

Why do living things age at different rates?

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A long life and slow aging are not the same measurement. An organism may die young because its environment is dangerous even if its risk of death has not increased much with age. Another may survive for a long time while showing a pronounced age-related decline. Before asking why species age differently, we need to decide whether we are comparing lifespan, the rate of functional change, or the changing risk of death among survivors.

This distinction matters especially across very different organisms. A familiar mammal's life history is not a universal template for a tree, an invertebrate or a clonally growing organism. The unit whose age is being measured can itself require a decision.

1. A risk conditional on having survived

Mortality at a specified age concerns individuals still alive at that age. It is different from the fraction of the original population that has already died. A survival curve can decline continuously even when the risk faced by each surviving individual is constant.

Consider an invented population in which each survivor has a 10 percent chance of dying during each yearly interval, independently of age. Starting with 1,000 individuals, the expected number after ten intervals is $1,000 \times 0.9^{10}$, or about 349. Many have died, yet the model includes no increase in risk with age. A falling survivor count alone therefore does not demonstrate demographic senescence.

Now compare a population whose yearly risk rises from one interval to the next. Its age pattern contains a different phenomenon, even if the two populations happen to have the same average lifespan. A single average compresses information that the age-specific pattern retains.

2. Diversity is an observation, not an exception to be erased

Jones and colleagues compared standardized age patterns of mortality and reproduction across diverse animals and plants. They found increasing, approximately constant, decreasing and more complex trajectories rather than a single universal shape. [1] This does not imply that any organism is immune to damage or can survive every hazard. It means that the familiar pattern of steadily rising adult mortality cannot simply be assumed for all life.

Comparative data also have limits. Species differ in how they are observed, whether records come from protected or natural conditions, and how age or stage is established. A curve reconstructed from limited observations is not equally certain at every point, especially when few old individuals remain. Those limitations should qualify the comparison without turning all patterns into unknowable quantities.

The useful result is a change in the explanatory demand. A theory must account for diversity, not merely explain one curve and label every other shape anomalous.

3. Damage, maintenance and organization

At the cellular level, aging research investigates interacting changes in genomes, proteins, organelles, signaling and tissue renewal. The updated hallmarks framework proposed by López-Otín and colleagues organizes twelve candidate processes, including genome instability, loss of protein maintenance, mitochondrial dysfunction and altered cellular communication. [2]

A framework of mechanisms is not a ranked list proving that the first item causes every other one. Processes can interact, and their importance can differ among tissues, organisms and conditions. An intervention that changes one measured feature may act through several routes. Establishing causal order is harder than documenting that multiple changes accompany age.

Repair also has a target and a cost. Maintaining one component does not automatically preserve the whole organism, just as replacing one part of a complex device does not establish that every other part remains sound. The analogy is useful only for the distinction between component maintenance and system-level performance; organisms also grow, remodel and respond in ways a passive machine does not.

4. Evolutionary and immediate explanations differ

An immediate explanation asks how damage develops or maintenance changes. An evolutionary explanation asks why inherited systems with those patterns persisted across generations. Identifying a molecular lesion does not by itself explain why a lineage evolved its particular investment in maintenance, growth and reproduction.

Life-history reasoning considers these allocations and the environments in which reproductive success occurs. It does not require a conscious decision by the organism or a universal program whose purpose is to make old individuals die. Nor can one infer a precise maintenance budget from lifespan alone. The same observed lifespan can arise through several combinations of physiology and external risk.

A proposed trade-off should therefore generate a comparison. Does changing allocation alter both the relevant reproductive outcome and the maintenance outcome? Are those effects consistent across environments? A plausible evolutionary story remains a hypothesis until the observations distinguish it from alternatives.

5. The comparison can change with the unit

For an organism that continually produces new modules, the age of a leaf, branch or stem need not equal the age assigned to the larger individual. Damage to one module and persistence of the lineage can coexist. A measurement of one part cannot automatically be converted into the aging rate of the whole.

The same issue appears within animals at a different scale. Different tissues can show different trajectories, and a blood-based measurement does not directly reveal every organ's state. An index may predict an outcome in a studied population while remaining incomplete as a mechanistic measure of aging.

Suppose a hypothetical marker falls after an intervention. That demonstrates a change in the marker under the tested conditions. It does not, without additional evidence, demonstrate longer life, preserved function or reduced disease across all tissues. The distinction between a surrogate measurement and the outcome of interest prevents a promising result from becoming an unsupported universal claim.

6. What we can reasonably conclude

Living things differ in their maintenance systems, development, ecological circumstances and life histories. Their observed aging patterns also depend on what is measured and on the population being followed. Comparative demography documents this diversity, while molecular frameworks identify interacting processes that can contribute to decline. [1,2]

The unresolved problem is how those processes combine to produce a particular trajectory. There is no single established cause that fixes every species' aging rate, and a result in one experimental organism is not proof of a human anti-aging intervention. A careful answer keeps lifespan, age-specific risk, cellular mechanisms and evolutionary explanations connected without treating them as synonyms.

Related reading

Article 033, “Why do we need sleep?”, considers recurring physiological maintenance and the evidence needed to establish its functions. It is an optional reciprocal comparison, not a claim that sleep determines every aging pattern.

References

- [1] O. R. Jones et al. Diversity of ageing across the tree of life. *Nature* 505, 169–173 (2014). <https://doi.org/10.1038/nature12789>
- [2] C. López-Otín, M. A. Blasco, L. Partridge, M. Serrano and G. Kroemer. Hallmarks of aging: An expanding universe. *Cell* 186, 243–278 (2023). <https://doi.org/10.1016/j.cell.2022.11.001>