Chapter 9

Limitations

Calculus and biology

Calculus is the branch of mathematics that is built on the premise that values and changes in those values can be divided up in ever smaller, indeed infinitely small (infinitesimal) pieces. Hamilton used calculus to devise models of infinitesimal age-specific changes, which is biologically unrealistic (Chapter 3). In Chapter 5, my co-authors and I have tried to improve on Hamilton's method by showing how Hamilton's indicators fit in Arthur's more general framework of perturbation analysis of scalar demographic metrics. The method calculates the change in fitness if mortality and fecundity are perturbed *across all ages*, rather than age-specifically. Also, we show how trade-offs can be modeled within this framework. Yet, it remains calculus applied to biology. Although the entire life history is perturbed at once in Chapter 5, i.e. all ages are affected, it is still done so in an infinitesimal manner: the perturbation function that we use is an infinite vector of age-specific changes that have some magnitude relative to each other, but are all infinitely small nevertheless. It is unlikely that this is the character of real biological perturbations, if only because living beings have a finite number of genes that together produce a very non-infinitesimal effect. Yet, Chapter 5 is instrumental in rejecting unjustified claims that have been made with reference to calculus, and as a linear approximation of real perturbations of life histories.

Age-structuring

Populations can be structured in many ways, both in empirical studies and in theoretical models [1]. Age is probably the only variable used for classification that in and of itself has absolutely no effect on anything at all. Without doubt, and without exception, age causes nothing. Its explanatory power results only from its correlation with yet-to-be-discovered underlying causal pathways. Still, it is intuitive to structure populations by age, specifically when one is interested in aging. Hamilton [2] showed how mathematically

convenient it is to derive sensitivities of fitness to age-specific changes. Theories of aging have largely been postulated in terms of age-specific changes [e.g.3,4,5, but see 6 and Chapters 3 and 4]. Still, in the end, age is just a 'parameterization': a way to describe a pattern. The parameterized pattern is independent of the parameterization, as is any calculation done on the pattern. Readers with mathematical background may liken this parameterization to the parameterization of a line integral: the result does not depend on the parameterization chosen to describe the line.

If one is not continuously aware of the fact that age is in principle irrelevant, analyzing age-structured models is playing with fire. It is easy to come up with some age-specific mathematical equation, but accounting for it biologically is much more challenging. One cannot freely dream up age-specific changes in vital rates without thinking about how these should come about, and about how changes in vital rates at different ages are related (Chapter 3). Books on age-structured population models [e.g.7] should come with a big warning that age-classification is essentially beside the point.

Models identify only sufficient conditions

Typically, evolutionary models of aging ask the question: suppose that the mechanistic constraints can be mimicked by these (relatively simple) equations, what mortality and fecundity patterns emerge? Would aging evolve, or not? When the model is good, it gives a set of parameters for which aging is predicted to evolve, and a set of parameters for which it is not. This means that *if* such equations and such parameter settings indeed mimicked real world phenomena, they could explain these phenomena. Or not. In terms of predicting real world phenomena, theoretical models construct only a *sufficient* condition for the predicted outcome (I am grateful to Dr. Giaimo for stating this so clearly). They are an instrument for conceptualizing thinking, and for assuring that reasoning is self-consistent. They do not, however prove anything about reality. Specifically, they do not produce a *necessary* condition. Sometimes it is tested whether models are 'consistent' with observed data [e.g. 8]. That is better than nothing, but it is also little more than nothing. What is shown is that the model can produce a pattern such as the one observed, but this is often trivial due to a great number of free parameters. It is risky to claim any 'success' of optimization models in explaining patterns [3], as many mechanisms, including many trade-offs, can produce the same pattern. This warning pertains also to the model in Chapter 7. It is unlikely that the mechanism that inspired the model, proposed in Chapter 4, is the only mechanism at play, and that all pattern like the patterns produced in Chapter 7 find their roots in the mechanisms laid out in Chapter 4.

References

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