vol. 191, no. 6 the american naturalist june 2018

How Life History Shapes Optimal Patterns of Senescence: Implications from Individuals to Societies

Natalie J. Lemanski* and Nina H. Fefferman

Department of Ecology and Evolutionary Biology, University of Tennessee, Knoxville, Tennessee 37916

Submitted December 15, 2016; Accepted December 19, 2017; Electronically published March 28, 2018

abstract: One evolutionary view of aging, the disposable soma theory, suggests that an organism's rate of senescence depends on the amount of energy invested in somatic maintenance. Since organisms have limited energy to allocate among growth, maintenance, and reproduction, the optimal amount of energy to invest in maintenance is influenced by the probability of death from extrinsic causes and the effect of somatic investment on survival. In eusocial animals, the disposable soma theory can be used to explain colonies' energy investment in the longevity of workers, who act as the somatic elements of a superorganism. There have been few theoretical considerations of how changes in the costliness of worker maintenance or in the effect of individual life span on group fitness influence a colony's investment in worker longevity. We develop a decision theory model to evaluate how changing the marginal costs and benefits of longevity and extrinsic mortality influence optimal worker life span in a social insect colony. Our model predicts that higher extrinsic mortality favors shorter life span. However, increased life span is favored when marginal benefits are an increasing function of longevity. In honeybees, this explains how greater somatic investment is sometimes favored despite high mortality. Our approach expands the disposable soma theory to make quantitative predictions about the selective pressures shaping senescence in social systems.

Keywords: evolution of aging, life history, senescence, disposable soma theory, honeybees.

Introduction

The phenomenon of senescence, the gradual deterioration in physiological function with age, seems clearly detrimental to fitness, yet is a ubiquitous feature throughout the tree of life. Furthermore, organisms differ greatly in both longevity and the pattern of senescence (Piraino et al. 1996; Miller 2001; Morbey et al. 2005). Understanding the evolutionary pressures that shape differences in longevity is one of the major challenges of life-history theory.

Evolutionary theory suggests that senescence occurs because of a decline in the force of selection with age (Medawar 1952; Williams 1957; Hamilton 1966). This decline in selection leads to a physiological decline in function with age because of an accumulation of late-acting deleterious mutations not eliminated by selection (Medawar 1952) or because of a trade-off between early life fecundity and later survival (Williams 1957; Nesse 1988). Disposable soma theory suggests a physiological mechanism for that trade-off; organisms have limited resources to allocate, so investing more energy in somatic maintenance means less available for reproduction (Kirkwood 1977). Selection should favor the optimal allocation of resources that maximizes fitness.

While evolutionary senescence theory was devised to explain senescence of individuals, this theory can also be used to understand the senescence of parts of a multicellular organism (e.g., Gardner and Mangel 1997) or of individuals in a functionally integrated superorganism, such as that of eusocial insect colonies (Seeley 1997; Lee 2003). Rather than maximizing individual reproductive value (Kozłowski 1993), selection in a superorganism acts on individual phenotypes to maximize their contribution to the fitness of the colony. For social insects, where workers have little to no direct reproduction, worker phenotypes should evolve to maximize their contribution to the colony's survival, growth, and reproduction (Lee 2003).

One original prediction of evolutionary senescence theory was that a higher probability of death from extrinsic causes (such as accidents or predation) should cause the evolution of faster senescence because the force of selection decreases more rapidly as the probability of surviving to older ages decreases (Medawar 1952; Williams 1957; Hamilton 1966). For a superorganism, higher extrinsic mortality should select for lower somatic investment and shorter life span of individuals. Another prediction is that there should be a trade-off between reproductive effort and longevity (Kirkwood and Austad 2000). In keeping with previous research (e.g., Abrams 1993), we assume for simplicity that extrinsic mortality is age independent, though we acknowledge that truly age-independent mortality may be rare in nature (Dowling 2012).

---

* Corresponding author; e-mail: natalie.lemanski@gmail.com.

ORCIDs: Lemanski, http://orcid.org/0000-0002-4685-5093; Fefferman, http://orcid.org/0000-0003-0233-1404.

Am. Nat. 2018. Vol. 191, pp. 756–766.
DOI: 10.1086/697225

There has been great interest in empirically testing these predictions. Experimental evolution studies have generally supported the prediction that higher extrinsic mortality leads to more rapid senescence (Gasser et al. 2000; Stearns et al. 2000) as well as the prediction of a trade-off between longevity and early fecundity (Rose 1984; Chippindale et al. 1993; Partridge et al. 1999; but see Partridge and Fowler 1992). In contrast, studies in wild populations have found mixed support for these predictions (Austad 1993; Holmes and Austad 1994; Keller and Genoud 1997; Bérubé et al. 1999; Wilkinson and South 2002; Reznick et al. 2004; Morbey et al. 2005; Ricklefs 2010; Healy et al. 2014).

There has been a great deal of work on refining the theoretical predictions of evolutionary senescence theory to help explain these conflicting findings. Contrary to Williams's original prediction, extrinsic mortality has no effect on senescence if a population is density independent or if density affects survival of all age classes equally (Abrams 1993; Caswell 2007). However, it does select for faster senescence if density dependence acts uniformly on fertility (Abrams 1993). In addition, empirical tests often use predation as a source of extrinsic mortality (e.g., Reznick et al. 2004). However, while theory assumes extrinsic mortality is age independent (Medawar 1952), predation is usually not random with respect to condition (Dowling 2012) and higher predation risk may therefore select for decreased, not increased, senescence (Chen and Maklakov 2012).

Furthermore, while previous models often assumed a linear effect of energy invested in reproduction on fertility or of energy invested in repair on somatic damage accumulation (Kirkwood and Rose 1991; Abrams and Ludwig 1995), there has been increasing recognition that the effect of energy investment in repair on mortality may be nonlinear (Cichoń 1997; Cichoń and Kozłowski 2000; Mangel and Munch 2005; McNamara and Buchanan 2005; Munch and Mangel 2006). For instance, Cichoń (1997) found that a higher efficiency of repair selects for a longer life span.

Just as energy invested in somatic repair may have a nonlinear effect on life span, in organisms or colonies that experience the senescence of parts, the life span of individual parts may have a nonlinear effect on the fitness of the whole organism or colony. The effect of extrinsic mortality on worker life span has been examined in social insects (Kramer and Schaible 2013). However, few theoretical studies have examined both nonlinear effects of somatic repair on individual life span and nonlinear effects of individual life span on fitness in social systems. Building on previous work, we seek to examine how the shapes of these relationships influence the evolution of longevity in a social context.

We here present a decision theory model examining optimal worker life span in a social insect colony based on (*a*) the risk of extrinsic mortality; (*b*) the effect of worker life span on colony productivity, defined as the colony's rate of acquisition of energetic resources (marginal benefits of life span); and (*c*) the effect of somatic investment in workers on worker life span (marginal costs of life span). Decision theory is a mathematical framework dealing with optimal decision making under risk or uncertainty (Peterson 2009) that has been widely applied in ecology (McNamara and Houston 1980; McNamara and Buchanan 2005; Nesse 2005; Bateson 2007). Decision theory is a logical framework for modeling individual workers as investments by a colony, with some risk (extrinsic mortality) and expected value (the workers' contributions to colony growth and reproduction); it can easily incorporate nonlinearity in the costs and benefits of the investment to determine the optimal investment level. This framework can also incorporate empirical data and predict how multiple factors interact to determine a colony's optimal investment in the life span of its colony members.

We examine this question using honeybees (*Apis mellifera*) as an excellent model system for expanding on evolutionary aging theory. Honeybee workers have extraordinary phenotypic plasticity in life span, influenced by season and behavioral role (Remolina et al. 2007; Münch and Amdam 2010). As predicted by disposable soma theory, the transition from in-hive work to foraging, with an accompanying increase in extrinsic mortality, also results in faster physiological senescence and a shorter life span (Rueppell et al. 2007; Dukas 2008). Worker senescence is under individual and social control (Amdam et al. 2005), making it an emergent property of the colony. Conveniently, measuring resource allocation among individual workers is much easier than measuring allocation among functional systems of an organism. Honeybee colonies are highly integrated units; although there is the potential for conflict, in the matter of colony survival, the interests of workers are largely aligned (Seeley 1997).

Methods

We model worker bees as an investment by the colony (the agent whose utility is being maximized). Selection should act on colonies to invest in somatic maintenance of workers in a way that optimizes the net contribution of its workforce to colony productivity, a reasonable proxy for colony fitness in honeybees because it determines how much energy can be allocated among the parent and all offspring colonies. We define the intrinsic life span as the average life span of a worker not killed by any extrinsic source of mortality. Intrinsic life span, n, is the variable being optimized in the model (model variables and parameters are defined in tables 1, 2). While this intrinsic life span is a deterministic product of resource investment, the realized worker life span will vary among individuals due to random extrinsic mortality. The average worker life span will be a function

Table 1: Model variables

Variable	Definition
n	Intrinsic worker life span, defined as the average life span of a worker not killed by any extrinsic (age-independent) source of mortality
P_n	Colony productivity resulting from an intrinsic worker life span of n days
B_n	Contribution to the colony of workers with intrinsic life span of n days
$\hat{B}_n$	Marginal benefit from increasing worker intrinsic life span from $n - 1$ to n days
C_n	Cost to the colony of producing workers with intrinsic life span n
$\hat{C}_n$	Marginal cost of increasing worker intrinsic life span from $n - 1$ to n days
I	Optimal worker intrinsic life span

of both intrinsic capacity for survival and random mortality.

We assume the colony can invest energetic resources to increase the durability of workers (more durable workers have a longer intrinsic life span). In honeybees, increasing the size of a worker's protein reserve results in an increased life span but imposes an increased cost because the worker must consume more pollen (Crailsheim et al. 1992; Amdam and Omholt 2002; Amdam et al. 2004; Alaux et al. 2010). Investing sufficient resources to produce workers with intrinsic life span of n days imposes a cost, which we call C_n. We then define the marginal cost, $\hat{C}_n$, as the increase in an average worker's resource consumption resulting from increasing its intrinsic life span from $n - 1$ to n days. In principle, we can empirically estimate the shape of the cost curve by measuring the impact of protein consumption on intrinsic life span. We can define total cost of workers with intrinsic life span n as

$$C_n = \sum_{1}^{n} \hat{C}_n.$$

We next define the marginal benefit, $\hat{B}_n$, as the increase in colony productivity resulting from an average worker's life span increasing from $n - 1$ to n days. In addition, workers have a daily probability of age-independent extrinsic mortality, m. The colony must pay an up-front cost, C_n, to produce workers of sufficient durability to have an intrinsic life span of n days, regardless of whether they are killed by extrinsic causes before reaching age n, but marginal benefits $\hat{B}_n$ are only realized once workers survive to age n. We can therefore define the expected payoff of workers with an intrinsic life span of n days as

$$P_n = \sum_{1}^{n} (\hat{B}_n(1 - m)^n) - \sum\nolimits_{1}^{n} \hat{C}_n.$$

We then manipulate the shapes of the cost-and-benefit functions to examine how their shapes affect the colony's optimal intrinsic worker life span. We model cases in which the benefit function is a linear (marginal benefits are constant with respect to age), exponential (marginal benefits are monotonically increasing with age), saturating (marginal benefits are monotonically decreasing with age), or sigmoidal (marginal benefits increase with age up to some point and then decrease) function of the intrinsic life span. We model the marginal benefits as a simple recursive function; this form was chosen arbitrarily as a convenient function that could take on various shapes by altering two parameters (see Cichoń 1997). When parameter $b_1 > 1$, the marginal benefits are decreasing, when $b_1 < 1$, the marginal benefits are increasing, and when $b_1 = 1$, the marginal benefits are constant with respect to n. When parameter $b_2 = 0$, $\hat{B}_n$ increases or decreases monotonically; when $b_2 > 0$, $\hat{B}_n$ increases and then decreases (B_n is sigmoidal). We define the marginal benefit function as follows:

Table 2: Model parameters

Parameter	Definition	Value
m	Daily probability of extrinsic worker mortality	0−.98
b_1	Parameter governing shape of benefit function (exponential, linear, or saturating)	.7−1.3
b_2	Parameter governing shape of benefit function (sigmoidal or nonsigmoidal)	0−.6
c_1	Parameter governing shape of cost function (exponential, linear, or saturating)	.7−1.3
$\hat{B}_1$	Initial marginal benefits of worker with intrinsic life span of 1	1−50
$\hat{C}_1$	Initial marginal costs of worker with intrinsic life span of 1	1
q	Ratio of initial marginal benefits ($\hat{B}_1$) to initial marginal costs ($\hat{C}_1$)	1−50

$$\hat{B}_n = \frac{\hat{B}_{n-1}}{b_1}(1 - b_2(n-1)).$$

Similarly, we model cases in which the cost function is linear (marginal costs are constant), exponential (marginal costs are increasing), and saturating (marginal costs are decreasing). When parameter $c_1 > 1$, the marginal costs are decreasing, when $c_1 < 1$, the marginal costs are increasing, and when $c_1 = 1$, the marginal costs are constant with respect to n. We define the marginal cost function as

$$\hat{C}_n = \frac{\hat{C}_{n-1}}{c_1}.$$

The exact values of C_n and B_n do not affect our general result, but their ratio may influence the optimal intrinsic life span. We define parameter q as the ratio of the initial value of $\hat{B}_n$ ($n = 1$) to the initial value of $\hat{C}_n$ ($n = 1$), and we examine various values of parameter q. We assign $\hat{C}_1$ a value of 1, and we define $\hat{B}_1$ as a function of $\hat{C}_1$ and q:

$$\hat{B}_1 = q\hat{C}_1.$$

Finally, we define the optimal intrinsic life span, I, as the point n where P_n is maximized:

$$I = \arg\max(P_n).$$

Results

Our model shows that when the costs and benefits of worker life span are linear with respect to life span, a decrease in extrinsic hazards increases the optimal intrinsic life span (fig. 1). This finding is consistent with existing senescence theory, which predicts that, all else being equal, longer life span should evolve when risks of accidental mortality are low (Kirkwood and Austad 2000).

We also find that increasing q, the ratio of initial marginal benefits to initial marginal costs, increases the optimal intrinsic life span (fig. 2). This means that increased worker life span should occur if the same degree of somatic maintenance can be achieved at a lower cost (higher efficiency of repair) or when protein is cheaper (when pollen is more abundant). We also expect increased worker life span to be optimal if the contribution per worker to colony productivity increases over its whole life span.

Extending beyond previous predictions, our model shows that a marginal cost function that increases with respect to life span favors a shorter intrinsic life span compared to when marginal costs are constant or decreasing (fig. 3). Increasing costs might occur when physical wear is multiplicative rather than cumulative (Cichoń 1997) or when one kind of wear increases other maintenance costs.

Figure 1: Effect of extrinsic mortality on optimal worker life span. As daily probability of death from extrinsic causes (parameter m) increases, the colony's optimal worker intrinsic life span decreases. We explored 50 values of m from 0 to 0.98. Other parameters were held constant at $q = 6$, $b_1 = 1$, $b_2 = 0$, and $c_1 = 0.95$.

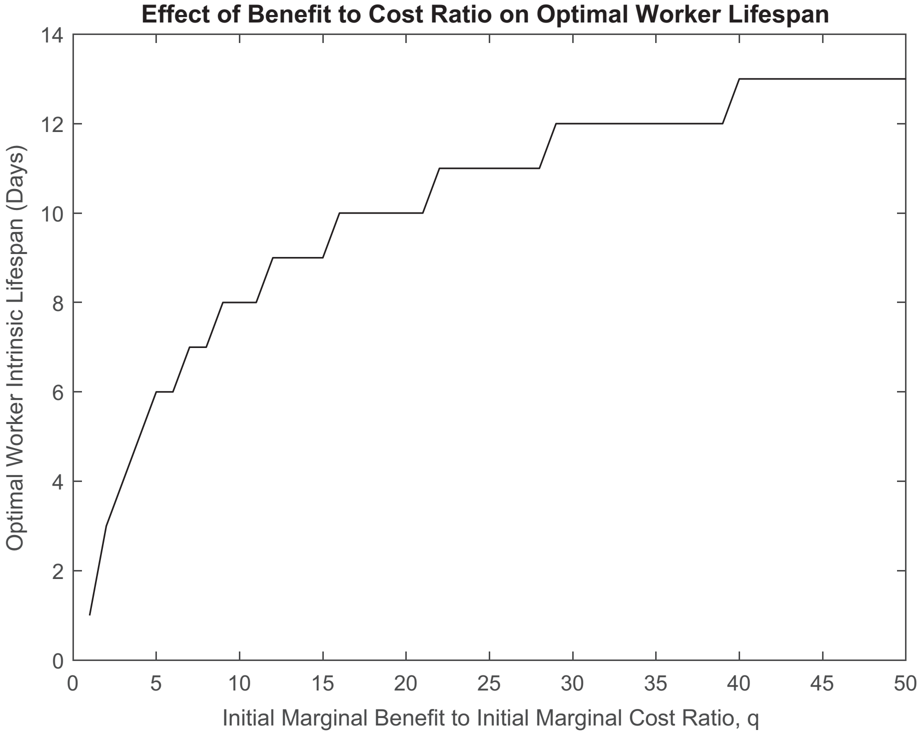

Figure 2: Effect of the benefit-to-cost ratio on optimal worker life span. Increasing parameter q, the ratio of the initial marginal benefits to initial marginal costs, increases the colony's optimal worker intrinsic life span. We explored 50 values of q from 1 to 50. Other parameters were held constant at $m = 0.1$, $b_1 = 1.1$, $b_2 = 0$, and $c_1 = 0.9$.

In addition, we find a marginal benefit function that increases with respect to life span favors longer intrinsic life span, while a decreasing marginal benefit function favors shorter intrinsic life span (fig. 4). Increasing marginal benefits occur when an individual's value increases with age. For a social insect worker, it may, for example, reflect an increase in an individual's contribution to the colony with age because of learning. For a solitary organism, it could reflect an increase in fecundity with age.

Discussion

Major evolutionary theories of aging agree that the fundamental cause of senescence is a decline in the force of selection with age. The disposable soma theory of aging explains senescence as a decline in physiological function caused by wear and tear that is allowed to accumulate rather than being repaired (Kirkwood and Rose 1991); the rate of senescence—and therefore life span—is a function of the amount of resources allocated to somatic repair rather than growth or reproduction (Kirkwood and Austad 2000). Natural selection acts on this allocation to maximize fitness.

In an organism or colony that experiences senescence of parts, selection acts on the level of somatic investment in individual parts to maximize the fitness of the whole organism or colony. In classic aging theory for single individuals, the unit of aging is the same as the unit of fitness. In a superorganism, while aging still occurs at the individual level, fitness is maximized at the colony level. The whole superorganism shares resources, and individuals have no direct reproduction. This contrasts with a population of individuals with different genotypes, in which selection acts on different allocation strategies directly to maximize individual reproduction. The present work thus differs from previous allocation models of senescence and allocation in heterogeneous populations in that, instead of modeling resource allocation between survival and reproductive effort in an individual, we assume resources are allocated among individuals to maximize colony fitness. Allocating less to an individual worker's survival does not directly benefit it but saves resources to be used on other workers, where the colony can get increased net benefit from the workers' contribution to the colony.

Much debate in evolutionary aging research concerns the role of extrinsic mortality in shaping the evolution of life span. Early evolutionary theories of aging predicted that greater extrinsic mortality selects for less allocation to maintenance and faster senescence (Medawar 1952; Hamilton 1966; Kirkwood 1977), while subsequent work suggests the

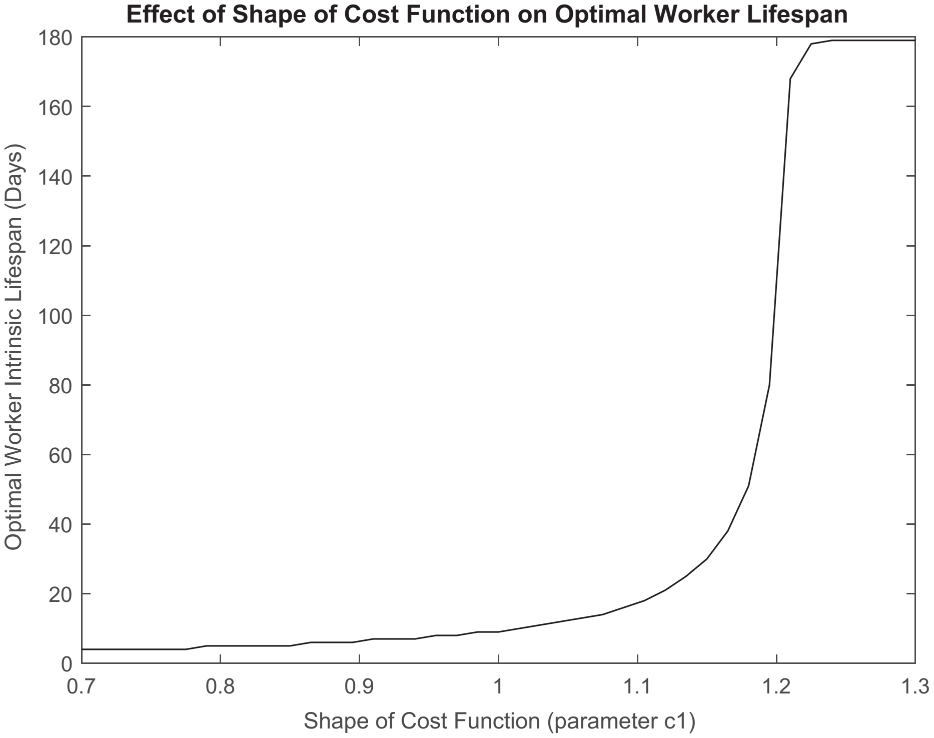

Figure 3: Effect of the cost function on optimal worker intrinsic life span. Parameter c_1 governs the shape of the marginal cost function; $c_1 = 1$ implies that the marginal cost is constant (costs increase linearly with worker life span), $c_1 > 1$ implies that marginal costs are decreasing (costs increase in a saturating way with life span), and $c_1 < 1$ implies that marginal costs are increasing (costs increase exponentially with life span). We find that the optimal worker life span decreases when marginal costs are increasing (i.e., when additional energetic investments produce smaller and smaller increases in worker life span) and increases sharply when marginal costs are decreasing. We examined 41 values of parameter c_1 from 0.7 to 1.3. Other parameters were held constant at $m = 0.1$, $q = 6$, $b_1 = 1.1$, and $b_2 = 0$.

relationship may be more complex (Law 1979; Abrams 1993; Baudisch 2011; Chen and Maklakov 2012).

There has also been increasing recognition that nonlinearity in the effects of somatic investment on fitness may be important to the evolution of life span (Cichoń 1997; Munch and Mangel 2006). Our model extends previous work by explicitly considering the effect of nonlinearities in both the energetic costs and the fitness benefits of changes in the intrinsic life spans of individuals in a social system.

We have chosen life span as the variable to optimize because it is the trait that, balanced against reproduction, most directly impacts fitness and because life span is a relatively easy dependent variable to empirically measure. However, an alternative formulation of our model that instead optimizes resource allocation, explicitly describing the effect of resource allocation on both individual life span and colony growth, would be equally valid and should produce a mathematically equivalent outcome unless there is no unique globally optimal solution.

We have chosen to focus on honeybees as a model system since they have a phenotypically plastic worker life span that is influenced by resource investment. In addition, the effects of resource investment on worker life span and the effects of worker life span on colony fitness are both measurable in honeybees, allowing us to estimate the shapes of benefit-and-cost curves. We have tailored this model to an example system to demonstrate how this modeling approach can make testable predictions about how ecological circumstances affect the evolution of life span.

Our model results agree with one of the main predictions of existing theory: that, all else being equal, higher extrinsic mortality should select for shorter intrinsic life span (fig. 1). This means that we expect colonies to invest fewer resources in workers that have a greater risk of death from external hazards such as predation. In honeybees, the largest change in extrinsic mortality occurs at the transition from in-hive work to foraging (Dukas 2008). As predicted, workers do experience reduced protein investment and, consequently, faster physiological senescence at the behavioral transition to foraging (Münch and Amdam 2010). Our model predictions are therefore consistent with the pattern of senescence associated with age polyethism in honeybees.

Our model also suggests that colonies should invest more in worker somatic maintenance when the benefit-to-cost ra-

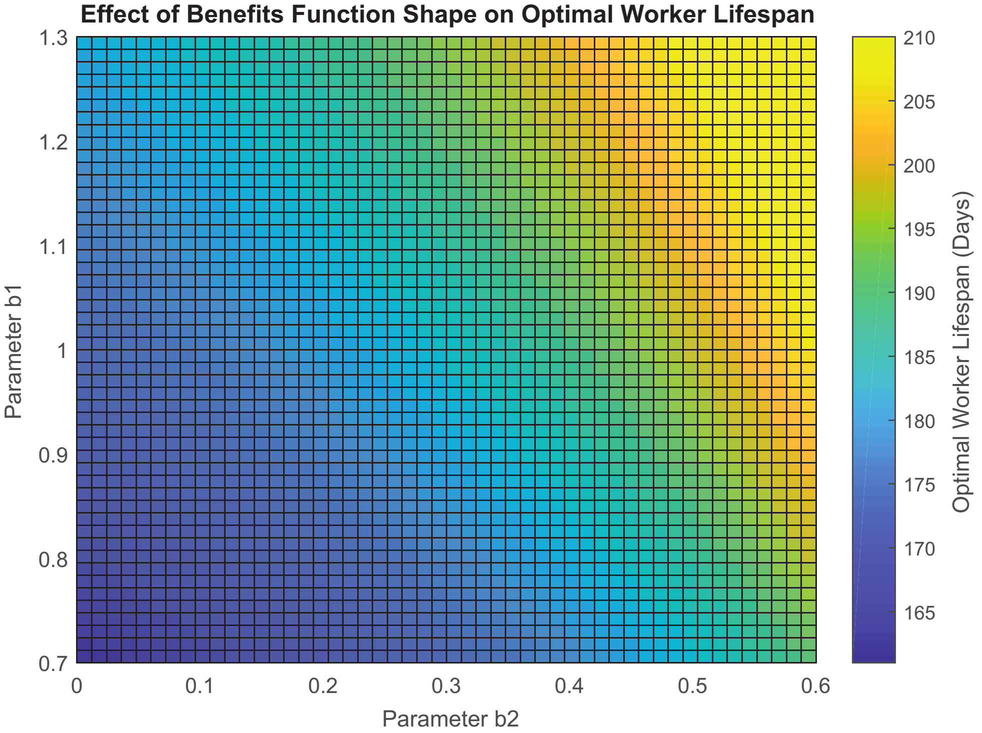

Figure 4: Effect of the benefit function on optimal worker intrinsic life span. Parameters b_1 and b_2 govern the shape of the marginal benefit function. Parameter $b_1 = 1$ means the marginal benefit is constant (benefits increase linearly with life span), $b_1 > 1$ means marginal benefits are decreasing (benefit function is saturating), and $b_1 < 1$ implies that marginal benefits are increasing (benefit function is exponential). Parameter b_2 governs whether the benefit function is sigmoidal: when $b_2 = 0$, the marginal benefits are monotonically increasing or decreasing, and when $b_2 > 0$, the marginal benefits increase to a certain point and then decrease (benefit function is sigmoidal). The optimal worker life span increases when marginal benefits are increasing ($b_1 < 1$) and decreases when marginal benefits are decreasing ($b_1 > 1$). We examined 51 values of parameter b_1 from 0.7 to 1.3 and 51 values of parameter b_2 from 0 to 0.6. Other parameters were held constant at $m = 0.11$, $q = 4$, and $c_1 = 0.95$.

tio increases, for example, when increased life span can be achieved at a lower cost (fig. 2). For honeybees, protein investment in workers should increase when pollen is more abundant. This prediction could be tested in honeybees by experimentally manipulating the amount or quality of protein available to colonies and measuring any changes in the intrinsic life span of adult workers. For other organisms, the return on investment in maintenance can change depending on the ecological circumstances. For instance, high resource abundance may favor phenotypes good at acquiring resources, allowing increased investment in reproduction without decreased investment in maintenance (Reznick et al. 2000).

Our model shows that an increasing marginal cost function selects for decreased intrinsic life span, compared to constant or decreasing marginal costs (fig. 3). Increasing marginal costs are likely to be a widespread pattern in nature because of the principle of low-hanging fruit: if there are multiple physiological mechanisms that can increase life span, organisms should first invest in pathways with the lowest unit cost. Increasing marginal costs can also occur when one kind of somatic damage makes other forms of maintenance costlier. For instance, wing wear, a major component of senescence in bees (Foster and Cartar 2011), probably increases the metabolic cost of flight; as a result, workers with greater accumulated wing wear would experience greater oxidative damage, which, if unrepaired, further accelerates senescence (Sohal and Weindruch 1996).

Our results also show that a pattern of increasing marginal benefits selects for greater intrinsic life span (fig. 4). If the marginal benefit function is constant, colonies should be indifferent between short-lived workers and long-lived workers so long as the total number of worker days stays the same. However, if the benefits of a worker are nonlinear with respect to age, colonies should prefer long-lived to short-lived workers when the benefits are increasing but prefer short-lived to long-lived workers when the benefits are decreasing. In honeybees, this suggests that workers should live longer when older bees are more valuable than younger bees; this situation occurs when learning increases an individual's value to the colony. For instance, in honeybees, older,

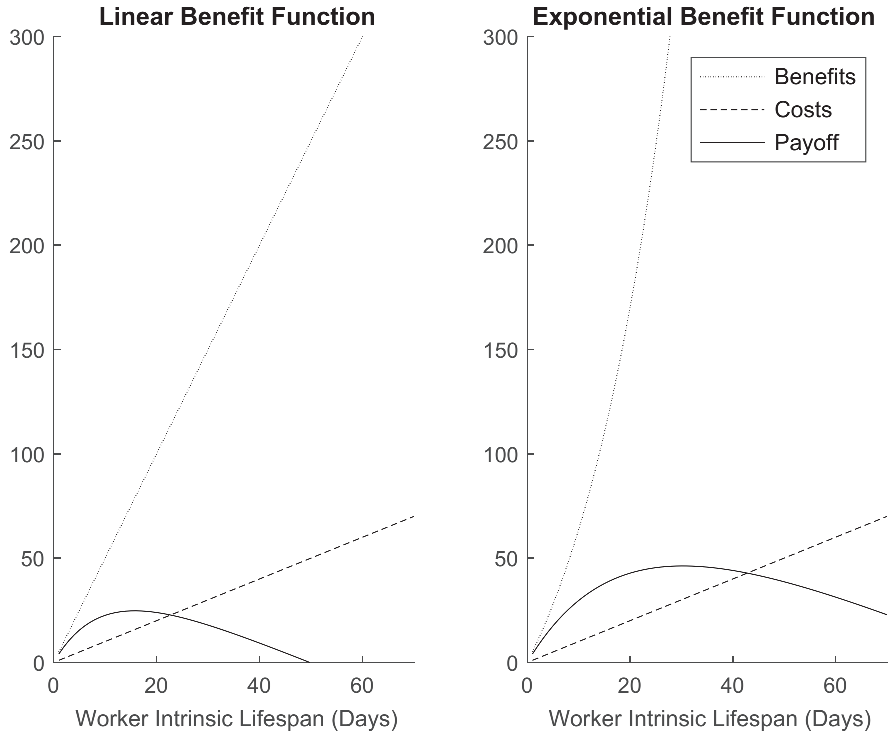

Figure 5: Effect of the benefit function on optimal worker life span in honeybees. As an application of this framework, we model the broodless period after swarming in honeybees as a change in the shape of the marginal benefit function. *Left*, optimal worker life span when the benefit function is linear. Parameter $b_1 = 1$. *Right*, during swarming, there is a threshold worker life span below which colony survival is low. The marginal benefit of each worker increases sharply above this threshold (benefits increase exponentially rather than linearly). An exponential benefit function results in increased optimal worker life span. Parameter $b_1 = 0.95$. Other parameters are held constant at $q = 5$, $c_1 = 1$, and $m = 0.1$.

more experienced foragers usually act as scouts during house hunting, making them especially valuable to the swarm during that period (Gilley 1998). Therefore, this model predicts long-lived workers to be more valuable at that time in the colony life cycle.

A pattern of increasing marginal benefits may also occur when there is a threshold effect or a minimum worker longevity that needs to be reached for a colony to survive a period where no new workers are produced. In honeybees, one such broodless period is winter, when workers are known to experience their slowest rate of aging (Amdam and Omholt 2002); another broodless period occurs immediately following reproductive swarming (Winston 1987). Our model predicts that changing the shape of the marginal benefit function alone can increase the optimal intrinsic worker life span even when extrinsic mortality is high, leading to the unusual prediction that honeybee workers may age slower rather than faster in colonies that have recently swarmed (fig. 5). This prediction could be tested empirically by comparing the protein status of workers from recently swarmed colonies to those from similarly sized colonies that have not swarmed; protein status could be measured as the level of stored vitellogenin, a lipoprotein that increases immune function and longevity in honeybees (Amdam et al. 2004; Seehuus et al. 2006) and that requires protein consumption to produce (Münch and Amdam 2010). Colonies should invest more in worker maintenance during swarming despite high extrinsic mortality because the benefit of long-lived workers exceeds the opportunity cost of additional lower-value, short-lived workers.

This work extends evolutionary senescence theory by providing a framework for examining how nonlinear costs and benefits affect the optimal life span in a social system. This model framework may be used with empirically estimated benefit-and-cost functions to make specific, testable predictions about how life span changes under different circumstances in organisms like the honeybee with adaptive plasticity as well as how life span evolves in different populations experiencing different ecological circumstances.

Acknowledgments

We would like to thank Drs. Peter Morin, Rachael Winfree, and David Tarpy for their valuable comments on the manuscript.

APPENDIX

Example Shapes of the Benefit Function Relating Worker Intrinsic Life Span to Colony Fitness

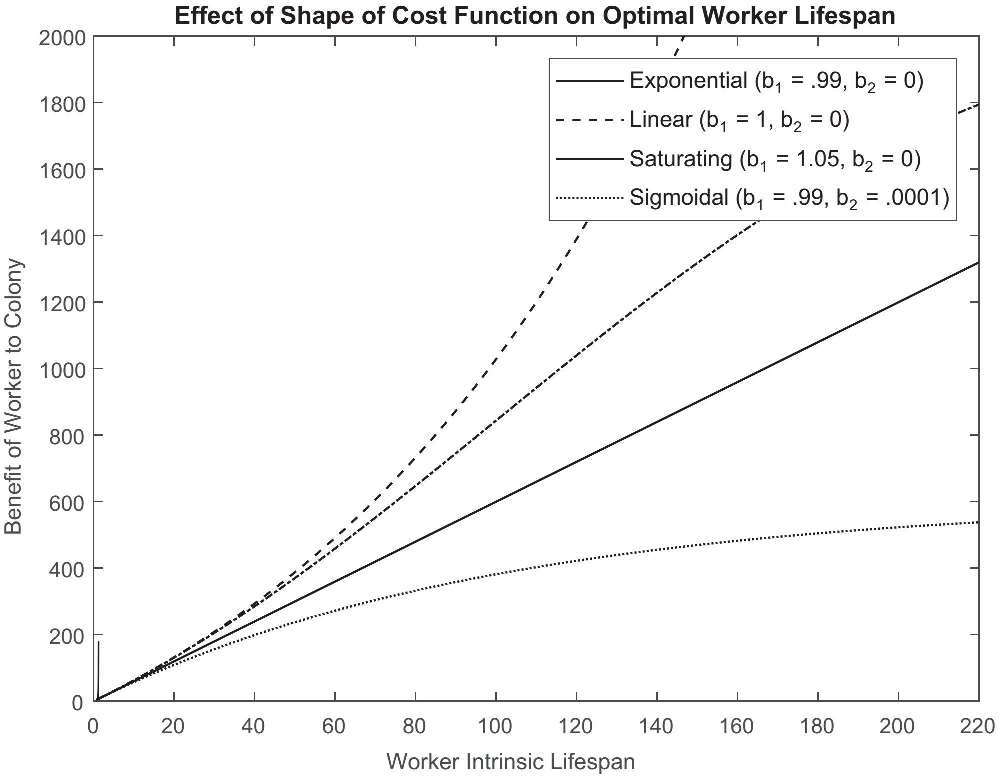

Figure A1: Four examples of benefit functions describing the benefit to the colony from workers having an intrinsic life span of *n* days. Parameters b_1 and b_2 determine the shape of the benefit function. When parameters $b_1 = 1$ and $b_2 = 0$, the benefit function is linear (solid line). When $b_1 > 1$ and $b_2 = 0$, the benefit function is saturating (dotted line). When $b_1 < 1$ and $b_2 = 0$, the benefit function is exponential (dashed line). When parameter $b_1 < 1$ and $b_2 > 0$, the benefit function is sigmoidal (dotted-dashed line).

Literature Cited

Abrams, P. A. 1993. Does increased mortality favor the evolution of more rapid senescence? Evolution 47:877–887.

Abrams, P. A., and D. Ludwig. 1995. Optimality theory, Gompertz' law and the disposable soma theory of senescence. Evolution 49:1055–1066.

Alaux, C., F. Ducloz, D. Crauser, and Y. Le Conte. 2010. Diet effects on honeybee immunocompetence. Biology Letters 6:562–565.

Amdam, G. V., A. L. T. O. Aase, S.-C. Seehuus, M. K. Fondrk, K. Norberg, and K. Hartfelder. 2005. Social reversal of immunosenescence in honey bee workers. Experimental Gerontology 40:939–947.

Amdam, G. V., and S. W. Omholt. 2002. The regulatory anatomy of honeybee lifespan. Journal of Theoretical Biology 216:209–228.

Amdam, G. V., Z. L. P. Simões, A. Hagen, K. Norberg, K. Schrøder, Ø. Mikkelsen, T. B. L. Kirkwood, and S. W. Omholt. 2004. Hormonal control of the yolk precursor vitellogenin regulates immune function and longevity in honeybees. Experimental Gerontology 39:767–773.

Austad, S. N. 1993. Retarded senescence in an insular population of Virginia opossums (*Didelphis virginiana*). Journal of Zoology 229: 695–708.

Bateson, M. 2007. Recent advances in our understanding of risk-sensitive foraging preferences. Proceedings of the Nutrition Society 61:509–516.

Baudisch, A. 2011. The pace and shape of ageing. Methods in Ecology and Evolution 2:375–382.

Bérubé, C. H., M. Festa-Bianchet, and J. T. Jorgenson. 1999. Individual differences, longevity, and reproductive senescence in bighorn ewes. Ecology 80:2555–2565.

Caswell, H. 2007. Extrinsic mortality and the evolution of senescence. Trends in Ecology and Evolution 22:173–174.

Chen, H. Y., and A. A. Maklakov. 2012. Longer life span evolves under high rates of condition-dependent mortality. Current Biology 22:2140–2143.

Chippindale, A. K., A. M. Leroi, S. B. Kim, and M. R. Rose. 1993. Phenotypic plasticity and selection in *Drosophila* life-history evolution. I. Nutrition and the cost of reproduction. Journal of Evolutionary Biology 6:171–193.

Cichoń, M. 1997. Evolution of longevity through optimal resource allocation. Proceedings of the Royal Society B 264:1383–1388.

Cichoń, M., and J. Kozłowski. 2000. Ageing and typical survivorship curves result from optimal resource allocation. Evolutionary Ecology Research 2:857–870.

Crailsheim, K., L. H. W. Schneider, N. Hrassnigg, G. Bühlmann, U. Brosch, R. Gmeinbauer, and B. Schöffmann. 1992. Pollen consumption and utilization in worker honeybees (*Apis mellifera carnica*): dependence on individual age and function. Journal of Insect Physiology 38:409–419.

Dowling, D. K. 2012. Aging: evolution of life span revisited. Current Biology 22:947–949.

Dukas, R. 2008. Mortality rates of honey bees in the wild. Insectes Sociaux 55:252–255.

Foster, D. J., and R. V Cartar. 2011. What causes wing wear in foraging bumble bees? Journal of Experimental Biology 214:1896–1901.

Gardner, S. N., and M. Mangel. 1997. When can a clonal organism escape senescence? American Naturalist 150:462–490.

Gasser, M., M. Kaiser, D. Berrigan, and S. C. Stearns. 2000. Life-history correlates of evolution under high and low adult mortality. Evolution 54:1260–1272.

Gilley, D. C. 1998. The identity of nest-site scouts in honey bee swarms. Apidologie 29:229–240.

Hamilton, W. D. 1966. The moulding of senescence by natural selection. Journal of Theoretical Biology 12:12–45.

Healy, K., T. Guillerme, S. Finlay, A. Kane, S. B. A. Kelly, D. McClean, D. J. Kelly, I. Donohue, A. L. Jackson, and N. Cooper. 2014. Ecology and mode-of-life explain lifespan variation in birds and mammals. Proceedings of the Royal Society B 281:20140298.

Holmes, D. J., and S. N. Austad. 1994. Fly now, die later: life-history correlates of gliding and flying in mammals. Journal of Mammalogy 75:224–226.

Keller, L., and M. Genoud. 1997. Extraordinary lifespans in ants: a test of evolutionary theories of ageing. Nature 389:958–960.

Kirkwood, T. B. 1977. Evolution of aging. Nature 270:301–304.

Kirkwood, T. B., and S. N. Austad. 2000. Why do we age? Nature 408: 233–238.

Kirkwood, T. B., and M. R. Rose. 1991. Evolution of senescence: late survival sacrificed for reproduction. Philosophical Transactions of the Royal Society B 332:15–24.

Kozłowski, J. 1993. Measuring fitness in life-history studies. Trends in Ecology and Evolution 8:84–85.

Kramer, B. H., and R. Schaible. 2013. Life span evolution in eusocial workers: a theoretical approach to understanding the effects of extrinsic mortality in a hierarchical system. PLoS ONE 8:e61813.

Law, R. 1979. Optimal life histories under age-specific predation. American Naturalist 114:399–417.

Lee, R. D. 2003. Rethinking the evolutionary theory of aging: transfers, not births, shape senescence in social species. Proceedings of the National Academy of Sciences of the USA 100:9637–9642.

Mangel, M., and S. B. Munch. 2005. A life-history perspective on short- and long-term consequences of compensatory growth. American Naturalist 166:E155–E176.

McNamara, J. M., and K. L. Buchanan. 2005. Stress, resource allocation, and mortality. Behavioral Ecology 16:1008–1017.

McNamara, J. M., and A. Houston. 1980. The application of statistical decision theory to animal behaviour. Journal of Theoretical Biology 85:673–690.

Medawar, P. B. 1952. An unsolved problem of biology. H. K. Lewis, London.

Miller, J. K. 2001. Escaping senescence: demographic data from the three-toed box turtle (*Terrapene carolina triunguis*). Experimental Gerontology 36:829–832.

Morbey, Y. E., C. E. Brassil, and A. P. Hendry. 2005. Rapid senescence in Pacific salmon. American Naturalist 166:556–568.

Münch, D., and G. V Amdam. 2010. The curious case of aging plasticity in honey bees. FEBS Letters 584:2496–2503.

Munch, S. B., and M. Mangel. 2006. Evaluation of mortality trajectories in evolutionary biodemography. Proceedings of the National Academy of Sciences of the USA 103:16604–16607.

Nesse, R. M. 1988. Life table tests of evolutionary theories of senescence. Experimental Gerontology 23:445–453.

———. 2005. Natural selection and the regulation of defenses: a signal detection analysis of the smoke detector principle. Evolution and Human Behavior 26:88–105.

Partridge, L., and K. Fowler. 1992. Direct and correlated responses to selection on age at reproduction in *Drosophila melanogaster*. Evolution 46:76–91.

Partridge, L., N. Prowse, and P. Pignatelli. 1999. Another set of responses and correlated responses to selection on age at reproduction in *Drosophila melanogaster*. Proceedings of the Royal Society B 266: 255–261.

Peterson, M. 2009. An introduction to decision theory. Cambridge University Press, Cambridge.

Piraino, S., F. Boero, B. Aeschbach, and V. Schmid. 1996. Reversing the life cycle: medusae transforming into polyps and cell transdifferentiation in *Turritopsis nutricula* (Cnidaria, Hydrozoa). Biological Bulletin 190:302–312.

Remolina, S. C., D. M. Hafez, G. E. Robinson, and K. A. Hughes. 2007. Senescence in the worker honey bee *Apis mellifera*. Journal of Insect Physiology 53:1027–1033.

Reznick, D. N., M. J. Bryant, D. Roff, C. K. Ghalambor, and D. E. Ghalambor. 2004. Effect of extrinsic mortality on the evolution of senescence in guppies. Nature 431:1095–1100.

Reznick, D. N., L. Nunney, and A. Tessier. 2000. Big houses, big cars, superfleas and the costs of reproduction. Trends in Ecology and Evolution 15:421–425.

Ricklefs, R. E. 2010. Life-history connections to rates of aging in terrestrial vertebrates. Proceedings of the National Academy of Sciences of the USA 107:10314–10319.

Rose, M. R. 1984. Laboratory evolution of postponed senescence in *Drosophila melanogaster*. Evolution 38:1004–1010.

Rueppell, O., C. Bachelier, M. K. Fondrk, and R. E. Page. 2007. Regulation of life history determines lifespan of worker honey bees (*Apis mellifera* L.). Experimental Gerontology 42:1020–1032.

Seehuus, S.-C., K. Norberg, U. Gimsa, T. Krekling, and G. V Amdam. 2006. Reproductive protein protects functionally sterile honey bee workers from oxidative stress. Proceedings of the National Academy of Sciences of the USA 103:962–967.

Seeley, T. D. 1997. Honey bee colonies are group-level adaptive units. American Naturalist 150(suppl.):S22–S41.

Sohal, R. S., and R. Weindruch. 1996. Oxidative stress, caloric restriction, and aging. Science 273:59–63.

Stearns, S. C., M. Ackermann, M. Doebeli, and M. Kaiser. 2000. Experimental evolution of aging, growth, and reproduction in fruitflies.

Proceedings of the National Academy of Sciences of the USA 97: 3309–3313.

Wilkinson, G. S., and J. M. South. 2002. Life history, ecology and longevity in bats. Aging Cell 1:124–131.

Williams, G. C. 1957. Pleiotropy, natural selection, and the evolution of senescence. Evolution 11:398–411.

Winston, M. L. 1987. The biology of the honey bee. Harvard University Press, Cambridge, MA.

Associate Editor: Jeff Leips
Editor: Alice A. Winn

“A scene in Cuba, with characteristic animals.” From the review of Wallace’s *Geographical Distribution of Animals* (*The American Naturalist*, 1877, 11:232–238).