Evolutionary Demography
Evolutionary Shaping of Demographic Schedules

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Darwin and Demography

Darwinian natural selection is a story about demographic success.

It makes sense to try to understand the age-specific patterns realized in demographic schedules from species to species in the light of evolution.
1. An overview of three strands of evolutionary demography:
   - Mutation Accumulation;
   - Stochastic Vitality;
   - Optimal Life Histories

2. Implications of a mathematical result about mutation accumulation.
   - A generalization is proved in the written paper.

3. Prospects for resolving a central open question.
   - A need for combining strands.


Part 1: The Three Strands: A Preview

- Mutation Accumulation: Similarities between flies and me.
- Stochastic Vitality: Differences between you and me.
- Optimal Life Histories: Differences between elephants and you.
FIGURE 2  Age-specific death rates for six species

A. Humans

B. Medflies

C. Nematodes

D. Wasps

E. Drosophila melanogaster

F. Beetles

NOTE: Dots indicate values directly calculated from data, and solid lines indicate estimates using frailty models. In each panel, the vertical dotted line indicates the estimated modal life span, and the vertical axis indicates the number of deaths per individual-OHM of exposure. (OHM is one-hundredth of the modal life span.) SOURCES: See Figure 1.
The rain it raineth every day.

William Shakespeare
The rain is the rain of not-so-good mutations, generation after generation.
The sunshine is natural selection, drying away the raindrops.
The woolen jacket is your genome.
The weight of wet is genetic load.
Sir Peter Medawar, 1951, An Unsolved Problem in Biology
Mutation Accumulation

Brian Charlesworth (2001), Journal of Theoretical Biology:

“The second is the ‘mutation accumulation’ theory, first proposed by Medawar, and which refers to the fact that deleterious alleles with effects restricted to late stages of life equilibrate at higher frequencies at mutation-selection balance than alleles that act earlier...”

- Large numbers of deleterious alleles each with small age-specific effects on survival.
- Natural selection weeds out more slowly bad alleles that only or mainly affect you when your days for procreating parenting and grandparenting are running short, leaving more at the equilibrium of “mutation-selection balance”.
Stochastic Vitality Encapsulates Individual Heterogeneity

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Evolutionary Demography
Differing genetic endowments and vagaries of development create heterogeneity in physiology and susceptibility to environmental shocks.

Unobserved heterogeneity fixed across life is called frailty, modeled by Vaupel, Manton, and Stallard (1979) with proportional hazards.

Stochastic vitality generalizes fixed frailty; “vitality” changes across life in a stochastic, usually Markovian process. When vitality hits a bottom threshold, you die.
Ken Manton, Anatoli Yashin, and others have high-dimensional representations of vitality in terms of a suite of physiological indicators, stochastic risk factor models.

Transition rates are estimated from longitudinal data sets like the Framingham studies.

Qualitative properties of stochastic vitality are studied with stylized models like a Brownian motion hitting a barrier.

Odd Aalen, David Steinsaltz, Steve Evans show that plateaus in hazard rates at advanced ages are a generic consequence of the mathematics of “quasi-stationarity”.
Life Histories Adapt to Optimize Returns on Investments

Table: Bank of Life: Statement for Account 666

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<th>Item</th>
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<th>Debits</th>
<th>Credits</th>
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</tbody>
</table>
Optimal Life History Theory is familiar from a long tradition in biology with organisms making adaptive tradeoffs over the life course to maximize reproductive success.

Tradeoffs could be programmed into the genome or implemented as dynamic behavioral responses. They could be found in genetic variation along the lines of “antagonistic pleiotropy” or they could be found in norms of reaction established by alleles that have gone to fixation.

Tom Kirkwood, proponent of the “disposable soma”, emphasizes investments in growth, maintenance, reproduction, and repair.
The demographic side of the enterprise, represented for instance by Hilly Kaplan, Arthur Robson, Annette Baudisch, Cyrus Chu, and Ronald Lee, emphasizes roles for intergenerational and intergroup transfers in social species. Analysis of returns to investments in different background environments give insight into differences, as between elephants and us. With respect to demographic schedules, by and large the constraints have to be invented to produce the intended shapes. The mathematics does not add predictive power...
Charlesworth (2001) assumed
- (A) a linear approximate theory for mutation accumulation, plus
- (B) mutant alleles with effects restricted to all ranges of later ages, plus
- (C) heavy constant background exogenous mortality

These could combine to imprint a Gompertz-like exponential pattern on mortality rates by age.

There are lots of proposals to explain Gompertz mortality schedules (many getting exponentials out by putting exponentials in).

In Charlesworth’s story, the exponential arises de novo, from a process that is highly generic and plausibly fairly ubiquitous.
The loss of net reproduction from one mutant allele reduces what is left to be lost by other mutant alleles.

The selective cost of a load of mutant alleles is less than the sum of the costs of each of them separately.

Thus natural selection has less force for weeding out mutant alleles over generations to preserve mutation-selection balance.

The theorem, extended in the written paper, shows that these interactions destroy the Charlesworth story.
Interactions Destroy the Equilibrium

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The interactions can be kept in check if effects of mutant alleles are not *restricted* to late ages but only *concentrated* at late ages, with some early-age signatures.

Such early-age signatures would in most cases imply late-age plateaus in mortality rates, such as are observed.
Human mortality rates have dropped a lot in “recent” times while genomes have only been changing a little.

Evolution shapes schedules only through effects on reproductive and nurturing ages, now no longer late in lifespans.

– mothers, grandmothers, fathers, grandfathers –

Why are mortality rates today in human populations not funny functions of age?
All strands of evolutionary demography face a challenge in positing ways for preserving regular shapes while levels hugely alter.

Sink or slide?

Are rates dropping age by age or is progress sliding back the same schedules to ever higher ages?

In an exponential Gompertz phase, there would not be an visible difference.
Sinking? Sliding?

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Age-specific effects of mutant alleles on mortality schedules could plausibly be imagined to operate through effects on transition rates for processes of stochastic vitality.

“Recent” human progress could plausibly be imagined to change the threshold of lethality as well as introducing favorable drift to vitality trajectories.

Probability models for financial meltdowns by Evans, Ettinger, and others provide new mathematical tools.

Under what assumptions would combinations of mutation accumulation, stochastic vitality, and moving thresholds let mortality rates slide back to later ages while preserving evolutionary signatures?