Gompertzian Mortality Curves Are A Natural Consequence of Biological Damage and Repair Mechanisms

Lloyd P. Goldwasser

Department of Demography, University of California, Berkeley

Exponentially increasing mortality rates during adulthood are a common feature of human populations as well as many species of animals. In humans this pattern is observed across countries and time periods; in animals they occur in species as diverse as rodents, birds, fruit flies, and nematodes. The ubiquity of this pattern suggests that it stems from fundamental biological processes rather than conditions or practices that are specific to our society.

Most explanations of the exponential increase in mortality rates invoke particular distributions of frailty or rates of aging. Such explanations are unsatisfying in part because the assumption of a given distribution, however plausible, in effect builds the conclusions into the model. Also, such models often invoke conditions or processes that are not likely to apply as widely as the phenomenon is observed.

I use a simple differential equation model to show that exponential increases in mortality rates can arise directly from basic biological processes of damage and repair. The key feature of the model is that the cellular and biochemical systems that repair damage are themselves produced and regulated by the very systems that they repair. Such systems include the genetic machinery that is common to all life. For instance, DNA is continually subject to damage as an organisms ages, and it simultaneously provides the template from which are synthesized the proteins that repair the damage. The DNA eventually cannot be repaired because the damage to it results in nonfunctioning repair enzymes: the damage is eventually self-catalyzing.

As with populations growing with a constant growth rate, self-catalysis leads to

exponential growth. In this case there is an exponential increase in damage to DNA and cellular components as the individual ages. One consequence may be an exponential increase in the probability of mortality due to that damage.

A levelling off of mortality rates seems to occur in humans, and in many species of animals there is a decline to lower mortality rates at the oldest ages. In this model, mortality plateaus may arise when there are relatively high levels of damage, because at that point new damage overlaps existing damage rather than affecting only intact components. For instance, further damage to DNA may affect proteins that have already ceased to function properly because the pool of functioning proteins has been reduced. Some additional process, though, must be involved in actual declines in mortality rates.