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Lifetime Expectancy

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Synonyms

Aging rate, Biological time, Life cycle, Life expectancy, Life history, Lifespan Longevity, Maximal longevity.

Definition

Lifetime expectancy can be defined as either the average or the maximal duration of the lifetime (lifespan) of individuals of an animal species after birth or eclosion. The average or typical (modal) duration of individual lifespan is dependent on a variety of factors, including habitat, habits, extrinsic mortality rates, and environmental variables, but still varies within the maximal known lifespan of the species.

Introduction

How long an animal lives is not a freely variable amount of time that depends simply on extrinsic, environmental factors. Fruit flies and lab worms live not much more than 3 weeks in the best of conditions; mice hardly ever reach 2 years of life even if kept fed, sheltered, and safe from all predators in controlled animal housing facilities; the healthiest humans are still not expected to live much more than one century. The fact that different animal species have characteristic maximal lifetime expectancies, and that these vary across species from under 1 day to over one century, strongly indicates that there are innate, biological determinants of longevity. This entry explores some of the current knowledge and theories about what factors determine lifespan, and therefore lifetime expectancy.

Life History, Biological Time, and the Pace of Life

How much of life goes by for different species within a similar period of time? Only 4 days separate the day when an individual *Caenorhabditis elegans* worm eclodes and when it is biologically capable of laying eggs and reproducing, and only under very rare circumstances and conditions will that worm individual still be alive 1 month later. In contrast, humans are still just about as helpless at 1 month as they were at 4 days of age, and it will take over a decade until they become biologically competent to reproduce. Although the biological bases of animal life are universal, life runs at a different *pace* for different species: *biological time* is not directly comparable across animals. The combined, absolute, and relative duration of time spent in each stage of life – embryonic development, infancy/postnatal growth, adolescence, sexually competent adult life, old age – is referred

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to as the *life history* of a species. How come biological time and therefore life history varies by four orders of magnitude across species remains one of the main fundamental unanswered questions in biology – although important insights have occurred recently.

The temperature at which the body functions and therefore at which all chemical reactions occur is of course a major variable across the enormous diversity of non-vertebrate animals, and also among fishes, amphibians, and non-avian reptiles. Broadly speaking, the warmer the temperature at which the body of an animal works, the faster its metabolic rate (as expected from simple thermodynamics), the faster the pace of life, and the shorter the lifespan of the animal. Similarly, metabolic rate per gram of body also decreases as animals become larger, and along with it the pace of life seems to slow down, and life expectancy increases. However, this relationship breaks down upon closer inspection. For instance, birds function at systematically higher body temperatures and have predictably higher metabolic rates than mammals of similar size, yet birds have a lifetime expectancy that is as much as 20 times that of rodents. Indeed, as explored further below, even when body temperature is removed from the equation by nature, life history remains enormously variable. Across endotherm, homeotherm animal species (birds and mammals), songbirds live systematically longer lives than primates, which in turn live systematically longer lives than non-primate mammals of similar body mass.

Despite the apparent regularities across the entire animal phylum as a whole, it is obvious already at the most superficial level of observation that different animal species live at different rates. There is enormous diversity in life, and not one single way or pace of living. What determines biological time for different animal species?

Extrinsic Factors

For decades, one favored explanation for the different life histories spanning four orders of magnitude across animal species, from one day to one century, was extrinsic mortality rates: the higher the likelihood of being predated upon on an average day, the stronger is the selective pressure for a species to develop quickly and reach sexual maturity earlier rather than later, which over generations would lead to lifetime acceleration as an adaptive feature driven by natural selection (Charnov 1993). As a consequence, longevity should increase as the probability of death caused by extrinsic factors decreases (Austad and Fischer 1991). In support of that proposition, there are known instances in which documented changes in predation risk have indeed been accompanied by changes in lifetime expectancy in the predicted direction within particular species. In the case of birds, flight has been evoked as a factor that significantly lowers predation risk and thus might account for the slower life history and longer life expectancy (Munshi-South and Wilkinson 2010) – although in that case *all* bats would be expected to be longer-lived than other mammals of similar size, which is not the case. Besides the scant experimental support, one major difficulty with this line of reasoning is that it presupposes that biological life histories are highly plastic, which is not compatible with available evidence. Most importantly, pinning the determination of lifetime expectancy down on extrinsic factors runs inevitably into a mathematical tautology (since increased longevity *is defined* as lower probability of death at any age) and circular reasoning that do not account for *how* diversity in biological lifetimes would arise in the first place, before any selection occurs. Another strong argument against extrinsically determined life histories is that there are striking regularities in lifetime expectancy across species, explored below, that make it far more likely that there are intrinsically, biologically determined mechanisms that set the pace of growth, maturation, and aging.

Intrinsic Factors: Rate-of-Living and Rate-of-Aging

One highly influential theory for an endogenous mechanism of setting the pace of life is the appropriately named Rate-Of-Living theory (Pearl 1928), according to which the duration of life of an organism is curtailed upon the exhaustion of a fixed quantity of something vital at a rate proportional to the metabolic rate. The value of this fixed

quantity would depend on an intrinsically, genetically determined "metabolic potential," and also on the rate of metabolism, which may be determined extrinsically (by environmental temperature, e.g., in the case of heterotherm animals) or intrinsically (in the case of endotherm animals).

As mentioned above, body temperature does affect all chemical reactions in an organism, including proteostasis (the balance between rates of protein synthesis, folding, damage, and disposal), and impact life history in *C. elegans* as expected, shrinking life history and shortening lifetime expectancy as populations are raised in warmer environments (Stroustrup et al. 2016). Shorter life histories as a whole, and lifetime expectancies, in particular, are predicted by models that presume progressive accumulation of damages to both proteins (Santra et al. 2019) and DNA (Hoeijmakers 2009), depending both on temperature and metabolic rate (West 2018). The process of accumulation of damages to proteins, DNA, lipids, and other vital molecules and structures is now widely considered as an underlying feature of aging. Thus, variation in lifetime expectancy across animal species and individuals is currently largely presumed to originate from variations in the rate of aging. At this point, however, variations in the rate of aging across species remain mostly an intuition not yet backed by strong experimental support. For example, while the rate of telomere shortening and presumably DNA repair ability is an excellent predictor of maximum longevity across a small sample of nine warm-blooded species (Whittemore et al. 2019), it is possible that it is the larger accumulation of DNA damages with a longer life that makes DNA repair ability appear higher in longer-lived species. It is difficult to tease apart cause and consequence in the generation of lifetime diversity from simple correlations.

Resilience to Aging

Recently, however, a novel variable brought into play did make simple correlations highly revealing by showing that body size and metabolic rate are not required to explain the wide range of variation in lifetime expectancy across warm-blooded animals, or the clade-specific differences (Herculano-Houzel 2019). The combination of a large dataset on life history with a novel dataset on the numbers of neurons that compose the brains of different species showed that across birds and mammals alike (not including bats, which were not available for analysis), variations in the estimated number of neurons in the pallium (cerebral cortex) account for as much as 74% of the variation in lifetime expectancy (measured as maximal longevity) across a sample of 750 species. Most importantly, clade-specific differences across birds, primates, and other mammals disappear; and after accounting for the number of pallial neurons in each species, residual analysis shows that body size and specific metabolic rate are mathematically irrelevant. Thus, the more the cortical neurons found in the adult brain of a warm-blooded species, the longer its life history, with sexual maturity reached only at older ages and expanded maximal longevity after reaching maturity – and humans are no exception (Herculano-Houzel 2019). It is too early to tell what lies underneath that correlation, and even if numbers of cortical neurons do have a determining role in life history and longevity or are simply a proxy for the true determinant factor, although it has been proposed that the correlation *is* causal through increased resilience to aging incurred with more postmitotic, cortical neurons in the brain (Herculano-Houzel 2019). Importantly, this proposition is well in agreement with the large body of evidence showing that in a wide range of animal species, and also across individuals, caloric restriction and exercise, the two manipulations that undisputedly extend longevity, also protect neurons in the cerebral cortex in a variety of ways.

Conclusion

Biological time differs across species, as evidenced by the fact that there is enormous diversity in lifetime expectancies across animal species, and that this diversity in turn originates from similarly enormous diversity in life histories. The pace of life does have some dependence on factors that affect metabolic rate, like body size and temperature, especially across heterotherm

animals. However, a new possibility to be investigated is that in homeotherms, the damages that define aging accumulate at similar rates, regardless of metabolic rate, and a new factor comes into play: the number of cortical neurons in the network that maintains the physiological integration of the body, in such a way that the larger that population of neurons is, the more resilient is the network, and so longer the body remains viable.

Cross-References

- ▶ Allometry
- ▶ Life History
- ▶ Life History (Various Clades)
- ▶ Senescence

References

Austad, S. N., & Fischer, K. E. (1991). Mammalian aging, metabolism, and ecology – Evidence from the bats and marsupials. *Journal of Gerontology, 46*, B47–B53.

Charnov, E. L. (1993). *Life history invariants. Some explorations of symmetry in evolutionary ecology.* Oxford: Oxford University Press.

Herculano-Houzel, S. (2019). Longevity and sexual maturity vary across species with number of cortical neurons, and humans are no exception. *Journal of Comparative Neurology, 527*, 1689–1705.

Hoeijmakers, J. (2009). DNA damage, aging, and cancer. *New England Journal of Medicine, 361*, 1475–1485.

Munshi-South, J., & Wilkinson, G. S. (2010). Bats and birds: Exceptional longevity despite high metabolic rates. *Aging Research Reviews, 9*, 12–19.

Pearl, R. (1928). *The rate of living*. London: University of London Press.

Santra, M., Dill, K. A., & de Graff, A. M. R. (2019). Proteostasis collapse is a driver of cell aging and death. *Proceedings of the National Academy of Sciences USA, 116*, 22173–22178.

Stroustrup, N., Anthony, W. E., Nash, Z. M., Gowda, V., Gomez, A., López-Moyado, I. F., Apfeld, J., & Fontana, W. (2016). The temporal scaling of Caenorhabditis elegans ageing. *Nature, 530*, 103–121.

West, G. (2018). *Scale: The universal Laws of life, growth, and death in organisms, cities, and companies*. New York: Penguin Press.

Whittemore, K., Vera, E., Martínez-Nevado, E., Sanpera, C., & Blasco, M. A. (2019). Telomere shortening rate predicts species life span. *Proceedings of the National Academy of Sciences USA, 116*, 15122–15127.