ALTRUISTIC AGING: THE EVOLUTIONARY DYNAMICS BALANCING LONGEVITY AND EVOLVABILITY

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Abstract. Altruism is typically associated with traits or behaviors that benefit the population as a whole, but are costly to the individual. We propose that, when the environment is rapidly changing, senescence (age-related deterioration) can be altruistic. According to numerical simulations of an agent-based model, while long-lived individuals can outcompete their short lived peers, populations composed of long-lived individuals are more likely to go extinct during periods of rapid environmental change. Moreover, as in many situations where other cooperative behavior arises, senescence can be stabilized in a structured population.

1. **Introduction.** In an evolutionary context, for a trait to be altruistic it must be both beneficial to others and costly to the altruist. Senescence, the deterioration associated with aging, easily fits the second test: dying from age-related causes is clearly more costly than not dying. This paper investigates situations where senescence might also satisfy the first criteria, where one organism's age-related deterioration aids the overall population. While from a classical viewpoint, altruism and cooperative behaviors more generally, seem as though they should be maladaptive, their prevalence across the biome has motivated significant research into scenarios which can select for cooperative behavior [9, 5, 3, 20, 16, 17, 21]. A portion of the prevalence of senescence may be attributable to the sort of structured populations that can stabilize cooperation.

There are several species that have negligible, or seemingly negligible aging, and at least one jellyfish, Turritopsis nutricula, which is believed to be functionally immortal [4]. There are theoretical reasons to believe that in some situations death rates should decrease with age, especially in species where greater sizes is especially competitive [22]. Nonetheless, senescence is widespread, and there are several well-established and non-contradicting evolutionary theories that attempt to explain why [15, 24, 13, 1, 18, 14].

The *mutation accumulation* theory of senescence states that selection pressure against mutations harmful in youth is typically greater than that of mutations harmful in old age. It thus stands to reason that genetic drift coupled with this difference in selection pressure can concentrate harmful mutations into being harmful

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in old age [15]. Another theory of senescence, the *antagonistic pleiotropy* theory, hypothesizes that senescence is the result of mutations which trade fitness in old age for fitness in youth [24]. The theory of the *disposable soma* presents senescence as the optimal balance between investing resources into maintaining an organism and investing in its future offspring [13].

While these theories have managed to make multiple, verified predictions, there are several empirical facts that present challenges to these theories. First, the existence of extremely long-lived organisms is counter to the predicted universality of these theories [6]. Second, the mutation accumulation and antagonistic pleiotropy theories suggest that extending organism life spans should require significant changes to many genes, but several simple single-gene knock-out experiments have been shown to drastically increase lifespans in some organisms [14]. On the other hand, it can be difficult to make simple predictions from these theories, as some of the processes associated with senescence have complex interactions with external mortality causes and population density [1].

Previous theories predict senescence because they assume that retaining youth is in some way expensive to the individual or its offspring. In contrast to previous evolutionary models of senescence, we do not assume any developmental trade-offs, or differing developmental trajectories and yet we nonetheless still predict senescence. Instead, we are interested in a different mechanism, slightly related to the fundamental but largely abandoned theory of August Weismann [23], namely: how dominance of older organisms in a rapidly changing environment can tether a population to a previously, but no longer, advantageous phenotype. Mechanistically: in a rapidly changing environment with limited resources, old age can become indicative of a misfit phenotype, death by old age can free up resources for fitter organisms, and if those fitter organisms are sufficiently related then age related death can be evolutionary advantageous.

To investigate this mechanism, we create an agent-based model where each organism tracks a numerical phenotype, their age, and the terminal age before which it dies of an age-related cause. These agents are simulated in discrete time, and undergo an age dependent and a phenotypic dependent mortality step, and a population dependent reproduction step. While the vast majority of parameters result in either a stable population or rapid extinction, along the critical boundary between these parameter regimes, populations with a finite terminal age survive longer than populations without a terminal age. However, in a mixed population, a subpopulation without a terminal age outcompetes its mortal rivals. Thus, it appears that in some parameter regimes senescence, as described by a terminal age, acts like an altruistic behavior: harmful to the altruistic individual but beneficial to the population.

Despite being empirically abundant, cooperative behaviors are notorious for requiring additional modeling assumptions to stabilize [9, 16]. In particular, there is an abundance of research into organisms engaged in explicit games such as prisoner's dilemma in the regime of weak selection pressure [21] and in structured populations such as stepping-stone populations [12] and stochastic island-migration models [7]. We extended our model from a well-mixed population to a set of islands where organisms can occasionally migrate from one island to another. On such an island model and with select parameter choices, we find that lower terminal age subpopulations can outcompete senescence-free subpopulations if migration is sufficiently slow–as to be expected of a cooperative behavior.

A somewhat similar result has been shown in other numerical studies, where stress related death, though costly to an individual, can be beneficial to a structured population [8] as can suicidal altruism [19]. However, in contrast to these papers, the proposed mechanism in this paper depends upon rapid change in the environment and population crashes and the results have implications for the senescence of otherwise successful organisms.

2. **An agent-based model of senescence.** To investigate the tradeoff between adaptability and longevity, we constructed a simple agent-based model. In our model, each agent i has three state variables: an age $a_i \in \mathbb{N}$, a heritable phenotype $x_i \in \mathbb{N}$, and a heritable terminal age $s_i \in \mathbb{N}$ at which time they would die of some age associated cause. These agents are members of a population with fixed carrying capacity K, and undergo age and phenotype based mortality and density dependent reproduction in discrete time. These agents are placed in a rapidly changing environment whose state is represented by the phenotype of maximum fitness, $X(t) \in \mathbb{N}$, which stochastically increments. Between the agents and the environment, the tradeoff of interest will be shown to be between individuals with a large terminal age effectively having a lower death rate and populations with a low terminal age having a greater diversity of phenotypes. In contrast to some models of senescence, in this model, an agent's terminal age has no direct cost to survival other than age-related death at the terminal age.

While age-related death is ultimately stochastic and largely indistinguishable from other mortality causes [1], to investigate the effect of age-related deterioration, we explicitly model mortality causes separately, breaking overall mortality into three parts: developmental/youth related causes, phenotype/environment fitness, and death by terminal age. First, we assume young agents have a reduced probability of survival $f(a_i)$, which represents factors such as inexperience, a developmental period, or other mechanisms as given in figure 1 top—where cumulative survival rates decrease for young ages but plateau for older ages. Second, agents whose phenotypes x_i differ from the ideal environmental phenotype $X(t)$ survive the environment with probability according to even, unimodal distribution $g(X - x_i)$, representing effects such as environmental stress or predation, figure 1 bottom. Finally, agents die when they reach their terminal age, possibly of some age associated disease, loss of requite competitiveness or other potential cause.

At each time step, the number of offspring is determined by the logistic equation with carrying capacity K, $rn(1-\frac{1}{K})$, where n is the number of individuals currently alive, r controls the rate of reproduction across the population and non-integer values are randomly rounded up or down. Each new agent has a chance η to mutate a different terminal age, either incrementing or decrementing their terminal age, and probability α of either incrementing or decrementing the phenotype of their parent.

The five stages in a single discrete time step are thus:

1. *Age advancement and terminal age check:* Each agent advances one year in age, $a_i(t + 1) = a_i(t) + 1$ and any agent older than their maximum age, $a_i(t+1) > s_i$, dies.
2. *Environmental change:* With probability ω, the environmental change parameter, the state of the world increments $X(t + 1) = X(t) + 1$, and otherwise remains unchanged.
3. *Age and environmental dependent mortality:* Each agent survives with probability equal to $f(a_i(t))g(X(t) - x_i)$.
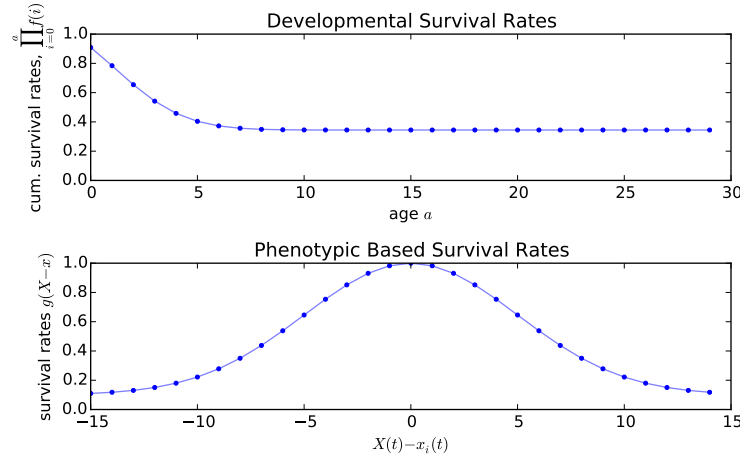
FIGURE 1.   For an agent with no environment/phenotype mismatch, the probability an agent survives until a given age decreases during youth and then holds steady until they reach their terminal age (top). Agents with a phenotype $x_i \neq X(t)$ have a probability less than one of surviving each time step.

4. *Reproduction:* The logistic equation is used to determine the number of new agents each of which has its sole parent chosen uniformly at random from the current alive population.

5. *Mutation:* Newly created agents undergo mutation, where s_i increments or decrements by one, each with probability $\frac{\eta}{2}$, and x_i increments or decrements by one, each with probability $\frac{\alpha}{2}$–agents are otherwise identical to their parent.

Unless otherwise mentioned, $\omega = 0.02$, and $\alpha = 0.03$. Populations are initialized with a uniform distribution of ages.

The key ingredients to our model are: agents that age, have a maximum terminal age, and which must evolve against a changing external environment in a population that can go extinct. Aside from these key ingredients, we have consciously avoided including elements of the model that would make longevity costly. For instance, it is not assumed that past some age agents cease reproducing. In fact, in comparison with agents with a lower terminal age, agents with a larger terminal age necessarily have a lower death rate and a higher number of expected offspring, and thus in a fixed environment, out-compete their lower terminal age competition.

We also consider a meta-population or 'island' version of the above model, where m copies of the above population each develop under a common, global environmental state $X(t)$, and agents occasionally migrate between islands. For total current population $\bar{n}$ split into $\bar{K}$ possible sites per island, the island version includes a 'Migration Stage' following mutation, where with probability $\mu \frac{\bar{n}}{m\bar{K}}$, a single agent attempts to move to a different island (if the destination island is completely full [rare] the migrant perishes).

3. **Simulations.** In order to investigate whether senescence can function as an altruistic behavior, we ran our agent-based model under a variety of different parameter regimes and initial conditions. As seen in figure 2, with the appropriate
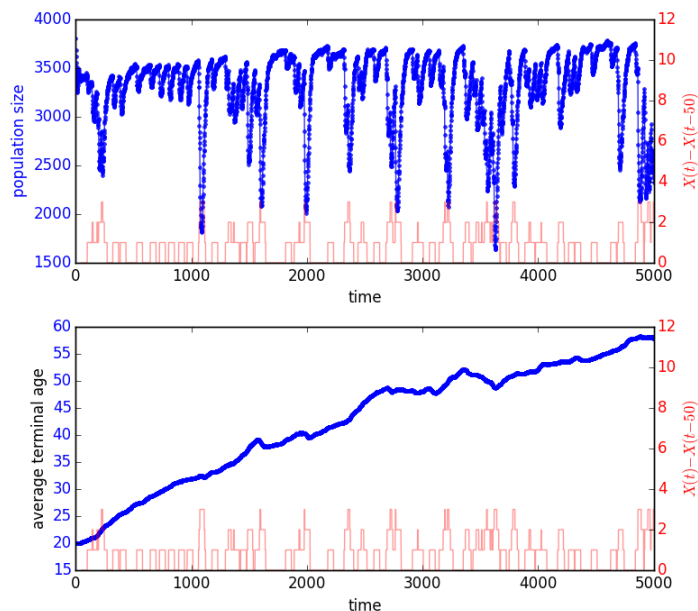
FIGURE 2. The critical parameter regime is characterized by occasional population crashes, which may or may not result in extinction (primary axis top) and coincide with changes in the environment $X(t)$ (secondary axis). When $\eta > 0$ (here $\eta = 0.25$) the maximum terminal age can mutate, where larger maximum ages are typical selected for (bottom). $K = 4,000$ and $s_i = 40 \forall i$.

choices in parameters, the stochastic change in the environment drives the population to undergo frequent and extreme population crashes. Principally, when $X(t)$, the state of the environment, increments multiple times over a short period of time, the population crashes and the agents with a mismatched phenotype rapidly die off, after which the population is able to recover. These cycles are easily identifiable when the population is plotted against the average difference of $\frac{1}{n} \sum_i (X(t) - x_i)$ as in figure 3. Interestingly, these population crash cycles tend to be organized around a reliable curve suggesting that the shapes of the age distribution and the phenotype distribution remain relatively predictable.

Eventually, a particularly bad population crash will result in extinction, but the expected time until an extinction depends on the particular choice of parameters. For these parameters in a critical regime, the average percent chance of a population going extinct as a function of the initial terminal age s_i is seen in figure 4. Notice, that populations with the lower terminal age are less likely to be driven to extinction than a higher terminal age. However, while it's the case that on the population level a higher terminal age increases the risk of extinction, inside a population the reduced death rate and higher lifetime fecundity of larger terminal ages leads shorter terminal age subpopulations to extinction, as in figure 5. The robustness of populations with a lower terminal age to environmental change is likely explained by their increased phenotype diversity, as seen in figure 6.

Thus, it appears that at the right parameter values, having a shorter terminal age can act analogously to an altruistic behavior, increasing the overall probability
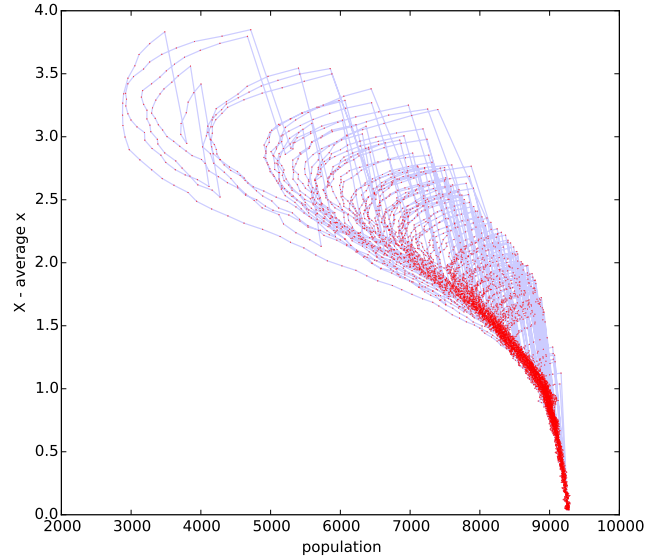
FIGURE 3. The population settles into a somewhat reliable relationship between total population and the average phenotype mismatch (initial transience not displayed). Parameters used: $K = 10,000$, $s_i = 40 \forall i$.
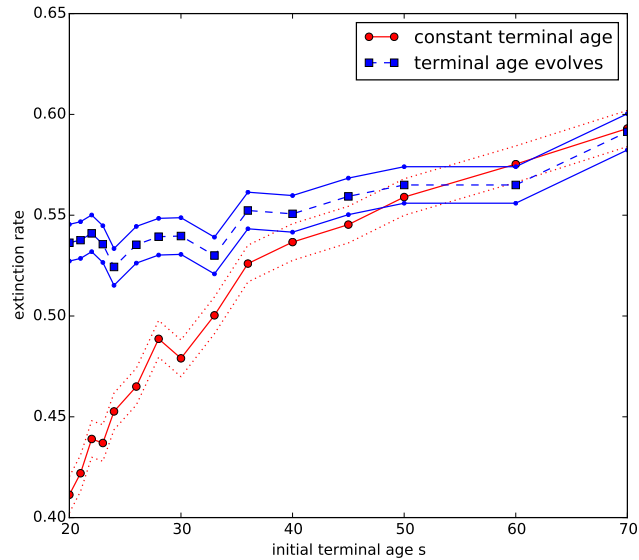

FIGURE 4. After $7,000$ time steps, populations with a large fixed terminal age are more likely to go extinct than those with a small terminal age. Allowing an agent's terminal age to mutate tends to increase the average terminal age and thus also the probability of extinction. $K = 1,000$ and the standard error of mean is displayed.
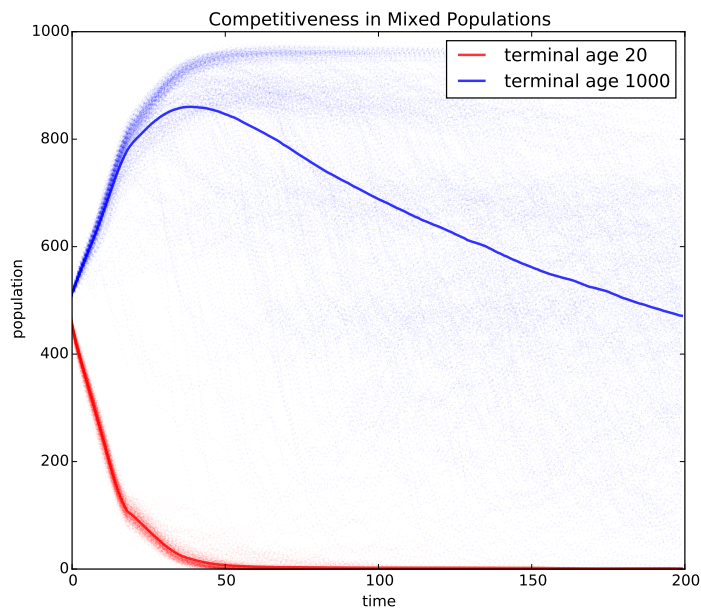
FIGURE 5. Out of 500 trials with an initial population split between $\frac{1}{2}$ with terminal age $1,000$ and $\frac{1}{2}$ with terminal age 20, the subpopulation with terminal age 20 was regularly out competed. The mean of the runs is highlighted.
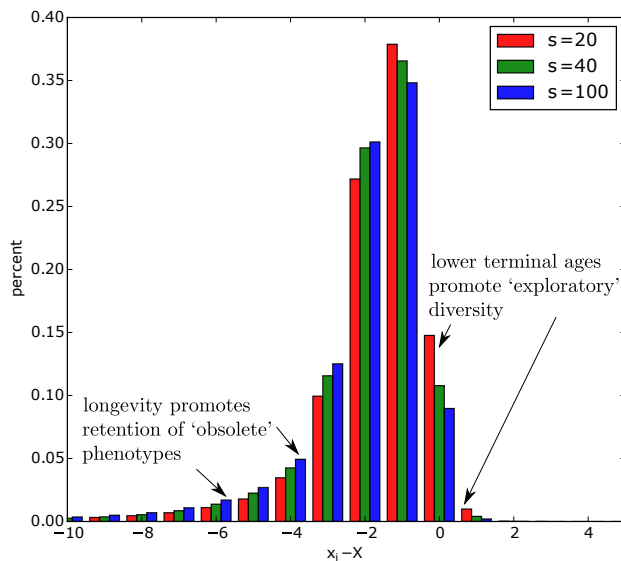

FIGURE 6. Sampled over many trials, populations with uniform, lower terminal ages are more likely to have the ideal phenotype $X(t)$ and even the potential future phenotype $X(t)+1$ than populations with larger phenotypes. $K = 1,000$ and results drawn across 500 runs, at each of $7,000$ different times.

of the population surviving but at an individual loss of competitiveness relative to having a larger terminal age.

However, while larger terminal ages are selected for in a single population, the opposite can be observed when similar agents are placed in an island model. Indeed, island models have been established as one setting which can stabilize cooperative behaviors. In this case, when the inter-island migration rate is appropriately set, the increased extinction probability for islands of agents with a larger terminal age can overcome the rate at which larger terminal aged agents invade and colonize other islands. For instance, in figure 7, sufficiently small migration rates μ give that the altruistic lower terminal age subpopulation does, on aggregate, outcompete the larger terminal age subpopulation.
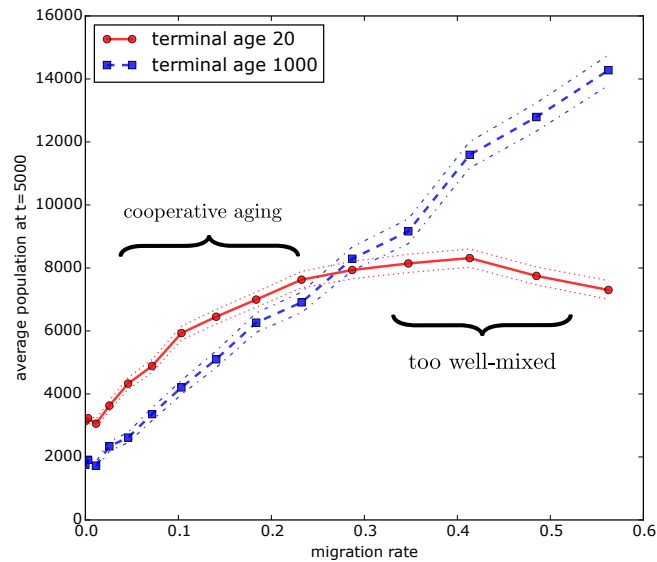

FIGURE 7. As the migration rate decreases, the populations with a lower terminal age begins to outcompete those with a longer terminal age. This was produced using 100 islands each with $K = 400$, and one third initially having populations with $s = 20$, another third with $s = 1000$ and the final third were initially barren. Otherwise this utilized the same parameters as figure 3.

Thus, under the appropriate parameter regime, having a terminal age acts as a cooperative behavior, both in how it behaves in a single population and how it can become selected for in an island setting.

4. **Discussion.** The cooperative aspect of senescence is sensitive to parameters, with the majority of parameter space resulting in either extinction, or stable populations. However, given that the external environment variable, $X(t)$ can represent co-evolving competitors, predators or prey, parameter regimes near the critical threshold between stability and extinction may represent the effects of self-organized criticality [2]. Meanwhile, the overall group selection dynamics should remain consistent for a variety of modeling choices, provided the right critical parameters are chosen.

In terms of modeling assumptions, there are several worth mentioning. Our use of a strict deterministic terminal age may seem less natural than a stochastic mechanism, where the probability of older agents dying from age-related causes is a function of both a_i and s_i. While such a choice would increase the parameter complexity, mathematically, an agent suffering a probability of death at each time step is the same as assigning the agent a fixed life span equal to the time step during which the agent would die. We believe that the effect of deterministically translating heritable traits into a terminal age is a reduction of noise and not a qualitative change in the cooperative nature of the dynamics. Additionally, the role of sexual reproduction may heighten the importance our proposed mechanism, particularly if sexual selection is correlated with age-related features such as size.

In the island model, the two competing forces were the propensity of longer terminal aged populations to locally go extinct and the ability of larger terminal aged agents to invade shorter terminal aged populations. It is possible that a stronger effect might be seen on a lattice model, or some other structured model. Similarly, adaptive migration may also favor senescence [10]. However, general comparisons of our island model to other migration based models of cooperation is complicated by the importance of local extinction events in our model. Indeed, having a large number of empty sites may lead to slightly different overall dynamics than migration models with fixed population sizes [10, 7].

The monotonicity of the changing environmental variable $X(t)$ seemingly precludes the recurrence of a trait in the population, though in practice, it only precludes the recurrence of a trait already present in the current population. For instance, whether $X(0) = 0$ and $X(1,000) = 100$ represent the same ideal phenotype or different ones is irrelevant to the model, because the probability that $x_i(1,000) = 0$ for any i is practically zero (for our choice of g). Nonetheless, there have likely been many settings with generally monotonic changes in the environment. For instance, the drastic environmental change from global warming will likely provide a generally monotonic set of challenges to species in many habits globally. Interestingly, it may be that one peculiar effect of global warming is an increase in age-related mortality, and a shortening of lifespans in the most stressed populations; though this prediction is likely common with both antagonistic pleiotropy and accumulated mutation theories under some modeling assumptions [1].

In contrast to other theories, cooperative senescence suggests a reason to expect traits explicitly designed to age an organism. For instance, the mechanisms of human aging that roughly resemble the programmed, biological clock mechanisms [11], may be partly explained by cooperative aspects of aging. However, there is no particular reason to restrict the cooperative argument to aspects of senescence that appear programmed, since in our model agents with any form of senescent traits have a group selection advantage. There may be aspects of senescence which have not only been selected for, but have further evolved into compact, modular genetic levers and thus appear programmed, but our model is not designed to make such predictions.

5. **Conclusion.** In contrast to previous studies of senescence, we do not assume that there is any individual benefit to traits linked to senescence. Instead, we have shown in numerical simulations that in a critical regime of rapid environmental or exogenous change which threatens the entire population, having a terminal age at which an otherwise healthy agent dies is cooperative. For modelers, this work

presents many possible extensions, as well as encourages a more theoretical treatment of the bifurcations associated with cooperative senescence as well as models which justify the critical parameter regime. For biologists, this suggests another possible explanation for some of the plethora of different patterns of senescence.

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