

Microbial ageing and longevity

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Supplementary Box 1 — Ecological conditions favoring the evolution of microbial aging (reproductive asymmetry) versus non-aging (reproductive symmetry).

Both microbial longevity and senescence can have evolutionary advantages, depending on the ecological circumstances. Mechanistically, longevity is characterized by processes of cellular repair and maintenance that directly combat damage, while senescence is characterized by accumulation of damage and the processes of asymmetric reproduction that sequester it¹ (see **Mechanisms of microbial aging** section). The relative costs and benefits of repair and maintenance versus asymmetric reproduction—reproduction-longevity tradeoffs—will determine whether senescence evolves^{2,3}. Combating damage expends ATP, time, and other resources⁴⁻⁷. Repair promotes longevity, but spending too much time and energy on damage repair could cause an organism to lose the opportunity to reproduce, perhaps due to extrinsic mortality or to insufficient energy resources remaining for reproduction.

Resource-rich environments support rapid rates of cellular activity, metabolism, growth, and reproduction, which as a byproduct can increase the rate of macromolecular damage. When macromolecular damage is high, repair is insufficient or too inefficient to cope with damage, and the evolution of asymmetric reproduction with its associated possibility of senescence can be favored^{2,3,8-13}. The benefits of the ability to quickly make use of resources for growth and reproduction in an energy-rich environment outweigh the cost of senescence.

On the other hand, the strategy of cellular repair excels in circumstances when damage is low enough to be kept in check^{3,13}. This would typically occur in stable, low-energy environments

where metabolic efficiency is advantageous and where slowed metabolic rates also slow the incidence of damage^{3,14–18}. As one might expect if low-energy environments select for repair and longevity, the long-lived microorganisms inhabiting ecosystems like the deep biosphere commonly exhibit signatures of cellular repair^{14,15,19}. Selection for repair and longevity in low-energy conditions may even provide an evolutionary basis for the observation that a low energy diet or dietary restriction extends lifespan in both unicellular and multicellular taxa^{20,21}.

References

1. Kirkwood, T. B. & Austad, S. N. Why do we age? *Nature* **408**, 233–238 (2000).
2. Chao, L. A model for damage load and its implications for the evolution of bacterial aging. *PLoS Genet.* **6**, e1001076 (2010).
3. Watve, M., Parab, S., Jogdand, P. & Keni, S. Aging may be a conditional strategic choice and not an inevitable outcome for bacteria. *Proc. Natl. Acad. Sci.* **103**, 14831–14835 (2006).
4. Mattoo, R. U. H. & Goloubinoff, P. Molecular chaperones are nanomachines that catalytically unfold misfolded and alternatively folded proteins. *Cell. Mol. Life Sci.* **71**, 3311–3325 (2014).
5. Rang, C. U., Proenca, A., Buetz, C., Shi, C. & Chao, L. Minicells as a damage disposal mechanism in *Escherichia coli*. *mSphere* **3**, (2018).
6. Ferenci, T. Trade-off mechanisms shaping the diversity of bacteria. *Trends Microbiol.* **24**, 209–223 (2016).
7. Lennon, J. T. & Jones, S. E. Microbial seed banks: the ecological and evolutionary implications of dormancy. *Nat. Rev. Microbiol.* **9**, 119–130 (2011).

8. Rang, C. U., Peng, A. Y., Poon, A. F. & Chao, L. Ageing in *Escherichia coli* requires damage by an extrinsic agent. *Microbiology* **158**, 1553–1559 (2012).
9. Erjavec, N., Cvijovic, M., Klipp, E. & Nyström, T. Selective benefits of damage partitioning in unicellular systems and its effects on aging. *Proc. Natl. Acad. Sci.* **105**, 18764–18769 (2008).
10. Koleva, K. Z. & Hellweger, F. L. From protein damage to cell aging to population fitness in *E. coli*: Insights from a multi-level agent-based model. *Ecol. Model.* **301**, 62–71 (2015).
11. Ackermann, M., Chao, L., Bergstrom, C. T. & Doebeli, M. On the evolutionary origin of aging. *Aging Cell* **6**, 235–244 (2007).
12. Chao, L., Rang, C. U., Proenca, A. M. & Chao, J. U. Asymmetrical damage partitioning in bacteria: a model for the evolution of stochasticity, determinism, and genetic assimilation. *PLoS Comput. Biol.* **12**, e1004700 (2016).
13. Lin, J., Min, J. & Amir, A. Optimal segregation of proteins: phase transitions and symmetry breaking. *Phys. Rev. Lett.* **122**, 068101 (2019).
14. Johnson, S. S. *et al.* Ancient bacteria show evidence of DNA repair. *Proc. Natl. Acad. Sci.* **104**, 14401–14405 (2007).
15. Orsi, W. D., Richards, T. A. & Santoro, A. E. Cellular maintenance processes that potentially underpin the survival of subseafloor fungi over geological timescales. *Estuar. Coast. Shelf Sci.* **164**, A1–A9 (2015).
16. Clegg, R. J., Dyson, R. J. & Kreft, J.-U. Repair rather than segregation of damage is the optimal unicellular aging strategy. *BMC Biol.* **12**, (2014).
17. Baig, U. I., Bhadbhade, B. J., Mariyam, D. & Watve, M. G. Protein aggregation in *E. coli*: short term and long term effects of nutrient density. *PLoS ONE* **9**, e107445 (2014).

18. Bradley, J. A., Amend, J. P. & LaRowe, D. E. Survival of the fewest: microbial dormancy and maintenance in marine sediments through deep time. *Geobiology* (2018).
doi:10.1111/gbi.12313
19. Jørgensen, B. B. & Marshall, I. P. G. Slow microbial life in the seabed. *Annu. Rev. Mar. Sci.* **8**, 311–332 (2016).
20. Adler, M. I. & Bonduriansky, R. Why do the well-fed appear to die young? A new evolutionary hypothesis for the effect of dietary restriction on lifespan. *BioEssays* **36**, 439–450 (2014).
21. Kapahi, P., Kaeberlein, M. & Hansen, M. Dietary restriction and lifespan: lessons from invertebrate models. *Ageing Res. Rev.* **39**, 3–14 (2017).