Appendix to George C. Williams' problematic model of selection and senescence: time to move on.

As we explain in our letter, Day & Abrams' (2020) interpretation of our message is incorrect, and this has apparently followed from confusion over terminology. Specifically, we point out that extrinsic mortality can be defined in terms of either its direct effects on age-specific mortality (such as the proximate effects of an experimental manipulation or the presumed *direct* action of an ecological threat) or the *total* effects on age-specific mortality and fertility (the *actual* changes of vital rates that are felt by the population). The latter can differ from the former due to ecological feedback, such as might be caused by changes in density pressures. For the most part, Day & Abrams build the arguments in their Appendix upon the premise that we adopt the former definition in the first part of our paper (Moorad et al. 2019), when in fact we use the latter. Consequently, their Appendix, for the most part, argues against a viewpoint that is not ours. This would include their model intended to demonstrate that one particular form of density-dependence can cause extrinsic mortality (in the *direct* sense of the word) to change how selection works. Given that our paper acknowledges in the section on density dependence that this can happen, the use of this model appears to us to succeed in only refuting a straw-man argument. We will say no more about this aspect of their Appendix, which makes up its first half, except to note that they refer to this narrative throughout its remainder.

Aside from this, we agree on most points that Day & Abrams have made regarding how selection is changed in response to the addition of extrinsic mortality. We do not share their enthusiasm for the techniques of Mylius & Diekmann (1995) because we feel that Hamilton's approach is sufficient, and from *our* perspective, it is simpler and more intuitive. The former is part of the ESS approach, and as such should generate the same predictions as those derived from Hamilton (Charlesworth 1980). Accordingly, we don't take issue with the use of these models *per se,* but we would object to suggestions that density-dependent population regulation somehow makes Hamilton' formulae inappropriate.

For the most part, the response from Day & Abrams could be chalked up simply to an unfortunate misunderstanding of terminology. However, there are comments made in the latter part of their Appendix that indicate disagreement and/or confusion on other points, and we feel that these warrant further discussion in the remainder of this Appendix.

<u>Density-dependent regulation cannot, by itself, cause changes in selection</u>

Day & Abrams correctly attribute the statement in the header to us, and we stand by it, but we note that some context is required to understand it properly. This is written in response to those who would invoke density-dependence by itself as a reason for why Williams' prediction appears to hold true in a particular situation. However, it is important to note that density-dependence with the addition of extrinsic mortality can lead to selection for late-life survival to be strengthened, relaxed, or unchanged, depending upon the ecological specifics (see Abrams (1993) or examples of each in Figure 1 of our response). Day & Abrams' model provides an example of a situation in which one sort of density-dependent regulation *does* change selection to favour more senescence when (direct) extrinsic mortality is added. We do not dispute that this is what they have demonstrated, and we recognize that this sort of ecological scenario *might* be widespread and important (but we believe that we lack sufficient information to know for sure). However, different density functions can lead to the opposite prediction (selection favours less senescence). More to the point, one can even imagine forms of density dependence in which there is *no* change in selection. To see how this might work, let us review Hamilton's formula for selection $\beta(x)$ for mortality at some age *x*,

$$\beta(x) = -\frac{\sum_{y=x+1}^{\infty} l_y m_y e^{-ry}}{\sum_{y=1}^{\infty} y l_y m_y e^{-ry}} \qquad [1]$$

Note that this is negative because it describes selection *for* a deleterious character (death). We imagine some change that reduces cumulative survival as a direct response to the environmental perturbation, but because population growth is density dependent, we do not allow *r* to change. If we chose to further constrain the model, we could specify that *r* is fixed to 0, and this would make the model equivalent to the condition usually imagined in the theory (e.g., Day & Abrams' model), but that is not necessary. Because survival is suppressed, fertility must be enhanced in order to satisfy the condition that *r* is unchanged. Adding extrinsic mortality (in the direct sense of the term) must decrease l_y, increase m_y, and leave e^{-ry} unaltered for all ages *y*. If we wish to adopt the usual simplifying assumption made by Day & Abrams and others that density supresses fertility by the same fraction at every age, then we would find that the fractional increase in m_y is *age-independent* but the fractional decrease in l_y is *age-dependent*. The product of these decreases with age faster after the perturbation than before, and this leads to a reduction of selection against late-life mortality.

However, let us replace one assumption with a biologically feasible assumption that the fertility of older individuals is more sensitive to density effects than that of younger individuals. Adding extrinsic mortality will decrease l_y in this case, but the loss of selection will be mitigated because the increase in m_y that is caused by the added mortality increases with age. With sufficient density-suppression of late life fertility, it is possible that late-life m_y increases to a sufficient degree to compensate exactly for the reduction in l_y; the *total* effects of extrinsic mortality on vital rates cancel out as $l_y m_y$ remains unchanged. Selection would not change in this case, even in the presence of density-dependent population regulation. Abrams (1993) mentions another scenario (survivorship is equally affected by density at all ages) that leads to no change in selection because the *direct* and *indirect* effects exactly cancel.

<u>On the relationship between age-distributions and selection</u>

Day & Abrams correctly attribute the following quote to us, "It has long been known that the addition of age-independent mortality can have, by definition, no effect on age-distributions. It follows that mortality that is truly independent of condition will not affect within- or among-age distributions of phenotypes." From this, they make two claims. The first is that our statement is incorrect, citing results from their model. We have already established that one's definition of age-independent mortality is important, and the definition of the term taken from the perspective of *total* effects makes our statement true (this corresponds to the "density-independent" condition as it is usually imagined). Their second claim is that it is irrelevant because a change in the age-distribution is neither necessary nor sufficient for a change in selection. This claim has some merit, because in the general case one can have changes in selection *without* changes in age structure. However, in the specific case of extrinsic mortality with density dependence that Day & Abrams envision, we might ask if such a change *is* both a necessary and sufficient condition for altered selection.

To explain, let us first note that there are *two* age-distributions that are relevant (in the simple asexual context) to this conversation. First, there is the distribution of ages of extant individuals,

$$P(x) = \frac{l_x e^{-rx}}{\sum_{y=1}^{\infty} l_y e^{-ry}} \qquad [2].$$

This is what people usually mean by the 'stable age structure'. However, there is also the distribution of ages of new parents,

$$Q(x) = l_x m_x e^{-rx} \qquad [3].$$

Note that $Q(x)$ is the relevant element in the summation that Hamilton uses to express the strength of selection against mortality (Equation 1). As it is the attenuation of this summation with increased age that describes the age-related loss of selection, one cannot change selection without changing $Q(x)$ (and *vice versa*). It is well-understood (Coale 1957, Charlesworth 1980, Moorad and Promislow 2010) that this distribution $Q(x)$ (and therefore the strength of selection) cannot change when the *total* effects of extrinsic mortality are age-independent.

Put into these terms, Day & Abrams' comment implies that one *can* change $Q(x)$ without changing $P(x)$; we acknowledge that point. Altering the age-specific fertility rates (say, by reducing early-age fertility and enhancing late-late fertility) *without* changing survival or population growth rates could accomplish this. However, since Day & Abrams' objections appear to focus on the specific case where *direct* extrinsic mortality is applied to populations that are under density-dependent population regulation, we might reasonably ask if their objection applies to these conditions. Let us describe how $P(x)$ and $Q(x)$ change in this situation. We increase mortality at every age by a factor $\Delta\mu$ and constrain the growth rate to remain the same. Following Day & Abrams' scenario, we increase fertility rates *by the same proportion* to compensate (we call the fractional increase *k*). The new distribution of new parents is

$$Q(x) = (1+k)m_x e^{-x\Delta\mu} l_x e^{-rx} \qquad [4].$$

Selection will change, and that change must follow from changes in *k* and $e^{-x\Delta\mu}$. As there appears to be no real debate about whether *selection* changes in this scenario (it does), we will leave this part of the discussion and determine whether these changes imply shifts in the age structure of extant individuals. Following the aforementioned logic, the new stable age distribution after the addition of extrinsic mortality becomes

$$P(x) = \frac{e^{-x\Delta\mu} l_x e^{-rx}}{\sum_{y=1}^{\infty} e^{-y\Delta\mu} l_y e^{-ry}} \qquad [5]$$

We can illustrate the effect of extrinsic mortality on a two-age class model and restricting *r* to be 0 (as in Day & Abrams' model). Here, the fraction of the two ages is

$$P(1) = \frac{1}{1+p_1 e^{-\Delta\mu}} \qquad [6a]$$

$$P(2) = \frac{p_1 e^{-\Delta\mu}}{1+p_1 e^{-\Delta\mu}} \qquad [6b],$$

where p_1 is the probability of survival from age 1 to age 2 (before the addition of extra mortality). Note that because $e^{-\Delta\mu}$ is maximized at 1 when $\Delta\mu = 0$, it is clear that the age distribution is shifted ever-more towards the younger age class as more mortality is added. Note that this does *not* happen in the case with extrinsic mortality defines the *total* effects (i.e., what is usually considered to be the result of the density-independent condition). It does not happen in this case, because the reduction in l_x is exactly compensated for by the increase in e^{-rx} in Equation 2. It should be clear from a comparison of the changes in $Q(x)$ and $P(x)$ that changes in one cannot occur without changes in the other because the shapes of both distributions are changed by the same factor $e^{-x\Delta\mu}$. We conclude that under the conditions explored by Day & Abrams, and despite their claims to the contrary, a change in the age distribution is both necessary and sufficient to change age-specific selection against

mortality. Our conclusion regarding the conditions considered by Day & Abrams is consistent with Charlesworth (1980),

"… demographic changes which leave the age-structure of the reproductive age-classes unchanged are unlikely to cause gene-frequency changes, unless they are due to a source of mortality or a factor affecting fecundity which is directed specifically at certain genotypes or at a specific group of ages of the reproductive individuals."

Comments on demographic stability

All of the derivations and expressions that we present assume that an equilibrium has been met with respect to vital rates, age structures, and population growth rates. Nonetheless, Day & Abrams have taken a comment that we made criticizing one particular study to indicate that we, "have not appreciated an important aspect of the mathematical foundation of all optimization models based upon the Euler-Lotka (EL) equation; namely, all mathematical analyses are valid only for the asymptotic state of the population … This is simply a mathematical fact and *it invalidates all of Moorad et al's arguments having to do with these transient effects* [our emphasis added]." Once again, we agree with most of what Day & Abrams believe (they do identify an important assumption that most models make), except where it deals with what we have said. Their criticism is lacking in detail as to where, exactly, these transgressions supposedly occur, but they do cite Box 3. There we had imagined that mortality has been added at one arbitrary age, and we have asked the question, "What are the consequences of adding mortality at one age to selection for mortality at all age classes?" With respect to the issue of demographic stability, this is no different from asking the same questions that have been asked (and answered) before, "What are the consequences of adding the same amount of mortality at all ages to selection for mortality at all age classes?" Specifically, we make a change to the l_x schedule that reflects the added mortality, we account for the effects of this change on the population growth rate, and then we modify Hamilton's description of selection to reflect these changes. This approach has been applied before (e.g., Charlesworth 1980, Caswell 2007) with no remonstrations.

It is difficult for us to understand the source of Day & Abrams confusion. Perhaps they are unclear about the meaning of "change" as it is used in this context. We will clarify our meaning here by imagining two equilibrium populations that are identical in all ways except that one has vital rates that reflects the added mortality and one that does not. The "change" in selection is the difference in selection gradients between the two. It should be obvious that there are no violations of assumptions of demographic stability in this case.

An alternative cause of their confusion could be our criticism of an assertion made by da Silva (2018) that *r* should be assumed to be zero in the general case because that is what the long-term growth rate must average out to be. This is incorrect for at least two reasons. The first reason, which we do not mention in our paper, is that this perspective does not include ecological feedbacks that might work to maintain a population growth rate equal to zero. However, if density is involved in this maintenance, then these feedbacks on vital rates need to be considered. We had mentioned a second reasons, was that da Silva's observations implies that population growth should be negative some of the time and positive some of the time. We were simply pointing out that a model, such as da Silva's, that assumes that *r* is *always* zero may not provide the same predictions as when it varies about zero. This is our only foray into a discussion of non-stable populations, and, despite Day & Abrams' assertion, we do not model this condition. We do, however, cite work that does consider this more complex situation properly (Caswell 2007, Caswell and Shyu 2017).

On the relationship between density dependent population regulation and stationary populations

Day & Abrams' views of our position on the previous topic appear to be confused with issues relating to density-dependence and population growth rates. For example, their criticism of our model in Box 3 appears to invoke density-dependence, but density-dependence is not part of our model. We challenged the common modelling practice of assuming that populations that are under density-dependent population regulation are stationary ($r = 0$) on the grounds that populations that grow or shrink must also be subject to the same sorts of ecological constraints. Apparently this has led Day & Abrams to conclude that this "appears to be part of the justification given in Moorad *et al.* for their belief that there is no difference in predictions between [density independence] and [density dependence]." This, of course, is a straw man argument based upon a distortion of our message, as we make clear here and in our original paper on p.525.

Nonetheless, we wish to clarify our position on the latter relationship between density-dependence and population growth rates because we think that this is important for understanding the robustness of current model predictions. In our paper, we suggested that the strategy of using only stationary populations to assess changes in selection with density dependence may have been implemented in order to ensure that fitness could be equated with lifetime reproductive success, and that might provide for simpler models. In retrospect, that assertion might be a bit harsh as we must acknowledge the point that $r = 0$ with density-dependence presents an ecological scenario that is attractive because it ensures that populations do not go extinct or grow to unreasonable size *without* running afoul of assumptions of population stability. However, we maintain our position that generalizing these models to accommodate *any* value for r in a stable population would be a positive development. It would be nice to know, for example, that the sort of model proposed by Day & Abrams predicted the same results in shrinking, stationary, or growing populations that are under density pressures. One way to approach this problem might be to choose an arbitrary r value that must be insensitive to the *direct* effects of a manipulation of vital rates. Because that value for r would be constrained by the conditions of the model, ecological feedback of those changes would lead to some particular *total* effect on the vital rates. The change in selection would then follow from selection gradients derived from Hamilton's expressions. This is what we have done above in our discussion of selection with density dependence, but if that is objectionable on the grounds that the suppressive effects of density don't realistically work that way, then r could be set to zero, and the same qualitative inferences that we make above still apply in this limited case.

Details of our Figure: All populations have 25 age classes. Fertility is zero throughout the first five (pre-adult) age classes. Mean fertility over the adult ages is the product of five and the gamma distribution with parameters $\alpha = 2, \beta = 0.2$. The shape of this function is illustrated in the center graph of the top row. All populations begin with Gompertz mortality, $(x) = Aexp(Bx)$, with $A = B = 0.05$. The slope parameter, B, was adjusted upwards until the Malthusian growth rate became approximately $r = 0.05$ ($B \cong 0.1872$). No ecological relationships are inferred at this point. In each of the cases below, an environmental perturbation was imagined that has the sole *direct* effect of increasing mortality by 0.1 at every age. The first column illustrates these direct effects, and they are equal across scenarios. The graph for the fourth scenario is scaled differently, and that will be explained below.

Scenario 1 (row **A**): Added age-independent mortality with density independence. The vital rates described above define the initial conditions (before the mortality is added). There are no indirect effects because there are no ecological feedbacks. The center graph illustrates age-specific fertility, but there is no change. There is no change in selection against mortality (right). This scenario and results are consistent with Abrams' prediction for the density-independent case (Abrams 1993).

Scenario 2 (row **B**): Added age-independent mortality with density dependence (through age-independent fertility). Fertility is suppressed from the previous conditions incrementally and by the same fraction until *r* converges with 0 from above. At this point, approximately 61% of age-specific mortality remains. These are the starting conditions. Age-independent mortality is added, and fertility is incrementally increased until *r* converges with 0 from below to reflect the relaxation of density pressure. The center graph illustrates the changes in fertility over the starting conditions (the *indirect* effects). Selection against adult mortality is supressed (right). This scenario and results are consistent with Abrams' 2nd enumerated prediction for the density-dependent case (Abrams 1993).

Scenario 3 (row **C**): Added age-independent mortality with density dependence (fertility is more affected in the old). Fertility is suppressed as described in Scenario 2, except that the incremental decline is proportional to the square of the age. Age-independent mortality is added, and fertility is incrementally increased (in the same squared-fashion) until *r* converges with 0 from below to reflect the relaxation of density pressure. The center graph illustrates the changes in fertility over the starting conditions (the *indirect* effects). There is a notable shift towards older fertility when compared to Scenario 2 (center, row **B)**. Selection against adult mortality is enhanced (right). This scenario and results are consistent with Abrams' 3rd enumerated prediction for the density-dependent case (Abrams 1993).

Scenario 4 (row **D**): Added age-independent mortality with density dependence (through age-independent mortality). Mortality was increased by increasing the '*A*' parameter until *r* converges with 0 from above ($A \cong 0.0728$). This represented the starting conditions. Age-independent mortality is added, and the leftmost graph on the fourth row represents the direct effects of this change. This is rescaled to reflect age-specific mortality (rather than cumulative survival), and the initial conditions with respect to mortality are slightly different from those in the first three Scenarios. Age-independence mortality is reduced until *r* converges with 0 from below to reflect the relaxation of density pressure. The center graph illustrates the change in age-specific mortality over the starting conditions (the *indirect* effects). Notice that the *direct* and *indirect* effects are equal and opposite. The *total* effects on vital rates are neutral, and the rightmost graph illustrates the obvious lack of change resulting for selection. This scenario and results are consistent with Abrams' 1st enumerated prediction for the density-dependent case (Abrams 1993).

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