

Interplay of Genetic and Actual Lifespans

Hwang *et al.* (HKR) [1] proposed a model of evolution contrasting genetic and actual lifespans in the presence of natural death and competitive death (crowding). Organisms mutate under a fixed bias toward longer or shorter genetic lifespans. Counterintuitively, HKR conclude that organisms live longer for mutational biases to shorter genetic lifespans (subcritical cases); for biases to longer genetic lifespans (supercritical cases) the actual lifespan is a constant independent of bias. We find, however, that their results are due to specific model assumptions. HKR assume that organisms that would produce offspring with genetic lifespans shorter than a nonzero artificial lower bound simply do not reproduce, which reduces the competitive pressure in subcritical cases and increases the actual lifespan of others. This assumption aside, the average of actual lifespans is asymptotically independent of the genetic lifespan and is determined only by competitive death in their model. This independence is itself sensitive to the model assumptions and is affected by various biologically justified model changes. Finally, HKR neglect to mention that, within a particular population, there is a positive correlation between genetic and actual lifespans.

We first note that the average age A used by HKR is a different quantity from the *true* average of actual lifespans (the average time between birth and death), which we call L hereafter. A equals L for a pure exponential age structure, which asymptotically occurs for supercritical and critical cases in HKR, but not for subcritical cases. While these two quantities have qualitatively similar dependence on mutational bias, the use of L makes it much easier to understand the origin of their results.

If the age structure is stationary, as in all the asymptotic regimes in HKR and model variants discussed below, the exact value of L is simply the total population divided by the total birth rate, i.e., $L = N / \sum_n C_n(0)$. The denominator is the sum over the birth rates for all genotypes. Calculating the denominator in HKR's model gives $L = N / (BN - b_- P_1)$, where P_1 is the number of organisms with the shortest genetic lifespan and b_- is the mutation rate to shorter genetic lifespans. This form shows that a nonzero value of P_1 caused by the bias to shorter genetic lifespans gives rise to an increase of L above the reference $L = B^{-1}$. $b_- P_1$ appears here because HKR prohibit offspring with genetic lifespans shorter than a nonzero artificial lower bound, 1. Organisms that would produce offspring violating this assumption simply do not reproduce, resulting in less competitive pressure and thus longer actual lifespans [this conclusion also applies to A since asymptotically $L \leq A$ in HKR as $L \sim \langle\langle (\gamma N + 1/n) \rangle\rangle^{-1}$ while $A \sim \langle\langle (\gamma N + 1/n)^{-1} \rangle\rangle$, where $\langle\langle \rangle\rangle$ is a weighted sum with weight P_n/N]. In other models this would not apply. For example, imagine modifying the genetic lifespan to approach zero geometrically using a natural death rate h^n ($0 < h < 1$, $-\infty < n < \infty$). In this model, for a station-

ary age structure, we obtain $L = B^{-1}$ regardless of bias. We prove the stationary age structure in subcritical cases (it is trivial for supercritical and critical cases) by showing that steady state is reached: if the characteristic value of n goes to $-\infty$ then N must approach zero, however, extinction is not possible (except for a trivial case $b, b_+ = 0$). Models with a geometric approach to a nonzero lower bound of genetic lifespans trivially give the same conclusion. This shows that the increase of the actual lifespan under bias to shorter genetic lifespans in HKR is due to an unjustified model assumption.

Aside from the effect of this assumption, the average of actual lifespans in HKR is decoupled from the genetic lifespan. This decoupling can be removed in various ways, for example, by assuming an immature (nonreproductive) period in proportion to the genetic lifespan, as is generally found in biology. With this assumption, since organisms with longer genetic lifespans must become older to make offspring, the total population decreases and the actual lifespans increase as the genetic lifespans increase. The genetic lifespans are bounded for a nonzero total population. This new model is obtained by modifying the definition of $C_n(0, t)$ in HKR to be $C_n(0, t) = b Q_n(t) + b_+ Q_{n-1}(t) + b_- Q_{n+1}(t)$, $Q_n(t) = \int_{\lambda n}^{\infty} C_n(a, t) da$, and $Q_0 = 0$, where λ is the ratio of the immature period to the genetic lifespan. Asymptotically $N \sim \sum_{n=1}^{\infty} Q_n \exp\{\lambda n(\gamma N + \frac{1}{n})\}$, which shows that N must approach zero if the characteristic value of n increases without bound. Such extinction may occur if λ is large. Otherwise, the characteristic value of n is bounded and a stationary age structure results. We calculate the asymptotic value of L to be $L \sim [\sum_{n=1}^{\infty} Q_n \exp\{\lambda n(\gamma N + \frac{1}{n})\}] / [B \sum_{n=1}^{\infty} Q_n - b_- Q_1]$. With nonzero N , L depends on the characteristic value of n , resulting, as expected, in a *positive* correlation between genetic and actual lifespans.

Finally, regardless of model variations, there is an interpretational problem with HKR's conclusions. They compare model worlds with different biases, but do not compare organisms competing in a particular world. The latter can be considered more significant in addressing the question they pose in their title, "Does good mutation help you live longer?". The asymptotic average age (= actual lifespan in this case) as a function of genetic lifespan n in HKR is $(\gamma N + 1/n)^{-1}$, which shows that there is a *positive* correlation between genetic and actual lifespans in a population.

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[1] W. Hwang *et al.*, Phys. Rev. Lett. **83**, 1251 (1999).