A dynamic threshold model for terminal investment

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Abstract

Although reproductive strategies can be influenced by a variety of intrinsic and extrinsic factors, life history theory provides a rigorous framework for explaining variation in reproductive effort. The *terminal investment hypothesis* proposes that a decreased expectation of future reproduction (as might arise from a mortality threat) should precipitate increased investment in current reproduction. Terminal investment has been widely studied, and a variety of intrinsic and extrinsic cues that elicit such a response have been identified across an array of taxa. Although terminal investment is often treated as a static strategy, the level at which a cue of decreased future reproduction is sufficient to trigger increased current reproductive effort (i.e., the terminal investment threshold) may depend on context, including the internal state of the organism or its current external environment, independent of the cue that triggers a shift in reproductive investment. Here, we review empirical studies that address the terminal investment hypothesis, exploring both the intrinsic and extrinsic factors that mediate its expression. Based on these studies, we propose a novel framework within which to view the strategy of terminal investment, incorporating factors that influence an individual's residual reproductive value beyond a terminal investment trigger – the *dynamic terminal investment threshold.*

Keywords: Residual reproductive value, life history evolution, condition-dependent reproductive investment, fecundity compensation, phenotypic plasticity

Acknowledgments: National Science Foundation IOS 16-54028 (SKS and BMS), Illinois State University Summer Faculty Fellowship and Faculty Research Award (SKS), National Institutes of Health 2R15HD076308-02A1 (SKS and CF Thompson)

Introduction

Investment in life history traits (i.e., growth, survival, and reproduction) can be constrained by limited resource availability (Calow 1979; Stearns 1992; Zera and Harshman 2001; Roff and Fairbairn 2007), genetic covariance and antagonistic pleiotropy between traits (Stearns 1989), or changes in the direction or strength of selection at different stages of life history (Schluter et al. 1991). These constraints may drive trade-offs (i.e. negative phenotypic or genetic associations) both between life history traits and within traits over time (Clutton-Brock et al. 1982; Reznick 1985; van Noordwijk and de Jong 1986; Stearns 1989; Roff 1992; Stearns 1992), such that investment cannot be simultaneously optimized for all traits at all times throughout an individual's lifetime. Selection acts within the bounds of these trade-offs to optimize investment strategies that maximize fitness within a particular context.

An especially salient trade-off is between reproductive effort and somatic defense (i.e., immunity) (Reznick 1985; Lochmiller and Deerenberg 2000; Zera and Harshman 2001; Zuk and Stoehr 2002; Lawniczak et al. 2007; Durso and French 2017). Investments in these traits can enhance fitness through their effects on reproduction and survival, but such investments inevitably entail evolutionary, maintenance, and deployment costs, which leads to an allocation trade-off between them (Schwenke et al. 2016). Evolutionary trade-offs arise from linkage or pleiotropy of the genes involved, and results in negative genetic covariance between traits. Negative genetic correlations have been demonstrated between reproductive effort and resistance to infection (e.g., Cotter et al. 2004; Simmons and Roberts 2005; Graham et al. 2010). Experimental evolution, with selection for either increased reproductive effort or resistance to infection, has resulted in coinciding decreases in resistance to infection and reproductive effort, respectively (e.g., Boots and Begon 1993; Zwaan et al. 1995; Luong and Polak 2007). Additionally, trade-offs can occur due to the immediate nutritional and metabolic costs of maintaining and utilizing these traits and their physiological linkage (Sheldon and Verhulst 1996; Lochmiller and Deerenberg 2000; Sadd and Schmid-Hempel 2009; Schwenke et

al. 2016); allocating resources towards defense against infection necessarily diverts resources away from reproductive effort and *vice versa*.

Given the evidence for trade-offs between reproduction and defense, the conventional view has been that individuals faced with a threat to self-integrity and longevity should change their life history investment pattern, shifting investment away from reproduction and towards defense and repair, thus ensuring their continued survival (Norris et al. 1994; Gustafsson et al. 1994; Svensson et al. 1998; Adamo et al. 2001; Jacot et al. 2004; Ahtiainen et al. 2005; Stahlschmidt et al. 2013). However, an alternative strategy is for individuals to increase investment in current reproduction when cued to a decreased likelihood of survival, at a cost of decreased somatic maintenance and future reproduction. Although this might at first seem counter-intuitive, evolutionary theory predicts that when an individual's expectation of future offspring (*residual reproductive value*) decreases upon its perception of increased mortality risk, investment in current reproduction should increase (Williams 1966). Within the context of life history theory, this has been termed the *terminal investment hypothesis* (Clutton-Brock 1984), with some authors also referring to the strategy as *fecundity compensation* (Parker et al. 2011). Terminal investment encompasses a broader range of potential changes in reproductive effort, and thus, we adopt this more general term in subsequent discussion.

The terminal investment hypothesis has received considerable attention since it was first proposed, but in a number of cases, the evidence is equivocal. At least part of this ambiguity may be due the framework within which the strategy of terminal investment has been addressed. The goal of this review is to: 1) synthesize the findings from previous empirical studies exploring the terminal investment hypothesis, and 2) propose an extended conceptual framework for a more nuanced interpretation of these findings. We propose that the strategy of terminal investment will exhibit a threshold in its expression, with this threshold being dynamic and dependent on an organism's internal state and extrinsic factors that together influence its expectation for future progeny (i.e., residual reproductive value).

The terminal investment hypothesis in review

Trade-offs concerning investment in life history traits, including reproduction and defense, are likely contingent on an individual's residual reproductive value. For example, if the chances of producing future offspring are high, individuals should invest in their current progeny at sub-maximal levels to optimize the trade-off between current and future reproduction. Conversely, if the chances of producing future offspring are low, individuals should increase investment in their current progeny (Williams 1966; Hirshfield and Tinkle 1975; Clutton-Brock 1984). Therefore, current reproductive effort and residual reproductive value are expected to exhibit negative covariance (Williams 1966; Hirshfield and Tinkle 1975; Pianka and Parker 1975). When a threat to future reproduction is raised consistently for all individuals globally, fixed strategies may evolve in populations, such as semelparity instead of iteroparity (Young 1990). However, in an environment where individuals face a spatial and temporal mosaic of varied levels of a threat to future reproduction, plastic strategies, such as terminal investment, will be advantageous.

The terminal investment hypothesis proposes that individuals facing a significant survival threat, and hence decreased residual reproductive value as a consequence of a truncated lifespan, should divert time, energy, and resources away from other life history traits (e.g., growth, maintenance or defense, and future reproduction) and towards current reproduction as a way of maximizing lifetime reproductive output (Williams 1966). The trade-off between current and future reproduction dictates that such an acceleration of reproductive effort would be sub-optimal within the context of a normal, undisrupted reproductive lifespan. Empirical studies have found support for terminal investment in numerous species in response to a real or simulated survival threat, with increases detected in various components of reproductive effort, including attractiveness of plastic epigamic traits in males, offspring production, and parental care (Tables 1-3).

Integral to the terminal investment hypothesis are the cues of reduced residual reproductive value, which can be considered terminal investment *trigger*s that an individual must be able to perceive to adaptively alter their reproductive investment. The type, timing, intensity, and predictability of these triggers are likely paramount to an individual's ability to implement a terminal investment strategy. Both intrinsic factors (e.g. age and nutrition-dependent condition) and extrinsic factors (e.g. contemporary food shortage, perceived predation risk, and infectious disease) can affect mortality rate, and consequently, residual reproductive value.

I. Intrinsic State

Both the probability of survival and the quantity and quality of offspring should be determined, at least in part, by an individual's internal state, potentially in interaction with current environmental conditions. In many organisms, likelihood of survival decreases the older an individual becomes (Type I survivorship), as does residual reproductive value (Pianka and Parker 1975). In addition, it is not surprising that the condition of an individual, as influenced by prior resource intake, will often affect reproductive investment (e.g., Wagner and Hoback 1999; Ohlsson et al. 2002; Warner et al. 2007; Fricke et al. 2008). This should be particularly pertinent in the case of capital breeders (Varpe et al. 2009), individuals that acquire their resources in advance, and then rely on stored energy reserves during reproduction (Drent and Daan 1980; Jönsson 1997). This dependency of reproduction on intrinsic state suggests that altered reproductive effort based on a perception of internal state could represent a form of terminal investment. Focusing primarily on age and nutrition-dependent condition, we highlight evidence from studies that explore alterations in reproductive effort brought about by intrinsic influences on residual reproductive value.

Age as an intrinsic cue for terminal investment

Age-related reproductive investment has been studied extensively (e.g., Gadgil and Bossert 1970; Hirshfield and Tinkle 1975; Pianka and Parker 1975; Pugesek 1983). Generally, reproductive effort is predicted to increase toward the end of the lifespan in species in which residual reproductive value decreases with age. This increase is hypothesized to arise from: i) decreased survival of low-performing reproducers, leading to overrepresentation of high-performing reproducers as cohorts age (Curio 1983; Forslund and Pärt 1995; Mauck et al. 2004); ii) age-related improvements in reproductive performance, as often accrues with increased breeding experience (Curio 1983); and iii) optimization of reproductive effort as individuals age, as predicted by life history theory (Williams 1966; Stearns 1992; Forslund and Pärt 1995). The last of these invokes a cost of reproduction. Based on the assumption that reproduction is costly (e.g., by decreasing future reproduction or survival) (Calow 1979; Reznick 1985; Alonso-Alvarez et al. 2004; Harshman and Zera 2007), this hypothesis predicts that young individuals, of high reproductive value or high future reproductive potential (Fisher 1930), should allocate less to current reproduction to ensure future reproductive opportunities, whereas older individuals, of low reproductive value, should allocate more to current reproduction. Within this framework of age-dependent terminal investment (Clutton-Brock 1984), selection favors older individuals that assume greater costs of reproduction, because future opportunities may be unavailable (Williams 1966). Overall, empirical evidence for the age-related reproductive patterns that are predicted by the cost of reproduction hypothesis is mixed (Table 1), but support for age-dependent terminal investment has been found in both sexes in various mammals, reptiles, and insects (Table 1; supplementary table S1). For example, queens of the ant *Cardiocondyla obscurior* have been shown to increase their rate of egg production with age, even months after mating (Heinze and Schrempf 2012).

An important obstacle to assessing age-dependent terminal investment is that it is difficult to disentangle a strategy of terminal investment from either of the other aforementioned hypotheses (i.e., differential survival of low- or high-performing reproducers or age-related

improvements in reproductive performance). The inability to perform empirical manipulations on fixed intrinsic parameters, such as age, means that positive relationships with reproductive effort cannot be conclusively attributed to an adaptive terminal investment strategy. For instance, while much of the early evidence for the terminal investment hypothesis comes from assessments of reproductive effort of large ungulates (e.g., Clutton-Brock et al. 1982; Maher and Byers 1987; Ericsson et al. 2001), several parameters that correlate with reproductive success (e.g., social dominance and experience) often increase with age (e.g., Coltman et al. 2002). On the other hand, if a reduction in reproductive success is observed with increasing age, this could simply be a consequence of somatic deterioration (i.e. senescence) rather than adaptive changes in reproductive effort (e.g., Loison et al. 1999; Weladji et al. 2002). Consequently, it is difficult to determine if changes in reproductive success as an individual ages are a result of increased reproductive effort consistent with a terminal investment strategy, or due to some other age-related behavioral or physiological manifestation (Pugesek 1981; Clutton-Brock et al. 1982). Tarwater and Arcese (2017) recently argued that future studies should consider both chronological age and time to death (independent of age) in assessments of age-related changes in reproductive effort. By separating these two factors, they observed both senescence (among old females) and terminal investment (among young females only) in song sparrows (*Melospiza melodia*). Interestingly, reproductive effort was highest for females in their last year of life only if they were 1 or 2 years old, even though this species can live beyond 5 years of age (Tarwater and Arcese 2017).

Nutrition-dependent condition as a cue for changes in reproductive effort

Variation in food availability is an important aspect of environmental heterogeneity. Allocation of limited resources lies at the heart of life history trade-offs (Stearns 1992; Roff 2002), with empirical manipulation of quantity and quality of nutrition being shown to influence trade-offs across an array of taxa (Hill and Kaplan 1999; Brown and Shine 2002; Lardner and

Loman 2003; Hunt et al. 2004; Kolluru and Grether 2005; Karell et al. 2007; Cotter et al. 2011). As energetically costly reproductive traits are constrained by the availability of adequate nutrition, most studies demonstrate that food limitation leads to decreased reproductive effort (Table 2; supplementary table S2). For example, cockroaches (*Nauphoeta cinerea*) reared on a low-quality diet regimen as juveniles exhibited a fixed phenotype as adults (i.e., one that could not be recovered with a change in diet), in which reproductive lifespan was significantly shorter than adults fed a high-quality diet as juveniles (Barrett et al. 2009). There is also evidence to suggest that low nutrition-dependent condition can also lead to terminal investment. In katydids (Simmons and Gwynne 1991), tree crickets (Brown 1997), and humped-winged grigs (Judge et al. 2011), all insect species in which males provide females with nuptial food gifts at mating, females held on a low-quality diet were more quick to remate than those held on a high-quality diet. Although the increased mating activity of females could represent a kind of "foraging effort" to offset nutrient limitation (direct benefit), it is equally consistent with a strategy of terminal investment due to the numerous genetic (indirect) benefits of polyandry (e.g., Fedorka and Mousseau 2002; Ivy and Sakaluk 2005). Additional evidence suggests that diet may also influence an individual's propensity to terminally invest in response to other extrinsic cues of reduced residual reproductive value (see "Interactions indicative of a dynamic terminal investment threshold" below).

II. Extrinsic Factors

While much initial theoretical and empirical work focused on the influence of intrinsic factors on residual reproductive value and, by extension, the likelihood of terminal investment, there has subsequently been a shift in focus to the extrinsic factors that elicit terminal investment. Residual reproductive value should be determined, in part, by the external environment, with the potential for perceived changes in extrinsic cues leading to the adaptive alteration of reproductive effort, including terminal investment. Extrinsic factors can positively or

234 negatively affect residual reproductive value, and do so either through a direct influence on
235 reproduction (e.g. castration, mate availability) or indirectly through an altered probability of
236 survival. Extrinsic factors that have been examined in this latter respect include variation in
237 predation risk (Korpimaki et al. 1994), and physical injury, including post-mating damage
238 (Morrow et al. 2003). However, most of the attention in this area has centered on exposure to
239 and infection by parasites and pathogens (Table 2).
240
241 *Parasite and pathogen infection as a trigger of life history changes*
242 The realization that parasites and pathogens could play major roles in the evolutionary
243 ecology of organisms (Hamilton 1980; Hamilton and Zuk 1982) precipitated their inclusion as
244 important drivers of life history strategies. At an ecological level, parasite infection is presumed
245 to have negative impacts on reproductive output and survival, with these fitness-related
246 consequences culminating in selection on hosts to either prevent or curtail infection, or to
247 mitigate any consequences of infection. For example, hosts can reduce the loss of fitness from
248 infection by upregulating their immune system. While the benefits of increased immune
249 investment in response to infection are obvious, the costs of upregulation often result in
250 restriction of resources that could be invested in reproduction. As highlighted more broadly
251 earlier, it is commonly predicted that infected individuals should exhibit decreased reproductive
252 effort due to a reallocation of resources towards defense (i.e. immunity). However, increasing
253 evidence suggests that some infected organisms instead increase their investment in
254 reproduction. While seemingly counter-intuitive, these results can be explained within a life
255 history framework via the terminal investment hypothesis.
256 Minchella and Loverde (1981) were among the first to discover parasite-induced
257 increases in reproductive effort in hosts, finding that snails (*Biomphalaria glabrata*) infected with
258 castrating trematodes (*Schistosoma mansoni*) exhibit transient increases in fecundity prior to
259 complete cessation of egg production due to the parasite-induced castration. This transient

increase resulted in fecundity compensation (or, terminal investment), thus decreasing the negative effects of a shortened reproductive lifespan associated with parasite infection in this system. Subsequently, many studies have explored infection-related changes in reproductive effort following both natural and artificial inoculation (Table 2; supplementary table S2).

Numerous studies report increases in reproductive effort following infection (Table 2), which is congruent with the predictions of the terminal investment hypothesis, yet overall a variety of outcomes have been found, sometimes even within the same study. For example, female deer mice (*Peromyscus maniculatus*) parasitized with the trematode parasite, *Schistosomatium douthitti* increase the expression of some reproductive traits (time to first reproduction and total litter mass), but not others (the time between consecutive litters, probability of litter cannibalism, litter size, litter sex ratio) (Schwanz 2008b; supplementary table S2). These results are intriguing with regard to the specifics of life history investment, but they make interpretation of overall life history strategies problematic, and unraveling contributions of individual traits would require multi-generational fitness measures. However, the trait-specific alterations of investment do provide some insight into potential constraints on the plasticity of reproductive traits following infection. An understanding of trait plasticity, in addition to the context within which a cue of reduced residual reproductive value is perceived, may help clarify equivocal findings (see “Dynamic terminal investment threshold” below).

Although most studies focus on responses in host traits, live pathogens and parasites used in the aforementioned studies cannot be regarded as passive bystanders. Shifts in host life history may be beneficial for parasite fitness, and therefore host responses may be a consequence of parasite manipulation (Minchella 1985; Sheldon and Verhulst 1996). Thus, it is important when interpreting findings to account for the fact that life history consequences of infection may be the result of selection on hosts, selection on parasites, or even non-adaptive side effects (Hurd 2001). Interestingly, however, several studies have found that individuals exposed to parasites (both with and without a subsequent infection), shift investment towards

current reproduction, consistent with predictions from the terminal investment hypothesis (e.g., Minchella 1985).

Non-pathogenic immune stimulation to test for infection-associated host life history shifts

To disentangle strategic shifts in life history by hosts from shifts due to parasite manipulation, many studies have employed measures to elicit an immune response in focal individuals without the confounding effects of pathogen proliferation and manipulation. The triggering of an immune response acts to simulate an infection that may signal reduced residual reproductive value to the host. Studies have utilized non-pathogenic immune-elicitors such as lipopolysaccharides (LPS), antigens, vaccines, sterile implants, and inactivated pathogens to act as a cue of pathogen or parasite infection, and then subsequently measured responses in various aspects of host reproductive effort (Table 2; supplementary table S2). Using this approach, any responses observed can clearly be attributed to changes in investment by the focal individual, rather than the result of parasite manipulation or the pathology of a real infection. Although several studies have documented outcomes that are consistent with a trade-off between investment in immune defense and reproduction, many others have documented increases in various components of reproductive effort in individuals following an experimental immune challenge, which is consistent with the predictions of the terminal investment hypothesis (Table 2; supplementary table S2). For example, male mealworm beetles (*Tenebrio molitor*) implanted with a nylon filament exhibit increased attractiveness of their sex pheromones, which are important for acquiring mates (Sadd et al. 2006).

Interestingly, some studies have investigated the influence of multiple infection-associated cues, which allows for a comparative analysis of how different stimuli are perceived as cues of reduced residual reproductive value, or that lead to differential responses. For example, Adamo (1999) assessed the effects of infection on oviposition in female crickets (*Acheta domesticus*), incorporating both live infections of the gram-negative bacteria *Serratia*

marcescens and the larvae of a parasitoid tachinid fly, *Ormia ochracea*, and inactive non-pathogenic immune-eliciting substitutes for each of the infections. Female crickets increased the number of eggs laid in response to both live *S. marcescens* and non-pathogenic LPS derived from *S. marcescens*. However, females did not alter their oviposition schedule when challenged with either live *O. ochracea* or its non-pathogenic substitute, Sephadex beads. These results suggest that changes in life history strategies, including those involving terminal investment, may be dependent on specific infection scenarios. Differential responses may be adaptive and related to how different infections change residual reproductive value, or may instead be subject to physiological constraints, such that only infections that trigger certain immune pathways act as terminal investment triggers.

With respect to the use of simulated infections to assess life history responses, an important methodological consideration is the incorporation of appropriate controls. Although sham controls are critical for identifying exact causal effects in any experiment, the inclusion of unmanipulated controls may be equally important, depending on the protocol of simulated infection used. However, studies often do not incorporate both unmanipulated and sham control treatments (supplementary table S2). The importance of both controls can be seen in the illustrative example of using an injection to deliver a non-pathogenic elicitor into the haemocoel of an insect, and subsequently measuring reproductive investment. A sham control injection of the vehicle alone is necessary to attribute any changes to the introduced elicitor. However, it is well known that cuticle wounding in insects leads to an immune response (Brey et al. 1993; Wigby et al. 2008), and thus, it is plausible that a sham control alone could result in an observable shift in reproductive effort (for example, see Altincicek et al. 2008). In this case, absence of an unmanipulated control that provides a baseline of reproductive effort could result in the conclusion that a particular organism does not exhibit terminal investment, when, in fact, it does.

III. The terminal investment threshold

The discussion above suggests that a strategy of terminal investment may be dependent on the form and intensity of the cue imposed. Historically, terminal investment has been approached as a static strategy, in which investigators have sought to determine if terminal investment does, or does not, occur in response to a specific cue believed to signal decreased residual reproductive value (i.e., a terminal investment trigger). Often the intensity of cues utilized is purposefully high, in an attempt to ensure that any potential response is triggered. Interestingly, more recent studies have incorporated a gradation in the intensity of these cues, which has shown that when individuals terminally invest, they often do so only at high cue intensities. For example, Hendry et al. (2016) found that asexual reproduction in pea aphids (*Acyrthosiphon pisum*) is affected by infection by the bacterium, *Pseudomonas syringae*, in a dose-dependent manner. Aphids exposed to low doses exhibited reduced reproduction relative to controls, presumably investing in defense against the pathogen (cost of immunity hypothesis), whereas those exposed to higher concentrations of bacteria exhibited the highest levels of reproduction (terminal investment). In this instance, individuals exposed to the highest dose of *P. syringae*, however, had the lowest reproduction, which is likely a consequence of the high live infection load leading to pathogenesis as this dose leads to high aphid mortality (Hendry et al. 2016). These results suggest that the intensity of the terminal investment trigger can be viewed as a threshold, one that reflects the relationship between the trigger and an individual's perceived residual reproductive value, which we refer to as the *terminal investment threshold* (Figure 1). Using the example of a pathogen infection, it may pay to invest in mitigation or clearance of the infection at low levels of infection, thus leading to a decrease in reproductive effort as a result of the cost of increased immunity. As the level of infection increases, the threat to longevity and future reproduction, both of which contribute to residual reproductive value, also increases. When the cue intensity reaches a tipping point at which investment in resistance against the infection is futile, infected individuals are predicted to fully

switch to a terminal investment strategy. The concept of a terminal investment threshold allows for a more quantitative assessment of terminal investment under a spectrum of cues that signal reduced residual reproductive value. Although such a threshold is illustrated here with respect to pathogen infection, it is relevant to a diversity of other cues associated with future reproductive potential. The exact threshold is presumed to have been optimized by selection, and is expected to differ between organisms and among the different cues that signal reduced residual reproductive value, thus potentially contributing to the equivocal findings across studies investigating terminal investment.

The dynamic terminal investment threshold

In addition to species-specific evolutionary or physiological constraints on life history plasticity, failure to uncover terminal investment in particular organisms could occur because the terminal investment threshold has not been exceeded. Furthermore, in the framework of a terminal investment threshold, it is highly likely that the tipping point is not static, but rather context dependent, leading to a *dynamic terminal investment threshold.*

It has been largely overlooked that the strategy of terminal investment, and the terminal investment threshold, may depend on the internal state of the organism or external environmental factors that are independent of the focal cue of reduced residual reproductive value (e.g. infection). Specifically, any extrinsic or intrinsic factor that influences baseline residual reproductive value beyond the threat posed by a potential terminal investment trigger may alter the severity of residual reproductive value reduction cued by a particular threat level and determine whether an individual adopts a terminal investment strategy (Figure 1). Indeed, many life history models have explored dynamic aspects of resource allocation (Perrin and Sibly 1993; Noonburg et al. 1998; Heino and Kaitala 1999), suggesting that trade-offs, and corresponding investment strategies, need not be static (Zera and Harshman 2001). Here, we

discuss evidence from previous studies in support of our proposed framework of a dynamic terminal investment threshold, and describe the specific factors that may influence it.

Interactions indicative of a dynamic terminal investment threshold

The relationship between individual age and residual reproductive value, with prospects of future reproductive opportunities diminishing as individuals move closer to the end of their lifespan (Williams 1966; Pianka and Parker 1975), makes age a highly relevant intrinsic factor upon which a dynamic terminal investment threshold to another threat cue might be contingent. More simply, age may determine the intensity of a second trigger that is required to elicit terminal investment. Due to the difference in residual reproductive value between young and old individuals, the intensity of a terminal investment trigger should be lower for older individuals (i.e., a lower terminal investment threshold than for younger individuals). Indeed, evidence of an age-dependent terminal investment threshold, as demonstrated by statistically significant interaction effects of age and treatment on reproductive effort, has been shown in previous studies (Table 3; supplementary table S3), even if these have not been explicitly situated within the framework of a dynamic terminal investment threshold. For example, Velando et al. (2006) demonstrated that the reproductive success of male blue-footed boobies (*Sula nebouxii*) declines with age. However, immune-challenged older males exhibited a 98% increase in reproductive output compared with old control males, whereas the reproductive success of immune-challenged younger males decreased relative to young control males. This significant interaction between age and another cue of reduced residual reproductive value (immune challenge) on the outcome of reproductive effort is indicative of a dynamic threshold in the propensity to terminally invest. Other studies have found similar significant interactions with age in birds, fish, and insects (Table 3). In some cases, extrinsic threat cues may not interact with age. For example, female burying beetles (*Nicrophorus vespilloides*) treated with inactivated bacteria (*Micrococcus lysodeikticus*) produced heavier broods compared with control females,

but this effect was observed regardless of female age (Cotter et al. 2010). However, further work using a spectrum of infection cues, including lower doses, would be required to determine whether the apparent absence of age-dependent terminal investment in this species is real, or is due instead to a relevant, but variable, infection cue threshold being exceeded in all age groups.

While age likely represents a widespread intrinsic factor underlying a dynamic terminal investment threshold, numerous other factors are likely to fine-tune the thresholds for focal triggers. For example, genetic differences in life histories and reproductive effort may also play an important role in determining an individual's propensity to terminally invest. Although this has not yet been tested explicitly, several studies have incorporated different clonal lines in the examination of reproductive effort following experimental manipulation of extrinsic mortality cues (e.g., the concentration of alarm cues) influencing residual reproductive value in both pea aphids (*Acyrthosiphon pisum*) and water fleas (*Daphnia magna*) (Table 3). These studies have revealed considerable variation in the response to these cues between lines and across treatments, demonstrating that a genotype-by-environment interaction may play a particularly important role in determining the terminal investment threshold. Superimposed on this genetic variation, the presence or absence of symbionts may also influence the terminal investment threshold, as these can modify the host's life history phenotype by causing numerous physiological, morphological, and even behavioral changes (e.g., Leonardo and Mondor 2006). Symbionts in aphids have been shown to significantly influence how hosts alter reproductive investment following a decrease in residual reproductive value (Barribeau et al. 2010). Interactions involving numerous other individual-level traits (e.g., body size, mating history, confidence of paternity) abound (Table 3; supplementary table S3).

In addition to intrinsic factors such as age and genotype, environmental factors that influence residual reproductive value may act as supplementary determinants of the propensity to terminally invest following exposure to a focal terminal investment trigger (Table 3). For example, when in isolation, captive zebra finches (*Taeniopygia guttata*) injected with LPS

engaged in classic ‘sickness behavior’ (e.g., lethargy, loss of appetite) relative to vehicle-injected controls, ostensibly to enhance survival in the face of an immune challenge; however, there was no effect of LPS injection on activity or time spent resting when in a group setting and in the presence of potential mates, despite similar underlying physiological responses to LPS in the two social settings (Lopes et al. 2012). Thus, multiple intrinsic and extrinsic factors, including the social environment and mate availability, can clearly interact to shape the propensity of individuals to increase mating activity in the face of a mortality cue.

At a coarse level, seasonal effects likely constitute an especially important extrinsic factor because they comprise both abiotic (e.g., photoperiod, temperature, precipitation) and biotic (food and/or mate availability, predator abundance) environmental factors that can influence reproduction. Indeed, many species exhibit seasonal variation in reproductive output, often to increase survival to a later, more favorable, season for breeding (Baker 1938; Cockrem 1995). It follows, then, that season may influence an individual’s terminal investment threshold, especially in seasonal breeders. A significant interaction between season and reduced residual reproductive value (specifically age) has been demonstrated for several reproductive traits (including reproductive allotment to clutch, clutch size, and offspring dry mass) in Western mosquitofish (*Gambusia affini*) (Billman and Belk 2014; Table 3; supplementary table 3). Specifically, younger fish decreased reproductive investment over the season, whereas older fish increased investment, suggesting that younger individuals adopt a strategy of reproductive restraint, whereas older individuals exhibit terminal investment (Billman and Belk 2014; (Billman and Belk 2014; Table 3; supplementary table 3). However, such a pattern may also be explained by experience, if older breeders are better at coping with poor environmental conditions or the reproduction-survival trade-off. Thus, disentangling the myriad factors influencing between-individual differences in reproductive effort requires an experimental approach.

Future avenues for investigating terminal investment

Although the current empirical evidence in support of a dynamic terminal investment threshold is compelling (Table 3; supplementary table S3), it is still fairly limited in scope. To better understand why evidence for terminal investment is often equivocal, or even conflicting, both among and within studies, future research should pay particular attention to the form and intensity of the focal cue of reduced residual reproductive value (i.e., the terminal investment trigger), other intrinsic and extrinsic factors that might further affect residual reproductive value, and the specific reproductive traits of interest that are measured. One pattern that seems to be emerging is that increases in reproductive effort are frequently observed in some traits, but not in others (supplementary tables S1-S3). One possible explanation for this is that traits may differ in their flexibility to respond to reduced residual reproductive value. Consequently, it is important to consider the plasticity of the reproductive traits of interest when seeking to document terminal investment. Similarly, this review highlights the importance of considering both the form and intensity of cues that signal reduced residual reproductive value. Therefore, further investigation into the propensity of certain cues to alter reproductive effort may prove illuminating. For instance, studies that incorporate both active and inactivated pathogens (Adamo 1999), different strains of pathogens (Sanz et al. 2001), or different cues altogether (Barribeau et al. 2010), can provide valuable information about how, and under what circumstances, individuals differentially respond. It is important to note that there may also be taxonomic constraints to the expression of terminal investment. For example, mammals or other groups with prolonged parental care may be the least likely to exhibit terminal investment (e.g., high risk of vertical transmission of pathogens during gestation and lactation, prolonged periods of offspring production and parental care necessitating parental survival beyond offspring production). Our incomplete understanding of these constraints may explain the lack of clear examples of terminal investment in within some groups.

492 Theoretical modeling of the evolution of plastic life history strategies can aid in the
493 discovery of the conditions under which terminal investment will be favored by selection. Only
494 recently have studies attempted to theoretically define these conditions (Gandon et al. 2002;
495 Bonds 2006; Javoiš 2013, Leventhal et al. 2014; Luu and Tate 2017). For example, Luu and
496 Tate (2017) examined the competing strategies of somatic maintenance and terminal
497 investment using a model in which investments in these traded off differentially with other life
498 history traits. They determined that the trade-off between reproduction and maintenance drives
499 directional selection for either terminal investment or maintenance, depending on the cost of
500 reproduction to an individual's survival, and that diversifying selection leading to coexistence of
501 divergent strategies is favored under particular conditions (i.e., when virulence of the pathogen
502 invoking a response is low and the cost of reproduction by the host is high) (Luu and Tate
503 2017). This study highlights further the context-dependent nature of both the evolution and
504 expression of terminal investment. For example, the bifurcation of strategies shown under
505 certain parameter values could lead to genotype-dependent terminal investment, as mentioned
506 earlier. Additional theoretical approaches are needed to expand predictions related to
507 thresholds of terminal investment triggers and dynamic terminal investment thresholds.
508 A major gap in the literature is the almost complete absence of testing for terminal
509 investment outside of animal taxa. There is no obvious *a priori* hypothesis for why terminal
510 investment should be taxonomically constrained, and thus broader taxonomic coverage might
511 provide additional novel and valuable insights, along with systems that might be more amenable
512 to further study. The potential for this is demonstrated by work on *Pseudomonas fluorescens*
513 (SBW25), which was found to exhibit transient increases in population growth rate induced by
514 lytic DNA phage (SBW25Φ2) binding, consistent with predictions of the terminal investment
515 hypothesis, (Poisot et al. 2013). However, this was accompanied by decreased size of daughter
516 bacterial cells, which may reflect constraints on terminal investment due to a trade-off between
517 number and quality of progeny. This is the only study of which we are aware that investigates

these inducible responses following reduced residual reproductive value in bacteria, although results from studies like these could have potentially important consequences for applied fields such as medicine and epidemiology. Indeed, recent work has demonstrated that parasites can adopt a terminal investment response to environmental stressors, including pharmacological treatments or host immune responses. For example, malaria parasites (*Plasmodium spp*) divert resources from within-host replication to the production of transmission stages (gametocytes) in response to high doses of antimalarial drugs (reviewed in Carter et al. 2013). Multicellular parasites have also been shown to increase immediate fecundity in harsh environments (e.g., nematodes in response to a sudden rise of pro-inflammatory cytokines of the host; Guivier et al. 2017).

Although age-related shifts in reproductive investment have been well studied in plants (e.g., Thomas 2011), seldom have tests of terminal investment been applied to these systems, despite their tractability and amenability to experimental manipulation. Root herbivory in mustard (*Sinapis arvensis*), for example, led to an increase in the number of visits per flower by pollinators (Poveda et al. 2003), analogous to changes in sexual attractiveness seen in animals facing a mortality cue (e.g., Sadd et al. 2006), whereas above-ground herbivory and a combination of above- and below-ground herbivory reduced reproductive output (Poveda et al. 2003). Thus, plant systems may provide a compelling arena in which controlled experiments can disentangle the numerous extrinsic and intrinsic influences on the terminal investment threshold.

A major obstacle in moving the field forward is the lack of knowledge concerning the mechanisms that precipitate terminal investment. Although potential mechanisms have been proposed for some systems (e.g., Bowers et al. 2015), this void needs to be filled, and likely requires greater integration of molecular and physiological approaches in studies of life history evolution. Advances may also be made by investigating other traits aside from reproduction that are influenced by strategic shifts in allocation toward competing life history demands. Although

544 evidence for terminal investment comes chiefly from changes in reproductive effort, the terminal
545 investment hypothesis predicts that increased reproductive effort following reduced residual
546 reproductive value also comes at a cost to investment in other life history traits, including growth
547 and survival. Mechanistic studies (i.e., those that assess the allocation of resources following
548 decreases in RRV) could also be particularly important for uncovering potential *cryptic terminal*
549 *investment*. For example, under some conditions (e.g., particularly advanced infection) it may be
550 impossible for individuals to increase reproductive investment relative to uninfected individuals
551 (e.g., due to a loss of homeostasis); however, their relative decrease in fecundity may be less
552 compared with individuals who do not terminally invest.
553
554 **Conclusions**
555 The strategy of terminal investment has received widespread support, and has been
556 documented across an array of taxa and evoked by a variety of cues that signal reduced
557 residual reproductive value. However, equivocal, and sometimes conflicting, results also
558 abound, and the various outcomes observed across studies may reflect, in part, the traits that
559 are measured, how the responses affect individual fitness, differences in methodology, and
560 system-specific constraints on plasticity. However, much of this ambiguity can be resolved
561 within the conceptual framework of a dynamic terminal investment threshold, which considers
562 both the internal state of the individual and extrinsic factors that determine the optimal response
563 to a mortality cue, situating this important life history decision within a more realistic backdrop of
564 environmental heterogeneity. The further characterization of the dynamic terminal investment
565 threshold is greatly in need of empirical studies that include multiple factors influencing residual
566 reproductive value along a graduated spectrum of cues that facilitate the detection of the
567 interactions indicative of a dynamic threshold.
568
569 **Conflict of Interest:** The authors declare that they have no conflict of interest.

570

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Table 1 Intrinsic decreases in individual residual reproductive value and overall evidence for terminal investment in specific studies. A more detailed version of this table can be found in the Supplementary Materials section (supplementary table S1)

Taxon	Species	Common name	Investigated factor	Sex	Overall evidence	Reference
bird	*Ficedula albicollis*	Collared flycatcher	Age	F	yes	Part et al. 1992
bird	*Larus californicus*	California gull	Age	F	yes	Pugesek 1981
bird	*Sula nebouxii*	Blue-footed booby	Age	M	yes	Velando et al. 2006
insect	*Hetaerina americana*	American rubyspot damselfly	Age	M	yes	González-Tokman et al. 2013
insect	*Cardiocondyla obscurior*	Ant	Age	F	yes	Heinze and Schrempf 2012
insect	*Achroia grisella*	Lesser wax moth	Age	F/M	yes	Lafaille et al. 2010
insect	*Gryllus texensis*	Texas field cricket	Age	F	yes	Shoemaker et al. 2006
insect	*Ostrinia scapulalis*	European corn borer moth	Age	M	yes	Thanda Win et al. 2013

reptile	*Chrysemys picta*	Painted turtle	Age	F	yes	Paitz et al. 2007
rodent	*Tamiasciurus hudsonicus*	North American red squirrel	Age	F	yes	Descamps et al. 2007
ungulate	*Oreamnos americanus*	Mountain goat	Age	F	yes	Côté and Festa-Bianchet 2001
ungulate	*Alces alces*	Moose	Age	F	yes	Ericsson et al. 2001
ungulate	*Ovis canadensis*	Bighorn sheep	Age	F	yes	Festa-Bianchet 1988
ungulate	*Kobus megaceros*	Nile lechwe	Age	F	yes	Bercovitch et al. 2009
fish	*Gambusia affinis*	Western mosquitofish	Age	F	see interaction table	Billman and Belk 2014
ungulate	*Bison bison*	American bison	Age	M	yes	Maher and Byers 1987
insect	*Nicrophorus vespilloides*	Burying beetle	Age	M	mixed	Benowitz et al. 2013
insect	*Nicrophorus orbicollis*	Burying beetle	Age	F	mixed	Creighton et al. 2009

primate	*Macaca mulatta*	Rhesus macaque	Age	F	mixed	Hoffman et al. 2010
ungulate	*Cervus elaphus*	Red deer	Age	F	mixed	Clutton-Brock et al. 1982
ungulate	*Dama dama*	Fallow deer	Age	M	mixed	Jennings et al. 2010
insect	*Nauphoeta cinerea*	Speckled cockroach	Juvenile diet quality	F	mixed	Barrett et al. 2009
bird	*Melospiza melodia*	Song sparrow	Chronological age / Years to death (YTD)	F	no / see interaction table	Tarwater and Arcese 2017
bird	*Ficedula hypoleuca*	Pied flycatcher	Age	F	no	Sanz et al. 2001
fish	*Syngnathus typhle*	Broad-nosed pipefish	Age	M	no	Billing et al. 2007
insect	*Allonemobius socius*	Southern ground cricket	Age	M	no	Copeland and Fedorka 2012
insect	*Glossina morsitans morsitans*	Tsetse fly	Age	F	no	Langley and Clutton-Brock 1998

insect	*Heliothis virescens*	Tobacoo budworm moth	Age	F	no	Staudacher et al. 2015
primate	*Pan troglodytes*	Common chimpanzee	Age	F	no	Fessler et al. 2005
insect	*Nicrophorus vespilloides*	Burying beetle	Age at first reproduction	F	no	Cotter et al. 2010
tree	*Quercus* spp.	Oak (8 species)	Time prior to death	NA	no	Koenig et al. 2017

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Table 2 Extrinsic decreases in individual residual reproductive value and overall evidence for terminal investment in specific studies. A more detailed version of this table can be found in the Supplementary Materials section (supplementary table S2)

Taxon	*Species*	Common name	Investigated factor	Sex	Overall evidence	Reference
amphibian	*Hyla japonica*	Japanese tree frog	Fungal pathogen (*Batrachochytrium dendrobatidis*)	M	yes	An and Waldman 2016
bird	*Delichon urbica*	House martin	Malaria infection *(Haemoproteus* or *Plasmodium* spp.)	F/M	yes	Marzal et al. 2008
insect	*Cardiocondyla obscurior*	Ant	Entomopathogenic fungus (*Metarhizium brunneum*)	F	yes	Giehr et al. 2017
insect	*Acyrthosiphon pisum*	Pea aphid	Oral exposure to bacteria (*Pseudomonas syringae*)	F/M	yes	Hendry et al. 2016
insect	*Drosophila nigrospiracula*	Fruit fly	Ectoparasitic mite (*Macrocheles subbadius*)	M	yes	Polak and Starmer 1998
insect	*Gryllus texensis*	Texas field cricket	Bacteria (*Serratia marcescens*)	F	yes	Shoemaker et al. 2006

snail	*Biomphalaria glabrata*	Freshwater snail	Trematode infection (*Schistosoma mansoni*)	F	yes	Blair and Webster 2007
rodent	*Peromyscus maniculatus*	Deer mouse	Trematode infection (*Schistosomatium douthitti*)	F	yes	Schwanz 2008a
bird	*Passer domesticus*	House sparrow	Newcastle virus vaccine	F	yes	Bonneaud et al. 2004
bird	*Troglodytes aedon*	House wren	LPS (from *Salmonella enterica*)	F	yes	Bowers et al. 2015
bird	*Somateria mollissima*	Common elder	SRBC	F	yes	Hanssen 2006
insect	*Acyrthosiphon pisum*	Pea aphid	HK bacteria (*Escherichia coli*)	F	yes	Altincicek et al. 2008
insect	*Nicrophorus vespilloides*	Burying beetle	Dead bacteria (*Micrococcus lysodeikticus*)	F	yes	Cotter et al. 2010
insect	*Gryllodes sigillatus*	Decorated cricket	HK bacteria (*Escherichia coli*)	M	yes	Duffield et al. 2015
insect	*Tenebrio molitor*	Yellow mealworm beetle	Nylon implant (single or two consecutive)	M	yes	Kivleniece et al. 2010

insect	*Tenebrio molitor*	Yellow mealworm beetle	Nylon implant (single or two consecutive)	M	yes	Krams et al. 2011
insect	*Tenebrio molitor*	Yellow mealworm beetle	LPS (from *Escherichia coli*)	M	yes	Nielsen and Holman 2012
insect	*Tenebrio molitor*	Yellow mealworm beetle	Nylon implant	M	yes	Sadd et al. 2006
rodent	*Peromyscus leucopus*	White-footed mouse	SRBC	M	yes	Derting and Virk 2005
rodent	*Phodopus sungorus*	Siberian hamster	LPS (undefined source)	M	yes	Weil et al. 2006
alveolate	*Plasmodium chabaudi*	Rodent malaria	Chloroquine (CQ) treatment of hosts (*Mus musculus musculus*)	NA	yes	Buckling et al. 1997
alveolate	*Plasmodium falciparum*	Human malaria	Chloroquine (CQ) treatment (*in vitro*)	NA	yes	Buckling et al. 1999

nematode	*Heligmosomoides polygyrus*	Intestinal roundworm	LPS-induced circulating pro-inflammatory cytokines within host (*Mus musculus musculus*)	F/M	yes	Guivier et al. 2017
amphibian	*Pseudophryne corroboree*	Southern corroboree frog	Fungal pathogen (*Batrachochytrium dendrobatidis*)	M	mixed	Brannelly et al. 2016
	Litoria verreauxii alpina	Alpine tree frog		F/M		
bird	*Ficedula hypoleuca*	Pied flycatcher	Protozoan (*Haemoproteus balmorali*)	F	mixed	Sanz et al. 2001
			Protozoan (*Trypanosoma* spp.)			
crustacean	*Daphnia magna*	Water flea	Microsporidian spores (*Glugoides intestinalis*)	F/M	mixed	Chadwick and Little 2005
plant	*Sinapis arvensis*	Mustard plant	Root herbivory (*Agriotes* sp.)	NA	mixed	Poveda et al. 2003
			Leaf herbivory (*Pieris rapae*)			

rodent	*Peromyscus maniculatus*	Deer mouse	Trematode infection (*Schistosomatium douthitti*)	F	mixed	Schwanz 2008b
snail	*Biomphalaria glabrata*	Freshwater snail	Trematode infection (*Schistosoma mansoni*)	F	mixed	Minchella and Loverde 1981
insect	*Acheta domesticus*	House cricket	Live bacteria or LPS (*Serratia marcescens*) Parasitoid tachinid fly (*Ormia ochracea*) Sephadex beads	F	mixed	Adamo 1999
insect	*Gryllus texensis*	Texas field cricket	Enforced running (blowing air on cerci) Predator (praying mantis, *Tenodera sinensis*) exposure	F	mixed	Adamo and McKee 2017
bacteria	*Pseudomonas fluorescens*	Rhizosphere bacteria	UV-inactivated lytic bacteriophage	NA	mixed	Poisot et al. 2013

bird	*Troglodytes aedon*	House wren	LPS (from *Salmonella enterica*)	F	mixed	Bowers et al. 2012
insect	*Heliothis virescens*	Tobacoo budworm moth	Dead bacteria (*Serratia entomophila*)	F	mixed	Staudacher et al. 2015
bird	*Ficedula hypoleuca*	Pied flycatcher	Diphtheria-tetanus vaccine	F	no	Ilmonen et al. 2000
insect	*Tenebrio molitor*	Yellow mealworm beetle	Tapeworm infection (*Hymenolepis diminuta*)	M	no	Worden et al. 2000
ungulate	*Ovis canadensis*	Bighorn sheep	Lungworm infection *(Protostrongylus* spp.)	F	no	Festa-Bianchet 1988
insect	*Anopheles gambiae*	African malaria mosquito	LPS (undefined source) Orally administered LPS	F	no	Ahmed et al. 2002
insect	*Gryllus camperstris*	Field cricket	LPS (from *Serratia marcescens*) Food availability	M	no	Jacot et al. 2004

insect	*Cyphoderris strepitans*	Sagebrush grig	LPS (from *Serratia marcesens*)	M	no	Leman et al. 2009
insect	*Tribolium castaneum*	Red flour beetle	Post-mating somatic damage	F	no	Morrow et al. 2003
	Callosobruchus maculatus	Cowpea weevil				
	Drosophila melanogaster	Fruit fly				
insect	*Euoniticellus intermedius.*	Dung beetle	LPS (from *Serratia marcescens*)	F	no	Reaney and Knell 2010
insect	*Gryllus texensis*	Texas field cricket	LPS (from *Serratia marcescens*)	F	no	Shoemaker and Adamo 2007
reptile	*Paroedura picta*	Madagascar ground gecko	Food availability	F	no	Kubička and Kratochvíl 2009

reptile	*Ctenophorus fordi*	Mallee dragon	LPS (from *Escherichia coli*)	F	no	Uller et al. 2006
bird	*Cyanistes caeruleus*	Blue tit	Malaria infection (*Haemoproteus* and *Plasmodium* spp.)	F/M	*see interaction table*	Podmokła et al. 2014
bird	*Sula nebouxii*	Blue-footed booby	LPS (from *Escherichia coli*)	M	*see interaction table*	Velando et al. 2006
fish	*Syngnathus typhle*	Broadnosed pipefish	Perceived predation risk (*Gadus morhua*)	M	*see interaction table*	Billing et al. 2007
insect	*Acyrthosiphon pisum*	Pea aphid	HK bacteria (genus *Enterobacter*) Alarm pheromone	both	*see interaction table*	Barribeau et al. 2010
insect	*Allonemobius socius*	Southern ground cricket	LPS (undefined source)	M	*see interaction table*	Copeland and Fedorka 2012
insect	*Hetaerina americana*	American rubyspot damselfly	Nylon implant	M	*see interaction table*	González-Tokman et al. 2013

988 Abbreviations: HK, heat-killed; LPS, lipopolysaccharides; SRBC, sheep red blood cells

Table 3 Evidence of interactions between factors influencing terminal investment. A more detailed version of this table can be found in the Supplementary Materials section (supplementary table S3)

Taxon	Species	Common name	Factors in investigated interaction		Sex	Interaction?	Reference
			X_1	X_2			
bird	*Melospiza melodia*	Song sparrow	Chronological age	Years to death (YTD)	F	yes	Tarwater and Arcese 2017
fish	*Gambusia affinis*	Western mosquitofish	Age	Season	F	yes	Billman and Belk 2014
insect	*Gryllodes sigillatus*	Decorated cricket	Age	HK bacteria (*Escherichia coli*)	M	yes	Duffield et al. in prep.
insect	*Drosophila nigrospiracula*	Fruit fly	Age	Ectoparasitic mite (*Macrocheles subbadius*)	M	yes	Polak and Starmer 1998
insect	*Ephippiger diurnus*	Chorusing	Age	Acoustic environments	M	yes	Rebar and

		bushcrick et		Geographic population			Greenfield 2017
crustacean	*Daphnia magna*	Water flea	Genotype	Bacteria (*Pasteuria ramosa*)	F/M	yes	Vale and Little 2012
insect	*Acyrthosiphon pisum*	Pea aphid	Genotype	HK bacteria (*Enterobacter cloacae*), gram-positive bacteria, fungus (*Erynia neoaphidis*)	F/M	yes	Leventhal et al. 2014
alveolate	*Plasmodium falciparum*	Human malaria	Chloroquine (CQ) treatment (*in vitro*)	Genotype	NA	yes	Bluckling et al. 1999
bird	*Passer domesticus*	House sparrow	Body size	Newcastle virus vaccine	F	yes	Bonneaud et al. 2004
bird	*Somateria mollissima*	Common elder	Individual quality	SRBC	F	yes	Hanssen 2006

amphibian	*Litoria rheocola*	Common mist frog	Fungal pathogen (*Batracho-chytrium dendrobatidis*)	Season Body condition index	M	yes	Roznik et al. 2015
fish	*Syngnathus typhle*	Broadnosed pipefish	Perceived predation risk (*Gadus morhua*)	Relative activity of female partner	M	yes	Billing et al. 2007
insect	*Belostoma flumineum*	Giant waterbug	Temperature	Clutch size	M	yes	Kight et al. 2000
insect	*Tenebrio molitor*	Yellow mealworm beetle	Nylon implant	Food availability	M	yes	Krams et al. 2015
insect	*Gryllus texensis*	Texas field cricket	Bacteria (*Serratia marcescens*)	Oviposition substrate	F	yes	Shoemaker et al. 2006
bird	*Ficedula hypoleuca*	Pied flycatcher	Age	Protozoan (*Haemoproteus balmorali*)	F	mixed	Sanz et al. 2001

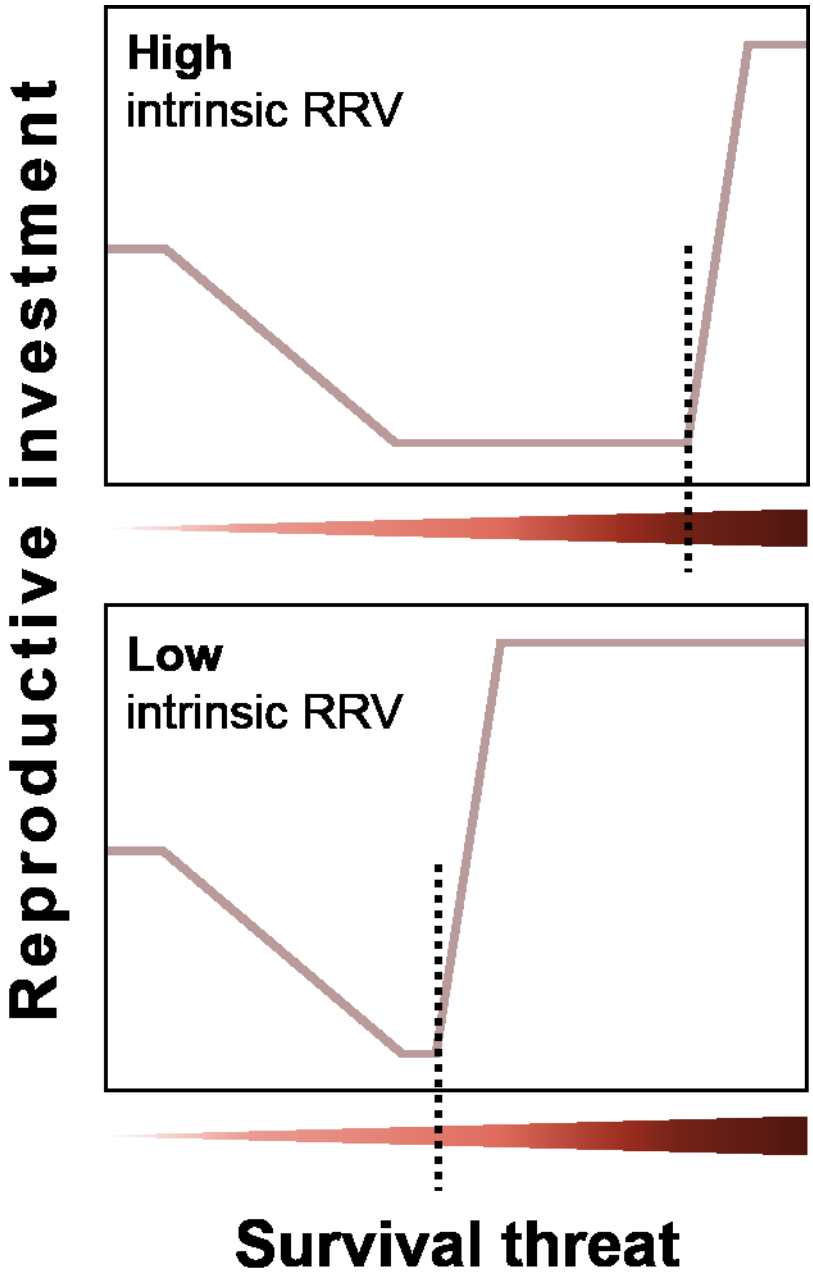
bird	*Sula nebouxii*	Blue-footed booby	Age	LPS (from *Escherichia coli*)	M	mixed	Velando et al. 2006
insect	*Nicrophorus vespilloides*	Burying beetle	Age	Paternity assurance	M	mixed	Benowitz et al. 2013
insect	*Allonemobius socius*	Southern ground cricket	Age	LPS	M	mixed	Copeland and Fedorka 2012
insect	*Hetaerina americana*	American rubyspot damselfly	Nylon implant	Age Body size	M	mixed	González-Tokman et al. 2013
insect	*Nicrophorus orbicollis*	Burying beetle	Age Number of previous reproductive attempts	Carcass size Resource availability	F	mixed	Creighton et al. 2009
insect	*Nicrophorus*	Burying beetle	Age at first reproduction	Prior reproductive investment	F	yes	Cotter et al. 2010

	vespilloides						
crustacean	*Daphnia magna*	Water flea	Genotype	Microsporidian spores (*Glugoides intestinalis*)	F/M	mixed	Chadwick and Little 2005
insect	*Acyrthosiphon pisum*	Pea aphid	Genotype	HK bacteria (genus *Enterobacter*)	F/M	mixed	Barribeau et al. 2010
				Alarm pheromone			
			Artificially established secondary symbionts	HK bacteria (genus *Enterobacter*)			
				Alarm pheromone			
bird	*Cyanistes caeruleus*	Blue tit	Malaria infection (*Haemo-proteus* and	Clutch size	F/M	mixed	Podmokła et al. 2014

			Plasmodium spp.)				
plant	*Sinapis arvensis*	Mustard plant	Root herbivory (*Agriotes* sp.)	Leaf herbivory (*Pieris rapae*)	NA	mixed	Poveda et al. 2003
insect	*Acheta domesticus*	House cricket	Age	Bacteria (*Serratia marcescens*)	F	no	Adamo 1999
insect	*Gryllodes sigillatus*	Decorated cricket	Genotype	HK bacteria (*Escherichia coli*)	M	no	Duffield et al. 2015
insect	*Gryllus camperstris*	Field cricket	LPS (from Serratia marcescens)	Food availability	M	no	Jacot et al. 2004
rodent	*Peromyscus leucopus*	White-footed mouse	Testosterone levels	SRBC	M	no	Derting and Virk 2005

992 Abbreviations: HK, heat-killed; LPS, lipopolysaccharides; SRBC, sheep red blood cells

Fig 1 Predictions based on intrinsic residual reproductive value (RRV) from the dynamic terminal investment threshold model. At low threat levels, individuals invest intermediately in reproduction to balance the reproduction-immunity trade-off. As a threat increases, investment in immunity increases to combat the threat. Thus, costs of immunity necessitate a decreased reproductive investment. At high threat levels, past where resistance is ineffective (terminal investment threshold, vertical dashed line), a terminal investment strategy of increased reproductive investment is predicted. Intrinsic RRV is expected to influence this threshold, with the threshold dropping as intrinsic RRV decreases.


High
intrinsic RRV
Low
intrinsic RRV
Reproductive investment
Survival threat

1002