Eusocial reproduction selects for longevity

Authors

Rafael D'Andrea[1]*, Charles D. Kocher[2]*, Bonnie Skiena[3], and Bruce Futcher[4]

1. Dept of Ecology and Evolution, Stony Brook University, NY11794
2. Laufer Center for Physical and Quantitative Biology, Stony Brook University, NY11794
3. 6 Storyland Lane, Setauket, NY 11733
4. Dept. of Microbiology and Immunology, Stony Brook University, NY11794

*Equal Contribution
Corresponding author: B. Futcher, bfutcher@gmail.com

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Author summary

We argue that eusocial populations have higher average ages than exponential ones, and grow more slowly and so have lower mortality from resource limitation. These two properties increase selection for longevity, which is further reinforced by unique "queen effects". The success of selection for longevity across eusocial species suggests the existence of multiple mechanisms of senescence—and multiple ways to evade it. This underscores that lifespan is a highly selectable and evolvable trait.

Author contributions

BF conceived of the study. BS and BF wrote and analyzed the health-points mortality computational model. RDA wrote and analyzed the Gompertz mortality computational model. CDK performed the analytical treatment. BF, RDA, and CDK planned the manuscript. BF wrote the manuscript with contributions from RDA and CDK. All authors contributed to manuscript edits and revisions.

Abstract

Naked mole-rats have exceptionally long lifespans and reproduce eusocially, with a single female—the queen—producing all offspring. Other eusocial species, such as bees, ants, and termites, also have long lifespans relative to their solitary kin. We propose that eusociality drives the evolution of longevity through three mechanisms: first, individuals in eusocial populations tend to be older than in non-eusocial populations, and thus genes favoring survival at later ages are more strongly selected; second, slower growth in eusocial populations reduces early mortality from resource scarcity, making longevity more important. Finally, the fitness benefits of long lifespan are stronger when the population's reproductive success rests on the survival of a single individual. Non-eusocial species reproducing in ways that partly access these benefits also have relatively long lifespans. We demonstrate these three mechanisms using mathematical models and computer simulations, highlighting longevity as an evolvable, selectable trait. Our results echo conclusions from Life History Theory but cast them in a new light.

Introduction

Humans and other animals have an exponentially increasing risk of death with age. In humans, the risk of dying increases by about 8% per year after sexual maturity, such that the risk of dying doubles about every 9 years. This exponential increase in mortality with age was described by Gompertz (1), and is typical of many animals (2-6), although exceptions are also seen (7). The mechanisms behind exponentially-increasing mortality are not known.

Among the apparent exceptions is the naked mole-rat, *Heterocephalus glaber*. This unusual mammal, the only species in its genus, lives in underground burrows in Africa, eating tubers, and sometimes eaten by snakes (8). Naked mole-rats reproduce eusocially: that is, a colony has only one reproductive female. Naked mole-rats have strikingly long lifespans of up to 40 years (9-11). Mammalian life spans are generally well-correlated with body size (12, 13), but naked mole-rats are roughly mouse-sized mammals that live about five-times longer than expected for their size (14, 15). For example, with a lifespan of about 40 years and a mass of about 35 grams, naked mole-rats live at least 10 times longer than *Mus musculus*, which has a lifespan of 1.5 to 4 years and a mass of about 20 grams. Recent studies suggest that naked mole-rats have a flat Gompertz curve with a slope near zero. That is, naked mole-rats apparently do not face a significantly increased chance of dying with older age (11, 16, 17).

Other eusocial species also have strikingly long lifespans (18-21). The 2,600 known species of termites are eusocial, and these have remarkable lifespans: for example, queens of the species *Macrotermes michaelseni* have a mean lifespan of about 18 years (18). Similarly, the mean lifespan of queens across ant species is 10 years, 11.5 years in termites, and 5.6 years in honeybees. By comparison, the mean lifespan of solitary species

is about 0.1 years (19). Lifespans in queens of eusocial stinging wasps range from about 100 to 10,000 days, compared to 10 to 100 days among solitary stinging wasps (22). Although workers live about one tenth as long as queens, they still commonly live 10-fold longer than their equivalents in non-eusocial species (22). For instance, in a eusocial honeybee species where queens live about 1,080 days, workers live about 180 days. In contrast, sawflies, a non-eusocial insect from another suborder of Hymenoptera, have lifespans of 7 to 9 days. We are not aware of any exceptions to the general rule that eusocial species have long lifespans.

Several researchers have studied this association between long lifespan and eusocial reproduction. It has been suggested that long lifespan causes eusocial reproduction (23, 24), and also that eusocial reproduction causes long lifespan (19); (see (25) for a discussion). The general argument for the latter is that species with eusocial reproduction have low extrinsic mortality (i.e., mortality from outside forces, such as predators, rather than internal factors such as cancer), which enables long lifespans and the selection of mutations promoting longevity. Specifically, it has been suggested that eusocial species are often found in a protected environment—a hive, or nest, or burrow—where individuals, especially the queens, are protected from extrinsic dangers (19, 26-28). Oster and Wilson described a colony of eusocial insects as "a factory within a fortress" (29). However, it has been questioned whether low extrinsic mortality is a complete explanation (30, 31), and other forces may be relevant (32).

Here we show that the dynamics of eusocial population growth causes selection for long lifespan regardless of sheltering from harsh environments. Eusocial reproduction leads directly to populations with higher average ages, and also to slower population growth which reduces early mortality from resource limitation. These cause selection for longevity. In addition, we suggest that funneling reproduction through a single individual, the queen, may on its own cause selection for longevity.

Results

Rationale: Figure 1A compares population growth under the usual reproductive strategy and the eusocial reproductive strategy. Temporarily ignoring death and resource constraints, the number of offspring grows exponentially with time under regular reproduction, but linearly under eusocial reproduction. Ignoring death and keeping other parameters constant, the median age in this regular-reproduction population is 0 (the age of newborns), while in the eusocial population, the median age is (queen lifespan/2). Thus, other things being equal, eusocial populations will tend to be smaller, with older individuals. As a shorthand, we will henceforth refer to eusocial reproduction as linear reproduction, and regular reproduction as exponential reproduction. Of course, eusocial/linear populations will eventually produce new colonies and in that sense will eventually reproduce exponentially. However, the parameters of reproduction of

individuals in a colony are different from those of reproduction of the colony itself, and can be dealt with separately—see Discussion.

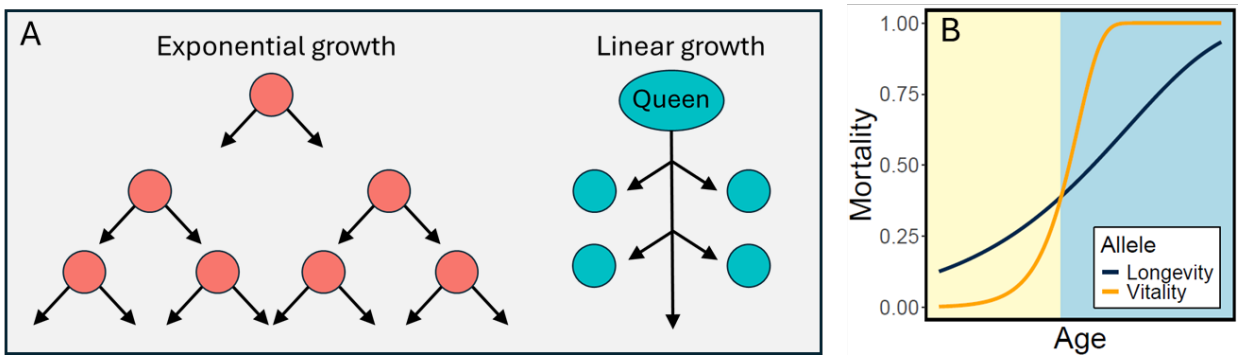

Figure 1. A. Diagram of population growth in normal vs eusocial reproduction. When all individuals reproduce, the population grows exponentially, and median ages are young (in fact, 0). When only the queen reproduces, the population grows linearly, and median ages are older ((queen age)/2). **B.** Gompertz mortality curve (see Equation 6) for two alleles. Lower values of α (high "vitality") benefit younger individuals (left side, yellow), while lower values of β (high "longevity") benefit older individuals (right side, blue). Parameters: Longevity allele ($\alpha = 0.125, \beta = 0.15$), Vitality allele ($\alpha = 0.002, \beta = 0.6$).

Age-dependent mortality in humans and other animals can be described by the Gompertz-Makeham law, where the hazard function at age n is $h(n) = \alpha e^{\beta n} + \gamma$. Parameter γ reflects the age-independent component of mortality. Parameter α sets mortality at age 0; that is, the baseline death rate before senescence, reflecting a young individual's resistance to death due to strength, health, or durability— a trait we call "vitality". The β parameter determines the steepness of the mortality curve, or how quickly survival decreases with age, which we refer to as "longevity". For instance, survival might decline with a half-life of 8 years, as in humans. A shallower curve implies increased longevity.

In Gompertz death (Fig. 1B), high α or β lead to high mortality (more accurately, high hazard). For convenience, we prefer to consider the inverse, survival. In what follows, our "vitality" parameter is related to the inverse of α, and our "longevity" parameter is related to the inverse of β. Thus, high vitality or high longevity lead to high survival.

Figure 1B compares Gompertz curves of two species, one of which (gold-colored line) has a relatively high vitality, and one of which (dark blue line) has a relatively high longevity. Fig. 1B shows that at young ages, the population with high vitality has lower mortality (yellow region of plot), while at old ages, the population with high longevity has lower mortality (blue region of plot). That is, youngsters "prefer" vitality, while oldsters "prefer"

longevity. With any reasonable parameters, the two lines must cross as age increases: before the cross, vitality is preferred, while after, longevity is preferred.

When Figs 1A and 1B are considered together, they illustrate two intuitive reasons why linear (i.e., eusocial) reproduction selects for longevity:

1. Age distribution. The median age in an exponential population will be younger than in an otherwise equivalent linear population. In fact, the median age in the exponential population of Fig. 1 (without death) is 0, the age of a newborn, while the median age in the linear population is (queen age)/2. For instance, after 8 generations, the median age in the exponential population would be 0, and in the linear population would be 4. Therefore, an exponential population tends to live on the left side of Fig. 1B, where vitality is preferred, while a linear population tends to live on the right side, where longevity is preferred. Therefore, selection for longevity will have a greater effect in a linear population.

2. Resource Pressure. Growing populations will eventually reach an equilibrium size at the carrying capacity of their environment. Exponential populations, with their high rate of reproduction, will push hard against this population cap, and resource limitation will be a major source of death. This death will often be at relatively young ages, set by lack of resources rather than by senescence. Successful alleles will therefore promote vitality. In contrast, linear populations, with a smaller capacity for reproduction, will push less hard against the population cap, and other sources of mortality such as senescence will be relatively more important, and hence more susceptible to natural selection. Successful alleles will promote longevity. The result will be that exponential populations will have shorter lifespans, and prefer vitality, while linear populations will have longer lifespans, and prefer longevity (see above, and Fig. 1).

Queen Effects. In addition to these two intuitive forces, there are "queen effects" arising from the channeling of reproduction through a single individual. For example, in a linear population, a mutant allele for increased longevity of the queen will spread because it will produce significantly more offspring than its wildtype allele—i.e., it will have higher fitness. In an exponential population, this allele would have a smaller advantage, since offspring of older females would form only a small portion of the next generation due to the much greater number of younger reproductive females. This is a selfish-gene effect; the allele for longevity has greater fitness than its alternative allele, but it does not increase the "fitness" of the population (assuming that upon death, a queen is replaced by some formerly sterile female). Other potential "queen effects" are described in the Discussion.

Approach: Williams' Alleles. To study these ideas, we used an approach inspired by G.C. Williams idea of "antagonistic pleiotropy" (33), in which one gene produces two antagonistic phenotypes. We note that both high vitality and high longevity can lead to long lifespan. We created "Williams' alleles", in which one gene has two effects in opposite directions, one effect on vitality, and the other on longevity (i.e., on the α and β parameters of the Gompertz curve). In our analytical approach, we balance vitality against

longevity, and find that in linear populations, longevity has relatively more importance than vitality, and the reverse in exponential populations. In the first of our numerical approaches, we compete two Williams alleles against each other in simulations. For instance, one allele, the vitality allele, might carry a relatively high value for vitality and a relatively lower value for longevity (e.g., vitality = 1000, longevity = 9), and a second, competing Williams' allele, the longevity allele, might be the converse (vitality = 900, longevity = 10). We then compete the two alleles and ask which allele is preferred. We find that over a wide range of parameters, whichever allele has the higher vitality (1000 vs 900) is preferred by exponential populations, while whichever allele has the higher longevity (9 vs 10) is preferred by linear populations.

Findings: Analytical approach. First, we mathematically modeled natural selection acting on populations with linear or exponential reproduction to explore the relative fitness advantages of longevity in each scenario. Many different combinations of vitality and longevity of a pleiotropic Williams' allele can yield the same fitness, since gains in one can offset losses in the other. We analytically mapped the combinations of vitality and longevity that maintain constant fitness to generate fitness nullclines. As Fig. 2 shows, the calculated nullclines for exponential and linear populations have very different slopes. In the exponential population, a small change in vitality requires a large change in longevity to offset it, whereas in the linear population, the same small change in vitality can be offset by only a moderate change in longevity—in other words, longevity is relatively more potent in the linear population. In comparative terms, this suggests that exponential populations "care" more about vitality while linear populations "care" more about longevity.

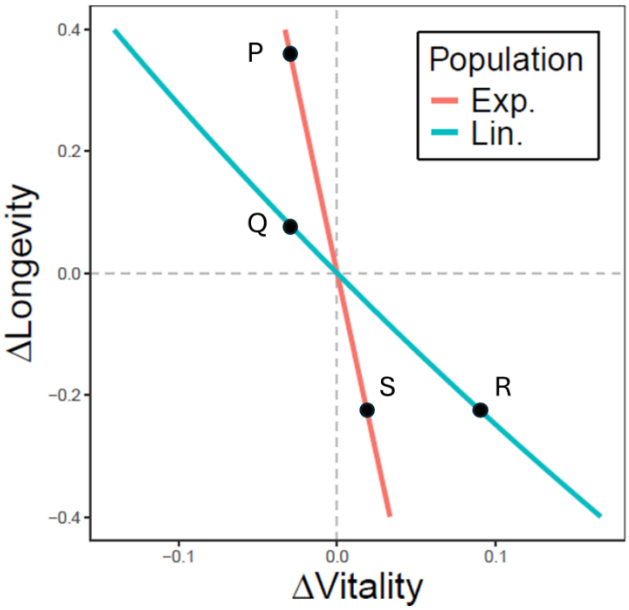

Figure 2: Calculated fitness nullclines (Methods: Equation 8) for alleles balancing vitality versus longevity in exponential (red) and linear populations (blue). Alleles on these nullclines all have equal fitness. The exponential population's steeper curve shows that it requires a larger gain in longevity to offset a given loss in vitality (point P) compared to the linear population (point Q). Conversely, the linear population requires a larger gain in vitality to offset a given loss in longevity (point R) compared to the exponential population (point S).

Numerical approaches. While these results are suggestive, the analytical treatment requires simplifying assumptions and bypasses the genetics of reproduction, thus precluding queen effects. Therefore, we used two independent agent-based models which

simulate dynamics in populations with exponential or linear reproduction. The models implemented Gompertzian death in entirely different ways as a safeguard against idiosyncratic results**.** We explored a variety of parameters in addition to vitality and longevity, including time to sexual maturity, probability of reproduction, litter size, the degree of resource limitation, and genetic dominance.

As above, these models used two competing Williams' alleles, one with a slightly higher vitality, the other with a slightly higher longevity. We competed these two Williams' alleles in populations, starting from an initial frequency of 50% each, and asked which allele "won" the competition by becoming fixed (i.e., rising to 100% frequency). This analysis confirmed that exponential populations favor vitality, while linear populations favor longevity (Fig. 3). This outcome held across a wide range of parameter combinations. Notably, when all parameters were identical between the two reproductive strategies, we found no scenario in which their preferences reversed.

When both reproduction types share the same parameter values, exponential populations are consistently much larger and composed of much younger individuals than linear populations (Fig. 3 A-C). This is primarily because rapid population growth in the exponential case quickly leads to resource limitation, bringing death upon individuals of all ages and curbing further population growth. This pattern holds across a range of parameter values and multiple resource limitation scenarios.

Example outcomes of our Williams' allele competition experiments are shown for exponential populations (Fig. 3D) and linear populations (Fig. 3E). We competed a vitality allele against a longevity allele—with the former providing 10% higher vitality but 10% lower longevity compared to the latter, with each initially comprising 50% of the population in both population types. The longevity allele fixes (i.e. reaches 100% frequency) more often in the linear population. Across 450 simulations of each population type, the longevity allele fixed in 58% of the linear populations but only 9% of the exponential populations (Fig. 3F). Results were similar regardless of which allele was dominant.

We note that the discrepancy in relative success of the two Williams' alleles between the two population types is likely even larger than suggested by these results. The linear population has a very small effective population size (Ne ~ 10, often less) due to the genetic bottleneck caused by a single reproductive female. This causes powerful genetic drift in the linear population, such that even strongly disfavored alleles can fix frequently. The difference in effective population sizes between the two kinds of populations makes it very difficult to calculate comparable selection co-efficients.

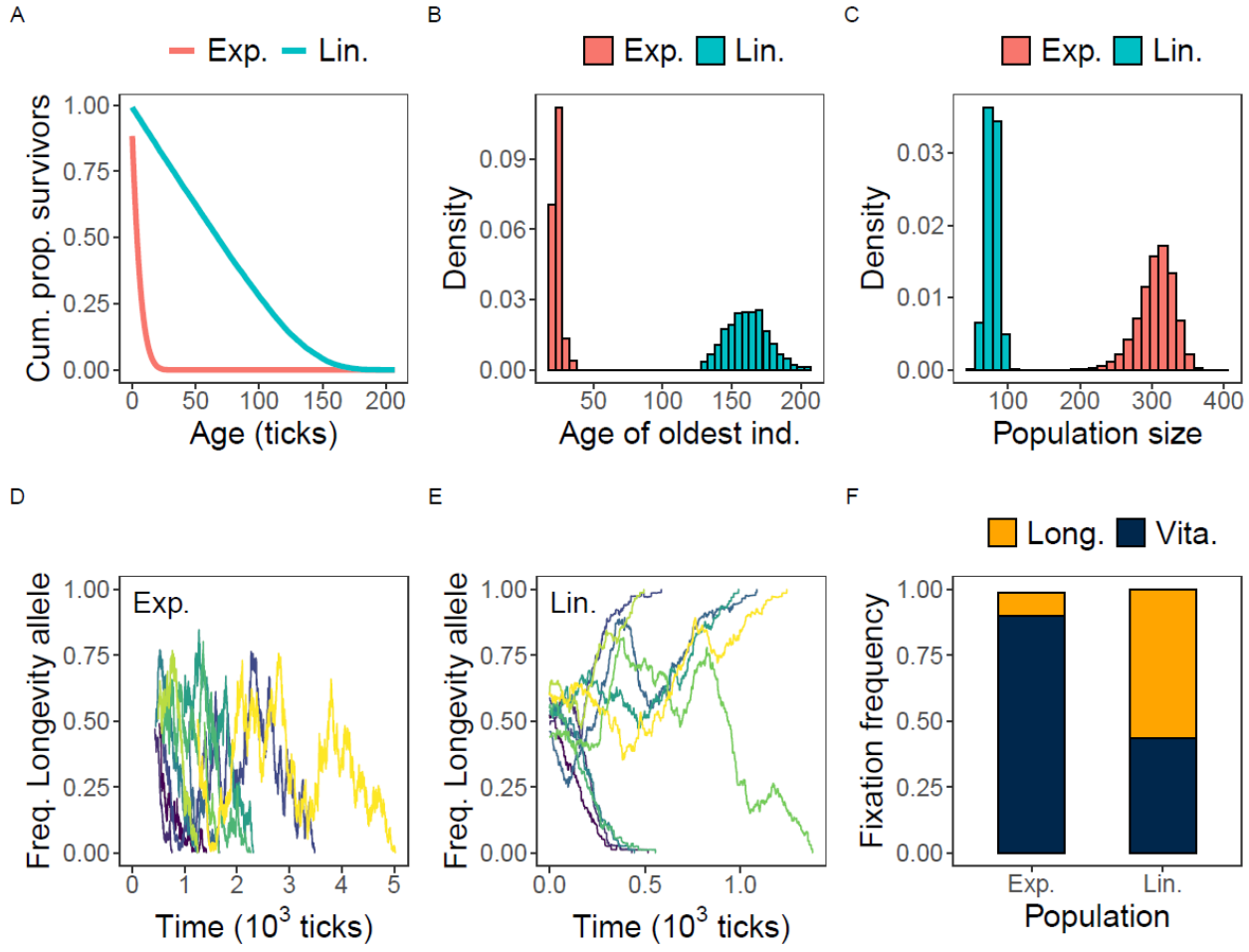

Fig. 3. **A**. Age distributions in the exponential (red) and linear (blue) populations, shown as the cumulative proportion of surviving individuals of each age over 450 simulations. **B**. Distribution of lifespans, shown as histograms of the ages of the oldest surviving individuals over 450 simulations. **C**. Distributions of population size. **D**. Time series of the relative frequency of the longevity allele for 9 simulations of the exponential population. **E**. Time series of the relative frequency of the longevity allele for 9 simulations of the linear population. **F**. Relative fixation frequencies of competing vitality (dark blue) and longevity (yellow) alleles across simulations of exponential and linear populations. The vitality allele had Gompertz parameters R9 HP1000 and the competing longevity allele had Gompertz parameters r10 hp900 (See Supplementary Information). Upper case denotes dominance. Fixation frequencies were calculated from 450 runs of the linear population and 444 runs of the exponential population (neither allele fixed in 6 runs). Similar results were obtained when dominance was reversed.

Queen Effects. We can adjust parameters so that a linear population matches the size and age structure of an exponential population. For example, increasing the queen's fecundity in the linear population boosts population size (and thereby shortens lifespans via resource limitation), whereas delaying sexual maturity, reducing the probability of reproduction, or shrinking litter size in the exponential population has the opposite effect. Indeed, natural variation in these life history traits across eusocial and non-eusocial populations can in principle neutralize the age-distribution and resource-pressure effects and thus mitigate longevity differences in nature. However, the populations still differ in that the linear population channels reproduction through a single individual.

To isolate the effects of channeling reproduction through a queen, we adjusted parameters in our Gompertz Mortality model (see Materials and Methods) to equalize population size and age distribution between linear and exponential populations. This allowed us to test how removing these differences influenced the competition dynamics of Williams alleles. Here, a wildtype Williams allele is invaded by a mutant, initially at frequency p_0, that conferred higher vitality but lower longevity. We measured the mutant's fixation rate f and calculated its fixation bias $\mathrm{FB} = f/p_0 - 1$, where FB = 0 indicates a neutral mutant whose vitality advantage exactly offsets its longevity disadvantage. To identify this neutral mutant in each population type, we varied the vitality advantage while keeping longevity loss fixed and determined the threshold at which FB changes sign. Our results show that in exponential populations, a lower vitality advantage is required to balance the longevity cost compared to linear populations (Fig. 4). Thus, even in the absence of age and resource pressure effects, the linear population shows a persistent albeit smaller predilection for longevity relative to the exponential population.

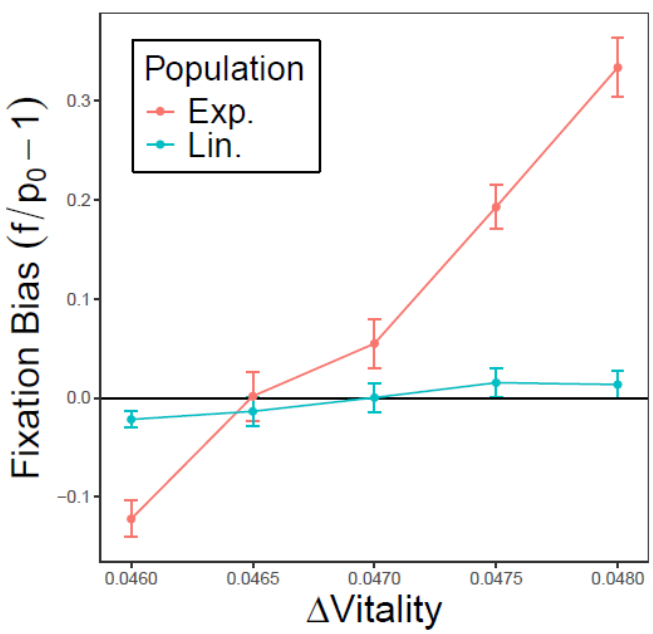

Figure 4: Fixation bias of an invading allele with higher vitality but lower longevity than the wildtype in exponential (red) and linear populations (blue). The x-axis shows the vitality difference between the invader and wildtype, while the y-axis shows f/p_0 -1, where f is the observed fixation probability and p_0 is the initial frequency of the invader. The intersection of each curve with y = 0 marks the vitality advantage required for the lower-longevity invading allele to match the wildtype. While both selection and effective population size influence the steepness of the curves, only selection affects the intersection point.

Note that while the intercepts of the curves with the neutral line indicate differences in selection for longevity between the two population types, their slopes are influenced by effective population size. The smaller effective size of the linear population results in a shallow slope, because of drift, effectively dampening the apparent strength of selection.

Discussion

We suggest that all else being equal, a switch in reproductive strategy from regular (exponential) to eusocial (linear) would change population structure: the population size would decrease, causing less pressure against the environmental resource cap, and the average age would increase. These changes would cause selection for increased longevity, especially in the queen. In contrast, a species switching from linear to exponential reproduction would experience larger, younger populations, more pressure against the resource cap, and less selection for longevity.

Consistent with this, life history strategies that slow down exponential reproduction tend to select for longevity (34). While linear reproduction is generally slower than exponential reproduction, the effects of linear reproduction on population size and age distribution can be mimicked by exponentially-reproducing species. For example, K-selected traits such as increased time to sexual maturity, decreased litter size, and increased time between litters would all cause smaller population sizes and lower resource pressure, higher average age, and increase selection for longevity (34). Possible examples are humans, whales, elephants, and the Greenland shark (35, 36).

Beyond age- and size-related effects, the channeling of reproduction through a single individual can generate “queen effects” that are unique to eusociality. Queen effects can result from at least three different mechanisms. First, an invading gene that increases queen longevity has a fitness advantage over the wildtype, as the queen monopolizes reproduction. In an exponentially reproducing population the same gene would confer a smaller advantage because the long-lived female’s offspring constitute a small fraction of the total population. Second, under linear reproduction, the queen’s alleles tend to fix due to the small effective population size. If the queen outlives other females, fixation becomes even more likely, reinforcing a selfish gene dynamic (i.e. the gene spreads regardless of its impact on population-level fitness). The effect is particularly pronounced when only a few males are reproductive, as seen in many eusocial species. By becoming a long-lived queen, an individual can effectively exclude competing alleles, ensuring the dominance of her own genetic lineage.

A third potential mechanism for the queen effect involves investment of resources in physical size and strength of the queen, improving her overall survival and reproductive capacity. In an exponential population, all females would have to be so endowed, incurring a larger resource cost. Once queen vitality is improved, the selective advantage of improved longevity increases. Queens that are larger, stronger, and longer-lived than

worker females are well-documented in bees, ants, wasps, termites, and naked mole rats (e.g., (37-39)). The concept is vividly displayed in fiction (40). Critically, whereas improved size and strength require resource investment and are thus limited to the queen, improved longevity might not require additional resources and thus might not incur a cost if expressed in the workers. Thus, longevity-promoting alleles that are initially selected in queens could come to be expressed throughout the population. In line with this reasoning, previous simulation work has shown that lifespan differences can evolve between queens and workers (31). In fact, in the eusocial termites and in Hymenoptera and in naked mole rats, both workers and queens tend to outlive their non-eusocial kin, with queens exhibiting particularly extended lifespans (e.g. (22, 41, 42)).

There is a substantial literature describing possible mechanisms for the long life of naked mole-rats. These mechanisms include increased hyaluronan (43), an activated INK4a-RB death senolytic death pathway (44), elevated glycogen, elevated expression of HIF-1α, and reduced succinate levels (45), resistance to oxidative stress (46), an improved immune system (47), an improved apolipoprotein A-I (48), extreme tolerance to hypoxia (49), improved regulation of splicing (10), mitochondrial anti-oxidant defenses (50), more efficient DNA repair (51, 52), improved proteostasis (53), better telomere maintenance (54, 55), and many other proposed mechanisms. Our work does not address mechanism, but gives a reason why longevity would be selected in the naked mole-rat, likely via many mechanisms.

Fig. 1A shows a population pyramid of exponential reproduction and rapid expansion. A broad, short pyramid would represent fast-reproducing species like mice, while a tall, narrow pyramid would represent slow-reproducing species like blue whales. As reproduction slows, populations become smaller with higher average ages, resembling linear reproduction and favoring longevity. We propose that linear (eusocial) reproduction is the limiting case of slow exponential reproduction, where both lead to older populations, reduced resource pressure, and stronger selection for long lifespan. (A related argument has been made by de Vries et al. (56).) An example of an exponentially-reproducing species with a tall, narrow reproductive pyramid is the Greenland shark (*Somniosus microcephalus*). It reaches sexual maturity at around 150 years, has an 18-year pregnancy, and is the longest-lived vertebrate known, with a lifespan of about 400 years (35, 36).

This “pyramid width” argument can be applied to eusocial species when they reproduce exponentially at the colony level. A queen produces new queens that establish colonies, but colony founding is slow, resulting in a tall, narrow reproductive pyramid. While little is known about naked mole-rat colony reproduction, that of honeybees is well studied (57). A successful hive typically produces about one new queen per year, or somewhat more, but high mortality (~25% survival for new colonies) limits expansion. If a hive produces one to two new colonies annually, this suggests a colony reproductive rate faster than humans but slower than mice. This fits with the queen bee’s lifespan (~6 years) falling between that of humans and mice, consistent with the pyramid width hypothesis.

Our perspective is consistent with life history theory (34, 57, 58) and can be framed as a life history argument. However, our approach is simple, relying on only a few population properties while producing large effects. For exponentially reproducing populations, our conclusions broadly match traditional life history predictions (e.g., "live fast, die young" vs. "slow and steady" strategies). However, the comparison with linear reproduction puts these strategies in a new light. Eusocial species such as ants, bees, wasps, and naked mole-rats do not obviously fit onto the traditional "live fast" vs "slow and steady" axis, but when analyzed as here, it can be seen that they are molded by some of the same underlying forces as "slow and steady" species, though in a non-standard way.

Our conclusions also align with previous research on the long lifespans of eusocial animals, which often attributes their longevity to lower extrinsic mortality, possibly due to sheltered environments such as hives or nests (19, 20). While we also invoke a kind of reduced extrinsic mortality, we propose that it arises primarily from slower population growth, which eases resource competition, rather than from fortress homes.

In summary, we argue that linear (eusocial) reproduction leads to higher population ages, and to reduced resource pressure, and these select for longevity. In addition, there are "queen effects" that also select for longevity. This selection for longevity is evidently successful. That selection for longevity is successful over such a wide variety of organisms argues that there are likely multiple mechanisms of senescence, and multiple mechanisms of evading senescence, which could differ between organisms. Lifespan would appear to be an evolvable trait, highly responsive to selection.

Methods

Analytical model

Our analytical model considers a contest between a Williams allele and a reference wild-type. We tracked the number of individuals of each phenotype at each age, following the discrete-time update equations:

$$A_0(t+dt) = X_A \qquad (1)$$

$$A_{n+1}(t+dt) = (1 - D_n)\, A_n(t) \qquad (2)$$

$$B_0(t+dt) = X_B \qquad (3)$$

$$B_{n+1}(t+dt) = (1 - d_n)\, B_n(t) \qquad (4)$$

Here, $A_n(t)$ and $B_n(t)$ are the abundances of individuals of age n carrying each allele at time t, and $X_{A,B}$ is the number of births of either type during a time interval dt, which depends on the abundances of both alleles at time t and the reproductive mode of the population (exponential or linear). Individuals of age n survive to age $n+1$ (measured in timesteps of dt) with probability $1 - D_n$ for wild-type allele A and $1 - d_n$ for Williams allele B. The death probabilities D_n and d_n, assumed density-independent for simplicity, increase with age according to the Gompertz-Makeham law:

$$h(n) = \lim_{dt \to 0} D_n/dt = \alpha e^{\beta n} + \gamma, \qquad (5)$$

where the positive constants α and β determine the mortality of newborns and how quickly mortality increases with age, respectively. The concepts of vitality and longevity as used in this study are thus inversely related to α and β, respectively. The age-independent component γ is related to the carrying capacity of the population (see Appendix 1 in the Supplementary Information). Correspondingly, we can find that the death probability, or the mortality, is

$$D_n = 1 - \exp\left[-\left(\frac{\alpha}{\beta}\right) \exp(\beta n) \left(\exp(\beta\, dt) - 1\right) - \gamma\, dt\right] \quad (6)$$

(see Equation S.18). It is this age-dependent component ($\gamma = 0$) of this quantity that is plotted in Figure 1B, with $dt = 1$ scaling the time units.

We characterize our Williams allele based on different choices for α and β, and the tradeoff between longevity and vitality is implemented via a tradeoff between those two parameters. That is, we set

$$\lim_{dt \to 0} d_n/dt = (1 - x)\alpha e^{(1-y)\beta n} + \gamma, \quad (7)$$

where x and y--the fraction change in α and β, respectively, from the reference wild-type--must be of opposite signs. If $x > 0$ and $y < 0$ (quadrant 4 of Fig 2), then allele B confers higher vitality and lower longevity than allele A; when the signs are switched (quadrant 2 of Fig 2),the opposite is true.

The winner of the competition between alleles A and B is determined by the difference in their respective finesses, given by equation 8 below (see Appendix 1 in the Supplementary Information for a proof):

$$\Delta F = \sum_{n=1}^{\infty} \prod_{i=0}^{n-1} (1 - D_i) - \sum_{r=1}^{\infty} \prod_{k=0}^{r-1} (1 - d_k) \quad (8)$$

This corresponds to the difference in survival to age n summed over all ages $n > 0$. If ΔF is positive, allele A wins. If it is negative, B wins.

Using SciPy's *fsolve* root-finding algorithm, we numerically searched the vitality-longevity parameter space to find the values of (x, y) for which $\Delta F = 0$. These roots correspond to points where both alleles have equal fitness. We did this procedure for a linear population and an equivalent exponential population (as described in Appendix 1 in the Supplementary Information), yielding the fitness nullclines in Figure 2. This analysis revealed that the linear population is more sensitive to longevity changes than the exponential population.

Numerical models

The Health Points and Insults model

This is an agent-based model in Python. Individuals are diploid male or female and sex frequencies average 50%. In exponential populations, mating between males and females is random. In eusocial/linear populations, there is only one mating female, the queen. Upon death of the queen, she is replaced randomly by some other female. Genes assort in the usual way by Mendelian genetics, and genes can be recessive or dominant. Time in the simulation proceeds in steps called "ticks".

The model balances births against deaths. Births are governed by parameters including time to sexual maturity, birth probability, and litter size. Deaths are governed by age-dependent mortality according to the Gompertz law and by a logistic equation that increases mortality with increased population size. As populations grow, density-dependent mortality increases, and populations come to an equilibrium size with births balancing deaths. We introduce various alleles that affect parameters of the Gompertz equation and study their effects. These "Williams alleles" influence the α and β parameters of the Gompertz equation in opposite directions. Typically, we compete one allele with high vitality (low α, high β) against another with high longevity (high α, low β) and observe which allele becomes fixed in a simulated population under linear or exponential reproduction. See Appendix 2 in the Supplementary Information for details.

The Gompertz Mortality model

To isolate the influence of the queen effects, we compared longevity and vitality alleles in exponential populations with similar size and age distribution. We simulated a population initially monomorphic for a wildtype Williams allele (see simulation details in SI). Once the age distribution reached a steady state, we introduced a specified number of individuals carrying a mutant allele which confers lower longevity but higher vitality (i.e. higher β and lower α) than the wildtype. We tested a range of invading alleles with the same reduced longevity but varying vitality. For each mutant allele, we repeatedly simulated its invasion and estimated the fixation probability as the proportion of simulations where the invader fixed. This observed fixation probability was then compared to the expected fixation probability of a neutral allele, equal to its initial frequency. We defined the fixation bias (FB) of the invading allele as FB = $f/p_0 - 1$, where f is the estimated fixation probability and p_0 is the initial frequency of the allele, calculated as the number of introduced mutant individuals divided by the total population size at the time of invasion. A positive value of FB indicates that the allele fixed more often than expected from neutrality, indicating positive selection; conversely for a negative FB. Thus, mutant alleles satisfying FB = 0 are those whose vitality boost exactly offsets their reduced longevity. Finding these alleles in both the exponential and linear populations gives a measure of the relative strength of selection for vitality versus longevity in each. This measure is robust to differences in effective population size between exponential and linear populations since, while effective population size affects fixation probability (and hence FB magnitude), only selection determines the bias from neutrality (FB sign).

Competing Interests

The authors declare no competing interests.

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