CHAPTER 39

PREDATOR–PREY INTERACTIONS: INTEGRATING FEAR EFFECTS

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Predator–prey interactions have shaped all life on earth. This underlying commonality is why so many diverse fields have developed parallel but, as of yet, largely independent research paths. Although progress within each field is clearly being made, such an enormous body of research conducted at all levels of biological organization in every animal taxa holds the promise that a truly transformative understanding of predator–prey interactions can be attained, if we can find the link that binds together seemingly disparate fields. The persistent threat of immediate violent death is a hallmark of predator–prey interactions and the "fear" this engenders is emerging as the common link—central to integrating ideas at all scales of research, from neuroscience to behavior, physiology, and developmental biology—that when incorporated into population and community ecology provides novel insights into how nature functions.

We consider predation risk effects to be a unifying theme because the fear of being killed by a predator is something virtually all animals must contend with, meaning this is likely a universal stressor. At the same time, we suggest that predator-induced fear is distinct from any other stressor that animals face because the potential outcome is so immediate, extreme, and irrevocable. Whereas failure to find food means the individual goes hungry or failure to find shelter might mean it goes cold, failure to avoid a predator means it is dead—its Darwinian fitness immediately drops to zero (Lima & Dill, 1990). Whereas most challenges that animals face (e.g., finding food or shelter) entail a continuum, the threat or risk of falling victim to a predator means that the outcome of each and every predator–prey interaction is instantaneous and profound; it is life or death (Boonstra, 2013). Given that the fitness cost of dying instantly in a predator attack is so disproportionate it is quite clear that all animals, across all taxa, must do whatever they can to avoid this outcome. This powerful evolutionary force has accordingly caused prey to develop myriad behavioral, neurobiological, physiological, and morphological antipredator defenses. Although it is well established that predator-induced fear leads to such responses in individuals, it is not so evident how fear can affect entire populations or resonate throughout the ecosystem.

The main purpose of this chapter is to provide an overview of the new thinking in how individual level, predator-induced responses can scale up to affect population dynamics in particular as well as ecosystems. Antipredator behaviors, for example, are exhibited by all animal species and are known to carry costs like reductions in food intake. However, part of the difficulty in understanding how antipredator behaviors might affect populations is that such effects on behavior are often considered fleeting, evident only at the time the predator is around. A momentary interruption of feeding would not be sufficient to affect overall demography. We suggest that, contrary to this traditional view of predator effects as fleeting, the effects on individual prey may generally be more long-lasting than virtually any other stressor, accordingly leading to long-term behavioral responses persistent enough to affect demography. Specifically, the learning and formation of fear memories can leave long-lasting

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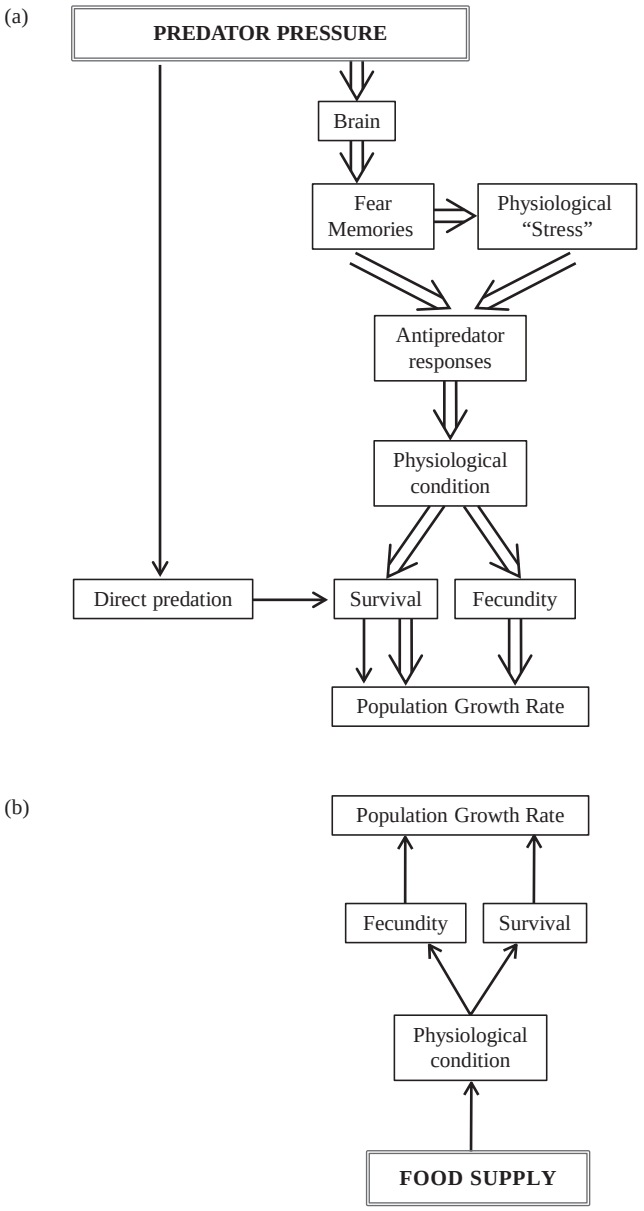

FIGURE 39.1. Schematic showing the pathways through which top-down predator limitation and bottom-up food limitation can affect populations. Panel A outlines the total impact that predators can have on prey populations. Arrows on the left show the traditional pathway whereby predators kill, thereby affecting prey survival only, which may then affect the population growth rate. Arrows on the right demonstrate how we conceptualize the fear or predation risk pathway. Predators and their cues stimulate the brain, which forms long-lasting fear memories that allow for predator recognition. This leads to a physiological "stress" response whereby the hypothalamic-pituitary-adrenocortical axis or other physiological coping mechanisms are set into motion. Fear memories and the stress response can set into motion antipredator responses which carry costs, such as predator-induced reductions in food intake, that are long-term and powerful enough to affect overall body condition, which in turn can affect both survival and fecundity (i.e., the birth rate), even if no prey are actually killed. Panel B highlights that alterations in food-intake powerful enough to affect body condition, survival, and fecundity can be evident but the mechanism ascribed has traditionally been the bottom-up food supply (the total amount of food in the environment available to each). That food-intake can affect demography is an important fact. That predator-induced reductions in food-intake can affect demography is part of the new thinking regarding predation risk effects. From "Diagnosing Predation Risk Effects on Demography: Can Measuring Physiology Provide the Means?" by L. Y. Zanette, M. Clinchy, and J. Suraci, 2014, *Oecologia, 176,* p. 638. Copyright 2014 by Springer-Verlag Berlin Heidelberg. Adapted with permission.

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effects on the brain that would continuously trigger antipredator behaviors when the threat of being killed is perceived as persistent. We briefly outline how predator-induced physiological stress, typically considered acute and fleeting, can also have chronic effects on prey. We then outline the long-lasting effects that fear has on development resulting in the long-term "memory of fear" passed on from parent to offspring, across generations. We next integrate these background ideas into population ecology, which has traditionally proposed that direct killing is the only way that predators can affect the number of individuals in the prey population (i.e., each prey killed reduces the prey population by one; see Figure 39.1a). We suggest that the total impact that predators have on prey populations is much greater than that, and we propose a second pathway involving predation risk effects (Figure 39.1a). Here, predators can induce chronic, long-term antipredator responses in prey, which carry costs on overall physiological condition that are powerful enough to affect prey demography (fecundity and survival) even if no individuals in the population are directly killed by predators. We end by describing how predator-induced fear can affect not only prey populations but also the prey of the prey, leading to what is known as a *trophic cascade*.

To be clear, our intention is not to suggest that predator-induced fear is a bad thing. On the contrary, fear has its uses. It is precisely the fear of predators that allows an individual to survive (and breed) another day. What we are saying is that antipredator defenses do carry costs that can be intense enough to affect survival and reproduction. The new understanding that the cost of fear (in terms of Darwinian fitness) can be very high is in fact generating new ideas about the significance of the adaptive benefits that fear must convey to outweigh such significant costs. For example, work on trophic cascades is providing evidence that when native predators are removed from an ecosystem their prey may become fearless, devoting the time they formerly spent being vigilant to eating, and this nonstop foraging can actually degrade the entire landscape and reduce overall biodiversity. Therefore, predator-induced fear has its uses, even at the scale of whole ecosystems.

BEHAVIORAL DEFENSES AGAINST PREDATORS

Of the possible antipredator defenses, the most commonly observed traits exhibited across all taxa are behavioral responses. The study of behavioral predator–prey interactions is immense and centers on the idea that although responses may be beneficial in terms of avoiding being killed, other activities are sacrificed, leading to some costs (see Chapter 40, this volume). One of the most well-established of these trade-offs is that scared prey eat less. Because animals cannot efficiently forage for food while avoiding becoming food at the same time, they must balance the benefits of safety against the costs of reduced food intake (Lima, 1998). Animals may respond to perceived predation risk by altering activity levels, space use, or temporal patterns of foraging and vigilance, any and all of which can alter the quantity or quality of food intake. Even animals at the top of the food chain (i.e., apex predators) show fear. Radio-tracking and camera trap studies make it evident that large carnivores (including the largest of all, tigers [*Panthera tigris*]) alter their foraging behavior to avoid humans, their principal predator (e.g., Carter, Shrestha, Karki, Pradhan, & Liu, 2012; Coleman, Schwartz, Gunther, & Creel, 2013; Cristescu, Stenhouse, & Boyce, 2013; Davis, Kelly, & Stauffer, 2011; George & Crooks, 2006; Ordiz, Kindberg, Sæbø, Swenson, & Støen, 2014; Smith, Wang, & Wilmers, 2015).

Most experimental studies evaluating the effects of fear on foraging have involved a researcher exposing a prey individual to a predator cue and measuring the cost as the reduction in time spent foraging in the subsequent few minutes. The underlying assumption in such studies is that the foraging cost of predation risk is acute and transitory, lasting only as long as the predator cue is present. We suggest that this view is no longer tenable for two reasons. First, a growing body of research shows that predation risk has long-lasting effects on learning, memory, and neurobiology, which of course drive behavior. Antipredator behavior, therefore, may be expected to persist much longer than just when the predator cue is present, and may instead persist so long as the memory of fear persists. Second, in the

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Fear and Population Ecology section of this chapter, we present the growing empirical evidence that fear does affect demography, which under most circumstances could only be the case if fear in fact leads to long-term changes in behavior.

LONG-LASTING EFFECTS OF FEAR ON THE BRAIN

Any animal exposed to any type of cue in a behavioral experiment will retain a memory of it over some duration. Whereas the effect on the animal's behavior may appear acute and transitory, lasting only as long as the cue is present, the effect on its memory likely lasts longer. Consistent with predator-induced fear being distinct, so too is the formation and retention length of fear memories. Because an animal has one chance to learn what to do if it successfully escapes a predator attack, and this memory is one it cannot afford to forget, new research suggests that the rapidity of fear learning and the longevity of fear memories are distinctively different from other types of learning and memory. Indeed, learning what can kill you should only require a single trial, and to be effective throughout one's life fear memories must be stable and long-lasting. Most of this research derives from work on laboratory rodents but elegant experiments on a variety of fish and invertebrates (Crook, Dickson, Hanlon, & Walters, 2014; Ferrari, 2014; Ferrari, Wisenden, & Chivers, 2010; Orr, Hittel, & Lukowiak, 2010) indicate that similar effects of fear on learning and memory are likely to be found in virtually all animal taxa.

Much of what we understand regarding the distinctiveness of fear learning and the longevity of fear memories derives from the biomedical literature using *animal model* research to better understand the human condition, as it relates primarily to posttraumatic stress disorder (PTSD; Clinchy et al., 2011; Clinchy, Sheriff, & Zanette, 2013; Daskalakis, Yehuda, & Diamond, 2013). PTSD is particularly relevant because a human who has experienced even a single life-threatening, traumatic event can form long-lasting memories in response. Indeed, PTSD is sometimes considered a disorder of memory whereby multiple cues, even innocuous ones, present at the time of the trauma are learned in an instant to be associated with the trauma, and can subsequently trigger intrusive memories of the event. This *one-trial learning* of multiple cues and the long-lasting nature of the memory leads to long-lasting behavioral and physiological responses that are frequently triggered well after the individual is out of the life-threatening situation (Daskalakis et al., 2013).

In recent years, the presentation of a predator or predator cues (e.g., showing a rat a cat or exposing a rat to cat odor) has become one of the principal stressors used in animal model studies of PTSD (Clinchy et al., 2011, 2013). The importance of using a predator is that the subject does not experience any physical pain (the cat cannot attack the rat) but it is experiencing something that is potentially life-threatening on a purely psychological basis. Previous methods would use stimuli like foot shock to simulate a traumatic event. However, exposing animals to something that is perceived as life-threatening better emulates the circumstances under which PTSD can form, and so provides a better animal model (Daskalakis et al., 2013). Behavioral and physiological assays demonstrate that even a single exposure to a predator can have long-lasting effects on anxiety-like behaviors and glucocorticoid levels in laboratory rodents. Of course, learning and memory occur in the brain and this body of research has also demonstrated multiple long-lasting effects of predator exposure on dendritic morphology and neuronal gene expression and activation (Adamec & Shallow, 1993; Armario, Escorihuela, & Nadal, 2008; Campeau, Nyhuis, Sasse, Day, & Masini, 2008; Masini et al., 2009; Mitra, Adamec, & Sapolsky, 2009; Roseboom et al., 2007; Rosen, Pagani, Rolla, & Davis, 2008; Schulkin, Morgan, & Rosen, 2005; Stam, 2007; Staples, McGregor, & Hunt, 2009; Takahashi, Chan, & Pilar, 2008). Moreover, recent discoveries deriving from research on the animal model of PTSD have demonstrated that not only is fear something that can be measured in the brain but that fear of predators can be specifically identified. Neural activity mapping and lesion studies on laboratory rodents have revealed that there are at least three *fear pathways* in the brain: fear of predators is processed by a distinct neural circuit

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independent of fear of an aggressive conspecific and fear of pain (reviewed in Gross & Canteras, 2012).

Researchers studying the predator model of PTSD have increasingly begun to suggest that predator exposure offers an additional advantage in attempting to understand PTSD, because long-lasting predator-induced fear and stress is ethologically and ecologically relevant, and it represents a valid experience applicable to animals in their natural environment (Cantor, 2009; Cohen, Kozlovsky, Richter-Levin, & Zohar, 2010; Roseboom et al., 2007; Staples et al., 2009). Wild animals are faced with the prospect of imminent violent death every moment of every day of their entire lives and so clearly any response to avoid immediate death would be fully adaptive, because dead animals do not reproduce.

Although we tend to think of predator–prey interactions as pertaining to other species, humans are still killed by large carnivores worldwide (e.g., Packer, Ikanda, Kissui, & Kushnir, 2005) and there is increasing recognition that many aspects of the human condition have been shaped by our evolutionary history as predators and prey. Such considerations have prompted some psychiatrists to begin discussing the evolution of PTSD as a response to predators (Cantor, 2009; Silove, 1998). Viewed in this way, PTSD may not be maladaptive in an evolutionary sense. What is unique about modern humans is that we can often escape life-threatening environments whereas other animals cannot because they cannot escape a world where others (their predators) are trying to kill them. Not being able to reverse the effects of a life-threatening trauma on memory and behavior is clearly adaptive for other animals because they are virtually certain to face cues signifying the trauma again and again (i.e., a predator trying to kill them). Living in a world where an acute fear for one's life does not reflect the environmental reality, as is the case for most modern humans, is a biologically extraordinary phenomenon which happened very recently in evolutionary terms. It is thus these extraordinary circumstances in which most modern humans exist that make PTSD pathological and so difficult to treat, because a virtually permanent rewiring of brain and behavior following a predator attack is highly adaptive for most other animals, as it must have been for humans over most of our evolutionary history.

One-trial learning documented in response to predator exposure in laboratory rodent studies has also been documented in flatworms, insects, fish, and amphibians (Ferrari, 2014; Ferrari et al., 2010). Repetition is not required for learning to occur precisely because it is a repetition of the life-threatening event that must be learned to be avoided (Ferrari, 2014). Fear learning and memories are quite different then from learning to respond to noxious prey items, for example. In the latter case, all animals will quickly learn to avoid food that tastes bad through conditioned taste aversion, whereby the food item is associated with subsequent illness, vomiting, and so forth. However, learning to avoid what makes you sick could take one trial or many, but in the end, the animal simply gets sick until it learns the association. An animal that fails to learn on the first trial to avoid or escape from a predator would more than likely die the next time around. Predator exposure that induces long-lasting memories has been shown in animals as simple as pond snails (*Lymnaea stagnalis*), which are only capable of forming a long-term memory of an operant conditioning task when exposed to a predator cue (Orr et al., 2010). The survival benefit of retaining a memory of trauma was recently shown in an elegant laboratory experiment on squid (*Doryteuthis pealei*) evading predatory fish (Crook et al., 2014). Following an initial trauma, squid that were not anaesthetized when traumatized (and so retained a memory of the trauma) had higher survival than those that were anaesthetized. Survival varied because the squid that possessed the memory of the trauma were warier and fled at greater distances when a predatory fish approached. Hypervigilance (wariness) and intrusive memories are hallmarks of PTSD (Daskalakis et al., 2013) and arguably convey a similar survival benefit to humans in life-threatening circumstances (Cantor, 2009; Clinchy et al., 2011; Silove, 1998).

LONG-LASTING EFFECTS OF FEAR AND STRESS PHYSIOLOGY

All vertebrates physiologically respond to challenges in the environment through the activation of the

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hypothalamic-pituitary-adrenocortical (HPA) feedback loop (see Chapters 19 and 23, this volume). Here, a stressor causes a cascade of hormone release initiated in the hypothalamus, which goes to the pituitary, which then directs the adrenal cortex to pump out glucocorticoids that circulate throughout the body, the levels of which negatively feeds back on the hypothalamus and pituitary, causing some inhibition of the entire process. Activation of the HPA axis has always been fundamentally linked to predation risk because it is at the core of the *fight-or-flight response*, which demands rapid mobilization of energy to one's muscles. Viewed in this way, physiological stress, as measured by this axis and overall glucocorticoid levels, has long been considered acute and fleeting because the evolutionary function of predator-induced fear and stress is to ensure immediate survival: the prey detects a predator; the prey freezes, flees, or fights; it survives or does not; and the event is over. If the animal survives, it returns to going about its business as if nothing had happened (Boonstra, 2013; Clinchy et al., 2011, 2013). Long-term activation of the HPA axis can and does lead to many negative effects, which can reduce survival and reproduction in the long-term (e.g., suppression in reproduction, the immune response, growth, and digestion; Boonstra, Hik, Singleton, & Tinnikov, 1998). Because of this, the traditional viewpoint in comparative endocrinology is that long-term activation (i.e., chronic physiological stress) cannot occur in nature because this would eventually result in death (Wingfield et al., 1998; Wingfield & Ramenofsky, 2011) and hence could not evolve because this would clearly be maladaptive. A growing body of literature, mainly spearheaded by ecologists working on predation risk effects in wild animals, has challenged this viewpoint (e.g., Boonstra, 2013; Clinchy et al., 2011, 2013).

Greater alertness and vigilance is clearly adaptive in readying the individual to flee from a predator (as demonstrated in the previous study on squid; Crook et al., 2014). The fact that fear memories may more or less permanently elevate alertness and vigilance similarly makes adaptive sense to ensure the individual is always ready to avoid a predator attack. Moreover, a large recent literature has documented that animals as diverse as fish, amphibians, reptiles, birds, and mammals all exhibit symptoms consistent with chronic stress in response to predation risk (reviews in Clinchy et al., 2013; Hawlena & Schmitz, 2010; Zanette, Clinchy, & Suraci, 2014). Though invertebrates do not have an HPA axis, physiological coping mechanisms evident in these groups also result in symptoms of chronic stress due to predation risk (Hawlena & Schmitz, 2010). The new thinking regarding the benefits of fear (e.g., Crook et al., 2014) helps resolve the apparent paradox whereby something that is evidently bad for you—chronic stress—can evolve. Animals in the wild can be chronically stressed and this may lead to their death, but so long as that death occurs at a later point in time than the death they would have suffered if they were not alert and responsive to predators, then chronic stress can be adaptive (Boonstra, 2013). In other words, anticipating that a predator could be around any corner might chronically activate the HPA axis, leading to downstream negative effects on survival and reproduction. However, staying alive a little longer, albeit in poor condition, maintains a fitness value greater than zero and, so, is better than being dead.

LONG-LASTING EFFECTS OF FEAR ON DEVELOPMENT

Many developmental biologists are also finding that responses to predator cues have lasting, often permanent effects on animals. In invertebrates and aquatic species (e.g., fish, tadpoles), predator exposure during development can lead to alterations in body shape and size that aid in escape, in addition to the development of body armature (e.g., spines) and chemical defenses. Such effects have long been known and there is a large literature on *inducible morphological defenses* (Tollrian & Harvell, 1999). These defenses are usually irreversible, presumably because the enormous amount of time and energy required to deconstruct an existing phenotype and reconstruct a new one sets significant limits on this type of plasticity (DeWitt, Sih, & Wilson, 1998; Hoverman & Relyea, 2007; but see Kishida & Nishimura, 2006). Other animals alter the timing of critical life-history traits under predator exposure,

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as is seen, for example, in those that undergo metamorphosis (reviewed in Benard, 2004), which of course once enacted cannot be reversed and so is permanent.

For terrestrial vertebrates, predator exposure may not induce the development of defensive spines, but long-term changes to the neural architecture of the brain (including dendritic spines) that lead to the formation of stable and long-lasting fear memories, described previously, could be considered an inducible morphological defense (Clinchy et al., 2013). Moreover, new research suggests that fear may lead to maternal effects that generate permanent, life-long consequences for developing offspring. For example, snowshoe hares (*Lepus americanus*) are extremely sensitive to predation risk and will fail to produce offspring when predation risk is naturally high or elevated with a manipulation (Sheriff, Krebs, & Boonstra, 2009). Offspring that are born to mothers stressed under high predation risk are themselves more stressed, smaller, and more vigilant (Sheriff, Krebs, & Boonstra, 2010). Evidence is emerging that epigenetic programming of the HPA axis may be a key driver in this inheritance from mothers to offspring (Lavergne, McGowan, Krebs, & Boonstra, 2014; see also Chapters 11 and 19, this volume). These traits may be what are required to survive in a high predator environment and it has been suggested that fear may cause mammal mothers to adaptively preprogram the behavioral and physiological traits of their offspring to help them better defend themselves against predators (Boonstra, 2013).

Prenatal stress in human mothers is associated with increased levels of anxiety, attention deficit/hyperactivity disorder, and conduct disorder in children (Glover, 2011). Recent research on maternal effects in wild animals (reviewed in Sheriff & Love, 2013) has prompted some researchers that study human development to consider whether increased childhood anxiety and similar disorders may have been evolutionarily adaptive (Glover, 2011; Nettle & Bateson, 2012). If our ancestors were regularly exposed to elevated levels of predation risk, then children born to stressed mothers might be better protected by being more alert, with increased vigilance and greater responsiveness (hyperactivity).

FEAR EFFECTS AND POPULATION ECOLOGY

Population ecologists monitor animal numbers and attempt to understand the factors that affect the key demographic parameters of births (i.e., fecundity or the number of propagules produced) and deaths, which together determine the population growth rate. These are the same parameters as those that define fitness and evolutionary ecologists are also interested in fecundity and survival. There are many factors that could potentially limit populations but two of the most well-studied are predators and food. Population limitation by predators is typically referred to as *top-down control*, whereas limitation by food supply is referred to as *bottom-up control*; each of these two factors has, historically, been considered independently. The traditional top-down view of predator limitation, which still persists in most ecology textbooks, is that the sole means by which predators can affect prey populations is through direct killing, thereby affecting only prey survival (see Figure 39.1a). In the case of bottom-up food limitation, populations are affected by the total amount of food in the environment available to eat, which can alter food-intake to such an extent that it can enhance (when food supply is plentiful) or degrade (food shortage) the overall physiological condition of individuals, thereby affecting both key demographic parameters, births and survival, and hence the population growth rate (see Figure 39.1b).

With respect to predator–prey interactions, the history of population ecology may be characterized as a (very) long process of recognizing the importance of antipredator defenses. As noted at the beginning of this chapter, simply counting the number of prey directly killed by predators long seemed sufficient to evaluate the ecological role played by predators. It would appear straightforward that the more prey that are killed, the faster the prey population declines, and the faster the predator population increases (because well-fed predators produce more offspring). Indeed, that the number of prey affects the number of predators, which in turn affects the prey, and so on was the principal consideration in the first mathematical models of predator–prey

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dynamics (Lotka, 1925; Volterra, 1926), and this has generally remained the case even today. Notwithstanding, early experiments on protozoa in petri dishes demonstrated that these models did not adequately capture the complexity of what happens in nature because the predator invariably ate the prey to extinction and then, with no food left, inevitably went extinct (Gause, 1934). The importance of antipredator defenses, and antipredator behavior in particular, was eventually recognized because it was only when prey were provided with a place to hide that the prey and predator populations could persist (Huffaker, 1958). Understanding antipredator defenses is thus critical to understanding how prey keep from being killed by predators, which will affect the population growth rate, though it has taken many decades to establish just how important this is to the population growth rate.

The benefits of fear are self-evident—an individual avoids being eaten. It is also self-evident that fear necessitates that the animal recognizes the predator as a threat and that the animal respond appropriately. There are many examples whereby failing to do so can affect populations, reducing prey population numbers and causing entire species to go extinct, as in cases where prey are confronted with a novel, unrecognizable predator whose range had expanded or had been introduced by humans (reviewed in Carthey & Banks, 2014). What is not so self-evident is that predator recognition and associated antipredator defenses may be so costly as to reduce the prey's reproduction or survival (Figure 39.1a) and this is in fact a topic of some debate. The traditional view persists that direct killing is the only way by which predators affect prey populations, and concomitantly, that predators can in no way affect the birth rate and cannot affect survival via mechanisms such as starvation. Instead, as mentioned, birth and starvation are viewed as being firmly in the domain of bottom-up food limitation alone (Figure 39.1b). One of the best established principles in behavioral ecology, however, is that scared prey eat less. Behavioral ecologists began to suggest in the 1980s that predator-induced fear may so greatly impair foraging that this could reduce the prey's ability to reproduce and increase its likelihood of starving to death (Abrams, 1984; Lima, 1998; Lima & Dill, 1990; Mangel & Clark, 1986; McNamara & Houston, 1987). Brown, Laundré, and Gurung (1999) called the integration of antipredator behaviors and population ecology the *ecology of fear* and research in this field has grown dramatically in the past decade or so.

The most direct means of testing whether predators can affect prey populations other than by direct killing entails eliminating direct killing, manipulating risk, and determining if prey reproduction and survival are affected. In the 1990s, such manipulations began to be conducted on invertebrate and aquatic species in experimental mesocosms. Here, direct killing by predators has often been actively eliminated by gluing shut (e.g., Peckarsky, Cowan, Penton, & Anderson, 1993; Schmitz, Beckerman, & O'Brien, 1997) or partially amputating (e.g., Nelson, Matthews, & Rosenheim, 2004) the mouthparts of invertebrate predators. These risky predators (Schmitz et al., 1997), which can intimidate but not kill (reviewed in Preisser, Bolnick, & Benard, 2005), are then placed together with invertebrate prey. Predation risk effects are then gauged by comparing prey populations in enclosures with and without predation risk. Other studies have used caged predators or predator odor (Kats & Dill, 1998; Paterson et al., 2013) to intimidate prey, again eliminating direct killing. Typically, such manipulations do find that predation risk alone affects demography, indicating that predation risk effects do exist at least in invertebrate and some aquatic systems. In fact, in a meta-analysis of this literature Preisser et al. (2005) concluded that in these systems predation risk effects "are generally as strong as or stronger than the effects of direct consumption [i.e., killing by predators]" (p. 507). Though most of these manipulations have been done in artificial enclosures, there is some evidence that they translate to the field as well (e.g., Peacor, Pangle, Schiesari, & Werner, 2012; Peckarsky et al., 1993).

Because of the logistical challenges of conducting manipulations at a large enough spatial scale over a long enough duration to affect the demography of free-living terrestrial vertebrates, few analogous manipulations have yet been conducted in terrestrial vertebrate systems. In one of the first reviews of this literature, Lima (1998) recognized this and called it a "terrestrial gap" in our knowledge of how

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predation risk may affect populations. For wildlife, animals will commonly lose body mass when exposed to predators or predator cues (Lima, 1986; Gosler, Greenwood, & Perrins, 1995; C. D. MacLeod, MacLeod, Learmonth, Cresswell, & Pierce, 2014; Pérez-Tris, Diaz, & Telleria, 2004), or otherwise alter their foraging patterns with effects on body condition (e.g., R. MacLeod, Lind, Clark, & Cresswell, 2007; R. MacLeod, MacLeod, et al., 2007), and some studies in terrestrial vertebrate systems have shown that such nutritional alterations may be strong enough to have demographic consequences (e.g., Creel & Christianson, 2008; R. MacLeod, Lind, et al., 2007). However, this and most other evidence that predation risk affects demography is based on natural contrasts of high and low risk, and in some cases, this has led to a great deal of contention as to whether predation risk effects on wildlife populations exist. For example, one of the most high-profile and hotly debated issues, far from being resolved, is whether the reintroduction of wolves to Yellowstone National Park in 1995 and 1996 has caused reductions in the population sizes of elk via predation risk (fear) effects. Elk have declined since wolves were reintroduced but the number of elk directly killed by wolves is too small to account for this, leading some to suggest that fear itself has contributed to the decline. Some involved in this debate argue that predation risk does affect the birth rate. Evidence in support comes from work by Creel, Christianson, Liley, & Winnie (2007) and Creel, Christianson, and Winnie (2011) showing that because scared elk eat less, their poor physiological condition simply does not allow them to bear offspring. Others, however, are quite adamant that no such predation risk effects exist (e.g., White et al., 2011; Middleton et al., 2013). Logically, it seems unlikely that predation risk effects on wildlife populations can be absent given that terrestrial vertebrates, like the invertebrates previously described, show long-term alterations in neurobiology, behavior, physiological stress, and development that may be expected to carry costs affecting demography just as they do for invertebrates. The experimental evidence is sparse, however, but mounting, and we will review what is known to date.

Some experimental evidence that predation risk affects fecundity and survival in wildlife comes from studies that have looked at the demographic effects of manipulating food availability at sites with fewer or more predators. The prediction from behavioral ecology is that the benefits of greater food availability (i.e., increased fecundity and survival) will only be realized when predation risk is low, because having fewer predators to worry about would allow prey to maximize food intake rates. When predation risk is high, however, food intake will plummet even if the bottom-up food supply is plentiful in the environment. Food and predators should thus interact in their effect on demography (Abrams, 1984; Lima, 1998; Lima & Dill, 1990; Mangel & Clark, 1986; McNamara & Houston, 1987). Experiments on snowshoe hares, arctic ground squirrels (*Spermophilus parryii*), and song sparrows (*Melospiza melodia*) have demonstrated just such an interaction. Krebs et al. (1995) conducted a two-factor predator by food manipulation and measured the effects on snowshoe hare densities over an 8-year period at Kluane National Park in Canada. Food was supplemented for some populations, predators were excluded from another using a 1 km^2 fence, another was provided supplemental food and protected with a fence, and several more populations served as nonmanipulated controls. Relative to controls, hare numbers increased three-fold and two-fold in the single factor manipulations, but 11-fold in the combination treatment, a synergistic effect. Arctic ground squirrels subject to these same experimental treatments demonstrated comparable demographic responses (Karels, Byrom, Boonstra, & Krebs, 2000). In a bifactorial experiment involving food supplementation and natural predator reduction conducted on song sparrows, Zanette, Clinchy, and Smith (2006) and Zanette, Smith, van Oort, and Clinchy (2003) documented an interactive effect on annual reproductive success (total offspring produced, which is a function of the birth rate—eggs laid—and the survival of those propagules to fledging). Relative to the controls, sparrows reared 1.1 extra offspring when food was added and 1.3 more when predation risk was low, but 4.0 extra offspring in the combination treatment. A later predation risk by food supplementation manipulation confirmed

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that song sparrow parents do reduce food-intake when predation risk is high even when the bottom-up food supply is unlimited for all (Zanette, Hobson, Clinchy, Travers, & Williams, 2013). Moreover, reduced food-intake by the song sparrows due solely to predation risk led to poor overall physiological condition, with effects on the birth rate (Travers, Clinchy, Zanette, Boonstra, & Williams, 2010; Zanette et al., 2013).

Manipulations involving risky but nonlethal predators could help resolve whether predation risk effects are even possible for wildlife populations; however, such manipulations are enormously challenging. Free-living wildlife of even modest size often roam over large areas, making it logistically difficult, though certainly not impossible, to conduct manipulations on entire populations with sufficient spatial replication. Moreover, it is a challenge to design a manipulation in which free-ranging, terrestrial vertebrate predators can intimidate but not kill their wildlife prey; disabling mouthparts, for example, is not an option. Nonetheless, of the six predation risk experiments conducted on wildlife of which we are aware (Eggers, Griesser, Nystrand, & Ekman, 2006; Fontaine & Martin, 2006; Hua, Sieving, Fletcher, & Wright, 2014; Sheriff et al., 2009; Travers et al., 2010; Zanette, White, Allen, & Clinchy, 2011), all point to the conclusion that predation risk effects do exist for wildlife.

Of the manipulations listed previously, the predation risk experiment that most closely parallels those conducted in experimental mesocosms was conducted by Zanette et al. (2011). Zanette and colleagues used protective measures at the nests of song sparrows to eliminate the direct killing of offspring. Predation risk was then manipulated throughout the breeding season using predator vocalizations broadcast for some populations, whereas others heard nonthreatening sounds. The researchers found significant effects on fecundity, because female song sparrows laid fewer eggs, in addition to effects on offspring survival, wherein a lower proportion of eggs and nestlings survived in the predator vocalization treatment. Continuous video surveillance at the nest confirmed that no deaths were due to direct killing, thereby unambiguously demonstrating predation risk effects. The net effect of predation risk was a 40% reduction in the total number of young produced per year. The effects on survival found in this manipulation were comparable to those found in song sparrow populations that exhibited naturally high and low levels of risk (Zanette et al., 2011), even when these populations had access to an unlimited, high quality, supplemental food source (Zanette et al., 2006). Thus, even though the predator playback manipulation relied on cues from a single modality (sound), it appeared to have simulated the demographic consequences that animals face when under naturally high levels of risk where predator cues would be relatively intense and multimodal (sound, sight, smell, and attacks). Moreover, the researchers demonstrated that adults exposed to high risk fed their nestlings less (i.e., predator-induced reductions in food intake), such that their nestlings were consequently lighter (i.e., in poorer condition) and a greater proportion died (Zanette et al., 2011), thereby linking antipredator responses to their physiological costs and demographic consequences.

Eggers et al. (2006) and Hua et al. (2014) did not actively eliminate direct killing but did manipulate perceived predation risk by broadcasting calls of predators. Eggers et al. demonstrated that predator (Corvid) call playbacks caused Siberian jays (*Perisoreus infaustus*) to lay fewer eggs in the first clutch of the season, and Hua et al. (2014) reported that broadcasting hawk calls reduced seasonal fecundity in Eastern bluebirds (*Sialia sialis*). In a study on several songbird species Fontaine and Martin (2006) experimentally reduced predation risk by removing predators and found an effect on egg mass, but not clutch size. Travers et al. (2010) demonstrated that female song sparrows that experienced frequent experimental nest predation laid smaller clutches because of predator-induced reductions in food-intake (Zanette et al., 2013). In the sole experiment on a mammal, conducted on captive snowshoe hares in large outdoor enclosures, Sheriff et al. (2009) showed that pregnant females exposed to a trained dog 1–2 min every other day before parturition were significantly less likely to give birth to offspring compared to control females.

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LONG-LASTING EFFECTS OF FEAR ON DEVELOPMENT AND ITS ROLE IN POPULATION ECOLOGY

The dynamics of animal populations will clearly be affected by the environmental conditions under which the parents breed in any given year. In population ecology, however, there is also the concept of *maternal/paternal* and *cohort effects*, wherein the population growth rate is not solely determined by current environmental conditions but also by conditions from the past. We have already described the long-lasting effects that fear can have on development, which can leave its imprint on offspring throughout their lives. Given that entire populations typically experience the same environmental conditions (e.g., high or low predation risk), this leads to the possibility that entire cohorts will be affected, which can then affect population numbers once those individuals recruit and become breeders themselves. For example, in poor weather years, assumed to be associated with a shortage of food, parents will rear offspring in relatively poor condition (e.g., low birth weight), who go on to rear fewer of their own offspring compared to those reared in good years with ample food. Importantly, the effect of early development on subsequent reproduction is evident even if food is plentiful in the years in which the initial cohort breeds (e.g., Albon, Clutton-Brock, & Guinness, 1987; Reid, Bignal, Bignal, McCracken, & Monaghan, 2003; reviewed in Lindström, 1999); in other words, favorable current conditions typically cannot compensate for poor early development.

Regarding predation risk effects, the best evidence that early development may affect later population sizes comes from work done on what is known as the *enigma of the extended low*. This is in reference to what are termed *cyclic populations* of snowshoe hares and voles that show a regular pattern of high and low numbers (peaks and troughs) over a number of years—10–11 years in the case of hares and 3–4 in the case of voles. The enigma is that once the population has reached the low phase, environmental conditions are actually ideal with few predators and lots of food, yet population numbers remain stubbornly low for years on end. The memory of fear transferred from parent to offspring has been proposed to be responsible. Maternal effects are evident in a wide range of animals (Lindström, 1999), and sometimes, the effects can be so long lasting that they take generations to dissipate from the population. For example, the decline phase of the snowshoe hare cycle is characterized by intense predation risk because predator numbers are at their peak and adult hare survival amounts to only 1% per year. This is life under a virtually certain death sentence. Boonstra, Hik, et al. (1998) showed that these adult hares exhibit symptoms consistent with chronic physiological stress in addition to degraded overall condition. Later, Sheriff et al. (2010) evaluated this cyclic variation in predation risk and found that female hares became physiologically stressed under high risk and produced offspring that were also more stressed, smaller, and vigilant. As noted, voles also cycle, and Boonstra, Krebs, and Stenseth (1998) brought wild meadow voles (*M. pennsylvanicus*) into the laboratory and bred them under ideal conditions to test if chronic predation risk had comparable effects to those found in hares. Voles captured during the low phase of the cycle produced many fewer offspring than those captured during the increase phase, and this attenuation of reproduction lasted three generations into the future.

The example of snowshoe hares during the decline phase dramatically illustrates our earlier point about the costs of fear being fully adaptive. If having fewer, smaller, more stressed offspring helps a mother hare and her offspring stay alive, they win—in evolutionary fitness terms—because virtually every other hare (99%) is dead. Moreover, this illustrates that such maternal effects can influence population dynamics and can do so over generations because these effects are long-lasting and cannot be instantly reversed, even when current environmental conditions are ideal.

FEAR AND FOOD CHAINS

Predator–prey interactions are well-known to have effects at several trophic levels down the food chain, extending beyond the effect that predators have on their prey. Here, predators are expected to have a positive, indirect effect on the food that their prey eats, not because the predator is directly interacting

with this lowest trophic level, but because of the predator's effect on its prey. This concept that the presence or absence of a predator can shape community structure is called a *trophic cascade* (Estes & Terborgh, 2010), and research has generally followed two paths. The first emphasizes direct killing. For example, the more zebras that lions kill, the fewer zebras there will be eating grass and there will be more grass. The second invokes predation risk effects whereby predator-induced changes in some trait (e.g., behavior, morphology, physiology) are sufficient to generate a trophic cascade. A trophic cascade caused by a change in prey traits, rather than prey numbers, is referred to as a *trait-mediated indirect interaction* (TMII). A *behaviorally-mediated trophic cascade* is one type of TMII specifically caused by the cascading effects that antipredator behavioral responses may have on the food web. In the latter case, the reasoning is that where predation risk is high, there ought to be more of the food that prey eat because the prey are too frightened to eat it. Because fearful prey may be expected to hide or congregate in safe habitats and avoid risky habitats, the result has been referred to as a *landscape of fear* (Laundré, Hernández, & Altendorf, 2001). Here, areas too risky for prey to venture will contain plenty of the prey's food and less risky spots will correspondingly be more depleted. In the absence of predators, one may expect prey to become fearless and devour food in any location at any time, thereby potentially degrading entire landscapes and negatively affecting overall biodiversity (e.g., Hebblewhite et al., 2005).

Trophic cascades have received an enormous amount of attention and are of great conservation importance, because many apex predators around the globe have been extirpated with devastating effects further down the food web (Ripple et al., 2014). Given the far-reaching effects that predators have on prey populations, and the prey (i.e., resource) of the prey, it has been suggested that these losses "may be humankind's most pervasive influence on nature" (Estes et al., 2011, p. 301). Manipulations conducted on invertebrate and aquatic species in experimental mesocosms have demonstrated not only that predation risk can affect the fecundity and survival of prey (as previously indicated) but also that these effects can have cascading consequences down food chains (Preisser et al., 2005). To date, the evidence that fear in wildlife may have cascading effects through whole ecological communities comes from natural contrasts (see reviews in Heithaus, 2008; Wirsing & Ripple, 2011) and remains controversial (e.g., Beschta & Ripple, 2013; Kauffman, Brodie, & Jules, 2013), because the critical experiments had not been done. A recent manipulation, however, confirms that the fear of large carnivores can generate a trophic cascade (Suraci, Clinchy, Roberts, Dill, & Zanette, 2016).

THE FUTURE OF FEAR

We have argued that fear can provide the common link integrating research and findings across a diversity of fields and taxa. There is clearly a need for more interdisciplinary research to bind these links together. We suggest one priority is to start conducting experiments testing the neurobiological effects of fear in wild animals. Such research would not only help validate the animal model of PTSD by establishing that findings in the lab are not an artifact of captivity, but would also expand our understanding of the etiology of PTSD given that "the extremity of the stressors faced by animals in the wild. . . emulate more closely the circumstances leading to PTSD in humans" (Matar, Zohar, & Cohen, 2013, p. 137). Demonstrating long-lasting neurobiological effects of fear in wild animals would also further our understanding of ecology because fear effects on birth and survival are easier to envisage if fear permanently transforms the brain. By the same token, more research on the developmental effects of fear ought to be a priority to better comprehend the etiology of childhood anxiety and the potential intergenerational effects of predators on prey ecology. With regards to ecology, clearly predation risk manipulations on free-living wildlife are necessary to assess the extent to which predation risk effects drive demography across taxa. Only in this way will we be able to gauge whether predation risk effects are generalizable across animal taxa and represent a basic, underlying, fundamental principal in ecology. Finally, there is an urgent need to experimentally test the effects of fear on food chains in terrestrial

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vertebrate systems, given current controversies concerning the conservation of large carnivores and the ecosystem services they may or may not provide (Estes et al., 2011; Marris, 2014; Ripple et al., 2014).

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