Derivation and inference on the bivariate age- and size-specific mortality.

Fernando Colchero[1], Annette Baudisch[2], Ralf Schaible[3].

[1] Department of Mathematics and Computer Science (IMADA) and Max-Planck Odense Center on the Biodemography of Aging (MaxO); [2] Max Planck Research Group on Modeling the Evolution of Aging, Max Planck Institute for Demographic Research; [3] Laboratory of Evolutionary Demography, Max Planck Institute for Demographic Research.

The term "senescence" refers to the physiological deterioration of an organism as it gets older, which results into a systematic increase in the age-specific risk of mortality and a decrease in the fertility rate with age. Evolutionary theories of aging all seem to agree that senescence is unavoidable in nature. However, species for which individuals keep growing as they get older (also known as "indeterminate growers") commonly show increases in fertility and declines in mortality with age. These species are thought to show negative senescence, a process that - in theory - entails physiological improvements through which individuals are capable of escaping senescence. Those opposing the notion of negative senescence argue that the real advantages behind larger sizes are only mechanical and not physiological, i.e. the actual mechanisms of senescence might still be at play, but they are overshadowed by the fact that larger individuals are less likely to be predated upon while they can produce more offspring because they have fewer physical constraints as they grow. The debate is far from solved; current mortality models cannot distinguish between the contributions of age and size to mortality since they tackle age- and size-specific mortality separately or as independent processes. We derive a model that expresses age- and size-specific mortality as a bivariate process from which the subsequent bivariate survival and probability density functions of ages and sizes at death can be calculated. We then constructed a Bayesian model to draw inference on these rates. Our results show that different effects of age and size on mortality can produce the impression of negative senescene where senescence is still present, while negative senescence can be masked by size effects. These methods should shed light into the debate and allow us to explore the existence and extension of negative senescence in wild populations.