

NOTE

DEVELOPMENTAL AXIOMS IN LIFE HISTORY EVOLUTION

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ABSTRACT

Life history theory is often invoked to make universal predictions about phenotypic evolution. For example, it is conventional wisdom that organisms evolve older ages at first reproduction because they have longer lifespans. We clarify that life history theory does not currently provide such universal predictions about phenotypic diversity. Using the classic Euler-Lotka model of adaptive life history evolution, we demonstrate how predictions about optimal age at first reproduction depend on prior, theoretical assumptions (i.e. axioms) about organismal development. These developmental axioms include the rates, forms, and tradeoffs involving growth or differentiation. Developmental innovations transform the biology underlying these axioms. Consequently, Euler-Lotka and related life history models do not make coherent predictions at macroevolutionary scales, where developmental innovations occur (e.g., across mammals, birds, or insects). By focusing on historical innovations instead of universal rules, life history theory can reconnect with flourishing research in evolutionary developmental biology.

KEYWORDS

Delayed maturity, devo-evo, evo-devo, optimization, recruitment, sociosexual development, tradeoffs

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1 *“Natural selection will always, ceteris paribus, favor rapid development; the sooner an organism*
2 *matures the less likely it is to die before maturing and reproducing...The development of longer*
3 *juvenile phases in a phyletic line must always be considered a price paid for some more*
4 *important development.”*

5 - Williams (1966a, pp. 87–88)

6

7 INTRODUCTION

8 Life history theory is one of the major predictive disciplines of evolutionary biology. The
9 theory predicts the evolution of phenotypes related to age, reproduction, and survival in terms of
10 variance in lifetime reproductive success (Williams 1966a; Roff 1992; Stearns 1992; Caswell
11 2000). Life history theory was formulated and tested with specific biological examples including
12 clutch size in songbirds (Lack 1947), spawning in salmon (Cole 1954), and predation in guppies
13 (Reznick et al. 2002). Yet characters such as “reproduction” and “survival” seem applicable to all
14 organisms, leading some branches of life history theory to make claims about universal tradeoff
15 rules (Reznick et al. 2000), universal phenotypic scaling laws (Charnov 1993; Burger et al.
16 2019), or universal “pace-of-life” axes for phenotypic variation (Pianka 1970). These claims
17 suggest models of optimized life history evolution via selection offer law-like predictions about
18 phenotypic diversity across large clades of organisms.

19 We argue current life history theory, by definition, offers no universal predictions about
20 phenotypic diversity across the tree of life. As an example, we focus on one axis of life history
21 evolution: age at first reproduction. We show how predictions of optimal age at first reproduction
22 rely on theoretical assumptions (i.e., axioms) about organismal development. However, major
23 changes in developmental processes have evolved within and among lineages. Developmental

24 evolution thus helps determine the course of adaptive life history evolution, even in cases when
 25 adaptive life history evolution does not determine the course of developmental evolution. This
 26 hierarchical relationship suggests life history research will be most empirically effective if it
 27 focuses less on universal rules, and more on the historical processes through which
 28 lineage-specific rules are generated and reconfigured. Our argument has deep precedents
 29 (Stearns 1982; Smith et al. 1985; Gould 2002, pp. 1037–1038), but our discussion clarifies how
 30 research into historical innovations forges new connections between phylogenetics,
 31 developmental biology, and life history theory.

32

33 **CONDITIONS FOR SELECTION ON DELAYED REPRODUCTION**

34 A primary goal of life history theory has been to understand “delayed reproduction,” or
 35 the fact that some organisms do not reproduce as quickly as possible (Cole 1954). In early verbal
 36 arguments, Williams recognized the evolution of delayed reproduction depends on facts about
 37 development (epigraph). Subsequent theory added rich quantitative depth to predictions of
 38 optimal age at first reproduction, especially in terms of body size and mortality (Wittenberger
 39 1979; Bell 1980; Caswell 1982; Stearns and Koella 1986; Kozłowski and Stearns 1989;
 40 Kozłowski 1992). However, these complicated mathematical approaches have made it harder to
 41 recognize key assumptions about organismal development.

42 Here, we demonstrate some fundamental features of life history evolution using the
 43 discrete Euler-Lotka model (Cole 1954; Stearns 1992; eq. 1).

$$1 = \sum_{x=\alpha}^{\omega} e^{-rx} B_x L_x \quad \text{Eq. 1}$$

44 This model maps age-specific reproduction and survivorship to population growth rate for a
 45 given genotype in a given environment (Table 1). The model assumes discrete age-classes, either

46 zero environmental variation or zero phenotypic plasticity, zero density dependence, stable age
 47 distribution, and exponential population growth rates. Changing these assumptions can have vast
 48 implications for life history theory, many of which have been thoroughly investigated (e.g.,
 49 Tuljapurkar 1990; Stearns 1992; Reznick et al. 2002; Caswell et al. 2018).

50 The Euler-Lotka model, and related formulations such as the Leslie matrix (Caswell
 51 2000), are used to make evolutionary predictions. A specific set of life history traits in a given
 52 environment— α , ω , B_x , L_x —yields a real number solution for population growth rate (r) such
 53 that different genotypes can be compared. Selection is predicted to favor the genotype with the
 54 highest r value (Stearns 1976).

55 Here, we show how different qualitative features of development prevent, or permit,
 56 selection for delayed reproduction. In the Euler-Lotka model, “development” means an increase
 57 in reproduction (B_x) or survival (component L_x) as a function of age. In reality, development
 58 involves diverse processes of somatic and extra-somatic organismal growth and differentiation
 59 over time; we discuss the consequence of such biological complexity below. Note that both
 60 quantitative and biological definitions make development the mirror-image of senescence, a
 61 classic topic in life history theory (Hamilton 1966; Charlesworth 2000).

62

63 *Impossible: static fecundity and survival*

64 Selection does not favor delayed reproduction in a lineage with no development. An
 65 example is given using a model of static fecundity ($B_x = b$) and static survival rates ($L_x = S^x$).
 66 This model, and the next one, are extraordinary in assuming newborns are identical to adults. We
 67 assigned example values for all parameters except α and r (Table 1). The genotype with the
 68 highest r is the one with the lowest α value, which reproduces most quickly (Fig. 1A).

69 This conclusion applies to all lineages with static fecundity and survival rates, including
 70 ones with high adult survival rates (Appendix). Despite conventional wisdom radiating from
 71 frameworks involving r - K or *fast-slow* life history spectra (Pianka 1970; see also Stearns and
 72 Rodrigues 2020), the Euler-Lotka model does not automatically predict that lineages with high
 73 fecundity should begin reproducing more quickly, nor that organisms with a longer lifespan
 74 should begin reproducing more slowly. A long lifespan is not a sufficient condition for the
 75 evolution of delayed reproduction.

76

77 *Insufficient: direct reproductive costs*

78 Bell (1980) frames the evolution of age at first reproduction in terms of the direct cost to
 79 reproduction, such as an energetic cost. Curiously, Bell's analysis also makes clear that such
 80 costs are insufficient for selection to favor delayed reproduction. We can model a direct cost by
 81 splitting survivorship into juvenile and adults survival rates, which apply before and after age at
 82 first reproduction respectively ($L_x = j^\alpha s^{x-\alpha}$). All else being equal, a direct cost is represented by
 83 $j > s$.

84 Example models involving only costs can still predict immediate reproduction (e.g., Fig.
 85 1B). A genotype that delays reproduction will pay the same cost eventually, but reap no
 86 intervening benefits, and can still die in the meantime. There are theoretical exceptions in the
 87 transient evolution of declining populations, where maintaining an immortal generation with no
 88 offspring (i.e., reaching $r = 0$) would beat any strategy that results in a declining population with
 89 offspring (Caswell 1982).

90

91 *Insufficient: Guaranteed development*

92 Development that proceeds freely throughout an organism's life—with no direct cost,
 93 opportunity cost, or tradeoff—is also insufficient for selection favoring delayed reproduction.
 94 For example, simple models of organismal development might involve a linear increase in
 95 annual fecundity ($B_x = dx$) or characteristic growth rate towards some asymptotic maximum
 96 fecundity ($B_x = F(1 - e^{-gx})$; Stearns and Koella 1986). Although fecundity is no longer static
 97 in these models, the benefits of development are still independent of age at first reproduction.
 98 Genotypes that delay reproduction develop to the same extent as those reproducing immediately,
 99 and merely incur the risk of dying before reproduction. As a result, selection still favors
 100 genotypes that reproduce as quickly as possible (Fig. 1C-D, Appendix).

101

102 *Sufficient: Developmental opportunities*

103 It finally becomes possible for selection to favor delayed reproduction when the risks and
 104 benefits of development are themselves linked to age at first reproduction. This link can occur in
 105 at least two ways within the Euler-Lotka model. The first involves increasing fecundity via
 106 guaranteed development (e.g., $B_x = dx$) combined with a cost to reproduction ($L_x = j^\alpha s^{x-\alpha}$,
 107 given $j > s$). In this case, the optimal age at first reproduction can be intermediate, balancing the
 108 benefits of development with the risks of mortality (Fig. 1E-F; Williams 1966a, pp. 88–89,
 109 177–178; Stearns 1992, pp. 128–135).

110 Tradeoffs are a core construct in life history theory (Lack 1954; Williams 1966b; Stearns
 111 1989; Reznick et al. 2000). In this model, however, development offers an increasing benefit
 112 relative to a static cost (s/j). Phrased in the terms of van Noordwijk and de Jong (1986),
 113 development here increases resource acquisition orthogonal to the axis of resource allocation in

114 the tradeoff between survival and reproduction. Selection can thus favor delayed reproduction
 115 not because an organism is caught up in a tradeoff *per se*, but rather because development
 116 provides the capacity to transform the terms of that tradeoff.

117 Alternatively, selection can also favor delayed reproduction given a direct tradeoff
 118 between development and reproduction. Consider a strict version of this tradeoff, in which
 119 fecundity develops only prior to the age at first reproduction (e.g., $B_x = d\alpha$). Here, the only cost
 120 to reproduction is the missed opportunity to develop. A genotype that delays reproduction can
 121 prolong development and benefit from a lifetime increase to annual fecundity (Fig. 1G-H).

122

123 *Hidden developmental processes*

124 In these simple Euler-Lotka model comparisons, qualitative assumptions about
 125 developmental processes provide the grounds for evolutionary predictions of age at first
 126 reproduction. Some modeling decisions obscure these developmental processes. First, juvenile
 127 mortality rates (j) often exceed adult mortality rates (s ; Williams 1966a). This is an assumption
 128 about development, in which survival rate not only increases as a young organism ages, but also
 129 outweighs any direct survival cost paid during reproduction itself. Second, there may be some
 130 absolute minimum threshold for maturation. Stearns (*pers. comm.*) raises a case in which
 131 genotypes share a fixed minimum body size for maturity but differ in somatic growth rate.
 132 Selection may favor the evolution of delayed reproduction if growing slower also means
 133 surviving better (e.g., foraging more slowly, but more safely; cf. Stearns 1992, pp. 124–125;
 134 Ebert 1994). In that example, the developmental assumption is that maturation occurs at a given
 135 body size. Third, density-dependent models predict the evolution of delayed reproduction based
 136 on different density-dependent effects for juveniles *vs.* adults (Reznick et al. 2002; Travis et al.

137 2023). Modeling any difference in the impact of density-dependence on young/prebreeding vs.
 138 old/breeding stages—whether through differences in predation rates, social interactions, resource
 139 dynamics, etc.—makes an axiomatic statement about development: young is different than old.
 140

141 DISCUSSION

142 Under the Euler-Lotka model, qualitative and quantitative predictions for optimal age at
 143 first reproduction depend on the properties of reproduction (B_x) and survivorship (L_x) functions
 144 (Fig. 1). Relative values of r , and therefore all evolutionary predictions, depend on at least three
 145 features of a given model: (1) the constituent parameter values of developmental functions, such
 146 as d , F , and g ; (2) the form of developmental functions, such as $B_x = dx$ or $B_x = F(1 - e^{-gx})$;
 147 and (3) the form of tradeoffs between development, survival, and reproduction, such as s/j or
 148 $B_x = d\alpha$.

149 These parameters, functions, and tradeoffs serve as the developmental axioms of any life
 150 history model. Under certain developmental axioms, including static survival and fecundity (Fig.
 151 1A, Appendix) or guaranteed development (Fig. 1C-D), it is impossible for the Euler-Lotka
 152 model to predict selection favoring delayed reproduction. It becomes possible for selection to
 153 favor delayed reproduction when development transforms a survival/reproduction tradeoff (Fig.
 154 1E-F) or when development itself trades off with reproduction (Fig. 1G-H). In all cases, relative
 155 values of r depend not only on the form of these tradeoffs, but also the form of developmental
 156 functions (e.g., Fig. 1G vs. H) and the parameter values within functions (Table 1). Constraining
 157 a set of developmental axioms is the only way to generate finite solutions in a life history
 158 optimization problem (Stearns 1976).

159 Yet, developmental axioms are only “constraints” in the sense they determine particular
160 axes of available evolution, not because they are themselves evolutionarily constrained (Gould
161 2002). For example, mammals teach us that developmental parameters evolve. Classic life
162 history paradigms assert fecundity—including both number and quality of offspring—scales with
163 adult body size (Pianka 1970; Western and Ssemakula 1982; Stearns 1992). Models of life
164 history evolution across mammals must therefore consider something like an F parameter (Table
165 1), asserting maximum possible fecundity as it relates to maximum functional body size.
166 Force-scaling laws dictate growth limits for leg bones in terrestrial mammals, setting physical
167 constraints on F (Biewener 2005). By evolving to live in the water, however, whales escape this
168 terrestrial size constraint (Goldbogen 2018). When researchers claim to derive “universal” life
169 history patterns for mammalian phenotypes, they thus predict either relative parent-offspring
170 body sizes with zero implications for absolute body sizes (e.g., Burger et al. 2019), or else
171 predict absolute body size by fitting different parameters for terrestrial and aquatic mammals
172 (e.g., Clauset 2013). The Euler-Lotka model can predict an optimized relationship between body
173 size and reproduction given F in mammals. But the Euler-Lotka model cannot make predictions
174 about the evolution of aquatic life, which redefines F in different lineages of mammals.

175 Birds teach us that developmental functions evolve. Recent phylogenetic studies
176 document the convergent evolution of sexual bimaturism in polygynous, lekking birds (Ancona
177 et al. 2020; Taylor and Prum 2023 [preprint]). In distinct lineages of lekking species such as
178 pheasants (Phasiandae), manakins (Pipridae), and bowerbirds (Ptilonorhynchidae), males provide
179 no parental care. Instead, they solicit mates with sexual displays at display territories (Bradbury
180 1981). Young males, but not females, must develop lek display sites and displays, although these
181 developmental processes are poorly understood (Collis and Borgia 1992; McDonald 2007;

182 Schaedler et al. 2021; Spezie and Fusani 2023). Parallel to the difference in developmental
183 demands, males in polygynous lekking species delay reproduction longer than females (Wiley
184 1974; McDonald 1993; Ancona et al. 2020; Taylor and Prum 2023 [preprint]). The Euler-Lotka
185 model could predict the sex-specific evolution of age at first reproduction in lekking birds, given
186 the unknown function that represents sociosexual development in young males. But the
187 Euler-Lotka model offers no predictions about the evolution of lekking, which redefines the
188 relationship between sociosexual development and reproduction.

189 Insects teach us that developmental tradeoffs also evolve. In ametabolous arthropods,
190 juveniles closely resemble adults, meaning the ecology of juveniles can closely resemble the
191 ecology of adults, and body size can continue to grow after maturity (Truman and Riddiford
192 1999; Rolff et al. 2019; Truman 2019). Hemimetabolous insects show a range of differences
193 between nymph and adult ecologies and adults do not grow after maturity. Holometabolous
194 insects show major differences between larval and adult ecologies and there is no growth after
195 pupation. The evolution of metamorphosis thus represents shifts in potential
196 survival/reproduction tradeoffs (via ecological changes at maturation) and
197 development/reproduction tradeoffs (via determinate size at maturity). Hemimetaboly and
198 holometaboly have singular, nested evolutionary origins in the insect phylogeny (Truman 2019).
199 Different models may predict evolution in lineages with particular developmental staging
200 (though not about insects, see life history models in Ernsting et al. 1993 on Collembola; Ebert
201 1994 on *Daphnia*). These models do not make predictions about the evolution of insect
202 metamorphosis, which redefines the models needed to predict evolution.

203

204 CONCLUSION

205 Qualitative and quantitative predictions of Euler-Lotka models depend on developmental
206 axioms, but developmental axioms, like any axiom by definition, do not depend on the
207 predictions of the Euler-Lotka model. The model makes no intelligible predictions about
208 phenotypic diversity at timescales where developmental axioms are free to evolve, such as across
209 mammals, birds, and insects, let alone across vertebrates, invertebrates, animals, or Life.

210 Stearns (2000) argued that classic life history theory failed to predict the observed
211 phenotypic diversity of life. The failure to translate “microevolutionary” predictions into
212 “macroevolutionary” ones was viewed as an empirical problem. Research efforts have thus
213 focused on more generalizable demographic models (e.g., de Vries and Caswell 2019),
214 exemplary empirical tests (e.g., Travis et al. 2023), and more complete comparative datasets
215 (e.g., Healy et al. 2019).

216 In contrast, we argue this failure has conceptual, not empirical, roots. The Euler-Lotka
217 and related life history models assume some kinds of evolution occur and others do not. Outside
218 of the model, the relative scope of “microevolution” and “macroevolution” is not clearly defined.
219 The model then defines a “microevolutionary” realm within its predictive scope: the adaptive
220 evolution of age-related allocation to survival and reproduction with respect to developmental
221 axioms. Simultaneously, the model defines a “macroevolutionary” realm: the evolution of
222 developmental axioms. Thereafter, microevolution involves predictable changes with respect to
223 static evolutionary rules, whereas macroevolution involves non-predictable changes to the rules
224 themselves. The model cannot bridge the gap between microevolution and macroevolution; the
225 model defines the gap.

226 The notion that developmental evolution affords (i.e., limits, generates, and establishes
227 conditions for) adaptive life history evolution is clear in Gould’s discussions of ontogeny and
228 “positive constraint” (Gould 1977, pp. 289–293, 2002, pp. 1025–1045) along with Stearns’ effort
229 to connect the adaptationist principles of optimization to the mechanistic principles of variation,
230 plasticity, and constraint (Stearns 1982). Gould, Stearns, and others began to bridge
231 developmental biology and life history theory at the famous 1981 Dahlem conference, but no
232 formal research program emerged (Love 2015). Nevertheless, informal research programs in
233 developmental life history theory are appearing. Rather than fitting data to models given
234 ahistorical evolutionary rules, such research aims to document the historical evolution of rules.
235 In recent examples, Beccari et al. (2023 [preprint]) investigate the diversification of mammal life
236 histories as clades of bats, whales, and monkeys established unique environments in trees, water,
237 and air. Kozłowski et al. (2020) highlight historical events in animal evolution (e.g., air sacs and
238 pneumatic bones in dinosaurs) that reconfigure the scaling between metabolic rate and body size.
239 Unlike standard comparative work in life history theory—which treats phylogenetic history like
240 statistical noise interfering with evolutionary patterns (Harvey and Pagel 1991)—this research
241 suggests the history of biological mechanisms is itself key to understanding life histories.

242 Across a disciplinary silo, evolutionary developmental biology (Gould 2002; Wagner
243 2014) is studying not only morphological rules (“*Baupläne*”), but also historical changes to those
244 rules (“innovations;” Wagner and Muller 2002), and the quantitative dynamics of evolution
245 within and across rulesets (“evolvability,” Wagner and Altenberg 1996). The challenge is to
246 reintroduce life history—time, age, reproduction, survival (Cole 1954)—to the hitherto
247 morphological concepts of innovation (Wagner and Muller 2002; Müller and Newman 2005).
248 Theoretical studies will help us understand how life history evolution proceeds when the process

249 of optimization under a ruleset unearths, or forecloses, evolvability to other rulesets. Empirical
250 studies can document when innovations in morphological, physiological, behavioral, social, or
251 cultural mechanisms have in historical fact come to route and reroute life history evolution.

252

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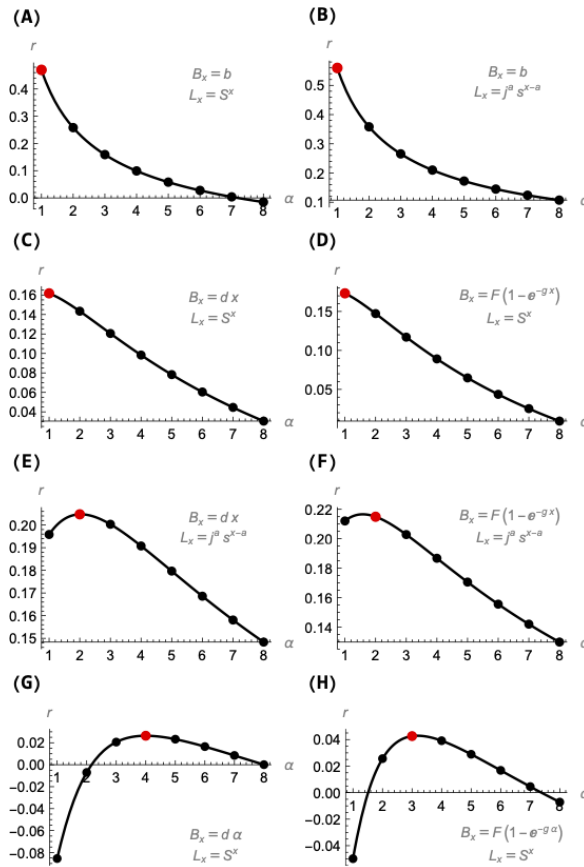
381 TABLES AND FIGURES

382 **Table 1.** Life history variables and parameters in discrete Euler-Lotka models. Example values

383 were used for predictions in Figure 1.

384

Variable	Definition	Bounds	Example values
r	Population growth rate (“fitness”)		
x	Age (or stage)	≥ 0	
α	Age at first reproduction	≥ 0	1, 2, ..., 8
ω	Age at last reproduction	$\geq \alpha$	30
B_x	Offspring produced at age x	≥ 0	
L_x	Survivorship to age x	$\geq 0, < 1$	
b	Static annual fecundity	≥ 0	1.00
d	Fecundity growth rate (linear)	≥ 0	0.15
g	Fecundity growth rate (von Bertalanffy)	≥ 0	0.10
F	Fecundity maximum (von Bertalanffy)	≥ 0	2.00
S	Survival rate (static)	$\geq 0, < 1$	0.80
j	Juvenile survival rate	$\geq 0, < 1$	0.95
s	Adult survival rate	$\geq 0, < 1$	0.80



385

386 **Figure 1.** Genotype growth rates, r , for different ages at first reproduction (α) according to
 387 discrete Euler-Lotka life history models. Optimum ages at first reproduction (red point) vary
 388 with preassigned parameter values (Table 1) and the developmental assumptions about
 389 reproduction, B_x , and survivorship, L_x . A): Static fecundity and survival. B): Static fecundity,
 390 survival cost to reproduction. C): Linear development in fecundity, static survival. D): von
 391 Bertalanffy development in fecundity, static survival. E): Linear development in fecundity,
 392 survival cost to reproduction. F): von Bertalanffy development in fecundity, survival cost to
 393 reproduction. G): Linear development in fecundity only before reproduction begins, static
 394 survival. H): von Bertalanffy development in fecundity only before reproduction begins, static
 395 survival.

APPENDIX

All else being equal, selection does not favor organisms that delay reproduction (Williams 1966). This statement can be proven with simple algebra for any reproduction (B_x), survivorship (L_x), and maximum lifespan (ω) values, as long as those values are not themselves functions of age at first reproduction (α). Equivalent statements can be made with partial differential equations for $dr/d\alpha$ (Bell 1980), linear algebra on the Leslie matrix (Tuljapurkar 1990), or the Lambert function (Lehtonen 2016).

Imagine two genotypes, both described under the discrete Euler-Lotka model (main text eq. 1). The only difference between genotypes is that Genotype I begins reproducing at $\alpha = A$, whereas Genotype II begins reproducing at $\alpha = A + 1$. No reproduction or survival terms shift as a function of α . Asserting a stable age distribution, Genotype II must shift (ϵ) in growth rate:

$$1 = \sum_{x=A}^{\omega} e^{-rx} B_x L_x \quad \text{Genotype I}$$

$$1 = \sum_{x=A+1}^{\omega} e^{-(r+\epsilon)x} B_x L_x \quad \text{Genotype II}$$

The right-hand sides of both genotype equations are equal:

$$\sum_{x=A}^{\omega} e^{-rx} B_x L_x = \sum_{x=A+1}^{\omega} e^{-(r+\epsilon)x} B_x L_x$$

Because all corresponding values of B_x and L_x are equal across genotypes, this rearranges:

$$e^{-rA} B_A L_A = \sum_{x=A+1}^{\omega} e^{-rx} B_x L_x (e^{-\epsilon x} - 1)$$

Given the Genotype I equation and biological definitions of reproduction and survivorship, the left-hand side is zero (if $B_A = 0$) or positive (if $B_A > 0$). The term $(e^{-\epsilon x} - 1)$ must be zero or positive for some value x . This condition is only possible if $\epsilon \leq 0$. The increase in α for Genotype II thus corresponds to a zero or negative contribution to r .

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