Trends in Ecology & Evolution

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Letter

George C. Williams' Problematic Model of Selection and Senescence: Time to Move on

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Moorad *et al*. [1] reinforces and elaborates on warnings made previously [2,3] that Williams' hypothesis that high extrinsic mortality selects for high rates of senescence should be treated with scepticism. A proper hypothesis should be based upon the best information available at the time, and for the case of the evolutionary theory of senescence, Hamilton's model of selection is superior to Williams' model. Hamilton's insights improved over Williams' insights by his appreciation for how fertility and juvenile survival schedules play a critical role in defining selection. While Williams' model is not articulated mathematically, it is clear that his hypothesis is motivated by his belief that the strength of selection against mortality specific to some age follows from the frequency of individuals that survived to that age [4]. However, Hamilton [5] was clear: the strength of selection is equal to the proportion of newborns that came from parents that survived to that age (see [1] or Equation 1 in the Appendix). Furthermore, Williams' explicitly states that juvenile mortality cannot affect the evolution of senescence, while Hamilton's formulae show us that it can. It should be emphasised that Williams himself came to appreciate that Hamilton's model describes the best way to think about how selection works [6].

Under limited conditions, predictions made by these models converge. In all other situations, Hamilton's predictions are more appropriate. We cannot think of any reason to motivate any test of modern evolutionary theory by appealing to Williams' hypothesis because Hamilton's superior motivating model is always available. Furthermore, tests that are motivated by Williams' hypothesis risk propagating Williams' flawed verbal model, which has a tenacious hold on the literature. The waters become muddied when modellers, such as Day and Abrams [7], cast their results as consistent with Williams' hypothesis when, in fact, the highly specific ecological conditions that they model do not resemble anything proposed by Williams' general model. While such claims are true technically, this way of thinking is problematic as it can lead to statements, such as this from Day and Abrams [7], that appear to justify Williams' verbal model:

> Williams' hypothesis continues to occupy the attention of evolutionary biologists … 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.

A hypothesis can be based upon a poor general model and make good predictions valid under special conditions. However, do these sorts of models warrant our attention when alternatives exist that are more logically sound and make predictions that are more general? For these reasons, we believe that while Williams' model of selection may hold historical interest, it has no place in modern discussions of ageing (NB – this objection has nothing to do with Williams' other insights on senescence [8]).

Day and Abrams objected to our statements pertaining to situations that correspond to where predictions from Williams and Hamilton converge. Specifically, they focus on a form of population regulation in which density supresses fertility equally at all ages. When age-independent mortality is added to such populations, fertility is enhanced due to the relaxation of ecological constraints baked into the model, and selection is changed as a result. They make several mischaracterisations of our views that warrant a response. We believe that these derive from confusion over terminology, specifically in the dual meanings of extrinsic mortality that we use in our attempt to synthesise a diverse field. This is discussed in our review where we consider a situation in which the distinction between definitions become important (p. 525). We take this opportunity to clarify our perspectives.

Extrinsic mortality can mean two different things when environmental changes can induce changes in vital rates through ecological feedbacks (Figure 1). (i) For some [7], extrinsic mortality is understood in the context of direct effects only. These are the proximate effects of a manipulation or treatment that raise mortality rates equally at all ages (Figure 1A–D). Here, the term does not consider knock-on effects caused by feedbacks that can alter mortality or fertility rates that might be called the indirect effects of extrinsic mortality. (ii) Others (including us) take the meaning of extrinsic mortality to pertain to a possible form of the total effects, or the summation of the direct and indirect effects. We believe a focus on total effects is relevant because these are the proximate determinants of Hamilton's predictions. Extrinsic mortality by this perspective means that the total effect of a manipulation is comprehensively described by an increase in age-independent mortality (only row A of A–D in Figure 1 qualifies).

The choice of definition has profound implications for how we might answer the deceptively simple question, 'How can the addition of extrinsic mortality alter selection?' We answered that extrinsic mortality cannot have an effect (A in

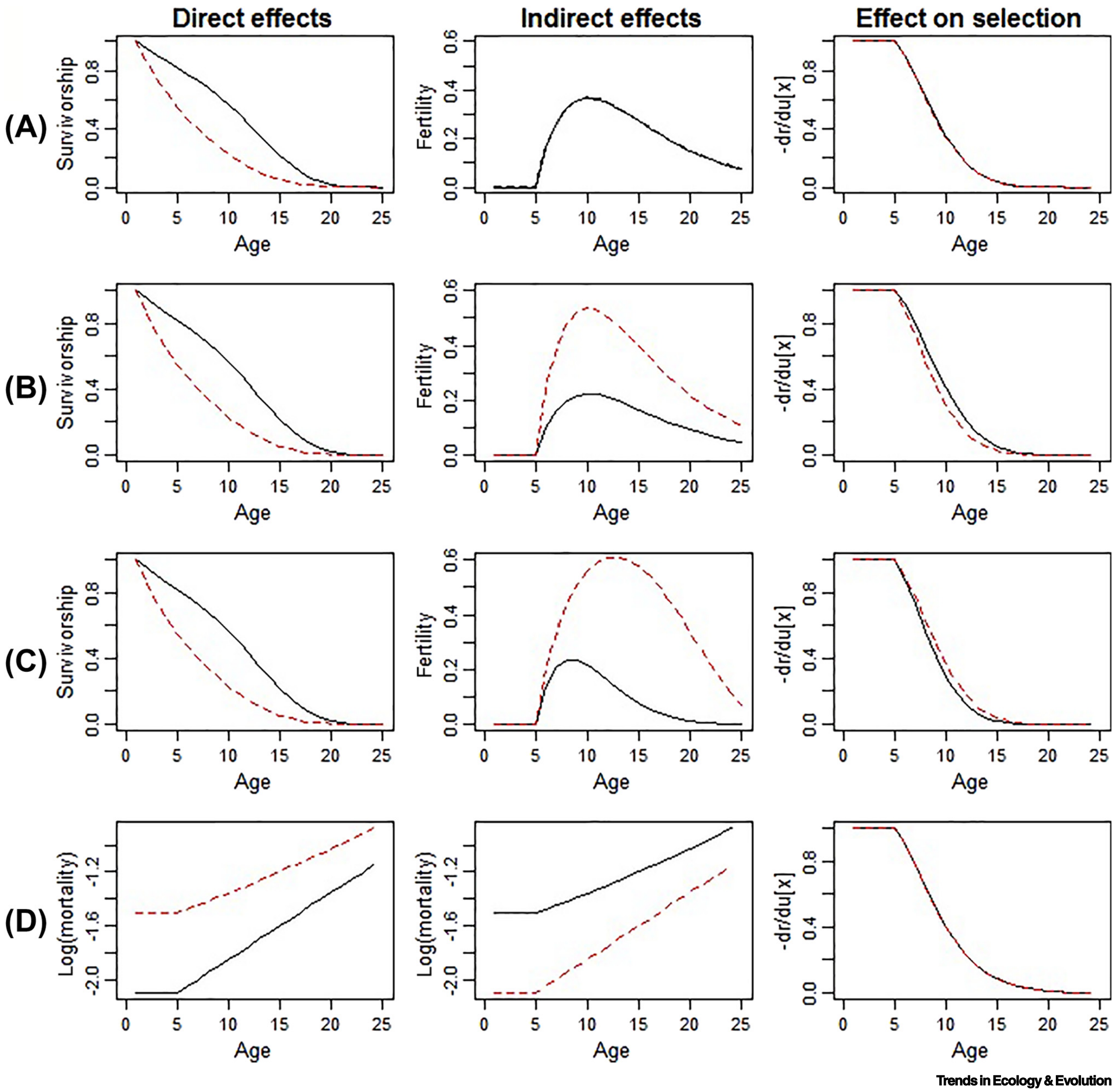

Figure 1. Consequences of Added Age-Independent Mortality: Direct Effects, Indirect Effects, and Changes in Selection. Four scenarios that correspond to cases discussed in [9]; we have illustrated how added age-independent mortality can affect vital rates directly versus indirectly. Rows correspond to different scenarios: (A) density independence; (B) density dependence through age-independent fertility; (C) density dependence (fertility is more affected in the old); and (D) density dependence through age-independent mortality (no total effects). Black lines indicate conditions before the added mortality and red lines indicate the conditions afterwards. For more details see the Appendix in supplemental information online.

Figure 1): mortality must be age dependent to matter. This is correct from our perspective. When Day and Abrams [7] ask this question, they interpret extrinsic mortality to apply to effects in the direct sense only. They invoke a particular model of density-dependent population regulation that causes fertility to increase when mortality is added (the indirect effects). They note that this will cause selection against late-life mortality to relax (B). This is correct, too, but their definition of extrinsic mortality used in the direct sense is equivalent to the simultaneous addition of age-independent mortality and

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fertility. We agree with Day and Abrams that the meaning of extrinsic mortality can be vague; future studies can clarify their use of the term by specifying its causal relationship with vital rates, as we do here.

Kozlowski *et al.* [9] share this confusion. This clarification should resolve the focus of both parties' objections and lay to rest any concern that we reject the role that density dependence might play in the evolution of senescence. On the contrary, we believe that this ecological feature could be important, but these studies should not be couched in terms of Williams' hypothesis; both for the reasons given above but also because different sorts of density dependence can lead to radically different model predictions, some of which are not consistent with Williams' hypothesis [10]. We agree wholeheartedly with Kozlowski *et al.* [9] that empirical investigation into the causes of selection as it relates to ageing should establish the nature of density dependence, and we believe that the survey that they describe is a valuable move in the right direction.

Day and Abrams [7] make technical criticisms in their Appendix to which we respond in our own Appendix (see supplemental information online).

Supplemental Information

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