



THE UNIVERSITY *of* EDINBURGH

Edinburgh Research Explorer

## George C. Williams' problematic model of selection and senescence

### Citation for published version:

Silvertown, J, Moorad, J & Promislow, D 2020, 'George C. Williams' problematic model of selection and senescence: Time to move on', *Trends in Ecology & Evolution*, vol. 35, no. 4.  
<https://doi.org/10.1016/j.tree.2020.02.001>

### Digital Object Identifier (DOI):

[10.1016/j.tree.2020.02.001](https://doi.org/10.1016/j.tree.2020.02.001)

### Link:

[Link to publication record in Edinburgh Research Explorer](#)

### Document Version:

Peer reviewed version

### Published In:

Trends in Ecology & Evolution

### General rights

Copyright for the publications made accessible via the Edinburgh Research Explorer is retained by the author(s) and / or other copyright owners and it is a condition of accessing these publications that users recognise and abide by the legal requirements associated with these rights.

### Take down policy

The University of Edinburgh has made every reasonable effort to ensure that Edinburgh Research Explorer content complies with UK legislation. If you believe that the public display of this file breaches copyright please contact [openaccess@ed.ac.uk](mailto:openaccess@ed.ac.uk) providing details, and we will remove access to the work immediately and investigate your claim.



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

2  
3 Moorad et al [1] reinforces and elaborates on warnings made previously [2, 3] that Williams'  
4 "hypothesis" should be treated with scepticism. A proper hypothesis should be based upon  
5 the best information available at the time, and for the case of the evolutionary theory of  
6 senescence, Hamilton's model of selection is superior to Williams'. Hamilton's insights  
7 improved over Williams' by his appreciation for how fertility and juvenile survival schedules  
8 play a critical role in defining selection. While Williams' model is not articulated  
9 mathematically, it is clear that his hypothesis is motivated by his belief that the strength of  
10 selection against mortality specific to some age follows from the frequency of individuals that  
11 survived to that age [4]. However, Hamilton [5] was quite clear: the strength of selection is  
12 equal to the proportion of newborns that came from *parents* that survived to that age (see  
13 [1] or Equation 1 in the Appendix). Furthermore, Williams' explicitly states that juvenile  
14 mortality cannot affect the evolution of senescence while Hamilton's formulae show us that  
15 it can. It should be emphasized that Williams himself came to appreciate that Hamilton's  
16 model describes the best way to think about how selection works [e.g., 6].

17  
18 Under limited conditions, predictions made by these models converge. In all other situations,  
19 Hamilton's predictions are more appropriate. We cannot think of any reason to motivate any  
20 test of modern evolutionary theory by appealing to Williams' hypothesis because Hamilton's  
21 superior motivating model is *always* available. Furthermore, tests that are motivated by  
22 Williams' hypothesis risk propagating Williams' flawed verbal *model*, which has a tenacious  
23 hold on the literature. The waters get muddied when modellers, such as Day & Abrams (DA)  
24 [7], cast their results as consistent with Williams' hypothesis when, in fact, the highly specific  
25 ecological conditions that they model do not resemble anything proposed by Williams'  
26 general model. While such claims are true *technically*, this way of thinking is problematic as  
27 it can lead to statements, such as this from DA [7], that appear to justify Williams' verbal  
28 *model*,

29  
30 "Williams' hypothesis continues to occupy the attention of evolutionary biologists ... It is true  
31 that for organisms with high evolutionarily unavoidable mortality, investment in repair and  
32 maintenance for ages that are seldom reached does not make sense."

33  
34 A *hypothesis* can be based upon a poor general model and make good predictions valid under  
35 special conditions. However, do these sorts of models warrant our attention when  
36 alternatives exist that are more logically sound and make predictions that are more general?  
37 For these reasons, we believe that while Williams' model of selection may hold historical  
38 interest, it has no place in modern discussions of aging (NB – this objection has nothing to do  
39 with Williams' other insights on senescence [see 8]).

40  
41 DA objected to our statements pertaining to situations that correspond to where predictions  
42 from Williams and Hamilton converge. Specifically, they focus on a form of population  
43 regulation in which density suppresses fertility equally at all ages. When age-independent  
44 mortality is added to such populations, fertility is enhanced due to the relaxation of ecological  
45 constraints baked into the model, and selection is changed as a result. They make several  
46 mischaracterizations of our views that warrant a response. We believe that these derive from  
47 confusion over terminology, specifically in the dual-meanings of "extrinsic mortality" that we

48 employ in our attempt to synthesize a diverse field. This is discussed in our review where we  
49 consider a situation in which the distinction between definitions become important (p. 525).  
50 We take this opportunity to clarify our perspectives.

51

52 “Extrinsic mortality” can mean two different things when environmental changes can induce  
53 changes in vital rates through ecological feedbacks (see Fig 1).

54 (1) For some [e.g., 7], ‘extrinsic mortality’ is understood in the context of *direct* effects only.

55 These are the proximate effects of a manipulation or treatment that raise mortality rates  
56 equally at all ages (e.g., Fig 1, **A-D**). Here, the term does *not* consider knock-on effects  
57 caused by feedbacks that can alter mortality or fertility rates which might be called the  
58 *indirect* effects of extrinsic mortality.

59 (2) Others (including us) take the meaning of ‘extrinsic mortality’ to pertain to a possible  
60 form of the *total* effects, or the summation of the direct and indirect effects. We believe  
61 a focus on total effects is relevant because these are the proximate determinants of  
62 Hamilton’s predictions. ‘Extrinsic mortality’ by this perspective means that the *total*  
63 effect of a manipulation is comprehensively described by an increase in age-independent  
64 mortality (only row **A** of **A – D** qualify).

65 The choice of definition has profound implications for how we might answer the deceptively  
66 simple question, “How can the addition of extrinsic mortality alter selection?” We answered  
67 that extrinsic mortality cannot have an effect (**A** and **D** in Fig 1): mortality must be age-  
68 dependent to matter. This is correct from our perspective. When DA [7] ask this question,  
69 they interpret extrinsic mortality to apply to effects in the *direct* sense only. They invoke a  
70 particular model of density-dependent population regulation that causes fertility to increase  
71 when mortality is added (the *indirect* effects). They note that this will cause selection against  
72 late-life mortality to relax (**B**). This is correct, too, but their definition of “extrinsic mortality”  
73 used in the *direct* sense is equivalent to the simultaneous addition of age-independent  
74 mortality *and* fertility. We agree with Day & Abrams that the meaning of “extrinsic mortality”  
75 can be vague; future studies can clarify their use of the term by specifying its causal  
76 relationship with vital rates, as we do here.

77

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

88

89 DA [7] make technical criticisms in their Appendix to which we respond in our own.

90

91 **Figure 1. Consequences of added age-independent mortality: direct effects, indirect effects,**  
92 **and changes in selection.** Four scenarios that correspond to cases discussed in [9]; we have  
93 illustrated how added age-independent mortality can affect vital rates *directly vs indirectly*.  
94 Rows correspond to different scenarios (**A** - density independence; **B** - density dependence

95 through age-independent fertility; **C** - density dependence (fertility is more affected in the  
96 old); and **D**) density dependence through age-independent mortality (no *total* effects). Black  
97 lines indicate conditions before the added mortality and red lines indicate the conditions  
98 afterwards. For more details see the Appendix.

99

100

## 101 **Literature Cited**

102

103 1. Moorad, J. et al. (2019) Evolutionary ecology of senescence and a reassessment of Williams'  
104 'extrinsic mortality' hypothesis. *Trends in Ecology & Evolution* 34 (6), 519-530.

105 2. Caswell, H. (2007) Extrinsic mortality and the evolution of senescence. *Trends in Ecology &*  
106 *Evolution* 22 (4), 173-174.

107 3. Wensink, M.J. et al. (2017) The rarity of survival to old age does not drive the evolution of  
108 senescence. *Evolutionary Biology* 44 (1), 5-10.

109 4. Williams, G.C. (1957) Pleiotropy, natural selection, and the evolution of senescence.  
110 *Evolution* 11 (4), 398-411.

111 5. Hamilton, W.D. (1966) Moulding of senescence by natural selection. *Journal of Theoretical*  
112 *Biology* 12 (1), 12-45.

113 6. Williams, G.C. (1999) The tithonus error in modern gerontology. *Quarterly Review of*  
114 *Biology* 74 (4), 405-415.

115 7. Day, T. and Abrams, P. (2020) Density dependence, senescence, and Williams' hypothesis.  
116 *Trends in Ecology & Evolution*.

117 8. Gaillard, J.M. and Lemaitre, J.F. (2017) The Williams' legacy: A critical reappraisal of his nine  
118 predictions about the evolution of senescence. *Evolution* 71 (12), 2768-2785.

119 9. Kozłowski, J. et al. (2020) Williams' prediction will often be observed in nature. *Trends in*  
120 *Ecology & Evolution*.

121 10. Abrams, P.A. (1993) Does increased mortality favor the evolution of more rapid  
122 senescence? *Evolution* 47 (3), 877-887.

123

124

125