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REVIEWS

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Letter

Density Dependence, Senescence, and Williams' Hypothesis

Troy Day[1,2,*] and Peter A. Abrams[3]

In 1957, George Williams [1] argued that higher, age-independent, adult mortality should be correlated with more rapid senescence. Charlesworth [2] and most subsequent studies have added the adjective 'extrinsic' to mean that the age-independent mortality was due to sources 'external' to the organism. We now know that neither age- nor condition-independent increases in extrinsic mortality are necessary for more rapid senescence to evolve, and analyses have also shown that higher mortality can lead to the evolution of slower senescence [3,4]. Indeed, there are empirical examples where an added source of mortality seems to have extended lifespan [5]. Nevertheless, Williams' hypothesis remains popular [6].

Recently Moorad *et al.* [7] (henceforth MPS) reviewed Williams' hypothesis and they made the strong claim (in at least ten different places throughout their paper) that Williams' hypothesis is wrong, flawed, and/or contains a conceptual error. They then claim to show that the hypothesis is wrong because, for an extrinsic source of mortality to affect senescence, it must act in an age-dependent fashion (and Williams' hypothesis involves age-independent mortality). This latter claim is also reiterated throughout their paper, where they say that '…formal theory shows that only mortality that is age-specific can influence the evolution of senescence…' and that 'mortality that is truly independent of condition…' cannot affect selection on senescence. This is further emphasized in an entire section of their paper titled 'Models that redefine extrinsic to mean something else', where they say that extrinsic mortality can affect selection only if '…one changes the meaning of "extrinsic" to mean age dependent'. There they also review Abrams [3] and Williams and Day [4], stating that these papers either support their view or, when they don't, it is because of differences in opinion ([7], p. 6).

The purpose of this letter is to point out that these conclusions are incorrect. Both Abrams ([3], p.882) and Williams and Day ([4], p.1482) have independently demonstrated that extrinsic mortality need not be age-dependent to drive the evolution of more rapid senescence. MPS mischaracterize [3] by incorrectly claiming that with '…age-independent density effects, Abrams' models found that the addition of extrinsic mortality had no effect…'. He did not. Likewise, [4] is mischaracterized by incorrectly claiming that any discrepancy between results comes from differences in opinion about how to measure fitness and that perhaps '…we need to examine whether [a] redefinition of fitness is justified'. In fact, these discrepancies are not a matter of opinion. In an online appendix (see supplemental material online), we provide a detailed analysis that illustrates this fact and we present a two-age model as a simple counterexample to MPS's claim that extrinsic mortality must be age-dependent in order to affect selection on senescence. This simple counterexample is nothing more than a special case of the general analysis already published in [3] and [4], but the restriction to two age-classes makes the analysis simpler.

One way to understand why density dependence is important in the evolution of senescence is to note that, roughly speaking, when a population is growing exponentially (i.e., no density dependence), the fitness consequence of a change in vital rates at age x is discounted by the probability of survival

to age *x* and by the population growth rate (because offspring produced earlier can, themselves, reap the rewards of exponential growth). When age-independent extrinsic mortality increases, the discounting through survival gets stronger (i.e., there is a smaller probability of reaching age *x*), while the discounting through population growth gets weaker (i.e., the exponential growth potential is reduced), such that these two effects exactly cancel and Williams' hypothesis does not hold. But when a population is regulated by density dependence (and so on average is not growing) the latter effect need not exactly cancel the former, even when all density and mortality effects act in an age-independent way.

Indeed, the predictions made by existing theory are completely unambiguous and worth reiterating. If a population is growing exponentially, then a change in age-independent mortality will not affect selection on senescence (i.e., Williams hypothesis is not valid). For populations subject to density dependence things are more complicated for two reasons. First, depending on the form of density dependence, population size might continually change over time in complex ways. At present, little theory speaks to this interesting case (see Appendix in the supplemental material online for further discussion). Second, a change in mortality might affect population density, which then feeds back to affect vital rates in other ways. Nevertheless, if density dependence leads to a constant equilibrium population size then predictions are still completely unambiguous. If all mortality and density effects are age-independent, then Williams' hypothesis is correct when density dependence acts solely through fertility and it is incorrect when density dependence acts solely through mortality. It is a fact, not an opinion, that age-dependency of external mortality is not required for Williams' hypothesis to be valid.

Some previous studies have made the same claim as MPS, suggesting that Williams' hypothesis is wrong irrespective of whether there is density dependence [8,9]. However, these studies have focused only on the case where density dependence acts solely through mortality and so their conclusions are actually in complete agreement with the above summary (see Appendix in the supplemental material online for further discussion).

Williams' hypothesis continues to occupy the attention of evolutionary biologists [6,10]. It is true that for organisms with high evolutionarily unavoidable mortality, investment in repair and maintenance for ages that are seldom reached does not make sense. Likewise, organisms that require a long time to mature must not deteriorate so rapidly that they never reproduce. Thus, we might expect a positive correlation between mean mortality rate and most measures of senescence in a diverse set of species. However, such a correlation is not very informative regarding the effect of mortality on the evolution of senescence in any particular lineage. The evolution of senescent traits in response to a new mortality source is influenced by many factors, including density dependence in all demographic rates, age-dependent effects of both altered density and the new mortality on existing mortality and fertility, and interactions between the mortality source and physiological changes caused by senescence [3,4]. In addition, senescence involves age-related changes in traits other than mortality, a fact that is often neglected. These changes in birth and growth rates are also likely to cause differential effects on the abundances of different age classes, further complicating evolutionary predictions about senescence. For example, when density dependence in fertilities is characterized by larger effects in more senescent individuals, Williams' hypothesis could again fail to hold.

In summary, to understand the effect of an environmentally imposed change in mortality (or fertility) on the evolution of senescence at least three types of measurements are needed: (i) the direct age-specificity of the change in demographic rates, where 'direct' means age-differences due to age-related factors other than senescence (a point also made by MPS); (ii) the interaction of existing senescence with the environmental factor (e.g., are there greater effects of the factor on individuals with greater senescent decline?); (iii) the density-dependent feedbacks from the environmental change on all life history parameters, including the interactions mentioned in point (ii). Future work should also reassess how to compare rates of senescence, because the trajectories of mortality (or other fitness parameters) versus age are not characterized by a single simple function [11,12]. In addition, it should expand theory to consider variable environments, where results are likely to differ from those of the equilibrium conditions assumed in this and most previous work.

Supplemental Information

Supplemental information associated with this article can be found online https://doi.org/10.1016/j.tree.2019.11.005.

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Letter

Williams' Prediction Will Often Be Observed in Nature

Jan Kozłowski [1,*], Oskar Burger,[2,3] and Maciej Jan Dańko[3]

Day and Abrams [1] (henceforth DA) criticize a recent paper by Moorad, Promislow, and Silvertown [2]. Moorad *et al.* [2] claim that the modern version of the Williams' hypothesis [3], that extrinsic mortality leads to faster senescence, is wrong. They argue that the Williams' prediction cannot be true because extrinsic mortality must be age dependent to affect senescence. However, DA show that this critique is an unjustified generalization. Indeed, age-independent extrinsic mortality has no effect on senescence if populations are growing exponentially or if an increase in density leads to higher age-independent mortality. DA show that when density-dependence acts uniformly on fecundity at all ages, age-independent extrinsic mortality affects senescence as Williams predicted. In fact, density dependence has the same effect if it acts on fecundity, juvenile mortality, or juvenile emigration [4,5], because these determine the number of juveniles entering the population. DA show, along with [4,5], that two different claims have been unjustly generalized; the first is that the original Williams' hypothesis is always true and the second is that it is always false.

Populations of vertebrates and some invertebrates are often more or less stable, regulated by mechanisms of density dependence. Due to the interaction with density dependence, a variety of relationships between extrinsic mortality and senescence are possible [4,5], and we should see evidence for the Williams' hypothesis often in nature. Indeed, our brief survey indicates that there are many well-known examples of density dependence acting on fecundity as well as on juvenile survival or emigration (the conditions under which Williams' hypothesis will hold if the mortality is truly age and condition independent). For example, Ricklefs [6] shows all of the following: (i) fecundity decreases with density in fruit flies (*Drosophila melanogaster*); (ii) a shortage of nesting sites was responsible for equilibrium population size in common terns (*Sterna hirundo*), which results in decreased average fecundity; (iii) the proportion of non-territorial males increases among males (probably young ones), which indicates increased migration, and the number of fledged juveniles per female as well as the proportion of surviving juveniles decrease with increasing density in song sparrows (*Melospiza melodia*); and (iv) an increase in the proportion of pregnant white-tailed deer (*Odocoileus virginianus*) following population reduction from hunting. Among ground squirrels (*Spermophilus parryii plesius*), mortality during hibernation increases with population density, possibly for all ages, and the weaning rate declines with density [7]. In a collection of 21 species of large herbivores, density-dependent effects were found acting on juveniles in 15 of them, on the age at first breeding in 12, on the number of offspring per female in 17, and on early juvenile survival in 17, whereas adult survival was affected in only one [8]. In another large herbivore data set, four of seven species showed evidence for density-dependent juvenile mortality, in one adult mortality was also affected, and in another only adult mortality was affected [9]. Among Mauritius kestrels (*Falco punctatus*), the number of fledglings and juvenile survival were sensitive to density [10]. Among willow tits (*Parus montanus*), fecundity as well as yearling and adult mortality are density dependent [11]. In an experimental manipulation of population density, juvenile mortality in the common lizard (*Lacerta* vivipara) was positively related to density, survival of adults remained unchanged, and females were smaller and less fecund at high density [12]. In fish, declines in resource availability that accompany higher densities generally change fecundity or recruitment probability through limitation of individual growth rate or survivability of juveniles, and density-dependent mortality is most likely to occur while fish are smaller than 10% of asymptotic length [13].

Our short survey shows that density dependence is likely to act most often on fecundity or mortality of juveniles. If so, senescence should commonly respond to the level of extrinsic mortality, at least among vertebrates. However, any credible study evaluating Williams' hypothesis should also establish the character of density dependence.

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