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Pathways to the density-dependent expression of cannibalism, and consequences for regulated population dynamics

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Abstract

Cannibalism, once viewed as a rare or aberrant behavior, is now recognized to be widespread and to contribute broadly to the self-regulation of many populations. Cannibalism can produce endogenous negative feedback on population growth because it is expressed as a conditional behavior, responding to the deteriorating ecological conditions that flow, directly or indirectly, from increasing densities of conspecifics. Thus, cannibalism emerges as a strongly density-dependent source of mortality. In this synthesis, we review recent research that has revealed a rich diversity of pathways through which rising density elicits increased cannibalism, including both factors that (a) elevate the rate of dangerous encounters between conspecifics and (b) enhance the likelihood that such encounters will lead to successful cannibalistic attacks. These pathways include both features of the autecology of cannibal populations and features of interactions with other species, including food resources and pathogens. Using mathematical models, we explore the consequences of including density-dependent cannibal attack rates on population dynamics. The conditional expression of cannibalism generally enhances stability and population regulation in single-species models but also may increase opportunities for alternative states and prey population escape from control by cannibalistic predators.

KEYWORDS

cannibalism, cannibalistic polyphenism, conditional behavior, density dependence, hunger, negative feedback, phenotypic plasticity, population regulation, prey population escape, stress

INTRODUCTION

Field ecologists almost universally view cannibalism as a prime example of a density-dependent mortality factor and, thus, as an important contributor to the regulation of cannibalistic populations (Fox, 1975; Ibánez & Keyl, 2010; Polis, 1981; Richardson et al., 2010; Ricker, 1954; Schausberger, 2003; Smith & Reay, 1991; Wise, 2006). Theoretical studies confirm that cannibalism can act as a strong regulatory force (e.g., Barabás et al., 2017; Ohlberger et al., 2020) but also that cannibalism can create other sorts of dynamics, including generation cycles (Briggs et al., 2000; Persson et al., 2004), alternative states (Persson et al., 2003), or other complex

dynamics, including chaos (Costantino et al., 1997; Higgins et al., 1997, reviewed by Claessen et al., 2004). In many cases, outcomes depend critically on the details of model structure and parameter values (e.g., Ohlberger et al., 2020) and sometimes on competitive and trophic interactions with other members of the community (e.g., Kohlmeier & Ebenhöh, 1995; Rudolf, 2007; Toscano et al., 2017).

Faced with the diversity of dynamics predicted by mathematical models, empiricists have conducted long-term, multigenerational field studies of the dynamics of diverse, cannibalistic animal populations (mites, insects, amphipods, crabs, leeches, fish, salamanders) and concluded that cannibalism is indeed a key regulatory influence (Andersson et al., 2007; Baskauf, 2003; Christie & Kraufvelin, 2004; Elliott, 2004; Grosholz et al., 2021; Moksnes, 2004; Persson et al., 2003; Persson & Elliott, 2013; Walde et al., 1992; Wissinger et al., 2010). Nevertheless, definitive empirical demonstrations of a regulatory role for cannibalism remain elusive, primarily because we lack an effective experimental manipulation of cannibalism that can be implemented over the long term in the field. There is no simple way to eliminate cannibalistic interactions without also eliminating the cannibal population itself. Thus, long-term studies of cannibalism rely strictly on observational methods, and evidence for population regulation is often open to multiple, competing causal explanations.

One thing we do know is that for cannibalism to contribute to the regulation of animal population densities, it must be a density-dependent process, generating increasing per-capita mortality as densities rise. A large body of empirical evidence, including both observational and experimental studies, has demonstrated that this is generally true: per-capita mortality rates from cannibalism rise, in many cases very strongly, with rising population density (Baskauf, 2003; Buddle et al., 2003; Elliott, 2004; Fincke, 1994; Fisher et al., 2021; Fox, 1975; Gillespie et al., 2020; Grosholz et al., 2021; Hannesson, 2018; Hopper et al., 1996; Houghton et al., 2017; Klotz & Wright, 2020; Moksnes, 2004; Oraze & Grigarick, 1989; Orr et al., 1990; Persson et al., 2003; Persson & Elliott, 2013; Polis, 1981; Strauss et al., 2016; Van Buskirk, 1989; Wagner & Wise, 1996; Wildy et al., 2001; Wissinger et al., 2010). These studies have, furthermore, demonstrated that there are multiple pathways through which cannibalism emerges as a density-dependent process. The goal of this review is to examine the surprisingly diverse processes that inject density dependence into cannibalistic interactions. Some of these processes have long been recognized and were carefully discussed in early reviews of cannibalism (Fox, 1975; Polis, 1981; Smith & Reay, 1991); we will review these more quickly and then describe in greater detail more recent studies that have uncovered novel and, in some cases, unexpected ecological processes, including interactions with other members of the community, that can strengthen the density dependence of cannibalism.

Collectively, this review highlights that per-capita cannibalism rates may be expected not only to increase with population density but also to accelerate with population density, at least in some cases. Because previous theoretical studies have not accounted for this acceleration, we use mathematical models to examine how it may impact ecological dynamics. These models suggest accelerating per-capita cannibalism rates can strengthen population stability and regulation but also lead to alternative states and prey escape from cannibalistic predators.

ORGANIZING THE SOURCES OF DENSITY-DEPENDENT CANNIBALISM

Density dependence in cannibalism emerges from three broad categories of sources: increasing density may (1) cause more frequent encounters between potential cannibals and vulnerable conspecific victims; (2) change the internal state of the cannibal, increasing its likelihood of initiating an attack, given an encounter; or (3) change the internal state of the victim, increasing the likelihood that an attack, once initiated, will be successful (Figure 1). If a potential cannibal can detect the greater vulnerability of a potential conspecific prey, this will also likely increase its willingness to mount an attack. Thus, factors promoting increased prey vulnerability will also frequently favor increased cannibal aggressiveness, creating broad overlap between the second and third sources of density-dependent cannibalism. We therefore treat these two sources of density-dependent cannibalism together. Cannibalism will reflect the product of encounters; the likelihood of initiating an attack, given an encounter; and the likelihood that an attack, once initiated, will be successful. Relevant aspects of the internal state of a potential cannibal could include aspects of physiology (e.g., hunger level, immunological state), development (size or stage), or morphology (in species that display the induction of cannibalistic morphs). The potential cannibal's informational state could also be important when the cannibal's experience shapes its expectation for (i) the future risk of competition, predation, or disease, (ii) the likelihood that an encountered conspecific is kin, or (iii) the likelihood that an encountered conspecific could mount a dangerous counterattack (Dong & Polis, 1992). Previous reviews on the dynamics

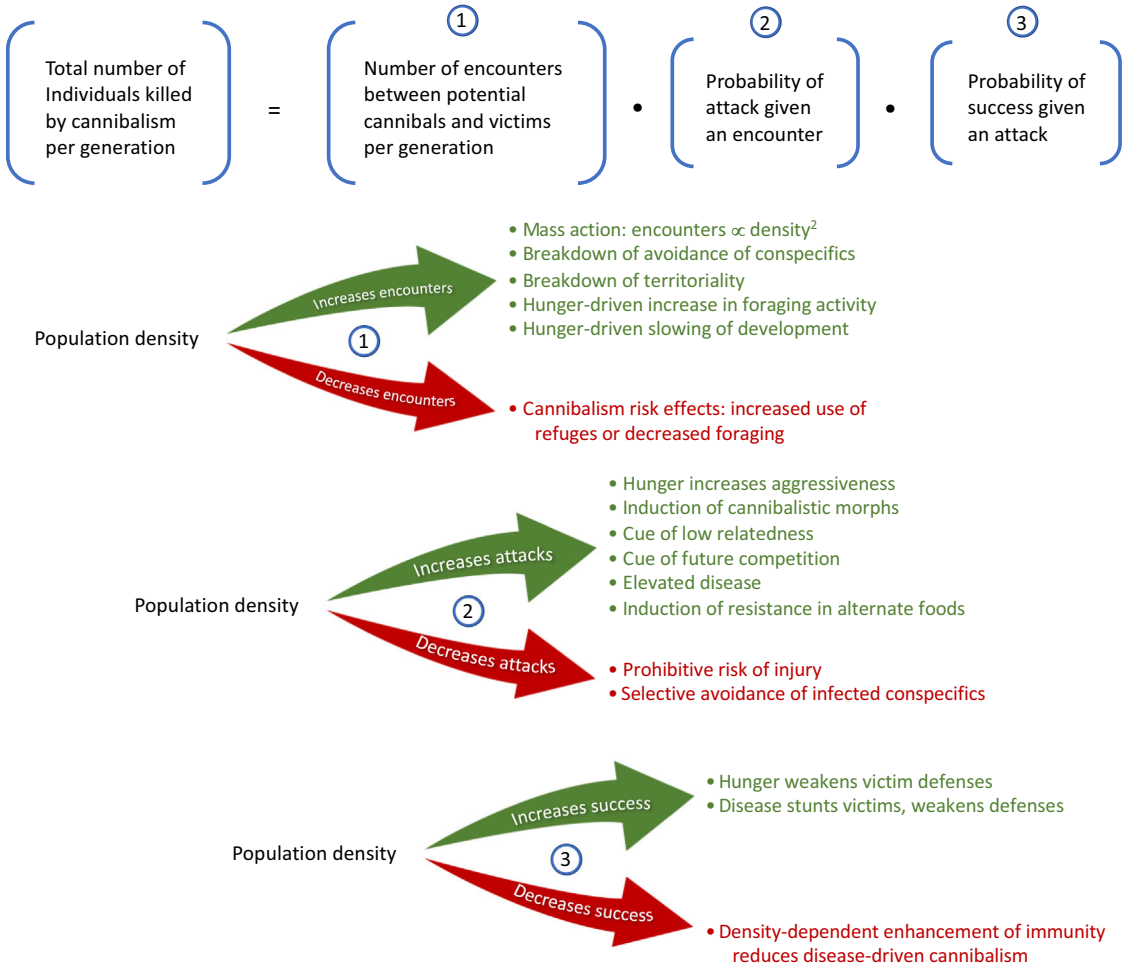

FIGURE 1 Sources of density dependence in mortality generated by cannibalism. Density can elevate cannibalism through multiple pathways, including those that promote encounters between conspecifics, elevate the likelihood that an encounter will lead to a decision to initiate an attack, and elevate the likelihood that an attack, once initiated, will lead to death of the victim.

of cannibalism have emphasized density dependence of encounters, with less attention given to factors shaping decisions to attack given an encounter and the outcome of an attack (but see Dong & Polis, 1992).

Any predatory attack, whether directed against a heterospecific or conspecific, is associated with costs and benefits. Cannibalistic attacks, however, are special, in that some costs may be unique or particularly elevated: (i) cannibalistic attacks risk loss of inclusive fitness if the victim is related to the cannibal (Pfennig, 1997); (ii) the cannibal may have an elevated risk of acquiring an infection, given that host-specific pathogens often move more readily between conspecifics than heterospecifics (Pfennig, 2000); and (iii) conspecifics may be armed with weapons similar to those of the attacking cannibal and may therefore be dangerous prey (Dong & Polis, 1992; Lund et al., 2016). These special costs act as brakes on the expression of cannibalism under many conditions, but if elevated density tends to release these brakes, we can expect greater density dependence of cannibalism expression. We now consider, first, the effects of density on encounter frequencies and, second, the effects of density on the likelihood of initiating an attack, given an encounter, and the probability that an attack, once initiated, will succeed.

EFFECTS OF DENSITY ON THE FREQUENCY OF ENCOUNTERS BETWEEN CONSPECIFICS

Foundational models of predator–prey interactions, like the Lotka-Volterra model, assume that the number of encounters between predators and prey will be proportional to the product of predator, P, and prey, N, densities. Thus, with all other things being equal and considering the short term (i.e., before any possible predator numerical responses to elevated prey availability),

we expect encounter frequency to rise linearly with increasing densities of either predators or prey (i.e., encounters $\propto$ density). We can extend this framework to the case of cannibalism. For the simplest model of so-called mass action, under which populations of cannibals are viewed as well-mixed particles moving randomly in space, and ignoring the stage structure of the cannibal population, we expect the frequency of encounters between conspecifics to increase as the square of density (i.e., encounters $\propto$ density^2; Andow et al., 2015) because rising cannibal density will often simultaneously elevate the densities of both the consumer and the victim subpopulations. This makes the large simplifying assumption that stage structure is relatively stable across different total population densities and, therefore, that overall density reflects the densities of both potential cannibals and victims. Encounter frequencies rising with the square of density create a powerful expected source of density dependence in many cannibalistic populations.

Territoriality and other systems of conspecific avoidance

The mass action assumption is, of course, often violated in nature. In many cases, to avoid costly exploitative and interference competition associated with sharing space with conspecifics, organisms actively detect and avoid conspecifics (Fox, 1975; Polis, 1981). For example, many consumers establish and defend territories. Similarly, many insects that use discrete patches of resources (e.g., an *Anthocharis cardamines* caterpillar that develops on a single flower head [Dempster, 1997] or a *Hyposoter horticola* parasitoid wasp that develops inside a host caterpillar [Couchoux et al., 2015]) recognize the presence of conspecifics, sometimes through the detection of marking pheromones deposited by ovipositing females, and subsequently avoid laying eggs in occupied patches (Nufio & Papaj, 2001; Roitberg & Prokopy, 1987).

Nevertheless, conspecific avoidance is often observed to break down as densities rise, leading to potentially sharp rises in cannibalism. In territorial systems, once the habitat is saturated with conspecifics, "excess" individuals may be at high risk of mortality due to cannibalism (e.g., Chapin & Reed-Guy, 2017; Moya-Laraño et al., 2002). Male plainfin midshipman fish compete for large intertidal rocks under which they excavate and defend nests; they attract females to these nests to lay eggs that they fertilize. Nest sites are limited, however, and when male densities exceed nest availability, intruding males take over nests from residents and then gradually cannibalize the eggs found there (Bose et al., 2019). In "solitary" parasitoids, a given host insect can support the development of at most one individual; when more than one egg is laid per host, called "superparasitism," the larvae engage in lethal chemical or physical interactions, with losers typically being consumed along with the host by the victor (Godfray, 1994). Superparasitism is generally avoided when overall levels of host exploitation are low to moderate but increases as parasitoid abundance rises relative to available hosts (Godfray, 1994; Pan et al., 2018; Rosenheim & Hongkham, 1996). Similar interactions are also observed in parasitoids where groups of individuals can develop successfully on a single host: cannibalism occurs when within-host density rises beyond the capacity of the host to support their development (Tena et al., 2009). The same density-driven increases in cannibalism are also observed in many herbivorous insects (Baskauf, 2003; Richardson et al., 2010). Cannibalism in these systems may therefore be nearly absent and, thus, density independent at lower (below saturation) densities and then rise, becoming strongly density dependent at higher densities.

Effects of hunger on encounter frequencies

Increasing density can elevate encounter frequency beyond the mass action expectations for two additional reasons. First, increasing densities may lead to growing intraspecific competition for food resources. The link between rising densities and food shortages is not expected to be universal; food shortages may be minimal in populations that are held well below their carrying capacities by predators or parasites (e.g., Rosenheim, 2001; Rosenheim et al., 1999), and in other cases key resources other than food can become limiting as population densities rise (e.g., nesting sites). Nevertheless, for many species, rising population densities will be tied to food scarcity. Literature reviews of the ecology of cannibalism underscore the importance of a shortage of food, or the eventual consequence of such a shortage, hunger (a changed motivational state driven by a shortfall of food), as central determinants of the incidence of cannibalism (Dong & Polis, 1992; Elgar & Crespi, 1992; Fox, 1975; Polis, 1981; Richardson et al., 2010; Romano & Zeng, 2017; Scharf, 2016; Schausberger, 2003; Wise, 2006). Hunger can increase cannibalism in part by increasing foraging behavior, which elevates encounter frequencies between conspecifics (Scharf, 2016). For example, the swarming and mass migration of Mormon crickets, *Anabrus simplex*, are driven by shortfalls of two essential nutrients: salt and protein (Simpson et al., 2006). A shortfall of protein in the diet leads to increased locomotion and, thus, increased opportunities for encounters between individuals. Given an encounter with a vulnerable conspecific, including individuals who are

molting, wounded, or freshly dead, cannibalism ensues. Similarly, hunger in the cannibalistic praying mantis *Tenodera sinensis* triggers a shift from a prey ambush foraging strategy to one based on prey pursuit, with mantids detecting and actively pursuing prey from greater distances; experiments demonstrated that either prey consumption or injections of the peptide insulin, which appears to function as a global indicator of satiety, reverse these effects (Bertsch et al., 2019).

Second, food shortages may slow the development of immatures, potentially lengthening the period of vulnerability to cannibal attack and thus increasing the likelihood of encounters with cannibals during an individual's lifetime (e.g., Strauss et al., 2016).

Opposing effects: Responses to predation risk

Opposing processes that offset some of the rise of encounter frequency with increasing cannibal density may also be important. Foremost among these is the possibility that potentially vulnerable individuals facing a population of cannibals may use chemical, visual, tactile, or other cues to recognize the elevated risk associated with high conspecific densities and modify their behavior to avoid dangerous encounters (i.e., a so-called risk effect; Culshaw-Maurer et al., 2020; Peacor et al., 2020). The most common expectation is for a decrease in foraging activity or an increase in the use of spatial refuges from potential cannibals (e.g., McPeek & Crowley, 1987; Sadeh et al., 2009). For example, in response to chemical cues associated with conspecific adults, larvae of the newt *Notophthalmus viridescens* reduce their activity (Mathis, 2003), and small larvae of the California newt *Taricha torosa* increase their use of refuges (Kats et al., 1994). Young-of-year larvae of the salamander *Ambystoma tigrinum* showed both of these avoidance behaviors (Wissinger et al., 2010). Although these responses may decrease encounters with dangerous conspecifics, the associated reductions in foraging opportunities may also slow growth, prolonging the period of exposure.

Other responses to the detection of elevated cannibalism risk are possible, too. For example, the small nymphal stages of the dragonfly *Plathemis lydia* exhibit increased movement in response to chemical or visual cues associated with larger conspecifics (Ferris & Rudolf, 2007); because these dragonflies are primarily sit-and-wait predators, this response may reflect an effort to leave the immediate area of enhanced risk. Larvae of the beetle *Tribolium freemani* normally molt to the pupal stage within 2 months, in the eighth or ninth instar; however, the pupal stage is vulnerable to cannibalism, and larvae held under crowded conditions remain in the larval stage for up to 6 months, undergoing additional molts up to the 14th instar (Ruang-Rit & Park, 2018). In other systems, accelerated rather than delayed development may reduce the risk of cannibalism. In the cane toad, newly hatched prefeeding larvae are highly vulnerable to cannibalism; in response to cues from cannibals, these larvae accelerate their development to reach the invulnerable tadpole stage (DeVore et al., 2021). Similarly, vulnerable hatchlings of the salamander *Hynobius retardatus* exhibited faster growth and development when held with cannibalistic conspecifics and exhibited faster swim speeds, enhancing their escape from cannibal attacks (Kishida et al., 2015). Thus, through different pathways, risk effects can reduce the frequency of dangerous encounters with cannibalistic conspecifics below mass-action expectations.

EFFECTS OF DENSITY ON THE LIKELIHOOD OF SUCCESSFUL ATTACK GIVEN AN ENCOUNTER BETWEEN CONSPECIFICS

Effects of hunger on cannibal aggressiveness

The special costs of cannibalism, including the risks of consuming kin, acquiring disease, and injury due to counterattack, mean that it is often optimal for an animal that encounters a conspecific to express cannibalism only conditionally (Dong & Polis, 1992). When potential benefits from cannibalism are relatively low, the costs may exceed the benefits, and opportunities to mount cannibalistic attacks are rejected. Only when benefits are especially high will they exceed the substantial costs and cannibalism be favored. Hunger is perhaps the commonest factor that elevates the benefits of cannibalism by making the nutrients acquired through cannibalism especially critical for the survival or reproduction of the cannibal (De Block & Stoks, 2004; Elgar & Crespi, 1992; Polis, 1981; Via, 1999). Recent studies across a wide diversity of taxa have added to the extensive literature summarized in the early reviews of cannibalism, demonstrating that shortages of alternate food or hunger are important causes of elevated cannibalism (e.g., Ahmad et al., 2015; Amaral et al., 2009; Bayoumy & Michaud, 2015; De Block & Stoks, 2004; Duarte et al., 2010; Gallucci & Ólafsson, 2007; Lukasik, 2010; Lund et al., 2016; Petersen et al., 2010; Pizzatto & Shine, 2008; Simpson et al., 2006; Van den Beuken et al., 2019; Vijendravarma et al., 2013). Petersen et al. (2010) manipulated food availability to

create hunger asymmetries between interacting pairs of the wolf spider *Pardosa prativaga*, with one individual satiated and the other starved; the starved individual increased its aggressiveness and willingness to take risks and emerged as the cannibal twice as often as the satiated individual. Cannibalism among ciliates and amoebae is likewise a response to the exhaustion of available bacterial prey; Bloomfield (2019) and Medina et al. (2019) explain how starvation in the social amoeba *Dictyostelium discoideum* triggers a sexual phase of the life cycle, including the formation of a macrocyst in which a newly formed diploid zygote cannibalizes thousands of surrounding cells.

Recent studies have begun to reveal the hormonal regulation of starvation-induced expression of cannibalism. Studying the larvae of the mosquito *Culex pipiens*, El Husseiny et al. (2018) found that well-fed individuals are never cannibalistic, whereas starved individuals are. Starved individuals showed strongly elevated titers of octopamine, a neurohormone known to be involved in the control of both responses to starvation and inter- and intraspecific aggression (Roeder, 2020; Yakovlev, 2018; Zhou et al., 2008). Experimental augmentation of octopamine levels in starved individuals led to further increases in cannibalism, whereas experimental administration of the octopamine receptor antagonist phentolamine suppressed cannibalism almost entirely (El Husseiny et al., 2018). Thus, remarkably, a single neurohormone, octopamine, appears to forge a physiological link between starvation and the enhanced aggression underlying cannibalistic attacks. The biogenic amine epinephrine plays the role of octopamine in vertebrates; it is structurally very similar to octopamine, and the octopamine receptor in invertebrates shares sequence similarity with the epinephrine receptor in vertebrates (Roeder, 2020). Epinephrine has been found to regulate both food intake and aggressive behavior, including cannibalism, in domestic chickens (part of the "fight or flight" response; Cheng et al., 2001, Dennis, 2016). This suggests that a common system of hormonal regulation may underlie hunger-associated cannibalism in many animals.

Effects of hunger on victim vulnerability

Whereas the early onset of hunger may increase motivation of cannibals to attack, prolonged hunger may have another effect: weakening an individual sufficiently that it is less likely to initiate a cannibalistic attack or less able to defend itself from attacking conspecifics (Petersen et al., 2010). Wong and Kölliker (2013) showed that a period of starvation for one member of a pair of newly hatched, first-instar European earwigs, *Forficula auricularia*, resulted in greater risk of falling victim to its well-fed conspecific. Enhanced victim vulnerability may favor cannibalistic attacks by increasing the likelihood of a successful attack while decreasing the risk of injury or effective counterattack.

Density-dependent induction of cannibalistic polyphenisms

In some cannibalistic taxa, certain individuals undergo a different developmental trajectory, producing a morphology that often includes larger body size but also sometimes enhanced development of structures used to attack, subdue, and ingest conspecifics (e.g., larger mouths or enhanced jaw musculature, teeth, or other fighting structures). Because this divergent development appears to be environmentally controlled, it is a polyphenism. Cannibalistic polyphenisms have been documented in diverse taxa, including protozoans (Banerji & Morin, 2009; Smith-Somerville et al., 2000; Waddell, 1992), mites (Lukasik, 2010; Van den Beuken et al., 2019), insects (Vijendravarma et al., 2013), amphibians (Hoffman & Pfennig, 1999; Levis et al., 2018; Nishimura, 2018), and fish (Amundsen, 2016), and is associated with greater expression of cannibalism. It is possible that such polyphenisms are more common than has been recognized; although in some cases the cannibalistic forms are conspicuous, in other cases the morphological differences may be subtle, even if still functionally significant. For example, the presence of a cannibalistic polyphenism in *Drosophila melanogaster*, one of the most intensively studied organisms in biology, went unrecognized until a study by Vijendravarma et al. (2013), who found that third-instar larvae raised on a strictly cannibalistic diet had mouth hooks with 20% more teeth, which they use to rasp holes in the integument of their conspecific victims.

Cannibalistic polyphenisms often develop in response to elevated conspecific density, hunger, or a diet rich in conspecifics. High densities of conspecifics elicit the development of cannibal forms ("macrostomes") in the ciliate protozoan *Tetrahymena vorax*; the availability of food appears not to be an important cue in this case (Banerji & Morin, 2009; Smith-Somerville et al., 2000). In this system, only the macrostome, with its enlarged mouth ("cytostome") and development of a large cytopharyngeal pouch, a vacuole large enough to accommodate a ciliate prey, is capable of ingesting and digesting conspecifics. In the tiger salamander *Ambystoma tigrinum*, density is also the prime elicitor of cannibalistic forms. Physical contact between young *Ambystoma* larvae is required to induce cannibalistic

forms; visual or chemical cues associated with conspecifics were insufficient in the absence of physical contact (Hoffman & Pfennig, 1999). Manipulations of conspecific density also elicited the development of cannibal forms in the Hokkaido salamander *Hynobius retardatus* (Nishimura, 2018). In some ciliates and amoebae, hunger, which is often a downstream consequence of high conspecific density, appears to trigger the shift to cannibalism and the production of giant cells that consume conspecifics and other protozoans instead of bacterial prey (Waddell, 1992).

The presence of a cannibalistic morph enhances physical asymmetries between potential cannibals and victims, even when they are members of the same cohort, thereby making cannibalistic attacks less dangerous and more profitable for cannibals. Because consumption of conspecifics can cause further gains in growth rates and increased elaboration of cannibalistic weapons, the potential for positive feedbacks appears to be widespread in systems with cannibalistic polyphenisms, with the endpoint being giant cannibals that can generate substantial mortality of conspecifics (Amundsen, 2016; Huss et al., 2010; Kishida et al., 2011; Persson et al., 2003). Cannibalistic polyphenisms, then, appear to be a major source of density dependence in cannibalism, largely as a result of elevated likelihood that an encounter between a vulnerable, noncannibalistic form and an aggressive, cannibalistic form will translate into a lethal attack.

Exceptions to the rule that cannibalistic forms are elicited under high-density conditions have been observed in several acarid mites. In these cases, cannibalistic forms are found only in males as part of a set of alternative male reproductive strategies in which males can either engage in lethal fights for access to females using their weaponized hind legs or engage in scramble competition without direct attacks among males (Stewart et al., 2018). The same weapons are, however, also used in the alternate context of cannibalistic attacks, yielding an important source of nutrition (Lukasik, 2010; Van den Beuken et al., 2019). In these acarid mites, the development of the cannibalistic polyphenism is strongly suppressed under high-density conditions (Radwan, 2001; Tomkins et al., 2004), apparently because fighting in high-density situations results in a too-costly escalation of the risk of injuries or death. Genetic determination of cannibalistic males has also been demonstrated in some acarid mites (Radwan, 1995).

Density as a cue of low relatedness

The risk of losing inclusive fitness through the consumption of kin is an important potential cost of cannibalism. Species that can recognize kin directly can minimize this cost (Pfennig, 1997). Species lacking direct kin recognition may, however, use indirect cues, including the local density of conspecifics, as a means of estimating the likely relatedness of conspecifics. Greater local densities may often signal lower risks from the consumption of kin, promoting cannibalism. Although not extensively studied, this may be a common source of density dependence in cannibalism. Observational and experimental studies of fish have shown that cannibalism of eggs by adult males is increased in the presence of other males, including sneaker males, and when a male takes over a nest from an egg-guarding conspecific; in all of these cases, the presence of other males signals a decreased likelihood of paternity of conspecific eggs (Bose et al., 2014, 2019; Gray et al., 2007; Manica, 2004). Similarly, adult females of the omnivorous insect *Geocoris pallens* cannot discriminate between their own eggs and eggs laid by conspecifics; females held in isolation rarely consume their own eggs, but the presence of a neighboring conspecific female triggers strongly elevated cannibalism (Law & Rosenheim, 2013). Field observations (Rosenheim, 2005) and experimentation (Law & Rosenheim, 2011) suggest that this strongly density-dependent cannibalism establishes an upper limit on density of *G. pallens*, even when prey resources are abundant.

Density as a predictor of future competition or mortality: Filial cannibalism

Filial cannibalism, the consumption of offspring by parents, can in some cases be a means of adjusting offspring number and quality to maximize final offspring success (Davenport et al., 2019). Thus, although filial cannibalism results in the immediate mortality of some offspring, it can be adaptive if it augments the survival or quality of those that remain. The consequences of this type of filial cannibalism for population growth rate are therefore expected to be positive, the opposite of the normal expectation. This type of filial cannibalism essentially represents an unusual type of parental care and, thus, falls somewhat outside the primary focus of this review; nevertheless, we briefly review density-dependent expression of this type of filial cannibalism.

Davenport et al. (2019) introduced a model showing that when population density affects offspring survival or reproductive success through density-dependent competition, disease, or predation, parents can favor cannibalizing some offspring to relax that subsequent density-dependent process. *Nicrophorus* spp. burying beetles appear to use current population density as a predictor of the future

intensity of competition likely to be experienced by their offspring and, thus, cannibalize more of their offspring to generate fewer, but larger, offspring, which can win contests over the possession of the small rodent carcasses used for reproduction (Creighton, 2005; Woelber et al., 2018). Similarly, sand gobies, *Pomatoschistus minutus*, appear to anticipate the action of density-dependent mortality factors when they cannibalize their eggs under high-density conditions (Klug et al., 2006). In both of these cases, cannibalism seems likely to promote, rather than slow, population growth.

Disease

Infectious disease is a challenge faced by virtually all organisms; as densities rise, opportunities for spread of most horizontally transmitted pathogens rise in parallel. Although some organisms anticipate the elevated risk of infection at high densities by displaying enhanced immune function (e.g., Murray et al., 2020), disease remains one of the most consistently density-dependent processes in nature (Schmid-Hempel, 2011). Disease also promotes cannibalism expression in numerous ways. Although our understanding of disease–cannibalism interactions is still developing, research has revealed several pathways through which disease is likely to create an underlying density dependence in cannibalism. First, in some cases, infected individuals may be weakened, increasing their vulnerability to attacks by conspecifics (Boots, 1998; Pfennig et al., 1999; Pizzatto & Shine, 2011; Williams & Hernández, 2006). In other cases, however, potential cannibals may avoid attacking infected conspecifics if they pose a risk of infection (e.g., Maák et al., 2020; Siva-Jothy et al., 2018). Second, infection may stunt host growth, augmenting within-cohort variation in size and thereby promoting cannibalism (Chapman et al., 1999; Elderd, 2019; Van Allen et al., 2017). Third, infections may kill the host, precipitating cannibalism (necrophagy; Collinge et al., 2006; Rudolf & Antonovics, 2007). Fourth, vertically transmitted pathogens may manipulate their host to increase its risk of being cannibalized, creating a mechanism for pathogen transmission to new host lineages (Patot et al., 2010; Varaldi et al., 2003; Varaldi & Lepetit, 2018). Fifth, uninfected hosts may cannibalize infected conspecifics as a means of limiting pathogen spread (Chouvenc & Su, 2012; Davis et al., 2018; Lehtonen & Kvarnemo, 2015; Rosengaus & Traniello, 2001; Van Allen et al., 2017; see also Posada-Florez et al., 2021). And sixth, infections may impose a nutritional stress on hosts, increasing their expression of cannibalism (Bunke et al., 2015, 2019; Rosenheim et al., 2019; Yan et al., 1994). The first five of these pathways involve the infected individual as the victim of cannibalism, whereas the last pathway involves the infected individual as the attacker. In Appendix S1, we examine in detail each of these pathways.

Induction of resistance in alternate food resources

Density-dependent expression of cannibalism can emerge when high densities of a cannibalistic consumer elicit the development of resistant phenotypes in the cannibal's heterospecific food resources, thereby promoting increased reliance on conspecifics as a source of food. This has been demonstrated in two quite different systems. First, Kishida and Nishimura (2004) showed that larvae (tadpoles) of the frog *Rana pirica* develop a predator-induced morphological defense: a wider "bulgy" morphology that restricts the ability of their gape-limited predator, larvae of the salamander *Hynobius retardatus*, to consume them. This response was graded in response to the local density of salamander larvae, with the defensive phenotype observed in response to close, but not more distant, cues from the predator. Salamander larvae faced with defended tadpoles consumed fewer tadpole prey and instead shifted to increased cannibalism and a stronger induction of cannibalistic morphs (Kishida et al., 2009). Second, Orrock et al. (2017) showed a parallel interaction in a plant–insect system: tomato plants, *Solanum lycopersicum*, exhibited a graded induced resistance in response to herbivory or the chemical elicitor methyl jasmonate, which decreased the quality of the plant's foliage to the caterpillar *Spodoptera exigua*. In response, caterpillars decreased their consumption of the host plant and increased their expression of cannibalism (but see also Elderd, 2019). Thus, if increasing densities of consumers favor stronger induced resistance in heterospecific food resources, these consumers may resort to more frequent attacks on conspecifics.

EFFECTS OF DENSITY DEPENDENCE ON POPULATION DYNAMICS

As shown in Figure 1, we expect mortality due to cannibalism to increase as the product of encounter frequency, the likelihood of initiating an attack given an encounter, and the likelihood that an attack, once initiated, will succeed. Under the mass action assumption, we expect encounter frequency to increase as (proportional to [population density]^2); as discussed earlier, in some cases we might expect encounter frequency to rise even faster than

this, for example, when hunger leads to intensified foraging activity. Furthermore, we have described a diversity of ecological pathways through which rising densities of a potentially cannibalistic species can lead to increasing probabilities of launching successful cannibalistic attacks given an encounter. Cannibalism is, therefore, expected to increase as the product of three functions, all rising with density. Whereas density-driven increases in encounters can rise without any clear bound, density-driven increases in the likelihood of mounting successful attacks cannot exceed the limit of 100%. All of these pathways produce a mortality process that is expected to rise rapidly with density, including, in some cases, terms in Taylor expansion that are proportional to (population density)^3. Although a mortality process that increases as the cube of population density might not be unique to cannibalism (e.g., a specialist predator that showed rapid numerical responses to prey populations and that exhibited a Type III functional response might generate mortality that increased as [prey density]^3, under some conditions), this does constitute an exceptionally strong form of density-dependent mortality, producing potentially powerful negative feedback on population growth rates. Here, we use mathematical models to explore the possible consequences of this powerful negative feedback on the predicted dynamics of cannibalistic populations.

In a review of over 30 mathematical models of cannibalism, Claessen et al. (2004) identified four consistent effects of cannibalism on population dynamics: (i) regulation of population size, (ii) destabilization of steady states that results in oscillatory or chaotic population dynamics, (iii) stabilization of species interactions, and (iv) creation of alternative states. The models reviewed by Claessen et al. (2004), however, assume that per-capita cannibalism rates are density-independent. Given the empirical evidence for density dependence in these rates, we examine the following question: What effects does density-dependence in per-capita cannibalism rates have on population dynamics? To address this question, we modify and analyze three models found in the Claessen et al. (2004) review. The first two models are the classical single species model of Ricker (1954) and a stage-structured extension of this classical model. These models provide insights into how density dependence in per-capita cannibalism rates impact (i) population regulation and (ii) stability. Furthermore, our analysis highlights how density-dependent cannibalism can modify the effects of environmental stochasticity on population regulation. The results for the stage-structured model are presented in Appendix S2. The third model is a cannibalistic variant of the classical McArthur-Rosenzweig predator–prey model due to Kohlmeier and Ebenhöh (1995). Modifying this model highlights how density dependence in per-capita cannibalism rates can (iii) stabilize species interactions and (iv) generate alternative ecological outcomes.

Incorporating density dependence in per-capita cannibalism rate

For classical mass-action models of cannibalism, the per-capita cannibalism rate (aN) is the product of a density-independent attack rate a and the population density N (top row in Figure 2). The resulting net mortality rate due to cannibalism is aN^2. In our models, we allow the attack rate $a(N)$ to be density-dependent. While most of our mathematical analysis only assumes the attack rate $a(N)$ is an increasing function of density N, our numerical explorations use two complementary functional forms with this property. The simpler form assumes the attack rate increases linearly with density. Specifically, $a(N) = a_{\min} + \beta N$, where $a_{\min}$ ins the minimal per-capita cannibalism rate and β determines how quickly per-capita cannibalism rates increase with density (middle row in Figure 2). For this functional form, the attack rate $a(N)$ increases without bound with the

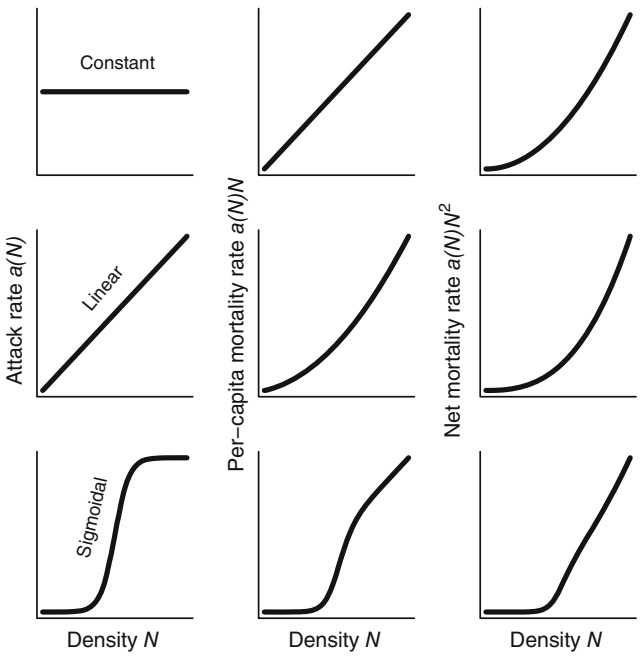

FIGURE 2 Different forms of attack rates and their impacts on per-capita cannibalism rates and net mortality rates due to cannibalism. Rows, from top to bottom, correspond to a density-independent attack rate a, a linearly increasing attack rate $a(N) = a_{\min} + \beta N$, and a sigmoidal attack rate $a(N) = a_{\min} + \frac{a_{\max} - a_{\min}}{1 + \exp(-\beta(N-\zeta))}$, respectively. Columns, from left to right, plot the attack rate, the per-capita cannibalism rate, and the net mortality rate due to cannibalism as functions of population density.

population density N. This might happen if searching activity driven by hunger increases with density. The second functional form is more appropriate if density dependence occurs in the probability of launching an attack or succeeding in an attack. In both of these cases, there is a maximal attack rate $a_{\max}$, and the functional form is

$$a(N) = a_{\min} + \frac{a_{\max} - a_{\min}}{1 + \exp(-\beta(N - \zeta))}$$

where ζ corresponds to the function's inflection point, and β determines how quickly per-capita cannibalism rates increase with density (bottom row in Figure 2). This latter functional form is like a Type III functional response for a switching predator: accelerating at lower densities and saturating at higher densities. For both functional forms of the attack rate $a(N)$, the per-capita cannibalism rate is an accelerating function of density, unlike the density-independent attack rate a (middle column in Figure 2).

Effects on lagged population dynamics: Regulation and stability

As the effects of negative feedbacks on population stability depend on whether the population exhibits delayed responses to changes in its density, we modified the simplest, discrete-time model of cannibalism, the Ricker equation (Ricker, 1954). In this model, the population density in generation t is N_t. Each individual produces λ offspring of which a fraction e^{-aN_t} escapes cannibalism; a is the attack rate. We modify this model to allow this per-capita rate, $a = a(N)$, to be density-dependent. Under these assumptions, the model becomes

$$N_{t+1} = N_t \lambda e^{-a(N_t)N_t}$$

When the intrinsic growth rate ($\ln \lambda$) is positive, the population will increase from low densities, allowing the population to persist. In the absence of cannibalism ($a = 0$), the population exhibits unbounded exponential growth. Density-independent cannibalism results in negative per-capita growth rates at high densities (i.e., $\ln\lambda - aN < 0$ for large N). This, coupled with a positive, intrinsic growth rate, ensures population regulation: a tendency for the population densities to decrease when reaching high densities and a complementary tendency of the population densities to increase when the population density is low (Chesson, 1982; Roth & Schreiber, 2014; Turchin, 1995).

The addition of density dependence in the attack rate strengthens population regulation. Why? On the one hand, density dependence results in lower attack rates at lower population densities. This results in more positive per-capita growth rates at lower population densities and, consequently, faster population recovery from low densities. Density dependence in the attack rate also leads to higher attack rates at high population densities. These higher attack rates result in more negative per-capita growth rates at high densities and, consequently, faster population declines from high densities. Thus, collectively, density dependence in the attack rates increases the speed at which the population moves away from low or high densities.

While increasing the strength of density-independent attack rates reduces the population's equilibrium density, it has no effect on stability (Claessen et al., 2004). In contrast, density dependence in attack rates strengthens the negative feedback at the system's equilibrium (Figure 3). More precisely, density dependence causes the derivative of the growth function $G(N) = N\lambda e^{-a(N)N}$ to become more negative at the equilibrium (slopes of colored curves at the dashed line in Figure 3a; Appendix S2). If the equilibrium is stable without density dependence, then weak density dependence will strengthen the equilibrium stability; the population will return to its equilibrium density more quickly following a disturbance (see blue curves in Figure 3a,b). However, if the density dependence near the equilibrium density is too strong, then density dependence can destabilize the system, leading to population cycling (red curve in Figure 3a,b) or chaos. Similar conclusions apply to the stage-structured Ricker model (Appendix S2).

When disturbances occur repeatedly (i.e., the population experiences environmental stochasticity), density dependence in attack rates typically strengthens population regulation by lowering the long-term mean population density (Figure 3c,d; Appendix S2). Specifically, when there are fluctuations in the intrinsic growth rate ($\ln\lambda$), the population approaches a stationary distribution (cf. Roth & Schreiber, 2014). At this stationary distribution, the average per-capita growth rate must equal zero, i.e., the population is tending to neither increase nor decrease in the long term (Benaïm & Schreiber, 2019; Schreiber et al., 2011). Thus, the average value of the intrinsic growth rate ($\overline{\ln\lambda}$) must be balanced by the average value of the net cannibalism rate $\overline{a(N)N}$. With density-independent attack rates, the fluctuations in the intrinsic growth rate have no effect on the long-term mean population density, i.e., $\overline{N} = \overline{\ln\lambda}/\overline{a}$. This outcome follows from the net mortality rate as a result of cannibalism's being linear in population density. This linearity

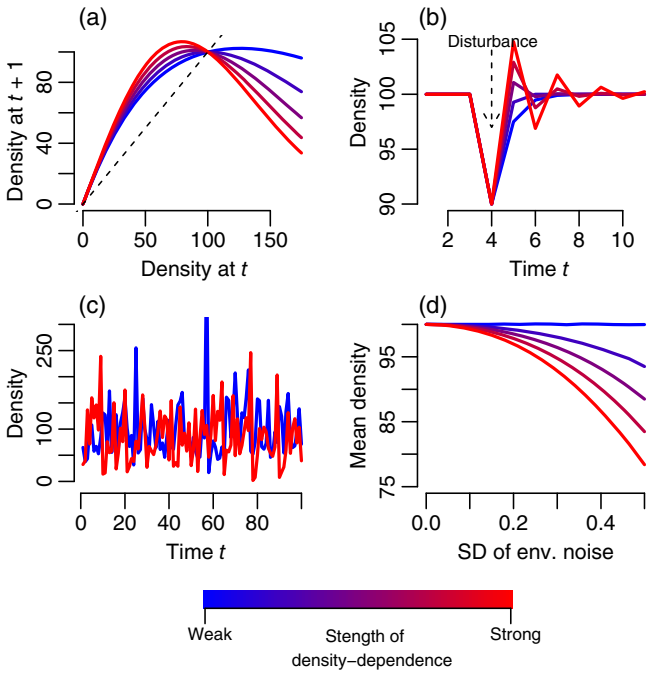

FIGURE 3 Effects of density dependence in per-capita cannibalism rates on stability and population regulation. (a) Relationship between population density in generation $t+1$ versus generation t. Colored curves correspond to different levels of density-dependent per-capita cannibalism rates. The one-to-one dashed line intersects these curves at the equilibrium densities for the system. (b) Population dynamics in response to one-time disturbance at time $t=4$; prior to disturbance, the population is at its equilibrium density. (c) Population dynamics with environmental fluctuations in intrinsic rate of growth $r=\ln\lambda$; only two levels of density-dependent cannibalism are shown. (d) Mean, long-term population density as function of standard deviation of fluctuations in λ. Parameterization: $\ln\lambda=0.8$, $a(N)=a_{\min}+\beta N$, with $\beta=0$ (blue), 0.003125, 0.006250, and 0.009375 (red) and $a_{\min}$ chosen to give equilibrium density $N=100$. (c, d) $\ln\lambda$ is normally distributed with mean 0.08 and standard deviations as shown.

breaks down with density-dependent per-capita cannibalism rates. For example, the function form $a(N)=a_{\min}+\beta N$ produces a per-capita cannibalism rate ($a_{\min}N+\beta N^2$) that is a convex function of population density. Hence, by Jensen's inequality, the fluctuations in population density (due to the fluctuation in $\ln\lambda$) result in higher per-capita cannibalism rates than in the absence of the fluctuations. Consequently, density-dependent attack rates lower the average population density (Figures 3d). The opposite can occur when attack rates become strongly saturated at higher population densities and these higher densities occur often. At these higher densities, the per-capita cannibalism rate can be concave as a function of population density, and fluctuations can increase in the average population density. However, we anticipate that this latter outcome will be shown to be less common in nature.

Effects on predator–prey dynamics: Stability, regulation, and alternative outcomes

To examine the impact of escalated cannibalism rates on species interactions, we consider the dynamics of a cannibalistic predator and its prey with densities N and P, respectively. Following Kohlmeier and Ebenhöh (1995), the prey has an intrinsic rate of growth r. The predator exhibits a Type II functional response (Holling, 1959) with attack rates a_N and a_P and handling times h_N and h_P for the prey species and cannibalism, respectively. We modify the model to allow the cannibalistic attack rate, $a_P=a_P(P)$, to be density-dependent. The predator converts consumed individuals with conversion efficiencies c_N and c_P to new offspring and has a per-capita mortality rate d. With our modifications, the model with overlapping generations is

$$\frac{dN}{dt}=rN-\frac{a_N NP}{1+a_N h_N N+a_P(P)h_P P}$$
$$\frac{dP}{dt}=\frac{P(c_N a_N N+(c_P-1)a_P(P)P)}{1+a_N h_N N+a_P(P)h_P P}-dP$$

When the cannibal's attack rate is density-independent, Kohlmeier and Ebenhöh (1995) found two threshold values of the per-capita cannibalism rates, $0<a_P^*<a_P^{**}$ (Appendix S3). When cannibalism is too strong ($a_P>a_P^{**}$), the predator is unable to regulate the prey, there is no equilibrium supporting both species, and both species quickly increase exponentially. When the cannibalism rate is too weak ($a_P<a_P^*$), the predator partially regulates the prey sufficiently to create an equilibrium supporting both species, but the equilibrium will be unstable, resulting in oscillations in both species' densities with ever increasing amplitude. In the presence of demographic stochasticity, one would expect one or both species to go extinct. Finally, when cannibalism is of intermediate strength ($a_P^*<a_P<a_P^{**}$), the predator regulates the prey and both species' densities approach a stable equilibrium.

Adding density dependence in the cannibal attack rate has two consistent effects on species interactions (Figure 4; Appendix S3). First, whenever cannibalism is sufficiently weak to produce an equilibrium supporting both species ($a_P<a_P^*$), density dependence always helps stabilize the equilibrium (Figure 4a vs. b). Second, density dependence can lead to alternative ecological

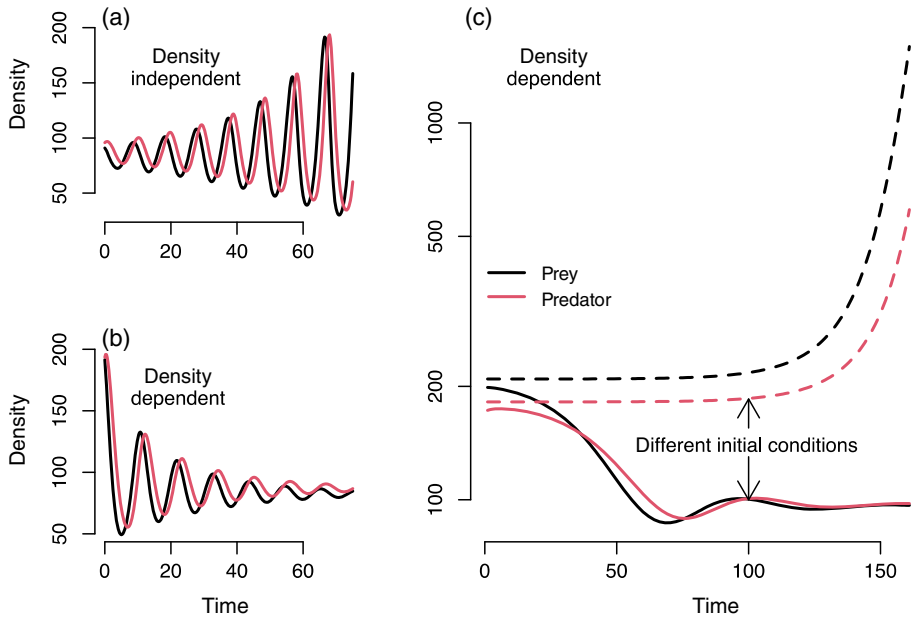

FIGURE 4 Density-dependent cannibalism stabilizes predator–prey interactions and generates alternative ecological outcomes. (a, b) Predator–prey dynamics with and without density dependence in per-capita cannibalism rate. Population trajectories of prey and predator are plotted in black and red, respectively. (c) Two predator–prey time series that only differ in initial densities of the two prey species. The solid pair approaches a stable equilibrium; the dashed pair exhibits unbounded growth of both species. Parameters: $r = a_N = h_N = h_P = c_N = \kappa = 1$, $d = 0.75$, $c_P = 0$, with $a_P = 0.8$ for the density-independent model and $a_P(P) = \frac{1.6}{1+\exp(-(P-1.18182))}$ for the density-dependent model. The a_p parameter is chosen such that the density-independent and density-dependent models share a predator–prey equilibrium.

outcomes. When predator and prey densities are not too high, the predator can regulate the prey and the species eventually coexist about a stable equilibrium (solid lines in Figure 4c). However, when predator or predator densities are too high, the predator is unable to regulate the prey and the species grows without bound (dashed lines in Figure 4c). Adding prey density dependence in this case results in the species' coexisting about an alternative stable equilibrium with high densities rather than unbounded growth (Appendix S3: Figure S1). Intuitively, density-dependent attack rates of cannibals can result in intermediate cannibalism rates at lower predator densities that promote stable coexistence and at the same time produce sufficiently high cannibalism rates at higher densities that disrupt regulation of the prey.

FUTURE DIRECTIONS

1. Although our literature review highlighted mechanisms through which per-capita mortality from cannibalism might be expected to accelerate with a rising density of the cannibal population, to our knowledge, no empirical study has attempted to determine whether such an acceleration occurs in nature. Hints of such an acceleration can be seen in some studies (e.g., Elliott, 2004, fig. 5b or Moksnes, 2004, fig. 2), but past research focused almost exclusively on establishing a strong underlying density dependence of cannibalism rather than attempting to describe the detailed form of the function. We see this as fertile ground for future research.
2. The possibility that a common system of hormonal regulation involving biogenic amine effector molecules may forge a widespread linkage between hunger and cannibalism expression in diverse animals warrants additional research.
3. Density-dependent induction of cannibalistic polyphenisms appears to be found in diverse taxa; additional research should examine how these cannibalistic polyphenisms influence the form of density-dependent mortality from cannibalism in the field.
4. Additional research should continue to expand our understanding of how interactions between a cannibal population and other members of the community, including disease organisms and heterospecific prey species, shape the resulting expression of density-dependent cannibalism.
5. It would be extremely valuable to devise an experimental means of manipulating the expression of cannibalism

over multiple generations in the field. Such manipulations would enable the first definitive tests of the role of cannibalism in the self-regulation of populations.

6. Our mathematical analysis highlights how accelerating per-capita cannibalism rates may act as a positive-feedback mechanism leading to alternative stable states. Prior work identified other positive-feedback mechanisms, including the direct effect of energetic gains from cannibalism (Cushing, 1991; Diekmann et al., 2003; Van den Bosch et al., 1988) and the indirect effect of reduced resource competition (Botsford, 1981; Fisher, 1987). The extent to which accelerating per-capita cannibalism rates amplify or dampen these other positive feedback mechanisms warrants future study.
7. The impacts of cannibalism on population dynamics often depend on the interplay between size-structure and time delays (Claessen et al., 2004). For example, this interplay can result in population cycles that alternate between periods with many dwarf-sized individuals and periods with few giant-sized individuals (Claessen et al., 2000). How cannibalism rates accelerate in a size-dependent manner and the implication of these accelerations for generation cycles remain to be understood.
8. Additional research should also examine other forms of population structure, including spatial or genetic structure or sexual dimorphisms.

CONCLUSION

Cannibalism can act as a mechanism of self-regulation because of the diverse ecological processes that make mortality from cannibalism density-dependent. For any predator, attacking and consuming heterospecific prey has obvious benefits but also some costs. However, attacking a conspecific has unique costs that go beyond those expected from heterospecific predation: Conspecifics may offer elevated risks of counterattack, are more likely to be sources of infectious pathogens, and may prove to be kin whose death will impose inclusive fitness costs on the attacker. For these reasons, many organisms that are capable of consuming conspecifics express some level of restraint, at least when environmental conditions are good. When, however, population density rises, conditions often deteriorate. Food resources may dwindle or become better defended, creating hunger; pathogens may spread, creating additional stress; and asymmetries of size and physiological vigor between pairs of conspecifics may grow. Finally, the balance may tip, with benefits of cannibalism exceeding the costs. Thus, cannibalism is often expressed as a partly or fully conditional behavior, expressed as rising population density induces stress. This creates strong negative feedback on the dynamics of single populations that can produce self-regulation but may limit the population growth of some predator populations sufficiently to allow their prey to escape from control. Thus, the conditional expression of cannibalism can make a central contribution to the density dependence in cannibalism-imposed mortality.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

Code (Schreiber & Rosenheim, 2022) is available in Figshare at https://doi.org/10.6084/m9.figshare.20024327.v1.

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REFERENCES

Ahmad, M., S. U. Chaudhary, A. J. Afzal, and M. Tariq. 2015. "Starvation-Induced Dietary Behaviour in *Drosophila melanogaster* Larvae and Adults." *Scientific Reports* 5: 14285. https://doi.org/10.1038/srep14285.

Amaral, V., J. Paula, S. Hawkins, and S. Jenkins. 2009. "Cannibalistic Interactions in Two Co-Occurring Decapod Species: Effects of Density, Food, Alternative Prey and Habitat." *Journal of Experimental Marine Biology and Ecology* 368: 88–93.

Amundsen, P.-A. 2016. "Contrasting Life-History Strategies Facilitated by Cannibalism in a Stunted Arctic Charr Population." *Hydrobiologia* 783: 11–9.

Andersson, J., P. Byström, D. Claessen, L. Persson, and A. M. de Roos. 2007. "Stabilization of Population Fluctuations Due to Cannibalism Promotes Resource Polymorphism in Fish." *American Naturalist* 169: 820–9.

Andow, D. A., J. R. Farias, R. J. Horikoshi, D. Bernardi, A. R. B. Nascimento, and C. Omoto. 2015. "Dynamics of Cannibalism in Equal-Aged Cohorts of *Spodoptera frugiperda*." *Ecological Entomology* 40: 229–36.

Banerji, A., and P. J. Morin. 2009. "Phenotypic Plasticity, Intraguild Predation and Anti-Cannibal Defences in an Enigmatic Polymorphic Ciliate." *Functional Ecology* 23: 427–34.

Barabás, G., M. J. Michalska-Smith, and S. Allesina. 2017. "Self-Regulation and the Stability of Large Ecological Networks." *Nature Ecology & Evolution* 1: 1870–5.

Baskauf, S. J. 2003. "Factors Influencing Population Dynamics of the Southwestern Corn Borer (Lepidoptera: Crambidae): A Reassessment." *Environmental Entomology* 32: 915–28.

Bayoumy, M. H., and J. P. Michaud. 2015. "Cannibalism in Two Subtropical Lady Beetles (Coleoptera: Coccinellidae) as a Function of Density, Life Stage, and Food Supply." *Journal of Insect Behavior* 28: 387–402.

Benaïm, M., and S. J. Schreiber. 2019. "Persistence and Extinction for Stochastic Ecological Models with Internal and External Variables." *Journal of Mathematical Biology* 79: 393–431.

Bertsch, D. J., J. P. Martin, G. J. Svenson, and R. E. Ritzmann. 2019. "Predatory Behavior Changes with Satiety or Increased Insulin Levels in the Praying Mantis (*Tenodera sinensis*)." *Journal of Experimental Biology* 222: jeb197673. https://doi.org/10.1242/jeb.197673.

Bloomfield, G. 2019. "Sex and Microcyst Formation in *Dictyostelium*." *The International Journal of Developmental Biology* 63: 439–46.

Boots, M. 1998. "Cannibalism and the Stage-Dependent Transmission of a Viral Pathogen of the Indian Meal Moth, *Plodia interpunctella*." *Ecological Entomology* 23: 118–22.

Bose, A. P. H., K. M. Cogliati, H. S. Howe, and S. Balshine. 2014. "Factors Influencing Cannibalism in the Plainfin Midshipman Fish." *Animal Behaviour* 96: 159–66.

Bose, A. P. H., M. J. Lau, K. M. Cogliati, B. Neff, and S. Balshine. 2019. "Cannibalism of Young Is Related to Low Paternity and Nest Take-Overs in an Intertidal Fish." *Animal Behaviour* 153: 41–8.

Botsford, L. W. 1981. "The Effects of Increased Individual Growth Rates on Depressed Population Size." *The American Naturalist* 117: 38–63.

Briggs, C. J., S. M. Sait, M. Begon, D. J. Thompson, and H. C. J. Godfray. 2000. "What Causes Generation Cycles in Populations of Stored-Product Moths?" *Journal of Animal Ecology* 69: 352–66.

Buddle, C. M., S. E. Walker, and A. L. Rypstra. 2003. "Cannibalism and
Density-Dependent Mortality in the Wolf Spider *Pardosa milvina* (Araneae: Lycosidae)." *Canadian Journal of Zoology* 81: 1293–7.

Bunke, M., M. E. Alexander, J. T. A. Dick, M. J. Hatcher, R. Paterson, and A. M. Dunn. 2015. "Eaten Alive: Cannibalism Is Enhanced by Parasites." *Royal Society Open Science* 2: 140369. https://doi.org/10.1098/rsos.140369.

Bunke, M., J. T. A. Dick, M. J. Hatcher, and A. M. Dunn. 2019. "Parasites Influence Cannibalistic and Predatory Interactions within and between Native and Invasive Amphipods." *Diseases of Aquatic Organisms* 136: 79–86.

Chapin, K. J., and S. Reed-Guy. 2017. "Territoriality Mediates Atypical Size-Symmetric Cannibalism in the Amblypygi *Phrynus longipes*." *Ethology* 123: 772–7.

Chapman, J. W., T. Williams, A. Escribano, P. Caballero, R. D. Cave, and D. Goulson. 1999. "Age-Related Cannibalism and Horizontal Transmission of a Nuclear Polyhedrosis Virus in Larval *Spodoptera frugiperda*." *Ecological Entomology* 24: 268–75.

Cheng, H. W., G. Dillworth, P. Singleton, Y. Chen, and W. M. Muir. 2001. "Effects of Group Selection for Productivity and Longevity on Blood Concentrations of Serotonin, Catecholamines, and Corticosterone of Laying Hens." *Poultry Science* 80: 1278–85.

Chesson, P. L. 1982. "The Stabilizing Effect of a Random Environment." *Journal of Mathematical Biology* 15: 1–36.

Chouvenc, T., and N.-Y. Su. 2012. "When Subterranean Termites Challenge the Rules of Fungal Epizootics." *PLoS One* 7: e34484. https://doi.org/10.1371/journal.pone.0034484.

Christie, H., and P. Kraufvelin. 2004. "Mechanisms Regulating Amphipod Population Density within Macroalgal Communities with Low Predator Impact." *Scientia Marina* 68(Suppl. 1): 189–98.

Claessen, D., A. M. de Roos, and L. Persson. 2000. "Dwarfs and Giants: Cannibalism and Competition in Size-structured Populations." *The American Naturalist* 155: 219–37.

Claessen, D., A. M. de Roos, and L. Persson. 2004. "Population Dynamic Theory of Size-Dependent Cannibalism." *Proceedings of the Royal Society of London B* 271: 333–40.

Collinge, J., J. Whitfield, E. McKintosh, J. Beck, S. Mead, D. J. Thomas, and M. P. Alpers. 2006. "Kuru in the 21st Century – An Acquired Human Prion Disease with Very Long Incubation Periods." *Lancet* 367: 2068–74.

Costantino, R. F., R. A. Desharnais, J. M. Cushing, and B. Dennis. 1997. "Chaotic Dynamics in an Insect Population." *Science* 275: 389–91.

Couchoux, C., P. Seppä, and S. van Nouhuys. 2015. "Behavioural and Genetic Approaches to Evaluate the Effectiveness of Deterrent Marking by a Parasitoid Wasp." *Behaviour* 152: 1257–76.

Creighton, J. C. 2005. "Population Density, Body Size, and Phenotypic Plasticity of Brood Size in a Burying Beetle." *Behavioral Ecology* 16: 1031–6.

Culshaw-Maurer, M., A. Sih, and J. A. Rosenheim. 2020. "Bugs Scaring Bugs: Enemy-Risk Effects in Biological Control Systems." *Ecology Letters* 23: 1693–714.

Cushing, J. M. 1991. "A Simple Model of Cannibalism." *Mathematical Biosciences* 107: 47–71.

Davenport, M. E., M. B. Bonsall, and H. Klug. 2019. "Unconventional Care: Offspring Abandonment and Filial Cannibalism Can Function as Forms of Parental Care." *Frontiers in Ecology and Evolution* 7: 113. https://doi.org/10.3389/fevo.2019.00113.

Davis, H. E., S. Meconcelli, R. Radek, and D. P. McMahon. 2018. "Termites Shape their Collective Behavioural Response Based on Stage of Infection." *Scientific Reports* 8: 14433. https://doi.org/10.1038/s41598-018-32721-7.

De Block, M., and R. Stoks. 2004. "Cannibalism-Mediated Life History Plasticity to Combined Time and Food Stress." *Oikos* 106: 587–97.

Dempster, J. P. 1997. "The Role of Larval Food Resources and Adult Movement in the Population Dynamics of the Orange-Tip Butterfly (*Anthocharis cardamines*)." *Oecologia* 111: 549–56.

Dennis, R. L. 2016. "Adrenergic and Noradrenergic Regulation of Poultry Behavior and Production." *Domestic Animal Endocrinology* 56: S94–S100.

DeVore, J. L., M. R. Crossland, and R. Shine. 2021. "Trade-Offs Affect the Adaptive Value of Plasticity: Stronger Cannibal-Induced Defenses Incur Greater Costs in Toad Larvae." *Ecological Monographs* 91: e01426. https://doi.org/10.1002/ecm.1426.

Diekmann, O., M. Gyllenberg, and J. A. J. Metz. 2003. "Steady-state Analysis of Structured Population Models." *Theoretical Population Biology* 63: 309–38.

Dong, Q., and G. A. Polis. 1992. "The Dynamics of Cannibalistic Populations: A Foraging Perspective." In *Cannibalism: Ecology and Evolution among Diverse Taxa*, edited by M. A. Elgar and B. J. Crespi, 13–37. Oxford: Oxford University Press.

Duarte, C., E. Jaramillo, H. Contreras, and K. Acuña. 2010. "Cannibalism and Food Availability in the Talitrid Amphipod *Orchestoidea tuberculata*." *Journal of Sea Research* 64: 417–21.

El Husseiny, I., H. Elbrense, T. Roeder, and S. El Kholy. 2018. "Hormonal Modulation of Cannibalistic Behaviors in Mosquito (*Culex pipiens*) Larvae." *Journal of Insect Physiology* 109: 144–8.

Elderd, B. D. 2019. "Bottom-Up Trait-Mediated Indirect Effects Decrease Pathogen Transmission in a Tritrophic System." *Ecology* 100: e02551. https://doi.org/10.1002/ecy.2551.

Elgar, M. A., and B. J. Crespi. 1992. "Ecology and Evolution of Cannibalism." In *Cannibalism: Ecology and Evolution among Diverse Taxa*, edited by M. A. Elgar and B. J. Crespi, 1–12. Oxford: Oxford University Press.

Elliott, J. M. 2004. "Contrasting Dynamics in Two Subpopulations of a Leech Metapopulation over 25 Year-Classes in a Small Stream." *Journal of Animal Ecology* 73: 272–82.

Ferris, G., and V. H. W. Rudolf. 2007. "Responses of Larval Dragonflies to Conspecific and Heterospecific Predator Cues." *Ecological Entomology* 32: 283–8.

Fincke, O. M. 1994. "Population Regulation of a Tropical Damselfly in the Larval Stage by Food Limitation, Cannibalism, Intraguild Predation and Habitat Drying." *Oecologia* 100: 118–27.

Fisher, M. E. 1987. "An Age-structured Fish Population Model with Coupled Size and Population Density." *Mathematical Biosciences* 86: 15–34.

Fisher, A. M., S. Le Page, A. Manser, D. R. Lewis, G. I. Holwell, S. Wigby, and T. A. R. Price. 2021. "Relatedness Modulates Density-Dependent Cannibalism Rates in *Drosophila*." *Functional Ecology* 35: 2707–16. https://doi.org/10.1111/1365-2435.13913.

Fox, L. R. 1975. "Cannibalism in Natural Populations." *Annual Review of Ecology and Systematics* 6: 87–106.

Gallucci, F., and E. Ólafsson. 2007. "Cannibalistic Behaviour of Rock-Pool Copepods: An Experimental Approach for Space, Food and Kinship." *Journal of Experimental Marine Biology and Ecology* 342: 325–31.

Gillespie, C. M., R. L. Mumme, and S. A. Wissinger. 2020. "Pond Drying Cues Promote Cannibalism in Larval *Anax junius* Dragonflies." *Freshwater Science* 39: 576–83.

Godfray, H. C. J. 1994. *Parasitoids: Behavioral and Evolutionary Ecology*. Princeton, NJ: Princeton University Press.

Gray, S. M., L. M. Dill, and J. S. McKinnon. 2007. "Cuckoldry Incites Cannibalism: Male Fish Turn to Cannibalism when Perceived Certainty of Paternity Decreases." *American Naturalist* 169: 258–63.

Grosholz, E., G. Ashton, M. Bradley, C. Brown, L. Ceballos-Osuna, A. Chang, C. de Rivera, et al. 2021. "Stage-Specific Overcompensation, the Hydra Effect, and the Failure to Eradicate an Invasive Predator." *Proceedings of the National Academy of Sciences USA* 118: e2003955118.

Hannesson, R. 2018. "Density-Dependent Growth and Cannibalism in Northeast Arctic Cod: Some Implications for Fishing Strategies." *Fisheries Research* 204: 1–7.

Higgins, K., A. Hastings, J. N. Sarvela, and L. W. Botsford. 1997. "Stochastic Dynamics and Deterministic Skeletons: Population Behavior of Dungeness Crab." *Science* 276: 1431–5.

Hoffman, E. A., and D. W. Pfennig. 1999. "Proximate Causes of Cannibalistic Polyphenism in Larval Tiger Salamanders." *Ecology* 80: 1076–80.

Holling, C. S. 1959. "Some Characteristics of Simple Types of Predation and Parasitism." *The Canadian Entomologist* 91: 385–98.

Hopper, K. R., P. H. Crowley, and D. Kielman. 1996. "Density Dependence, Hatching Synchrony, and within-Cohort Cannibalism in Young Dragonfly Larvae." *Ecology* 77: 191–200.

Houghton, R. J., C. Wood, and X. Lambin. 2017. "Size-Mediated, Density-Dependent Cannibalism in the Signal Crayfish *Pacifastacus leniusculus* (Dana, 1852) (Decapoda, Astacidea), an Invasive Crayfish in Britain." *Crustaceana* 90: 417–35.

Huss, M., T. van Kooten, and L. Persson. 2010. "Intra-Cohort Cannibalism and Size Bimodality: A Balance between Hatching Synchrony and Resource Feedbacks." *Oikos* 119: 2000–11.

Ibánez, C. M., and F. Keyl. 2010. "Cannibalism in Cephalopods." *Reviews in Fish Biology and Fisheries* 20: 123–36.

Kats, L. B., J. A. Breeding, and K. M. Hanson. 1994. "Ontogenetic Changes in California Newts (*Taricha torosa*) in Response to Chemical Cues from Conspecific Predators." *Journal of the North American Benthological Society* 13: 321–5.

Kishida, O., and K. Nishimura. 2004. "Bulgy tadpoles: inducible defense morph." *Oecologia* 140: 414–21.

Kishida, O., G. C. Trussell, K. Nishimura, and T. Ohgushi. 2009. "Inducible Defenses in Prey Intensify Predator Cannibalism." *Ecology* 90: 3150–8.

Kishida, O., G. C. Trussell, A. Ohno, S. Kuwano, T. Ikawa, and K. Nishimura. 2011. "Predation Risk Suppresses the Positive Feedback between Size Structure and Cannibalism." *Journal of Animal Ecology* 80: 1278–87.

Kishida, O., A. Tezuka, A. Ikeda, K. Takatsu, and H. Michimae. 2015. "Adaptive Acceleration in Growth and Development of Salamander Hatchlings in Cannibalistic Situations." *Functional Ecology* 29: 469–78.

Klotz, A. M., and J. P. Wright. 2020. "Impacts of Female Body Size on Cannibalism and Juvenile Abundance in a Dominant Arctic Spider." *Journal of Animal Ecology* 89: 1788–98.

Klug, H., K. Lindström, and C. M. St. Mary. 2006. "Parents Benefit from Eating Offspring: Density-Dependent Egg Survivorship Compensates for Filial Cannibalism." *Evolution* 60: 2087–95.

Kohlmeier, C., and W. Ebenhöh. 1995. "The Stabilizing Role of Cannibalism in a Predator-Prey System." *Bulletin of Mathematical Biology* 57: 401–11.

Law, Y.-H., and J. A. Rosenheim. 2011. "Effects of Combining an Intraguild Predator with a Cannibalistic Intermediate Predator on a Species-Level Trophic Cascade." *Ecology* 92: 333–41.

Law, Y.-H., and J. A. Rosenheim. 2013. "Presence of Conspecific Females Motivates Egg Cannibalism Owing to Lower Risk of Filial Cannibalism." *Animal Behaviour* 85: 403–9.

Lehtonen, T. K., and C. Kvarnemo. 2015. "Infections May Select for Filial Cannibalism by Impacting Egg Survival in Interactions with Water Salinity and Egg Density." *Oecologia* 178: 673–83.

Levis, N., A. J. Isdaner, and D. W. Pfennig. 2018. "Morphological Novelty Emerges from Pre-Existing Phenotypic Plasticity." *Nature Ecology & Evolution* 2: 1289–97.

Lukasik, P. 2010. "Trophic Dimorphism in Alternative Male Reproductive Morphs of the Acarid Mite *Sancassania Berlesei*." *Behavioral Ecology* 21: 270–4.

Lund, J. O., S. A. Wissinger, and B. L. Peckarsky. 2016. "Caddisfly Behavioral Responses to Drying Cues in Temporary Ponds: Implications for Effects of Climate Change." *Freshwater Science* 35: 619–30.

Maák, I., E. Tóth, M. Lenda, G. Lorinczi, A. Kiss, O. Juhász, W. Czechowski, and A. Torma. 2020. "Behaviours Indicating Cannibalistic Necrophagy in Ants Are Modulated by the Perception of Pathogen Infection Level." *Scientific Reports* 10: 17906.

Manica, A. 2004. "Parental Fish Change their Cannibalistic Behaviour in Response to the Cost-to-Benefit Ratio of Parental Care." *Animal Behaviour* 67: 1015–21.

Mathis, A. 2003. "Use of Chemical Cues in Detection of Conspecific Predators and Prey by Newts, *Notophthalmus viridescens*." *Chemoecology* 13: 193–7.

McPeek, M. A., and P. H. Crowley. 1987. "The Effects of Density and Relative Size on the Aggressive Behaviour, Movement and Feeding of Damselfly Larvae (Odonata: Coenagrionidae)." *Animal Behaviour* 35: 1051–61.

Medina, J. M., P. M. Shreenidhi, T. J. Larsen, D. C. Queller, and J. E. Strassmann. 2019. "Cooperation and Conflict in the Social Amoeba *Dictyostelium discoideum*." *The International Journal of Developmental Biology* 63: 371–82.

Moksnes, P.-O. 2004. "Self-Regulating Mechanisms in Cannibalistic Populations of Juvenile Shore Crabs *Carcinus maenas*." *Ecology* 85: 1343–54.

Moya-Laraño, J., J. M. Orta-Ocaña, J. A. Barrientos, C. Bach, and D. H. Wise. 2002. "Territoriality in a Cannibalistic Burrowing Wolf Spider." *Ecology* 83: 356–61.

Murray, R. L., S. Tah, J. Koprivnikar, L. Rowe, and S. J. McCauley. 2020. "Exposure to Potentially Cannibalistic Conspecifics Induces an Increased Immune Response." *Ecological Entomology* 45: 355–63.

Nishimura, K. 2018. "An Interaction-Driven Cannibalistic Reaction Norm." *Ecology and Evolution* 8: 2305–19.

Nufio, C. R., and D. R. Papaj. 2001. "Host Marking Behavior in Phytophagous Insects and Parasitoids." *Entomologia Experimentalis et Applicata* 99: 273–93.

Ohlberger, J., Ø. Langangen, I. J. Winfield, and Y. Vindenes. 2020. "The Importance of Variation in Offspring Body Size for Stability in Cannibalistic Populations." *Oikos* 129: 59–69.

Oraze, M. J., and A. A. Grigarick. 1989. "Biological Control of Aster Leafhopper (Homoptera: Cicadellidae) and Midges (Diptera: Chironomidae) by *Pardosa ramulosa* (Araneae: Lycosidae) in California Rice Fields." *Journal of Economic Entomology* 82: 745–9.

Orr, B. K., W. W. Murdoch, and J. R. Bence. 1990. "Population Regulation, Convergence, and Cannibalism in *Notonecta* (Hemiptera)." *Ecology* 71: 68–82.

Orrock, J., B. Connolly, and A. Kitchen. 2017. "Induced Defences in Plants Reduce Herbivory by Increasing Cannibalism." *Nature Ecology & Evolution* 1: 1205–7.

Pan, M.-Z., T.-X. Liu, and C. Nansen. 2018. "Avoidance of Parasitized Host by Female Wasps of *Aphidius gifuensis* (Hymenoptera: Braconidae): The Role of Natal Rearing Effects and Host Availability?" *Insect Sci.* 25: 1035–44.

Patot, S., J. Martinez, R. Allemand, S. Gandon, J. Varaldi, and F. Fleury. 2010. "Prevalence of a Virus Inducing Behavioural Manipulation near Species Range Border." *Molecular Ecology* 19: 2995–3007.

Peacor, S. D., B. T. Barton, D. L. Kimbro, A. Sih, and M. J. Sheriff. 2020. "A Framework and Standardized Terminology to Facilitate the Study of Predation-Risk Effects." *Ecology* 101: e03152.

Persson, L., and J. M. Elliott. 2013. "Population Variation and Individual Maximum Size in Two Leech Populations: Energy Extraction from Cannibalism or Niche Widening?" *Oecologia* 172: 119–27.

Persson, L., A. M. de Roos, and A. Bertolo. 2004. "Predicting Shifts in Dynamics of Cannibalistic Field Populations Using Individual-Based Models." *Proceedings of the Royal Society of London B* 271: 2489–93.

Persson, L., A. M. de Roos, D. Claessen, P. Byström, J. Lövgren, S. Sjögren, R. Svanbäck, E. Wahlström, and E. Westman. 2003. "Gigantic Cannibals Driving a Whole-Lake Trophic Cascade." *Proceedings of the National Academy of Sciences USA* 100: 4035–9.

Petersen, A., K. T. Nielsen, C. B. Christensen, and S. Toft. 2010. "The Advantage of Starving: Success in Cannibalistic Encounters among Wolf Spiders." *Behavioral Ecology* 21: 1112–7.

Pfennig, D. W. 1997. "Kinship and Cannibalism." *Bioscience* 47: 667–75.

Pfennig, D. W. 2000. "Effect of Predator-Prey Phylogenetic Similarity on the Fitness Consequences of Predation: A Trade-off between Nutrition and Disease?" *American Naturalist* 155: 335–45.

Pfennig, D. W., J. P. Collins, and R. E. Ziemba. 1999. "A Test of Alternative Hypotheses for Kin Recognition in Cannibalistic Tiger Salamanders." *Behavioral Ecology* 10: 436–43.

Pizzatto, L., and R. Shine. 2008. "The Behavioral Ecology of Cannibalism in Cane Toads (*Bufo Marinus*)." *Behavioral Ecology and Sociobiology* 63: 123–33.

Pizzatto, L., and R. Shine. 2011. "You Are What you Eat: Parasite Transfer in Cannibalistic Cane Toads." *Herpetologica* 67: 118–23.

Polis, G. A. 1981. "The Evolution and Dynamics of Intraspecific Predation." *Annual Review of Ecology and Systematics* 12: 225–51.

Posada-Florez, F., Z. S. Lamas, D. J. Hawthorne, Y. Chen, J. D. Evans, and E. V. Ryabov. 2021. "Pupal Cannibalism by Worker Honey Bees Contributes to the Spread of Deformed Wing Virus." *Scientific Reports* 11: 8989. https://doi.org/10.1038/s41598-021-88649-y.

Radwan, J. 1995. "Male Morph Determination in Two Species of Acarid Mites." *Heredity* 74: 669–73.

Radwan, J. 2001. "Male Morph Determination in *Rhizoglyphus echinopus* (Acaridae)." *Experimental and Applied Acarology* 25: 143–9.

Richardson, M. L., R. F. Mitchell, P. F. Reagel, and L. M. Hanks. 2010. "Causes and Consequences of Cannibalism in Noncarnivorous Insects." *Annual Review of Entomology* 55: 39–53.

Ricker, W. E. 1954. "Stock and Recruitment." *Journal of the Fisheries Research Board of Canada* 11: 559–623.

Roeder, T. 2020. "The Control of Metabolic Traits by Octopamine and Tyramine in Invertebrates." *Journal of Experimental Biology* 223: jeb194282. https://doi.org/10.1242/jeb.194282.

Roitberg, B. D., and R. J. Prokopy. 1987. "Insects that Mark Plants." *Bioscience* 37: 400–6.

Romano, N., and C. Zeng. 2017. "Cannibalism of Decapod Crustaceans and Implications for their Aquaculture: A Review of its Prevalence, Influencing Factors, and Mitigating Methods." *Reviews in Fisheries Science & Aquaculture* 25: 42–69.

Rosengaus, R. B., and J. F. A. Traniello. 2001. "Disease Susceptibility and the Adaptive Nature of Colony Demography in the Dampwood Termite *Zootermopsis angusticollis*." *Behavioral Ecology and Sociobiology* 50: 546–56.

Rosenheim, J. A. 2001. "Source-Sink Dynamics for a Generalist Insect Predator in a Habitat with Strong Higher-Order Predation." *Ecological Monographs* 71: 93–116.

Rosenheim, J. A. 2005. "Intraguild Predation on *Orius tristicolor* by *Geocoris* Spp. and the Paradox of Irruptive Spider Mite Dynamics in California Cotton." *Biological Control* 32: 172–9.

Rosenheim, J. A., and D. Hongkham. 1996. "Clutch Size in an Obligately Siblicidal Parasitoid Wasp." *Animal Behaviour* 51: 841–52.

Rosenheim, J. A., D. D. Limburg, and R. G. Colfer. 1999. "Impact of Generalist Predators on a Biological Control Agent, *Chrysoperla carnea*: Direct Observations." *Ecological Applications* 9: 409–17.

Rosenheim, J. A., N. A. Booster, M. Culshaw-Maurer, T. G. Mueller, R. L. Kuffel, Y. H. Law, P. B. Goodell, et al. 2019. "Disease, Contagious Cannibalism, and Associated Population Crash in an Omnivorous Bug, *Geocoris pallens*." *Oecologia* 190: 69–83.

Roth, G., and S. J. Schreiber. 2014. "Pushed beyond the Brink: Allee Effects, Environmental Stochasticity, and Extinction." *Journal of Biological Dynamics* 8: 187–205.

Ruang-Rit, K., and Y. Park. 2018. "Endocrine System in Supernumerary Molting of the Flour Beetle, *Tribolium Freemani*, under Crowded Conditions." *Insect Biochemistry and Molecular Biology* 101: 76–84.

Rudolf, V. H. W. 2007. "Consequences of Stage-Structured Predators: Cannibalism, Behavioral Effects, and Trophic Cascades." *Ecology* 88: 2991–3003.

Rudolf, V. H. W., and J. Antonovics. 2007. "Disease Transmission by Cannibalism: Rare Event or Common Occurrence?" *Proceedings of the Royal Society B* 274: 1205–10.

Sadeh, A., M. Mangel, and L. Blaustein. 2009. "Context-Dependent Reproductive Habitat Selection: The Interactive Roles of Structural Complexity and Cannibalistic Conspecifics." *Ecology Letters* 12: 1158–64.

Scharf, I. 2016. "The Multifaceted Effects of Starvation on Arthropod Behaviour." *Animal Behaviour* 119: 37–48.

Schausberger, P. 2003. "Cannibalism among Phytoseiid Mites: A Review." *Experimental and Applied Acarology* 29: 173–91.

Schmid-Hempel, P. 2011. *Evolutionary Parasitology: The Integrated Study of Infections, Immunology, Ecology, and Genetics*. Oxford: Oxford University Press.

Schreiber, S. J., M. Benaïm, and K. A. S. Atchadé. 2011. "Persistence in Fluctuating Environments." *Journal of Mathematical Biology* 62: 655–83.

Schreiber, S., and J. Rosenheim. 2022. "R Code for 'Pathways to the Density-Dependent Expression of Cannibalism, and Consequences for Regulated Population Dynamics." Figshare. https://doi.org/10.6084/m9.figshare.20024327.v1.

Simpson, S. J., G. A. Sword, P. D. Lorch, and I. Couzin. 2006. "Cannibal Crickets on a Forced March for Protein and Salt." *Proceedings of the National Academy of Sciences USA* 103: 4152–6.

Siva-Jothy, J. A., K. M. Monteith, and P. F. Vale. 2018. "Navigating Infection Risk during Oviposition and Cannibalistic Foraging in a Holometabolous Insect." *Behavioral Ecology* 29: 1426–35.

Smith, C., and P. Reay. 1991. "Cannibalism in Teleost Fish." *Reviews in Fish Biology and Fisheries* 1: 41–64.

Smith-Somerville, H. E., J. K. Hardman, R. Timkovich, W. J. Ray, K. E. Rose, P. E. Ryals, S. H. Gibbons, and H. E. Buhse, Jr. 2000. "A Complex of Iron and Nucleic Acid Catabolites Is a Signal that Triggers Differentiation in a Freshwater Protozoan." *Proceedings of the National Academy of Sciences USA* 97: 7325–30.

Stewart, K. A., T. P. G. Van den Beuken, F. T. Rhebergen, J. A. Deere, and I. M. Smallegange. 2018. "Evidence for a Third Male Type in a Male-Dimorphic Model Species." *Ecology* 99: 1685–7.

Strauss, T., D. Kulkarni, T. G. Preuss, and M. Hammers-Wirtz. 2016. "The Secret Lives of Cannibals: Modelling Density-Dependent Processes that Regulate Population Dynamics in *Chaoborus crystallinus*." *Ecological Modelling* 321: 84–97.

Tena, A., A. Kapranas, F. Garcia-Marí, and R. F. Luck. 2009. "Larval Cannibalism during the Late Developmental Stages of a Facultatively Gregarious Encyrtid Endoparasitoid." *Ecological Entomology* 34: 669–76.

Tomkins, J. L., N. R. Lebas, J. Unrug, and J. Radwan. 2004. "Testing the Status-Dependent ESS Model: Population Variation in Fighter Expression in the Mite *Sancassania Berlesei*." *Journal of Evolutionary Biology* 17: 1377–88.

Toscano, B. J., V. Hin, and V. H. W. Rudolf. 2017. "Cannibalism and Intraguild Predation Community Dynamics: Coexistence, Competitive Exclusion, and the Loss of Alternative Stable States." *American Naturalist* 190: 617–30.

Turchin, P. 1995. "Population Regulation: Old Arguments and a New Synthesis." In *Population Dynamics: New Approaches and Synthesis*, edited by N. Cappuccino and P. W. Price, 19–40. Cambridge, MA: Academic Press.

Van Allen, B. G., F. P. Dillemuth, A. J. Flick, M. J. Faldyn, D. R. Clark, V. H. W. Rudolf, and B. D. Elderd. 2017. "Cannibalism and Infectious Disease: Friends or Foes?" *American Naturalist* 190: 299–312.

Van Buskirk, J. 1989. "Density-Dependent Cannibalism in Larval Dragonflies." *Ecology* 70: 1442–9.

Van den Beuken, T. P. G., L. W. Stockwell, and I. M. Smallegange. 2019. "Et Tu, Brother? Kinship and Increased Nutrition Lower the Incidence of Cannibalism in Male Bulb Mites." *Animal Behaviour* 152: 45–52.

van den Bosch, F., A. M. de Roos, and W. Gabriel. 1988. "Cannibalism as a Life Boat Mechanism." *Journal of Mathematical Biology* 26: 619–33.

Varaldi, J., and D. Lepetit. 2018. "Deciphering the Behaviour Manipulation Imposed by a Virus on its Parasitoid Host: Insights from a Dual Transcriptomic Approach." *Parasitology* 145: 1979–89.

Varaldi, J., P. Fouillet, M. Ravallec, M. López-Ferber, Z. M. Boulétreau, and F. Fleury. 2003. "Infectious Behavior in a Parasitoid." *Science* 302: 1930.

Via, S. 1999. "Cannibalism Facilitates the Use of a Novel Environment in the Flour Beetle, *Tribolium castaneum*." *Heredity* 82: 267–75.

Vijendravarma, R. K., S. Narasimha, and T. J. Kawecki. 2013. "Predatory Cannibalism in *Drosophila melanogaster* Larvae." *Nature Communications* 4: 1789. https://doi.org/10.1038/ncomms2744.

Waddell, D. R. 1992. "Cannibalism in Lower Eukaryotes." In *Cannibalism: Ecology and Evolution among Diverse Taxa*, edited by M. A. Elgar and B. J. Crespi, 85–101. Oxford: Oxford University Press.

Wagner, J. D., and D. H. Wise. 1996. "Cannibalism Regulates Densities of Young Wolf Spiders: Evidence from Field and Laboratory Experiments." *Ecology* 77: 639–52.

Walde, S. J., J. P. Nyrop, and J. M. Hardman. 1992. "Dynamics of *Panonychus ulmi* and *Typhlodromus pyri*: Factors Contributing to Persistence." *Experimental & Applied Acarology* 14: 261–91.

Wildy, E. L., D. P. Chivers, J. M. Kiesecker, and A. R. Blaustein. 2001. "The Effects of Food Level and Conspecific Density on Biting and Cannibalism in Larval Long-Toed Salamanders, *Ambystoma macrodactylum*." *Oecologia* 128: 202–9.

Williams, T., and O. Hernández. 2006. "Costs of Cannibalism in the Presence of an Iridovirus Pathogen of *Spodoptera frugiperda*." *Ecological Entomology* 31: 106–13.

Wise, D. H. 2006. "Cannibalism, Food Limitation, Intraspecific Competition, and the Regulation of Spider Populations." *Annual Review of Entomology* 51: 441–65.

Wissinger, S. A., H. H. Whiteman, M. Denoël, M. L. Mumford, and C. B. Aubee. 2010. "Consumptive and Nonconsumptive Effects of Cannibalism in Fluctuating Age-Structured Populations." *Ecology* 91: 549–59.

Woelber, B. K., C. L. Hall, and D. R. Howard. 2018. "Environmental Cues Influence Parental Brood Structure Decisions in the Burying Beetle *Nicrophorus marginatus*." *Journal of Ethology* 36: 55–64.

Wong, J. W. Y., and M. Kölliker. 2013. "The More the Merrier? Condition-Dependent Brood Mixing in Earwigs." *Animal Behaviour* 86: 845–50.

Yakovlev, I. K. 2018. "Effects of Octopamine on Aggressive Behavior in Red Wood Ants." *Neuroscience and Behavioral Physiology* 48: 279–88.

Yan, G., L. Stevens, and J. J. Schall. 1994. "Behavioral Changes in *Tribolium* Beetles Infected with a Tapeworm: Variation in Effects between Beetle Species and among Genetic Strains." *American Naturalist* 143: 830–47.

Zhou, C., Y. Rao, and Y. Rao. 2008. "A Subset of Octopaminergic Neurons Are Important for *Drosophila* Aggression." *Nature Neuroscience* 11: 1059–67.

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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