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 should evolve older ages at first reproduction if 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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"Natural selection will always, ceteris paribus, *favor rapid development; the sooner an organism matures the less likely it is to die before maturing and reproducing…The development of longer juvenile phases in a phyletic line must always be considered a price paid for some more important development."*

- Williams (1966*a*, pp. 87–88)

INTRODUCTION

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

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

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

CONDITIONS FOR SELECTION ON DELAYED REPRODUCTION

A primary goal of life history theory has been to understand "delayed reproduction," or the fact that some organisms do not reproduce as quickly as possible (Cole 1954). Following verbal arguments by Williams (epigraph), subsequent theory added rich quantitative depth to predictions of optimal age at first reproduction, especially in terms of body size and mortality (Wittenberger 1979; Bell 1980; Caswell 1982; Stearns and Koella 1986; Kozłowski and Stearns 1989; Kozłowski 1992). However, these complicated mathematical approaches have made it harder to recognize key assumptions about organismal development.

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

$$\mathbf{1} = \sum_{x=\alpha}^{\omega} e^{-rx} B_x L_x \qquad \textit{eq. 1}$$

This model maps age-specific reproduction and survivorship to population growth rate for a given genotype in a given environment (Table 1). The model assumes discrete age-classes, either zero environmental variation or zero phenotypic plasticity, zero density dependence, a stable age

distribution, and an exponential population growth rate. Relaxing these assumptions can have vast implications for life history theory, many of which have been thoroughly investigated (e.g., Tuljapurkar 1990; Stearns 1992; Reznick et al. 2002; Caswell et al. 2018)**.**

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

Here, we show how different qualitative features of development prevent, or permit, selection for delayed reproduction. In biological reality, development involves diverse processes of somatic and extra-somatic organismal growth and differentiation over time. In the Euler-Lotka model, "development" means an increase in reproduction (B_x) or survival (component L_X) as a function of age. This definition positions development as the inverse of senescence (i.e., an age-related decline in reproduction or survival), which is a more classic topic in life history theory (Hamilton 1966; Charlesworth 2000).

Insofar as explicit discussions about the role of development in life history evolution already exist, those discussions tend to treat ontogeny as important in terms of plasticity (i.e., how does the environment perturb or stimulate an organism's eventual phenotype?) or constraint (i.e., what set of phenotypes might an organism, or lineage, eventually obtain?; see especially valuable contributions in Stearns 1982; Maynard Smith et al. 1985; West-Eberhard 2003; González-Forero 2024). In contrast, we focus on the evolutionary consequences of development in the sense that organisms undergo structural processes of change and differentiation—plastic or

105 otherwise, functional or constrained, perturbed or not—that fundamentally establish their age-
106 related capacities to survive and reproduce.
107
108 *Impossible: static fecundity and survival*
109 In the confines of the Euler-Lotka model, selection does not favor delayed reproduction
110 in a lineage with no development. An example is given using a model of static fecundity ($B_x = b$)
111 and static survival rates ($L_x = S^x$; eq. 2).

$$\mathbf{1} = \sum_{x=\alpha}^{\omega} e^{-rx} b S^x \qquad \textit{eq. 2}$$

112 This model, and the next one, are extraordinary in assuming newborns are identical to
113 adults. We assigned example values for all parameters except α and *r* (Table 1). The genotype
114 with the highest *r* is the one with the lowest α value, which reproduces most quickly (Fig. 1A).
115 This conclusion applies to all lineages with static fecundity and survival rates, including
116 ones with high adult survival rates (Appendix). Despite conventional wisdom radiating from
117 frameworks involving *r-K* or *fast-slow* life history spectra (Pianka 1970; see also Stearns and
118 Rodrigues 2020), the Euler-Lotka model does not automatically predict that lineages with high
119 fecundity should begin reproducing more quickly, nor that organisms with a longer lifespan
120 should begin reproducing more slowly. A long lifespan is not a sufficient condition for the
121 evolution of delayed reproduction.
122
123 *Insufficient: direct reproductive costs*
124 Bell (1980) frames the evolution of age at first reproduction in terms of the direct cost to
125 reproduction, such as an energetic cost. Curiously, Bell's analysis also makes clear that such

costs are insufficient for selection to favor delayed reproduction. We can model a direct cost by splitting survivorship into juvenile and adults survival rates, which apply before and after age at first reproduction respectively ($L_x = j^{\alpha}s^{x-\alpha}$; eq. 3). All else being equal, a direct cost is represented by $j > s$.

$$\mathbf{1} = \sum_{x=\alpha}^{\omega} e^{-rx}(b)(j^{\alpha}s^{x-\alpha}) \qquad \textit{eq. 3}$$

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

Insufficient: Guaranteed development

Development that proceeds freely throughout an organism's life—with no direct cost, opportunity cost, or tradeoff—is also insufficient for selection favoring delayed reproduction. For example, simple models of organismal development might involve a linear increase in annual fecundity ($B_x = dx$; eq. 4) or characteristic growth rate towards some asymptotic maximum fecundity ($B_x = F(1 - e^{-gx})$; Stearns and Koella 1986).

$$\mathbf{1} = \sum_{x=\alpha}^{\omega} e^{-rx}(dx)S^{x} \qquad \textit{eq. 4}$$

Although fecundity is no longer static in these models, the benefits of development are still independent of age at first reproduction. Genotypes that delay reproduction develop to the same

145 extent as those reproducing immediately, and merely incur the risk of dying before reproduction.
146 As a result, selection still favors genotypes that reproduce as quickly as possible (Fig. 1C-D,
147 Appendix).
148
149 *Sufficient: Developmental opportunities*
150 It finally becomes possible for selection to favor delayed reproduction when the risks and
151 benefits of development are themselves linked to age at first reproduction. This link can occur in
152 at least two ways within the Euler-Lotka model. The first involves increasing fecundity via
153 guaranteed development (e.g., $B_x = dx$) combined with a cost to reproduction ($L_x = j^{\alpha}s^{x-\alpha}$,
154 given $j > s$; eq. 5).

$$\mathbf{1} = \sum_{x=\alpha}^{\omega} e^{-rx}(dx)(j^{\alpha}s^{x-\alpha}) \qquad \textit{eq. 5}$$

155 In this case, the optimal age at first reproduction can be intermediate, balancing the benefits of
156 development with the risks of mortality (Fig. 1E-F; Williams 1966*a*, pp. 88–89, 177–178;
157 Stearns 1992, pp. 128–135).
158 Tradeoffs are a core construct in life history theory (Lack 1954; Williams 1966*b*; Stearns
159 1989; Reznick et al. 2000). Note, however, the curious role that tradeoffs play in this model.
160 Development offers an increasing benefit relative to a static cost (s/j). Phrased in terms of van
161 Noordwijk and de Jong (1986), development here increases resource acquisition orthogonal to
162 the axis of resource allocation in the tradeoff between survival and reproduction. In other words,
163 selection operates not because an organism is caught up in a tradeoff *per se*, but rather because
164 development may act to transform the terms of that tradeoff throughout the life of an organism.

165 In addition, selection can favor delayed reproduction given a direct tradeoff between
166 development and reproduction. Consider a strict version of this tradeoff, in which fecundity
167 develops only prior to the age at first reproduction (e.g., $B_x = d\alpha$; eq. 6).

$$\mathbf{1} = \sum_{x=\alpha}^{\omega} e^{-rx}(d\alpha)S^x \qquad \textit{eq. 6}$$

168 Here, the only cost to reproduction is the missed opportunity to develop. A genotype that delays
169 reproduction can prolong development and benefit from a lifetime increase to annual fecundity
170 (Fig. 1G-H).
171
172 *Hidden developmental processes*
173 In these simple Euler-Lotka model comparisons, qualitative assumptions about
174 developmental processes provide the grounds for evolutionary predictions of age at first
175 reproduction. Some modeling decisions obscure these developmental processes. First, juvenile
176 mortality rates (j) often exceed adult mortality rates (s; Williams 1966*a*). This is a common
177 assumption about development, according to which survival rate not only increases as a young
178 organism ages, but also outweighs any survival cost paid for reproduction itself.
179 Second, there may be some absolute minimum threshold for maturation. Stearns (*pers.*
180 *comm.*) raises a case in which genotypes share a fixed minimum body size for maturity but differ
181 in somatic growth rate. Selection may favor the evolution of delayed reproduction if growing
182 slower also means surviving better (e.g., foraging more slowly, but more safely; cf. Stearns 1992,
183 pp. 124–125; Ebert 1994). In that example, the developmental assumption is that maturation
184 occurs at a given body size.

185 Third, beyond the Euler-Lotka model, density-dependent life history models make
186 predictions based on different density-dependent effects for juveniles *vs.* adults (Reznick et al.
187 2002; Travis et al. 2023). Modeling any difference in the impact of density-dependence on
188 young/prebreeding *vs.* old/breeding stages makes an axiomatic statement about development:
189 young is different than old. Such differences could presumably be tied to variation in juvenile
190 capacities with respect to predator avoidance, foraging competition, social competition, etc.
191 However, it is common for practitioners to assume, but not specify, some ontogenetic process.
192
193 **DISCUSSION**
194 Under the Euler-Lotka model, qualitative and quantitative predictions for optimal age at
195 first reproduction depend on the properties of reproduction (B_x) and survivorship (L_x) functions
196 (Fig. 1). Relative values of r, and therefore all evolutionary predictions, depend on at least three
197 features of a given model: (1) the constituent parameter values of developmental functions, such
198 as d, F, and g; (2) the form of developmental functions, such as $B_x = dx$ or $B_x = F(1 - e^{-gx})$;
199 and (3) the form of tradeoffs between development, survival, and reproduction, such as s/j or
200 $B_x = d\alpha$.
201 These parameters, functions, and tradeoffs serve as the developmental axioms of any life
202 history model. Given certain developmental axioms, including static survival and fecundity (Fig.
203 1A, Appendix) or guaranteed development (Fig. 1C-D), it is impossible for the Euler-Lotka
204 model to predict selection favoring delayed reproduction. It becomes possible for selection to
205 favor delayed reproduction when development transforms a survival/reproduction tradeoff (Fig.
206 1E-F) or when development itself trades off with reproduction (Fig. 1G-H). In all cases, relative
207 values of r depend not only on the form of these tradeoffs, but also the form of developmental

functions (e.g., Fig. 1G *vs*. H) and the parameter values within functions (Table 1). Constraining a set of developmental axioms is the only way to generate finite solutions in a life history optimization problem (Stearns 1976).

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

Birds teach us that developmental functions evolve. Recent phylogenetic studies document the convergent evolution of sexual bimaturism in polygynous, lekking birds (Ancona et al. 2020; Taylor and Prum 2023 [preprint]). In distinct lineages of lekking species such as pheasants (Phasiandae), manakins (Pipridae), and bowerbirds (Ptilonorhynchidae), males provide

no parental care. Instead, they solicit mates with sexual displays at display territories (Bradbury 1981). Young males, but not females, must develop lek display sites and displays, although these developmental processes are poorly understood (Collis and Borgia 1992; McDonald 2007; Schaedler et al. 2021; Spezie and Fusani 2023). Parallel to the difference in developmental demands, males in polygynous lekking species delay reproduction longer than females (Wiley 1974; McDonald 1993; Ancona et al. 2020; Taylor and Prum 2023 [preprint]). The Euler-Lotka model could predict the sex-specific evolution of age at first reproduction in lekking birds, given the unknown function that represents sociosexual development in young males. But the Euler-Lotka model offers no predictions about the evolution of lekking, which redefines the relationship between sociosexual development and reproduction.

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

1994 on *Daphnia*). These models do not make predictions about the evolution of insect metamorphosis, which redefines the models needed to predict evolution.

CONCLUSION

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

Stearns (2000) argued that classic life history theory failed to predict the observed phenotypic diversity of life. The failure to translate "microevolutionary" predictions into "macroevolutionary" ones is often viewed as an empirical problem: ecology is complicated, all models are wrong, etc. Research efforts have thus focused on more generalizable demographic models (e.g., de Vries and Caswell 2019), exemplary empirical tests (e.g., Travis et al. 2023), and more complete comparative datasets (e.g., Healy et al. 2019).

In contrast, we argue this failure has conceptual, not empirical, roots. The Euler-Lotka and related life history models assume some kinds of evolution occur and others do not. One makes this assumption even when one assumes environments are stable, ecologies are simple, and measurements are perfect. Outside of the model, the relative scope of "microevolution" and "macroevolution" is not clearly defined. The model then defines a "microevolutionary" realm within its predictive scope: the adaptive evolution of age-related allocation to survival and reproduction with respect to developmental axioms. Simultaneously, the model defines a "macroevolutionary" realm: the evolution of developmental axioms. Thereafter, microevolution

involves predictable changes with respect to static evolutionary rules, whereas macroevolution involves non-predictable changes to the rules themselves. The model cannot bridge the gap between microevolution and macroevolution; the model defines the gap.

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

Across a disciplinary silo, evolutionary developmental biology (Gould 2002; Wagner 2014) is studying not only morphological rules ("*Baupläne*"), but also historical changes to those rules ("innovations;" Wagner and Muller 2002), and the quantitative dynamics of evolution

within and across rulesets ("evolvability," Wagner and Altenberg 1996). The challenge is to reintroduce life history—time, age, reproduction, survival (Cole 1954)—to the hitherto morphological concepts of innovation (Wagner and Muller 2002; Müller and Newman 2005). Theoretical studies will help us understand how life history evolution proceeds when the process of optimization under a ruleset unearths, or forecloses, evolvability to other rulesets. Empirical studies can document when innovations in morphological, physiological, behavioral, social, or cultural mechanisms have in historical fact come to route and reroute life history evolution.

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438 **Table 1**. Life history variables and parameters in discrete Euler-Lotka models. Example values
439 were used for predictions in Figure 1.
440

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	≥ 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)	≥ 0, < 1	0.80
j	Juvenile survival rate	≥ 0, < 1	0.95
s	Adult survival rate	≥ 0, < 1	0.80

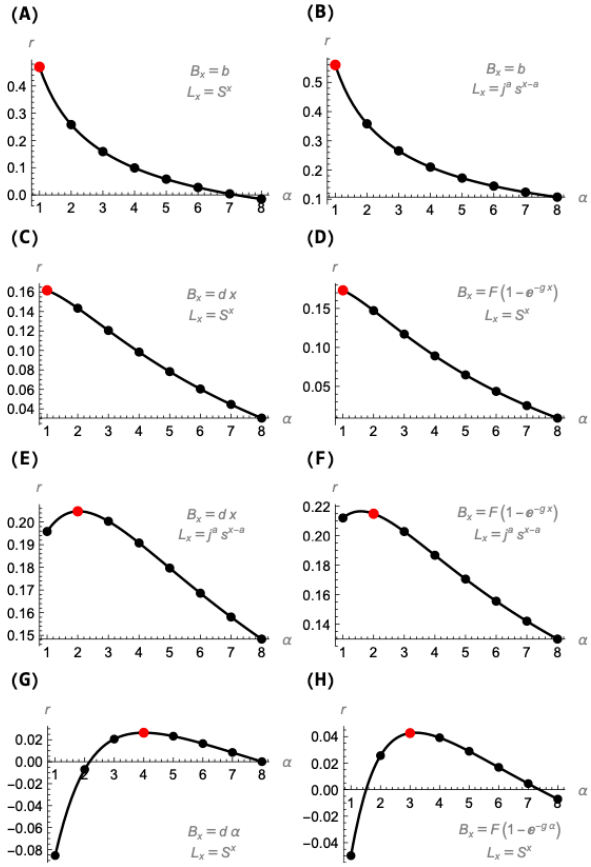

Figure 1. Genotype growth rates, *r*, for different ages at first reproduction (α) according to discrete Euler-Lotka life history models. Optimum ages at first reproduction (red point) vary with the developmental assumptions about reproduction (B_x) and survivorship (L_x), along with variation in pressing parameter values (not shown, see Table 1). A): Static fecundity and survival. B): Static fecundity, survival cost to reproduction. C): Linear development in fecundity, static survival. D): von Bertalanffy development in fecundity, static survival. E): Linear development in fecundity, survival cost to reproduction. F): von Bertalanffy development in fecundity, survival cost to reproduction. G): Linear development in fecundity only before reproduction begins, static survival. H): von Bertalanffy development in fecundity only before reproduction begins, static 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 will thus shift in growth rate by some amount (ε):

$$\mathbf{1} = \sum_{x=A}^{\omega} e^{-rx} B_x L_x \qquad \textit{Genotype I}$$

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

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

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

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

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

Given the Genotype I equation and biological definitions of reproduction ($\boldsymbol{B_x \geq 0}$) and survivorship ($\boldsymbol{0 \leq L_x \leq 1}$), the left-hand side is zero (if $\boldsymbol{B_A = 0}$) or positive (if $\boldsymbol{B_A > 0}$). The

term ($\boldsymbol{e^{-\epsilon x} - 1}$) must therefore be zero or positive for some value *x*. This condition is only possible if $\epsilon \leq \mathbf{0}$. The increase in α for Genotype II thus corresponds to a zero or negative contribution to *r*.