapt well to most conditions, eat a variety of plants, and have high population growth rates. Rabbits certainly have direct effects on both the vegetation and on other grazing species that are competitively inferior. Less well appreciated and probably more insidious, however, are the indirect effects they can have on native wildlife via apparent competition and hyperpredation (Courchamp et al. 1999, 2000, Norbury 2001). Because they can withstand predation and still increase in numbers, rabbits sustain high cat numbers even when native prey for the cats are sparse. For example, on the sub-Antarctic island of Macquarie, introduced cats persisted with parakeets for more than 60 years. However, within 20 years of rabbit introduction the parakeet was extinct – and other native prey reduced in numbers – after introduced rabbits increased cat numbers. Likewise, in New Zealand, both cats and introduced stoat populations are supported by rabbits, and highly endangered native grand and Otago skinks suffer elevated predation as a result. The effects are worst when rabbit density fluctuates, because the sustained predator community switches to skinks most ferociously when rabbit numbers temporarily decrease. The moral of the story is that to reduce effects of invasive predators in these cases we must also deal with the rabbits. Control of rabbits needs to be sustained, because if it is tentative, allowing rabbits to bounce back in repeated pulses, the predator suite could switch to native fauna during rabbit lows and cause even more damage.

A different type of enemy-mediated apparent competition called **hyperpredation** occurs with the introduction of both a predator and an introduced prey that is able to sustain or increase its numbers in the face of predation. The ability of the introduced prey to numerically withstand predation increases numbers of the predator, which in turn decimates a native prey. Thus, a native prey species declines with the arrival of an introduced prey, but the mechanism is not competition but rather hyperpredation via apparent competition (Box 8.2).

A striking example of hyperpredation initiated by humans involves cats (the hyperpredator) and cat food (the cat's reliable "prey"). Cat food can maintain both domestic and semi-feral farm cats at densities far higher than native carnivores (Woods et al. 2003, Kays & DeWan 2004). In Great Britain the cat population of approximately 9 million is about 20 times that of stoats and weasels and more than 30 times that of foxes. Cat numbers in the US total perhaps 80–100 million owned, stray, or feral cats. Because of their numerical response (coupled with a functional response of tens to hundreds of wild birds and mammals killed per cat), free-roaming cats have caused local extinctions of native birds and mammals throughout the world (Mack et al. 2000, Risbey et al. 2000).

Awareness of enemy-mediated apparent competition can lead to better management decisions that may not be obvious (e.g., Box 8.2). A classic case involved proposals to remove feral pigs from the California Channel Islands (US), both because the pigs have badly damaged the islands' native vegetation and have supported through hyperpredation increased numbers of introduced golden eagles, which in turn prey on the endemic and endangered island foxes. Although the pig removal would seem to be a straightforward and sensible plan, eradicating pigs without also reducing the eagles could actually trigger fox extinction because eagles will likely kill more foxes as pigs decline (Courchamp et al. 2003).

Functional responses of predators

The **functional response**, or **kill rate**, describes the number of prey killed per predator per unit time. As prey numbers increase, the kill rate could respond (or not) in many different ways. Although predator–prey theorists have categorized a variety of functional

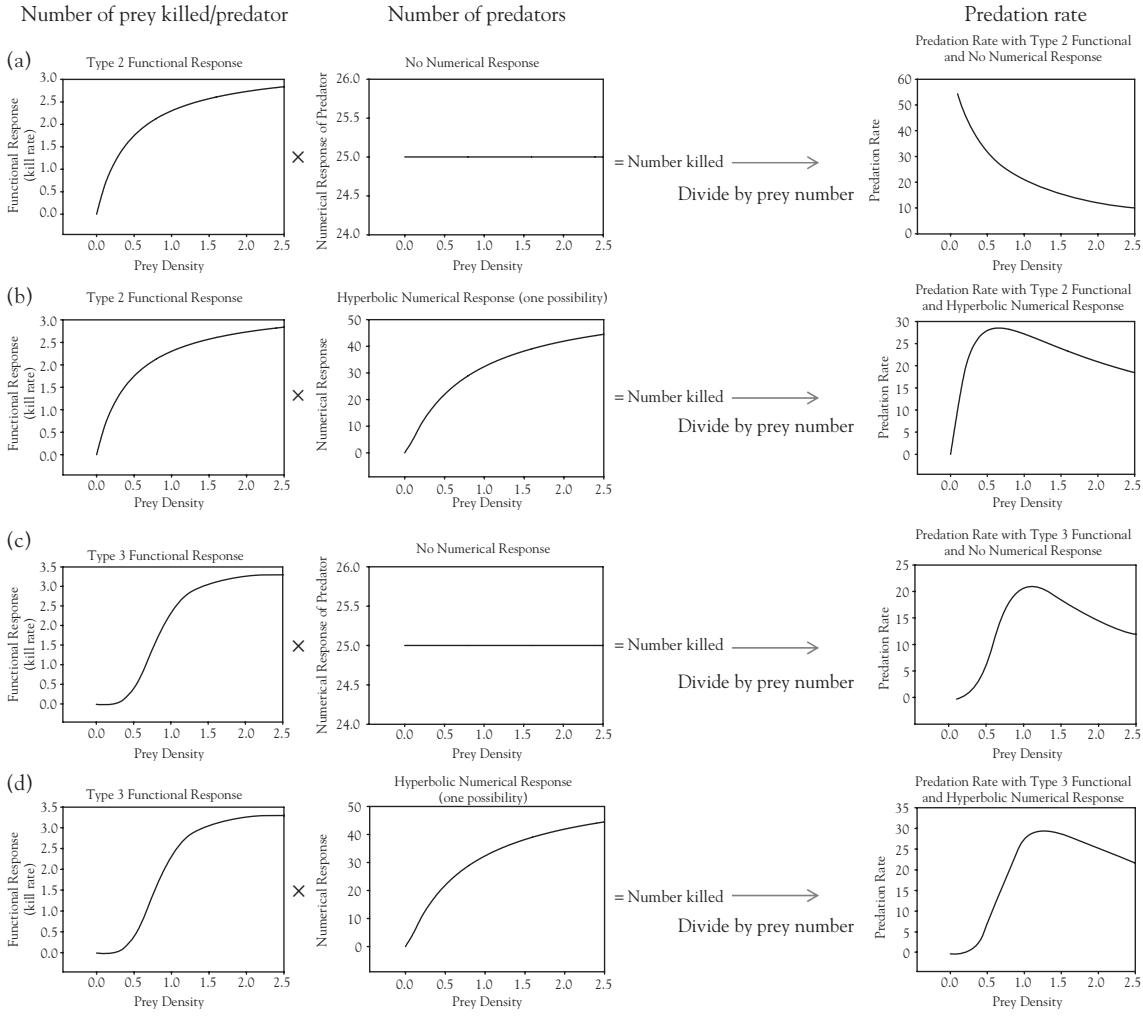

Fig. 8.1 Examples of how the overall predation rate on a prey (right panels) are affected by functional and numerical responses. Each row of panels represents a functional response (Type 2 or Type 3) multiplied by a numerical response (none or hyperbolic); the product of those is the number of prey killed, which is then divided by prey number to give the predation rate (% prey population killed by predators per year) across different prey densities. (a) Type 2 functional response and no predator numerical response; (b) Type 2 functional response and hyperbolic predator numerical response; (c) Type 3 functional response and no predator numerical response; (d) Type 3 functional response and hyperbolic predator numerical response. In (c) and (d), the predator pit is the prey densities to the left of the hump of the predation rate curve (last column).

responses, we will focus on the most common two, named by Holling (1959) as **Type 2** and **Type 3** functional responses. These are shown in the panels on the left of Fig. 8.1; Type 2 is a hyperbolic curve whereas Type 3 is sigmoidal. We are ignoring a Type 1 functional response, a straight-line relationship between prey density and number of prey killed per predator, for which it is hard to come up with biologically realistic mechanisms.

For any functional response, the kill rate must always flatten at a maximum because, as a bad guy in a bad movie might say: "There's only so much time to

kill." A predator must burn **search time** to locate prey and **handling time** to pursue, kill, and eat the prey; these set an upper limit for how many prey can be killed per unit time. The functional response can also be limited by **satiation** where a full stomach takes away the motivation to eat more. However, the kill rate can exceed what would be expected based solely on satiation (Kruuk 1972, Short et al. 2001): four or fewer red foxes killed up to 230 adult black-headed gulls in one night, eating fewer than 3% of them; in two separate instances in Australia a single introduced fox killed 11 wallabies and 74 penguins over several days, eating almost none of the victims; up to 19 spotted hyenas killed 82 Thomson's gazelle and badly injured 27 more in one night, eating only 16% of the kill.

Such seemingly heinous acts by predators raise intense emotions in humans because the gratuitous killing can seem to be an immoral waste of life. Why do predators do it? In some cases, when predators encounter easily accessible domestic prey or arrive in a system with naïve wild prey they initiate **surplus killing**, whereby animals are killed but not eaten. The **henhouse syndrome**, leading to surplus killing, is an almost inevitable result of a high-performance predator confronted with an easy target (Box 8.3). A striking case of **partial prey consumption** that comes with surplus killing can be found in brown and black bears eating salmon, where bears consume less of each fish when more fish are available. Furthermore, all fish and fish parts are not equal: unspawned fish – with higher muscle quality – are eaten more than spawned-out fish, with high-energy parts like brains and eggs preferentially consumed (Gende et al. 2001). Box 8.3 explains why surplus killing is not driven by bad morals but rather is the inevitable result of the behavioral development of predators.

In addition to surplus killing via the henhouse syndrome, **excessive killing** beyond immediate energetic needs may be an adaptive strategy for foraging over a longer time period. One compelling illustration can be found with least weasels (Jedrzejewska & Jedrzejewska 1989) that kill and consume bank vole prey approximately in proportion to their energetic needs each day during the summer and fall, but kill (and cache in their nests) more than they need as the Polish winter cold descends. When temperatures get really cold weasels stop hunting and instead eat out of their cache, a highly adaptive trait facilitating survival through cold winters (not unlike the nuts in a squirrel's hoard).

Box 8.3 The henhouse syndrome: surplus killing by predators

The behavioral programming of the act of predation can lead to the killing of far more prey than necessary to fulfill energetic demands. Often called the henhouse syndrome because it can happen when a predator gets into a chicken coop, surplus killing arises from the ethology of predation. Each of the four behaviorally distinct behaviors involved in predation (search, pursue, kill, and consume) are independently reinforced (Kruuk 1972). That is, the animal is rewarded not just by completing the whole predation act – eating the prey – but also by successfully carrying out each of the four behavioral components independently. (Think about why this must be true: for a young predator to learn its craft, where most early attempts fail to culminate in a prey in the belly, there must be positive reinforcement, or psychological encouragement, for performing each stage on the way to consuming the prey item.) A decrease in time spent performing any one or more of these behaviors will elevate the functional response. Normally, each step is time-consuming because the arms race adaptations of most prey challenge predators at each step of the search–pursue–kill–consume process. However, if the predator is presented with an unusual case where search and pursuit are made ridiculously easy – say the prey is penned or ecologically naïve – the predator can simply perform the act of killing again and again and again. The predators are not morally bereft, nor are such killers a case of problem or rogue individuals. For a predator faced with available prey, trivial costs of killing, and little risk of injury, there simply is no adaptive reason why it should stop killing, regardless of whether the prey are eaten.

Similarly, coyotes in the Yukon of Canada cache entire carcasses of nearly half of their snowshoe hare kills in early winter, and return to eat most of them over the next few months of deep winter even when they are covered by half a meter of snow (O'Donoghue et al. 1998). Of course, a carcass killed but not eaten by a predator is not wasted in an ecological sense because scavengers and decomposers will consume it. In fact, some scavengers depend on excess kill, as when common ravens treat gunshots as a dinner bell and fly towards the sound with the expectation of finding a 70-kg elk gut pile to scavenge from a successful hunter (White 2005).

Regardless of whether the upper limit to the kill rate comes from satiation from a full belly or from simply running out of time due to search and handling time required during surplus killing, an upper limit or flattening of the functional response must occur. What drives the other differences in the shape of the Type 2 and Type 3 curves? Again, satiation, and search and handling time play a role, as well as behaviors such as prey switching, predator learning, and prey escape strategies. For example, the left-hand side of the Type 3 curve, showing an increasing kill rate with increasing prey density, can be caused by predators developing a **search image**, learning to recognize, subdue, and consume the prey. A newly acquired search image can cause the predator to switch to a prey as it becomes more numerous. The increasing kill rate in the Type 3 curve can also come from prey behavior: if prey use camouflage or safe hiding places that are limited in number, a larger proportion of prey will be taken as prey numbers increase.

The functional response curves distill predator responses as a function of prey density (and so are often called **prey-dependent** models). A countercurrent to functional response curves plotted against prey density has emphasized that the kill rate depends on lots of things other than prey numbers. As we have seen, the kill rate can be affected by context – evolutionary background and age structure as well as habitat and weather conditions – and also by other species including alternate prey and predators. One alternative way of capturing some of these other influences on functional response has been to plot the kill rate not against prey number but rather against the ratio of prey to predator population sizes (Abrams & Ginzburg 2000). Such **ratio-dependent** predator–prey models can be useful complements to the traditional ones based only on prey density, helping us to understand the factors other than prey numbers that affect kill rates in wild populations (Vucetich et al. 2002).

Ultimately, the shape of the functional response curve for any predator–prey system is a manifestation of how well the predator and the prey are doing in the arms race. Prey strive to minimize the functional response by defense and escape strategies, while predators improve their search image and decrease travel and processing time between kills.

Total predation rate

Having discussed the numerical and functional responses, we can next ask how these responses affect the overall number of prey killed and the predation rate per time. As we saw in equation (8.1), the number of prey killed is the product of the number of predators and how many prey each predator kills: the numerical response times the functional response. In turn, the number of prey killed divided by prey abundance gives the predation rate per unit time.

Let us work through a specific example, based on a wolf/moose predator–prey system (Messier 1994, 1995). Suppose that at a moose density of 2 moose/km^2 we have:

- wolf functional response = 9 moose killed/wolf per year;
- wolf numerical response = 42 wolves/1000 km^2 = 0.042 wolves/km^2.

From the numerical and functional response at 2 moose/km^2:

- total number of prey killed = $9 \times 0.042 = 0.38$ moose killed/km^2 per year;
- total predation rate = (0.38 moose killed/km^2 per year) / 2 moose = 0.19, or 19% of the moose in the area killed by wolves per year.

These calculations of predation rate, building off numerical and functional responses, can be extended across a range of prey densities to provide insights of how the predation rate might regulate prey population growth (Fig. 8.1). If the functional response is Type 2 and the predator numerical response is unaffected by density of a target prey species – perhaps because the predator is subsidized by other prey – then the predation rate depends only on the kill rate (functional response) divided by the prey density (Fig. 8.1a).

The outcome is biologically profound, because the Type 2 kill rate creates a predation rate that decreases

as the prey density increases, translating to positive density dependence in prey survival across all prey densities. For a large or increasing prey population, this means that predation of this sort will not regulate prey numbers; as prey numbers increase, the predation rate decreases so that the survival rate increases (that is the positive density dependence). However, where prey numbers are small or declining, the positive density dependence can create Allee effects (Chapter 7): declining small populations endure proportionately higher predation so that survival is lower, spiralling the small population toward extinction. Recall again the endangered island foxes mentioned in the last section as victims of hyperpredation by invasive golden eagles sustained by exotic feral pigs (see also Chapter 7). Pig numbers allow the eagles to persist relatively independently of fox numbers, so as foxes declined the predation rate remained high even as foxes declined toward extinction (Angulo et al. 2007). In short, a Type 2 functional response for a predator whose numbers are unaffected by the prey could cause positive density dependence leading to Allee effects in small prey populations and lack of regulation in larger populations.

A Type 2 functional response coupled to a hyperbolic numerical response (Fig. 8.1b) can create a narrow window of very small population sizes where density dependence is negative, **stabilizing** the decline of small populations. However, if the hyperbolic numerical response is moved upward, as would be expected in a multiprey system where the predator could sustain itself independently of the prey being considered (so the intercept on the Y axis of the numerical response is > 0), then the total predation rate curve becomes – like Fig. 8.1(a) – **destabilizing** at all prey densities (Messier 1995). This brings us to the generalization that potentially severe Allee effects (destabilizing positive density dependence) due to predation are likely when the functional response is Type 2 and when the predator numbers are limited by factors other than the prey in question (Sinclair et al. 1998, Gascoigne & Lipcius 2004). Both of these conditions are common in wildlife populations.

By contrast, the Type 3 kill rate will tend to impose a broader "refuge" of negative density dependence at low prey densities (Fig. 8.1c, d), thereby regulating prey density by increasing prey survival (decreasing predation rate) for declining small prey populations; again the predation rate is nonregulatory at larger prey numbers. This prey density threshold below which predation is regulatory is sometimes called a **predator pit**, the idea being that if environmental stochasticity or stressors decrease the prey to numbers less than the hump on the predation rate graphs, prey will be able to persist with the predator but would be unable to increase in numbers due to the higher predation rate as prey density increases.

Predators can kill a lot of prey through numerical and functional responses, which can lead to a high predation rate. However, perhaps counterintuitively, a high predation rate does not necessarily mean that predators will limit prey population growth. Why not? Because two other factors must still be considered before concluding the regulatory effect of predators on prey. First, mortality due to predation may be compensated for or it may be on top of other mortality factors. Second, which age or stage class gets killed matters for prey population growth. We will explore each of these next.

FACTOR 2. DETERMINING HOW PREDATION AFFECTS PREY NUMBERS: COMPENSATION

When Paul Errington started studying predation on muskrats and bobwhite quail in the mid-1940s, the theory of predation in wildlife biology was simple: predators kill prey, so the removal of predators should mean more prey. Errington (1946) challenged that dogma. Behaviors such as territoriality may limit population size for many prey, making certain individuals (e.g. social subordinates) vulnerable to dying from disease or starvation if they are not killed by predators. Errington (1956) called these individuals the "doomed surplus," surely one of the most compelling phrases of ecological jargon of all time. Taylor (1984:28) notes that "by reducing predators to the ecological equivalent of garbage collectors, Errington undoubtedly served to forestall the conscious eradication of a number of carnivorous birds and mammals from North America."

Although it may be disconcerting to think about a doomed surplus in a population, the phrase makes it easy to realize that mortality due to predation may be at least partly **compensatory**. The mortality arising from predators killing the doomed surplus will be compensated for with lower mortality from other sources, say due to weather. Let us use H to describe the annual mortality rate due to predation, S_0 to describe the survival rate in the absence of predation, and S_A to

describe annual survival. If predation is completely compensatory, merely replacing other forms of mortality, then the annual survival rate will be unaffected by the predation rate (H), so that $S_A = S_0$. In a classic example, red grouse in Scotland that do not obtain territories in the autumn absorb nearly all of the mortality for the population. When a territory holder dies, a nonterritorial bird that would likely have died quickly takes its place, maintaining survival and keeping density steady even when predators remove a large number of grouse (Jenkins et al. 1964). Likewise, an ambitious experimental study of predation on mule deer in southeast Idaho (with over 1000 radiocollared deer to determine mortality) found that fawn mortality from malnutrition replaced fawn mortality from coyotes when coyotes were removed, so that mule deer population growth was unaffected by coyote predation (Hurley et al. 2011). Compensation in survival can only go so far, because predation mortality can only be fully compensatory if it does not exceed other nonpredation-related mortality sources (i.e. $H \leq (1 - S_0)$).

By contrast, if predation operates as a fully **additive** form of mortality, so that it acts independently of other forms of mortality, then overall survival (S_A) requires not being killed by predators ($1 - H$) and surviving through everything else (S_0). By the laws of probability[2]

$$S_A = S_0(1-H) \tag{8.2}$$

For example, if $S_0 = 0.6$ (meaning that the background prey survival rate is 60%) and $H = 0.7$ (meaning that 70% of the prey are killed by predators while 30% survive predation), then survival in the presence of additive mortality is $S_A = S_0(1 - H) = 0.6 \times (1 - 0.7) = 0.6 \times 0.3 = 0.18$, or 18%.

The critical distinction is that under compensatory mortality the overall survival (S_A) is unaffected by the level of predation while under additive mortality S_A declines linearly as the predation rate increases. Of course, survival is just one vital rate, so we could also ask whether compensation of predator mortality occurs at the level of population growth. To answer this, we must know whether predator mortality is compensated for by increases in other vital rates such as reproduction or immigration into a depredated population. Some of the most obvious examples of increasing reproduction to compensate for predation come from multiple clutches in birds. Mallard ducks rarely double brood (produce a second clutch after hatching ducklings), but if their nest is depredated they typically renest, and can do so for up to five times in one season if nests are preyed upon repeatedly (Hoekman et al. 2006). Compensation for predation also occurs by immigration. For instance, despite humans killing more than 50% of an introduced red fox population each year as part of an effort to protect endangered birds in California the foxes persisted, in part because up to half of the population comprised immigrants coming in from neighboring populations (Harding et al. 2001). Because compensation of predator mortality can occur not only through survival (when the doomed surplus are taken) but also through increased reproduction and immigration, some populations can sustain high predation rates without suffering decreased λ.

The occurrence of compensatory mortality has intense management implications. If predator mortality is compensatory at the level of prey population growth, then predator programs will ultimately be ineffective because a high or low predation rate would have little effect on prey dynamics (Côté & Sutherland 1997, Banks 1999). By far the greatest interest in compensation of predator-caused mortality centers on the harvest of wildlife by humans as predators, so I will wait until Chapter 14 to explore further intricacies of compensatory mortality. For now, I will leave you with the general understanding that the predation rate alone cannot predict whether predators will reduce the numbers or dynamics of a prey population; fully compensatory predation will not affect prey dynamics at all, even if the predation rate is high, while fully additive mortality from predation will decrease population growth. Having established two of the factors determining the effect of predators on their prey – the predation rate and compensation – we will next explore the third main factor, the age or stage of the prey killed.

[2] The word *additive* is sometimes confusing, since equation (8.2) shows multiplication. However, if you use the same laws of probability and the same numbers from the example but apply them to mortality (1-survival) the additivity becomes clear. The probability of dying from a predator (0.7) or from other sources (0.4) is, by the laws of probability for an inclusive "OR" operation: $0.7 + 0.4 - (0.7 \times 0.4) = 0.82$. (The parenthetical bit subtracts the probability of dying from both causes because you can only die once.) Because $(1 - 0.82) = (1 -$ probability of dying$) = 0.18 =$ probability of surviving, we see the same answer as derived in the text.

FACTOR 3. DETERMINING HOW PREDATION AFFECTS PREY NUMBERS: WHO GETS KILLED

The characteristics of the individuals killed by predators can influence the effect of predation on prey population dynamics in multiple ways. First, all individuals are not equally killable. As one example, in Utah prairie dogs exposed to high predation from red foxes and nothern goshawks, several sex–age classes were especially prone to being killed: juveniles, adult males (preoccupied with mating activities), pregnant females, recent immigrants, and adults at the edge of the colony (Hoogland et al. 2006). For another example, American pronghorn on the National Bison Range of Montana currently face a single substantial predator, the coyote, which kills approximately 90% of fawns in their first year but cannot kill adults (Byers 1997).[3]

The second way that who gets killed can affect predation dynamics is in the extent to which the predation mortality can be compensated; for instance, hatchling mortality in birds might be relatively easily compensated for by multiple additional clutches, whereas less latitude may exist to compensate for adult mortality.

Finally, as we have seen, all age classes and vital rates are not created equal in their effects on prey population growth. We can assess whether a given predation rate is likely to affect the prey's population growth rate by calculating reproductive values and performing sensitivity analyses, as in the last chapter. The bottom line is that the effect on population growth of the number of prey killed will depend on the stage class of the killed prey.

A good example expands the shearwater case study (Box 8.1). Using a matrix-projection model and an LSA-style approach incorporating uncertainty to explore how the λ value of Hutton's shearwater would vary across management changes, Richard Cuthbert and colleagues (Cuthbert et al. 2001, Cuthbert & Davis 2002) found that small changes in adult survival affect population growth more than even fairly large changes in chick or fledgling survival. Because stoats prey on chicks more than adults and the highest mortality risk for adults occurs away from the breeding ground where stoats are. Therefore, the management recommendation was to divert attention away from stoat predation on chicks and instead focus on minimizing the smaller level of stoat predation on adults and on other adult mortality sources, such as by-catch of shearwaters from ocean fishing.

Other examples abound where the effects of predators have been elucidated by formal analysis of which age or stage of prey is being killed. Although cheetah cubs are heavily preyed upon by lions and hyenas, an LSA sensitivity analysis incorporating both mean vital rates and their likely changes under management found that management focusing solely on reducing predation on cubs would be less effective than actions to increase – even slightly – survival of adults (Crooks et al. 1998). Likewise, although the short-necked turtle in Australia is enduring high predation from introduced red foxes, with rates exceeding 95% in some areas, the turtles would actually be better served by management to reduce adult mortality, which is much lower than nest predation but contributes more to turtle population growth (Spencer & Thompson 2005). Finally, you may recall from Chapter 7 that breeding-ground vital rates for mallards, which are often driven by predation, influence population growth more than vital rates in the nonbreeding season, which includes harvest by hunters.

SUMMARY

The question of whether predators control prey looms large in applied population biology, with implications ranging from whether predator reduction will protect endangered prey or increase ungulate prey for hunters, to whether introduced predators are likely to decimate their prey. To answer any of these questions with a broad yes or no would be ecologically naïve. Rather, we can answer the question for any particular case by assessing three primary details.

First, we need to know the predation rate or the percentage of the prey population killed by predators. The predation rate is the number of prey killed divided by prey abundance; the number of prey killed is the product of the numerical and functional responses. The numerical response describes the number of predators as prey numbers change. Multiple predator species can complicate the numerical response because reduction of one predator could increase the numerical response of other predators due to competitive

[3] Byers (1997) argues that the remarkable adaptations of adult pronghorn for speed (approaching 100 km/h) are a "ghost of predation past," when Pleistocene predators including cheetahs and hyenas would have preyed on adults.

release or trophic cascades. Multiple prey also complicate the predator numerical response through apparent competition or hyperpredation, where one prey sustains high numbers of a predator which in turn affects other prey species.

The other component affecting the number of prey killed is the functional response, or kill rate; that is, the number of prey killed per predator per unit time. The kill rate must plateau due to time available to search for and handle prey. However, it may well exceed immediate energetic requirements if surplus killing occurs or if kills are cached to be used over longer time periods. Complex behaviors and feedbacks between predator and prey determine the shape of the functional response curve, with predator learning and prey escape behavior playing roles. A Type 2 functional response curve will tend to create an Allee effect in small prey populations, decreasing survival as prey numbers decrease; by contrast a Type 3 response tends to stabilize small prey numbers. Ratio-dependent models are an alternative to functional response plotted against prey density.

Even for a certain predation rate, two other details must be known to determine whether predation will affect a prey's population dynamics. First, we must know whether the predation mortality is compensated for. Compensation occurs via lower mortality in other parts of the year, lower mortality in other life stages, and/or by increased reproduction or immigration. If predation mortality is compensated for, then predation is unlikely to affect prey density. By contrast, under additive predator mortality, survival declines and is not compensated for through reproduction or immigration.

Finally, the effect of predators on a prey population will depend on who gets killed. Because all age or stage classes are not equal in their vulnerability to predation, in their ability to compensate for mortality, or in their effect on population growth rate, massive predation can occur on certain age classes with very little impact on population growth. Alternatively, small additive mortality rates from predation imposed on age classes with a high reproductive value and/or making up a large proportion of the population can substantially lower population growth.

Predation is awe-inspiring, bone-chilling, and a major driver of population dynamics for many wildlife species. The predation rate by age class and the extent to which mortality due to predation can be compensated will vary over time and space, affected by weather, habitat changes, parasites and diseases, and other factors. By measuring these factors over space and time, the effect of a predator on a prey can be resolved.

FURTHER READING

Errington, P.L. (1946) Predation and vertebrate populations. *Quarterly Review of Biology* **21**, 144–77; 221–45. A true classic, filled with insights that continue to be timely even now.

Taylor, R.J. (1984) *Predation*. Chapman and Hall, New York. This slim volume rings with an engaging style that packs in an enormous amount of theory, math, and applied thoughts on predation.