NOTE

DEVELOPMENTAL AXIOMS IN LIFE HISTORY EVOLUTION

Liam U. Taylor[1]* and Richard O. Prum[1,2]

[1] Department of Ecology and Evolutionary Biology and [2]Yale Peabody Museum, Yale University, New Haven, CT 06511, USA

*liam.taylor@yale.edu

Running Title: *Developmental life history*

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

ACKNOWLEDGEMENTS
We are grateful to Stephen Stearns and Daniel Stadtmauer for feedback on the manuscript and years of stimulating discussion. This work was supported by an NSF GRFP grant (#DGE1752134) for L.U.T. and the W.R. Coe fund from Yale University.

Main text word count: 2,972

CONTENTS

"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 (Stearns 1982; Smith et al. 1985; 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). In early verbal arguments, Williams recognized the evolution of delayed reproduction depends on facts about development (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 demonstrate some fundamental features of life history evolution using the discrete Euler-Lotka model (Cole 1954; Stearns 1992; eq. 1).

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

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, stable age distribution, and exponential population growth rates. Changing 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 specific set of life history traits in a given environment—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 the Euler-Lotka model, "development" means an increase in reproduction (B_x) or survival (component L_X) as a function of age. In reality, development involves diverse processes of somatic and extra-somatic organismal growth and differentiation over time; we discuss the consequence of such biological complexity below. Note that both quantitative and biological definitions make development the mirror-image of senescence, a classic topic in life history theory (Hamilton 1966; Charlesworth 2000).

Impossible: static fecundity and survival

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

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

Insufficient: direct reproductive costs

Bell (1980) frames the evolution of age at first reproduction in terms of the direct cost to 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 ($L_x = j^{\alpha} s^{x-\alpha}$). All else being equal, a direct cost is represented by $j > s$.

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 ($B_x = dx$) or characteristic growth rate towards some asymptotic maximum fecundity ($B_x = F(1 - e^{-gx})$; Stearns and Koella 1986). 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 extent as those reproducing immediately, and merely incur the risk of dying before reproduction. As a result, selection still favors genotypes that reproduce as quickly as possible (Fig. 1C-D, Appendix).

Sufficient: Developmental opportunities

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

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

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

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

Hidden developmental processes

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

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

DISCUSSION

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

These parameters, functions, and tradeoffs serve as the developmental axioms of any life history model. Under certain developmental axioms, including static survival and fecundity (Fig. 1A, Appendix) or guaranteed development (Fig. 1C-D), it is impossible for the Euler-Lotka model to predict selection favoring delayed reproduction. It becomes possible for selection to favor delayed reproduction when development transforms a survival/reproduction tradeoff (Fig. 1E-F) or when development itself trades off with reproduction (Fig. 1G-H). In all cases, relative 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 timescales 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 was viewed as an empirical problem. 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. 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 trees, water, and air. 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.

LITERATURE CITED

Ancona, S., A. Liker, M. C. Carmona-Isunza, and T. Székely. 2020. Sex differences in age-to-maturation relate to sexual selection and adult sex ratios in birds. Evolution Letters 4:44–53.

Beccari, E., P. Capdevila Lanzaco, R. Salguero-Gómez, and C. Pérez Carmona. 2023. Mammal life history strategies are strongly associated with environmental realms. BioRxiv [preprint].

Bell, G. 1980. The Costs of Reproduction and Their Consequences. The American Naturalist 116:45–76.

Biewener, A. A. 2005. Biomechanical consequences of scaling. Journal of Experimental Biology 208:1665–1676.

Bradbury, J. W. 1981. The Evolution of Leks. Pages 138–169 *in* R. D. Alexander and D. W. Tinkle, eds. Natural Selection and Social Behavior. Chiron Press, New York.

Burger, J. R., C. Hou, and J. H. Brown. 2019. Toward a metabolic theory of life history. Proceedings of the National Academy of Sciences 116:26653–26661.

Caswell, H. 1982. Life History Theory and the Equilibrium Status of Populations. The American Naturalist 120:317–339.

———. 2000. Matrix population models. Sinauer, Sunderland.

Caswell, H., C. de Vries, N. Hartemink, G. Roth, and S. F. van Daalen. 2018. Age ×

stage-classified demographic analysis: a comprehensive approach. Ecological Monographs 88:560–584.

Charlesworth, B. 2000. Fisher, Medawar, Hamilton and the Evolution of Aging. Genetics 156:927–931.

Charnov, E. L. 1993. Life history invariants: some explorations of symmetry in evolutionary ecology. Oxford University Press, Oxford.

Clauset, A. 2013. How large should whales be? PloS one 8:e53967.

Cole, L. C. 1954. The population consequences of life history phenomena. The Quarterly review of biology 29:103–137.

Collis, K., and G. Borgia. 1992. Age-related effects of testosterone, plumage, and experience on aggression and social dominance in juvenile male satin bowerbirds (*Ptilonorhynchus violaceus*). The Auk 109:422–434.

de Vries, C., and H. Caswell. 2019. Stage-Structured Evolutionary Demography: Linking Life Histories, Population Genetics, and Ecological Dynamics. The American Naturalist 193:545–559.

Ebert, D. 1994. A Maturation Size Threshold and Phenotypic Plasticity of Age and Size at Maturity in *Daphnia magna*. Oikos 69:309–317.

Ernsting, G., C. Zonneveld, J. A. Isaaks, and A. Kroon. 1993. Size at Maturity and Patterns of Growth and Reproduction in an Insect with Indeterminate Growth. Oikos 66:17–26.

Goldbogen, J. A. 2018. Physiological constraints on marine mammal body size. Proceedings of the National Academy of Sciences 115:3995–3997.

Gould, S. J. 1977. Ontogeny and phylogeny. Harvard University Press, Cambridge, Massachusetts.

———. 2002. The structure of evolutionary theory. Harvard University Press, Cambridge, Massachusetts.

Hamilton, W. D. 1966. The moulding of senescence by natural selection. Journal of Theoretical Biology 12:12–45.

Harvey, P. H., and M. D. Pagel. 1991. The comparative method in evolutionary biology (Vol. 239). Oxford University Press, Oxford.

Healy, K., T. H. G. Ezard, O. R. Jones, R. Salguero-Gómez, and Y. M. Buckley. 2019. Animal life history is shaped by the pace of life and the distribution of age-specific mortality and reproduction. Nature Ecology & Evolution 3:1217–1224.

Kozłowski, J. 1992. Optimal allocation of resources to growth and reproduction: implications for age and size at maturity. Trends in ecology & evolution 7:15–19.

Kozłowski, J., M. Konarzewski, and M. Czarnoleski. 2020. Coevolution of body size and metabolic rate in vertebrates: a life-history perspective. Biological Reviews 95:1393–1417.

Kozłowski, J., and S. C. Stearns. 1989. Hypotheses for the production of excess zygotes: models of bet-hedging and selective abortion. Evolution 43:1369–1377.

Lack, D. 1947. The significance of clutch-size. Ibis 89:302–352.

———. 1954. The Natural Regulation of Animal Numbers. Clarendon Press, Oxford.

Love, A. C. 2015. Conceptual Change and Evolutionary Developmental Biology. Pages 1–54 *in* A. C. Love, ed. Conceptual Change in Biology, Boston Studies in the Philosophy and History of Science (Vol. 307). Springer Netherlands, Dordrecht.

McDonald, D. B. 1993. Demographic consequences of sexual selection in the long-tailed manakin. Behavioral Ecology 4:297–309.

318 ———. 2007. Predicting fate from early connectivity in a social network. Proceedings of the
319 National Academy of Sciences 104:10910–10914.
320 Müller, G. B., and S. A. Newman. 2005. The innovation triad: an EvoDevo agenda. Journal of
321 Experimental Zoology Part B: Molecular and Developmental Evolution 304B:487–503.
322 Pianka, E. R. 1970. On r- and K-Selection. The American Naturalist 104:592–597.
323 Reznick, D., M. J. Bryant, and F. Bashey. 2002. r- and K-selection revisited: The role of
324 population regulation in life history evolution. Ecology 83:1509–1520.
325 Reznick, D., L. Nunney, and A. Tessier. 2000. Big houses, big cars, superfleas and the costs of
326 reproduction. Trends in Ecology & Evolution 15:421–425.
327 Roff, D. A. 1992. Evolution of life histories: theory and analysis. Chapman & Hall, New York.
328 Rolff, J., P. R. Johnston, and S. Reynolds. 2019. Complete metamorphosis of insects.
329 Philosophical Transactions of the Royal Society B: Biological Sciences 374:20190063.
330 Schaedler, L. M., P. H. L. Ribeiro, and L. T. Manica. 2021. Phenotype networks reveal
331 differences between practice and courtship displays in swallow-tailed manakins. Animal
332 Behaviour 171:29–39.
333 Smith, J., R. Burian, S. Kauffman, P. Alberch, J. Campbell, B. Goodwin, R. Lande, et al. 1985.
334 Developmental Constraints and Evolution: A Perspective from the Mountain Lake
335 Conference on Development and Evolution. The Quarterly Review of Biology
336 60:265–287.
337 Spezie, G., and L. Fusani. 2023. Sneaky copulations by subordinate males suggest direct fitness
338 benefits from male–male associations in spotted bowerbirds (*Ptilonorhynchus*
339 *maculatus*). Ethology 129:55–61.
340 Stearns, S. C. 1976. Life-history tactics: a review of the ideas. The Quarterly review of biology

51:3–47.

Stearns, S. C. 1982. The role of development in the evolution of life histories. Pages 237–258 *in* Evolution and Development: Report of the Dahlem Workshop on Evolution and Development Berlin 1981, May 10–15. Springer Verlag, Berlin.

———. 1989. Trade-Offs in Life-History Evolution. Functional Ecology 3:259–268.

Stearns, S. C. 1992. The Evolution of Life Histories. Oxford University Press, New York.

———. 2000. Life history evolution: successes, limitations, and prospects. Naturwissenschaften 87:476–486.

Stearns, S. C., and J. C. Koella. 1986. The evolution of phenotypic plasticity in life-history traits: predictions of reaction norms for age and size at maturity. Evolution 40:893–913.

Stearns, S. C., and A. M. M. Rodrigues. 2020. On the use of "life history theory" in evolutionary psychology. Evolution and Human Behavior, Current debates in human life history research 41:474–485.

Taylor, L. U., and R. O. Prum. 2023. Social context and the evolution of delayed reproduction in birds. BioRxiv [preprint].

Travis, J., R. D. Bassar, T. Coulson, A. Lopez-Sepulcre, and D. Reznick. 2023. Population regulation and density-dependent demography in the Trinidadian guppy.

Truman, J. W. 2019. The Evolution of Insect Metamorphosis. Current Biology 29:R1252–R1268.

Truman, J. W., and L. M. Riddiford. 1999. The origins of insect metamorphosis. Nature 401:447–452.

Tuljapurkar, S. 1990. Delayed reproduction and fitness in variable environments. Proceedings of the National Academy of Sciences 87:1139–1143.

van Noordwijk, A. J., and G. de Jong. 1986. Acquisition and Allocation of Resources: Their

Influence on Variation in Life History Tactics. The American Naturalist 128:137–142.

Wagner, G. P. 2014. Homology, genes, and evolutionary innovation. Princeton University Press, Princeton.

Wagner, G. P., and L. Altenberg. 1996. Perspective: complex adaptations and the evolution of evolvability. Evolution 50:967–976.

Wagner, G. P., and G. B. Muller. 2002. Evolutionary innovations overcome ancestral constraints: a re-examination of character evolution in male sepsid flies (Diptera: Sepsidae). Evolution and Development 4:1–6.

Western, D., and J. Ssemakula. 1982. Life history patterns in birds and mammals and their evolutionary interpretation. Oecologia 54:281–290.

Wiley, R. H. 1974. Evolution of social organization and life-history patterns among grouse. The Quarterly review of biology 49:201–227.

Williams, G. C. 1966*a*. Adaptation and natural selection. Princeton University Press, Princeton.

———. 1966*b*. Natural Selection, the Costs of Reproduction, and a Refinement of Lack's Principle. The American Naturalist 100:687–690.

Wittenberger, J. F. 1979. A Model for Delayed Reproduction in Iteroparous Animals. The American Naturalist 114:439–446.

TABLES AND FIGURES

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

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	≥α	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

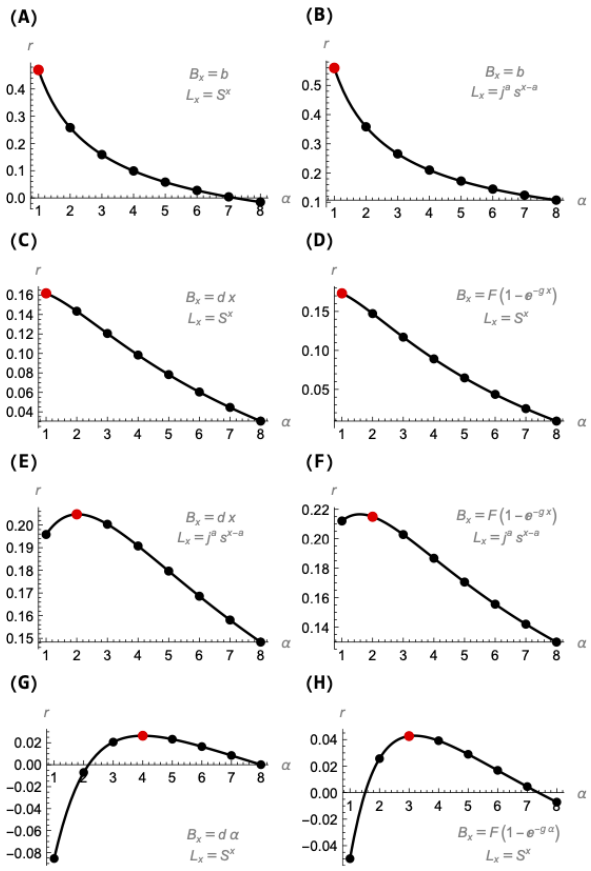

Figure 1. Genotype growth rates, *r*, for different ages at first reproduction (α) according to discrete Euler-Lotka life history models. Optimum ages at first reproduction (red point) vary with preassigned parameter values (Table 1) and the developmental assumptions about reproduction, B_x, and survivorship, L_x. 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 must shift (ε) in growth rate:

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

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

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

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

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*.

LITERATURE CITED

Bell, G. 1980. The Costs of Reproduction and Their Consequences. The American Naturalist 116:45–76.

Lehtonen, J. 2016. The Lambert W function in ecological and evolutionary models. Methods in Ecology and Evolution 7:1110–1118.

Tuljapurkar, S. 1990. Delayed reproduction and fitness in variable environments. Proceedings of the National Academy of Sciences 87:1139–1143.

Williams, G. C. 1966. Adaptation and natural selection. Princeton University Press, Princeton.