



Richard Price: The Schedules of Mortality

1 RICHARD PRICE: LIFE AND WORKS

Into a gabled-roofed structure of two stories, solid yet unadorned, on the 23rd day of February 1723, came Richard Price, son of an austere religious dissident living under the *restraint of the gospel* (Cone 1952; p. 9). *Observations on the Importance of the American Revolution and the Means of Making It a Benefit to the World*, a pamphlet written in 1785 wherein Price lauded Americans as existing in the space between primitive barbarity and debauched decadence, proved Price a principled cosmopolitan, rather than a parochial partisan. Price maintained a warm correspondence with Arthur Lee, Joel Barlow, Benjamin Franklin, John Adams, Thomas Jefferson, Benjamin Rush, Josiah Quincy, Ezra Stiles, and other American revolutionaries, while concurrently communicating with many an Earl, Viscount, and Prime Minister with a heavy hand on the levers of governmental power within Britain. As he extolled the genius, and appreciated the friendship of other leading men of his age, so he was extolled for his “excellent understanding...boldness and freedom of thinking” and appreciated for “the purity of his views, and the simplicity of his manners.” (Cone 1952; p. 4). Condorcet estimated Price as, “one of the formative minds of the century”¹ (Cone 1952; p. 5). A strong liberal and Unitarian Minister, Richard Price, earned a reputation as a “voluminous writer on religion, morals, politics, and mathematics”² (Price 1903). Even dissenting contemporaries at odds with Price’s political radicalism conceded his *goodness, gentleness, humility, honesty,*

sincerity, and *disinterestedness* (Cone 1952; pp. 2–3). In short, he was a good man in a great age.

Through a lifetime of intellectual accomplishment, his study of life and its insurance is remembered (Mayhew 2014). Standing upon the shoulders of the Reverend Thomas Bayes, John Graunt, Sir William Petty, and the Astronomer Edmund Halle, Price studied mortality and insurance most extensively in the following 1783 publication: *Observations on reversionary payments on schemes for providing annuities for widows, and for persons in old age; on the method of calculating the values of assurances on lives; and on the national debt*. To this was added, *An Essay on the Population of England, From the Revolution to the Present Time*. Both works went through multiple editions and were expanded with appendixes. At large, Price was trying to glean broad themes on life and death as they differed between city and countryside, men and women, past and present, war and peace, fatherland and colony, plenty and famine, health and disease.

Like any scholar, no matter how ingenious, Price had his critics. Among contemporaries, Reverend John Howlett criticized Price for applying what was essentially a proto-Malthusian calculus, reminiscent of a mercantilist vision of economics applied to human demographics. Price, Howlett seems to charge, looked at population as a balance sheet, whereupon gains to one side of the ledger were made out of losses from the other. Contrasting with Price's belief that London's population grew at the cost of depopulating and impoverishing the countryside, Howlett compared London to the *mouth* of the nation, the feeding of which only distributes nutriment to the rest of the body. Howlett cited the growth of Bristol, Birmingham, Liverpool, and Manchester as evidence that London grew with, and not at the expense of, the nation at large. Even Price's straightforward interpretation of war as a drain on population was likewise stood on its head by Howlett. While conceding to Price that war, especially when considering non-combatant casualties of famine and disease, can function to decrease the population, Howlett argued war is apt to stimulate population growth among the farmers and manufacturers that keep men in the field.³

2 INSURING AGAINST MORTALITY

Aided by the haughty conceit of hindsight, one could continue on in the vein of Howlett, finding ever more faults in the accuracy of Price's data and the soundness of his conclusions. Yet, that would miss the

point—the point that Robert J. Mayhew (2014; p. 30) makes. Mayhew, though a biographer of Malthus, capably explains the significance of Price’s work on mortality:

When Richard Price stood on the podium on November 4, 1789, he was one of the most respected and revered scholars of his age, that reputation the result of four decades of patient inquiry. The full range of his achievement has been well analyzed...., but one key element of that achievement, one motif in his great melody was his work on population and insurance. In many ways, Price’s core concern—to quantify mortality rates to allow for successful life insurance schemes based on mathematically robust tables of life expectancy— sounds prosaic enough, but it in fact fitted into a far more grandiose socioreligious vision of revolutionary and Utopian progress.

Mayhew’s provision of context is all important. Reading Price’s work on mortality is rarely inspiring, but becomes so when situated within this larger “socioreligious vision.” Price’s *vision* came of the intersection of religion and reason made possible by the waning of the latter and the rising of the former during the *Enlightenment*. In illustration of the point, we find Price’s interpretation of God’s plan, not to be submission to divine will or ascetic neglect of self in preparation for a transcendent afterlife, but to encompass the advancement of liberty, dignity, free will, individuality, and intelligence as a means of improving the worldly present. It can thereby be understood how, in service of the same God, Price studied tables of mortality, whereas his forbearers mortified the flesh.

The underlying conditions actuating this transition from sacred to secular, or one might more properly say from theism to deism, are detailed by McNeill (1998; pp. 262–263), the world historian featured in Chapter 9. McNeill, in his *Plagues and Peoples*, describes an early modern Europe wherein “sudden and unexpected death remains a real and dreaded possibility in everyone’s life experience.” Again and again, McNeill hammers home the point that epidemic plague introduced erratic unpredictability into human lives—a reality which purportedly encouraged traditional religious belief and devotion to a personal God.⁴ By way of contrast, during the Enlightenment, small medical advances combined with unplanned ecological adjustments, to *relax the dominion of epidemic disease over human minds and bodies*.⁵ The observations of McNeill and Mayhew, as they are, respectively, related to the

Enlightenment generally, and Price as an actor within that age, insightful as they are, have been limited to descriptions of passively determinant forces. As will be developed within the subsequent section, however, something more biological and active may have been afoot. Pursuant to this possibility, we take up three points in this coming section: Most prosaically and importantly, we are first obliged to explain how mortality relates to life history. Second, we argue that declining mortality risk across millennia of human history occasioned the slowing of life history, a precondition for achieving the *high civilization* characteristic of the Enlightenment. Third, we argue that the Enlightenment not only was partially made possible by reduced random mortality, but was itself partially responsible for further reducing mortality and slowing life history.

3 INTRINSIC VERSUS EXTRINSIC MORTALITY

Within the evolutionary literature, a distinction is made between types of mortality⁶ (Stearns and Hoekstra 2005)—the logic of which Price would have valued. On the one hand, there is *extrinsic mortality*, mortality which can be neither predicted nor controlled; on the other hand, there is *intrinsic mortality*—mortality which can be predicted and controlled (Ellis et al. 2009; Griskevicius et al. 2011). The epidemic death occasioned by plague when it first afflicted Europe and smallpox newly introduced into the New World Amerindian populations are examples of highly extrinsic mortality. On the other hand, *cold winters* (Lynn 1991; Rushton 2000) exemplify intrinsic mortality, in that they are easily predicted and their accompanying mortality risks can be mitigated or even nullified with sufficient preparation (Hertler 2016). These contrasting types of mortality have opposite effects on life history. In the main, extrinsic mortality evokes *r*- or *fLH*-selecting evolution, whereas intrinsic mortality evokes *K*- or *sLH*-selecting evolution. The excessive random death imparted by extrinsic mortality sources retards fitness-relevant returns to protracted gestation, extended juvenile periods, parental care, deferred reproduction, and related *K*- or *sLH*-selected developmental features. Such *K*- or *sLH*-selected strategies, pursued amidst extensive extrinsic mortality would, on average, cause an organism to die before it reproduced. Therefore, the reduction in extrinsic mortality is a necessary condition for the slowing of life history speed; but, importantly, it is not sufficient. *Only intrinsic mortality actively slows life history.* Take, again, the cold winter. This source of intrinsic mortality actively

drives K - or sLH -selected evolution because provisioning, saving, collecting, storing, future-oriented thought, parental investment, intelligence, conscientiousness, and corresponding K - or sLH -selected behaviors distinctly reduce the probability of death for parent and offspring. To conclude by way of an analogy, liken mortality to an absolute number. As the number alone specifies size, the sign specifies direction; so it is with mortality. The absolute rate of mortality effects evolutionary speed, but only by knowing whether mortality is extrinsic or intrinsic, can we know the direction the evolutionary process will take along the life history spectrum.

Now to qualify this dichotomy with appropriate nuance: First, as might be imagined, some mortality risks fall out along a continuum, even while others are more categorically situated. The second point, building on the first, is that these classifications are not immutable for all time (Hertler 2017). More accurately, a mortality risk can sometimes transition from extrinsic to intrinsic, or otherwise fall further down a continuum of controllability. For humans, predicting and controlling mortality risks, and thereby changing them from extrinsic to intrinsic, have been achieved through a complicated history of *gene-culture coevolution*. All traditional sources of human mortality, *predation*, *starvation*, *violence*, and *war*, have become less acutely extrinsic, drifting down the aforementioned continuum of controllability over the course of human evolutionary history. Consider predation first. Partially as an outgrowth of cognitive evolution at the species level, prior to civilization, and enabling the achievement of that state, mankind already had several defenses against predation, a source of mortality that remains extrinsic for most species. Fire and weapons were undoubtedly instrumental. And then there was safety in numbers. Aggregation is a common anti-predatory defense employed, for instance, by herds of ungulates (Levin 2009). As mentioned, however, predation is only one source of extrinsic mortality. Starvation is another. Exchanging nomadic hunting of thinning herds for shepherding flocks of domesticates, while at the same time substituting the gathering of wild foodstuffs for the harvesting of cultivated crops, brought starvation further down the mortality gradient,⁷ making it less common and more preventable. Relatedly, as civilized life and social order augmented, the immanent destruction of nomadic brigandage on settled communities was exchanged for the slow and sustainable tribute to a warrior caste, guaranteeing the industrious husbandman would reap some proportion of what he had sown. Still later, life became insulated

from internal violence via laws and law enforcement. Thereafter, life became further insulated from disease through inoculation and sewage systems. The foundational achievement of civilization, and a precondition⁸ for high culture, is, in these many ways, the ability to stave off mortality and to make its coming more predictable.

These many reductions in extrinsic mortality risks, with the concomitant imposition of intrinsic mortality risks, were instrumental in slowing human life histories, with consequent evolution of longer lives, larger brains, as well as augmented intelligence, parental investment, education, enculturation, cooperation, restraint, conscientiousness, and future-oriented investments. In our view, these were prerequisites out of which the biological capital was wrought, necessary to create the person and productions of men like Richard Price. *Human Accomplishment* documents the effect, with author Charles Murray describing eighteenth-century London as “jammed with men of immense accomplishment...” (Murray 2003; p. 48). By way of illustration, Murray can easily point to men such as Adam Smith, Edmund Burke, James Fox, Edward Gibbon, and Oliver Goldsmith. “By the late 1720s,” Murray (2003; p. 50) writes, “England’s combination of economic prosperity, social stability, and civil liberties had no equivalent anywhere on the continent.” This is nothing if not the signature of an unprecedented slowing of life history.⁹

Going further afield, one might say that the Enlightenment was both a *product of past evolution* and a *driver of future evolution*. Price and contemporaries achieved insight into, and an increasing mastery over, nature, not excluding human nature. Faster and more fully than ever before, humans in this time and place became their own selective agents. Not from the top-down consciousness of one man or mind, but from the bottom-up as an organic aggregate, humans anthropogenically engineered their selective regime rather than passively suffering under it. The further reduction in extrinsic mortality was part and parcel of this larger process. By way of example, consider the small practical advances in social order occasioned by Benjamin Franklin, as discussed in Hertler (2017; pp. 39–40):

They [Enlightenment era Americans] were trying, and largely succeeding, in creating predictable and orderly environments that were conducive to their slow life history strategy. In actualizing this selfish imperative, they helped the communities they lived in. By way of example, consider the thirty odd years that Benjamin Franklin graced early 18th century

Philadelphia. Exemplifying the slow life history, Franklin reacted against correlates of extrinsic mortality such as disease, the robbery of his home, the threat of the frontier, the specter of foreign invasion, and the burning of the southern end of town, by agitating for hospitals and refuse removal, instituting a night watch and lighted streets, organizing and serving in the state militia, submitting the Albany Plan of Union, and promoting fire brigades and fire insurance.

Being an Enlightenment scion in the mold of his good friend Franklin, Price did his share, as citizen and scientist, to further the cause. As progressive citizen, Price strove to make rulers accountable to the office they held and the people they served. This was part of a larger process of curtailing capricious and despotic abuses that, in earlier times, deprived subjects of life and the resources necessary to sustain and perpetuate life. So, where early civilization witnessed the rise of a ruling class that protected the populace from the extrinsic threat of nomadic raiders, by the eighteenth century, the civilizing process progressed such that it began to protect the populace from its protectors.

Then, as mentioned, Price functioned more directly like a cog in this progressive Age of Reason in the character of a social scientist studying mortality. Life insurance schemes arising from the researches of Price, and those like *John Graunt* (1676) who came before him, were a slightly different species of guarantee. Life insurance was the guarantor of the lineage when the life had failed.¹⁰ Life insurance was what rendered unto the family line, what all the other guarantees had failed to render unto the insured. From a genetic perspective, guaranteeing a life or a lineage amounted to something not far from being one and the same. Those genetics lost with the parent's life, because of the proffered dividend, were more likely to live on in the children of the deceased. Life insurance is the guarantee, in a real sense, of *parental effort*, a *K*- or *sLH*-selected marker. The *K*- or *sLH*-selected expend much of their lifetime and energy in rearing their young, whether by direct investments such as holding, lactation, and teaching, or by indirect investments such as collecting and hunting, or its modern-day equivalents, earning and providing. The life insurance dividend, often taken on the life of the breadwinner, then serves to supplement the loss of indirect parental effort, and the theretofore irreplaceable resources lost to the offspring with the loss of the provider.

Condensing the foregoing discussion to its utmost, we interpret the study of mortality by Price during the Enlightenment as an unconscious, though active, expression of niche construction by a K - or sLH -selected person within a K - or sLH -selecting social system, having the effect of further slowing life history toward the K - or sLH -selected end of the life history spectrum.

4 COMPARATIVE EVIDENCE

It is principally incumbent upon us to proffer evidence of mortality's relationship to life history. To this end, consider the following selection of animals catalogued by Hertler (2017; p. 29), all of which show unto-ward longevity, a signature of slow life history:

Creosote and Yucca of the Mojave Desert (Sussman et al. 2014; Bellingham and Sparrow 2000), the many succulent species within the Grand Canyon of Arizona (Bowers et al. 1995), cave salamanders (Speakman and Selman 2011), Siberian actinobacteria (Sussman et al. 2014), and the great majority of Antarctic plant species (Green et al. 2007)...Quahog clams (Bodnar 2009; Philipp and Abele 2009), tortoises and turtles (Gibbons 1987), elephants (Wiese and Willis 2004), arboreal primates (van Schaik and Isler 2012), bats (Wilkinson and South 2002; van Schaik and Isler 2012), and birds, specifically parrots and cockatoos (Young et al. 2012).

Not incidentally, the outsized lifespan of these organisms is paired, in every instance, with an uncommon degree of insulation from unpredictable mortality. The first five listed are insulated from unpredictable mortality by the harshness of the ecologies in which they live. Alternatively, the latter six examples all gain a measure of protection, specifically from the extrinsic mortality risk of predation, via a developed defense. As for the clam, turtle and tortoise, that defense is a shell. The elephant has its formidable bulk, allowing an adult to walk safely amidst a pride of lions, while the arboreal primate takes to the trees where ground predators cannot follow.

Bolstering these observations, and following from classical predictions (Medawar 1957; Williams 1957), is Holmes and Austad's (1994) article entitled, *Fly Now, Die Later: Life-History Correlates of*

Gliding and Flying in Mammals. This publication establishes the life history effects of flying via mortality reduction, as does Wilkinson and South's (2002) comparative study of bats with analogous terrestrial rodents. Moreover, Reznick¹¹ et al. (2004) observed deferred senescence in guppy populations insulated from extrinsic mortality risks, whereas Rauser et al. (2009) reported experimental studies establishing the predicted causality underlying this correlation (Hertler 2017). Similarly, experimental populations of bacteria link extrinsic mortality to rapid aging (Rauser et al. 2009). Further still, experimentally parasitized fruit flies show more rapid senescence, paired with a compensatory rise in mating effort (Polak and Starmer 1998), both of which are *r*- or *fLH*-selected traits (Hertler 2017). Later, many other studies would use this model genetic organism to demonstrate the effects of mortality on longevity and similar trade-offs predicted by life history theory (Travers et al. 2015; Gasser et al. 2000). As with *Drosophila*, extrinsic mortality drives longevity and related life history evolutionary effects among social insects (Negroni et al. 2016), including ants (Keller and Genoud 1997), bees (Rueppell et al. 2007), and termites (Keller 1998). Similar effects are found across a swath of sampled amphibians (Johnson et al. 2012) and terrestrial animals (Ricklefs 2010). Lastly, life history theory also explains the sex-based longevity differences (Kruger and Nesse 2006) and sex ratio statistics (Leimar 1996; West and Sheldon 2002; Collin 2006) that so confounded Price (Cone 1952).¹² In short, extrinsic mortality's effect on evolved longevity, a hallmark biological marker of the slow life history, is established via the weight of convergent theoretical, biogeographic, observational, and experimental evidence across taxa.

Chapter 16 demonstrates how individual traits, such as longevity, are in fact integrated into a life history complex. As for our more general thesis relating to civilization as a *product* and *driver* of *K*- or *sLH*-selecting evolution, this can only be implicitly developed in subsequent chapters: (1) Chapter 6 relates mortality to population density, the other driver of life history speed; (2) Chapter 9 describes why life history did not slow uniformly across all geographical areas; (3) Chapter 14 treats government as it is relevant to life history; and (4) Chapter 15 correlates life history evolution with the development of civilization outside of the specific role of mortality.

NOTES

1. Not unlike other towering intellectuals of the age, Price at once played the part of “a penetrating philosopher of questions of free will and determinism, an eloquent apostle of liberty, [and] a leading mathematician and Fellow of the Royal Society” (Mayhew 2014; p. 30).
2. As characterized by Walter Ashburner, son of Samuel Ashburner of Boston, in an introduction to *Letters to and From Richard Price 1767–1790*, published in 1903 by John Wilson & Son.
3. When Howlett begins to discuss migration to colonial possessions, he most precisely defines the relationship between population loss and replacement. Mass emigration does of course detract from the population of the mother country, but not without the counterbalance of early marriage and augmented fecundity. Those left behind have more ample means of substance in direct consequence of excess population being siphoned off. Howlett rests upon the authority of Benjamin Franklin, quoting his 1751 essay on *The Peopling of Countries*, wherein the population of a country is likened to a *polypus* (an archaic appellation for polyp); such that one can “take away a limb, its place is soon supplied; cut it in two, and each deficient part shall speedily grow out of the part remaining.”
4. What is most interesting, not only in that it is a feat of synthesis, but in its implicit description of Price’s life and work, is the following connection made by McNeil (1998, pp. 262–263):

The retreat of plague and malaria and the containment of small-pox were thus essential preparations for the propagation of deistic opinions of the kind that became fashionable in advanced circles in the eighteenth century. A world in which lethal infectious disease seldom seized a person suddenly in the prime of life no longer stood so much in need of belief in Divine Providence to explain such deaths.

5. This is a close paraphrase and reordering of McNeil’s words, the original of which is as follows: “Before the findings of the astronomers and mathematicians of the seventeenth century could become a basis for a popularized world view, therefore, epidemic disease had also to relax its dominion over human minds and bodies.”
6. A terminological word of caution: The contrasting terms *extrinsic* and *intrinsic* mortality are differently used elsewhere to denote aging from without and aging from within; for an example of such unrelated usage see Koopman et al. (2015).
7. This claim is made for settled agriculture as it benefited mankind in the fullness of time, across locales, and at large. No doubt, many earlier

- agriculturalists had a less varied and more impoverished diet than their hunting and gathering counterparts.
8. The word *precondition* is here used advisedly. It is important to understand that the reduction in extrinsic mortality only enables the evolution of a slow life history, but it is the introduction of intrinsic mortality that compels such an evolutionary course.
 9. This theme will be explored in a future work, roughly titled, *Evolutionarily Explaining the European Enlightenment*. Though we are here focusing on extrinsic mortality reductions, these are paired with agricultural surplus, economic growth, and demographic increase over millennia of adaptation to civilization, group selection, and sexual selection, as well as altering mortality regimes.
 10. Though it largely fell outside the scope of Chapter 4, Baker (1999), in his *Fraternity among the French peasantry: Sociability and voluntary associations in the Loire Valley, 1815–1914*, describes small-scale community organizations that reduced extrinsic mortality risks from fire, starvation, famine, and crop failure by distributing risk across all community members.
 11. See Reznick et al.’s original study for nuances, caveats, and qualifications of interest that are beyond the scope of the present review.
 12. Price’s curiosity on this score is described by Cone (1952; p. 47): “Price’s data revealed it [sex ratio] as 20:19, while Dr. William Derham found it to be 14:13. The female life expectancy was greater, however, because the male led a more hazardous and irregular life, and had ‘some particular delicacy’ of constitution. Eleven years later Price still puzzled over these phenomena. He sent to the Royal Society a paper from Dr. Joseph Clarke of the Dublin Lying-in Hospital. The facts were as Price had stated them in 1775. Dr. Clarke, who had become interested in these matters from reading Price’s articles, argued that the male fetus was larger than the female. Therefore it required more nutrition and was more liable to injury at birth.”

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