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Research Article

The contributions of stochastic demography and social inequality to lifespan variability

Hal Caswell

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Abstract

BACKGROUND
Individual lifespans differ. Some of those differences are due to heterogeneity, some to stochasticity. Some of the heterogeneity is due to socioeconomic, physiological, or environmental differences; some to unobserved latent factors. All of these are, from time to time, called inequality.

OBJECTIVE
This paper aims to clarify the relations between heterogeneity, stochasticity, inequality of opportunity, and inequality of outcome in a wider context than has yet been attempted.

METHODS
A population is divided into groups differing in their demographic rates. Markov chain or life table methods provide the moments of longevity for each group. A mixing distribution describes the relative abundance of groups. The variance in longevity is partitioned into within-group and between-group components. The approach applies to longevity, healthy longevity, lifetime reproductive output, and other outcomes.

CONCLUSIONS
Important socioeconomic factors make only a small contribution to the variance in longevity, most of which is due to individual stochasticity. Some exceptions, in laboratory studies of insect populations and interspecies comparisons in biodemography, are explored.

CONTRIBUTION
Recognizing the role of stochasticity clarifies the source and the implications of this important source of variance.

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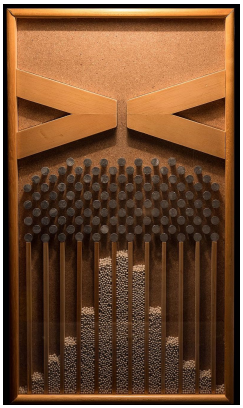
1. Introduction

A series of identical metal balls fall through a hole at the top of a box. Impelled downward by gravity, they encounter a series of identical pins, and bounce, each with identical odds, to the left or the right. The process repeats, over and over, until each ball arrives at its final resting place along the bottom of the box. The device (Figure 1) is called a Galton box. It is an archetypal example of a stochastic process that creates a reproducible statistical result: The distribution of the balls is a realization of a binomial distribution; if the box is made sufficiently deep and enough balls are sampled, the arrangement converges in distribution to the normal distribution.

Figure 1: A Galton box, showing the distribution of identical balls having passed through 10 rows of identical pins.



Source: Wikimedia Commons under a CC-BY-SA license. A video of the box in action is available at `https://commons.wikimedia.org/wiki/File:Galton_box.webm`

A subset of balls at the bottom of the Galton box have moved far to the right. But, to the best efforts of the maker of the box, these balls are identical in every way to the balls that didn't move to the right. They have no inherent 'right-wardness.' If they were extracted from the rightmost end of the distribution and replaced at the top of the box, they would end up distributed from left to right exactly as the first set of balls. There is no inequality among the balls in their tendency or ability to move to the right, or the left.[2]

[2] The Galton box is more than an entertaining desk toy or a classroom demonstration of the binomial distribution. In the 19th century, a set of linked boards functioned as an analog computer for Galton to demonstrate 'laws of heredity' (Galton 1877). By suitably modifying the spacing of the pins, the box can generate other distributions; e.g., a lognormal instead of a normal distribution, serving as a motivation for a model of traffic

A set of identical Swedish women are born at age 0. Impelled by the passage of time, they progress to age 1, age 2, and so on. Upon reaching any given age, every individual encounters an identical risk of mortality. They bounce off this risk, either dying or progressing to the next age. The process repeats until each individual arrives at her final resting place at some age. Figure 2 shows the distribution of the ages at death, or the longevity, of these individuals, subject to the Swedish schedules of mortality in 1891 and 2007. The Swedes who enjoyed long lives, progressing to the far right hand end of the age axis are identical to the Swedes who died early, and accumulated at the left hand end of the axis. There is no inherent 'long-livedness' among these individuals. There is no more inequality among these women in their tendency for long or short lives than there is among the balls to move left or right in the Galton box.

In producing this distribution for Swedes, the Galton box has been replaced by a different but equally stochastic machine: an absorbing Markov chain. The equations for this chain are familiar in demography; if $\mu(x)$ is the mortality rate at age x, then the survivorship function is

$$\ell(x) = \exp\left(-\int_0^x \mu(s)ds\right) \tag{1}$$

and the distribution of longevity is

$$f(x) = \ell(x)\mu(x). \tag{2}$$

In this calculation, every individual experiences exactly the same risk of mortality at each age, and the uncertain fate of an individual is the result of continued exposure to those risks. The variation among the Galton box balls in their final location, or among Swedish women in the age at death calculated from a life table calculation of longevity, is called individual stochasticity (Caswell 2009). This is variation arising from the outcome of stochastic processes to which a set of individuals are identically subject.

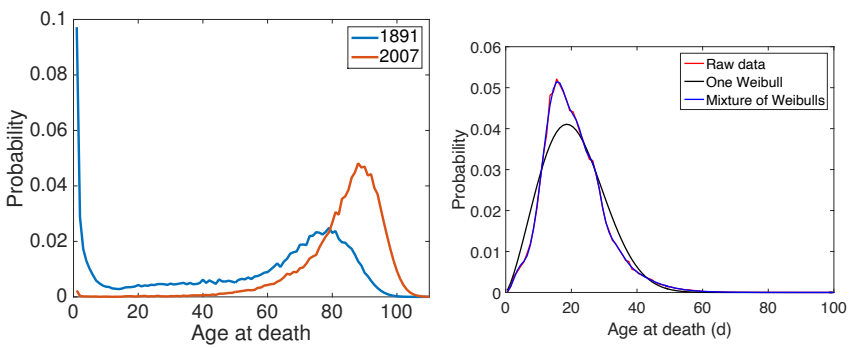
> "Statistically speaking, human life is a random experiment and its outcome, survival or death, is subject to chance. If two people were subjected to the same risk of dying (force of mortality) during a calendar year, one might die during the year and the other survive. If a person was allowed to relive the year he survived the first time, he might not survive the second time." (Chiang 1979)

flow (Li et al. 2010). Lorenz (1993) and Ekeland (1993) invoked the Galton box, albeit in different ways, as a prototypical example of a physical system producing complex and unpredictable results. And Popper (1982: p. 100), following an argument of the physicist Alfred Landé, uses a simple version of the Galton box as a devastating critique of determinism and subjectivist interpretations of probability.

> "Like all men in Babylon, I have been proconsul; like all, I have been a slave. I have known omnipotence, ignominy, imprisonment . . . I owe this almost atrocious variety to an institution which other republics know nothing about, and which operates among them imperfectly and in secret: the lottery." (Borges, 1962 *The Lottery in Babylon*)

But, certainly Swedish women, or any other group of organisms, are not identical. If we actually observed ages at death of a cohort, rather than calculating them as a Markov process, the result would include this fact. Such observations are common in laboratory studies of animals. Figure 2 shows such data for the observed distribution of adult longevity in a large laboratory life table study of the medfly *Ceratitis capitata*. The shape of this distribution, and its statistics, including its variance, reflect both the stochastic nature of the mortality process and whatever inequality in mortality may exist among the individual insects. Animal and plant demographers sometimes have such data; human demographers rarely do. We will return to this example later.

Figure 2: **Left: The distribution of the age at death for Swedish females in 1891 and 2007, computed from a Markov chain realization of the period life tables. Right: The distribution of age at death for a cohort of over one million medflies, showing the observed data, the fit of a single Weibull distribution, and the fit of a mixture of Weibull distributions; see Section 5.2**



Source: Hartemink and Caswell (2018) under a CC-BY license.

1.1 Social inequality and its demographic consequences

If we think of inequality as differences in the rates to which individuals are subject, it is absent from the Galton box and from life table calculations of longevity. But no one

can doubt that social, environmental, physiological, and economic inequality exist. Economic inequality, in income and wealth, has many demographic consequences and has been increasing in many countries for decades (e.g., Piketty and Saez 2014; Atkinson 2015; Therborn 2014). Many other aspects of social life also differ among individuals, producing inequality in education, religious identity, occupation, neighborhood deprivation, and more.

There is a large literature on ways to measure socioeconomic inequality (Atkinson 2015). Some focus on properties of the tail of the distribution (e.g., the share of income or wealth owned by the top 10% or top 1%). Others use properties of the whole distribution (entropy, variance, the Gini coefficient). Some are derived in terms of hypothetical transfers of income or wealth from one part of the distribution to another.

Here I will focus on the variance because it is independent of such assumptions, is a familiar basic statistical quantity, and can be decomposed into components measuring the contribution of different sources (indeed, it was introduced for just such purposes). In addition, many of the common indices of inequality are highly correlated with each other, and the choice of other indices would not change the main conclusions here.

1.2 Opportunity and outcome

Economists distinguish equality of opportunity from equality of outcome (e.g., Atkinson 2015). In our context, differences in the demographic rates to which individuals are exposed are an inequality of opportunity; someone subject to higher mortality has less opportunity for a long life than someone subject to lower mortality. Differences in longevity, due to individual stochasticity, among individuals exposed to the same demographic rates are an inequality of outcome despite equality of opportunity.

The choice between providing everyone with the same opportunity or guaranteeing everyone the same outcome is fraught with philosophical and political priorities, and has been discussed at length (e.g., Sen 1992; Therborn 2014; Atkinson 2015). The discussions often invoke ideas about outcomes that depend, in some way, on the efforts or agency of the individuals.[3] Among individuals with equal opportunity to study, say, medicine, those that choose to study harder will have a better outcome than those who do not. It is unclear, to me at least, how outcomes that are truly stochastic (like the balls in the Galton box or the Swedes in a life table calculation) fit into such arguments, but Anderson (1999) discusses the issue at length.

[3] Atkinson (2015: p. 9), for example, describes the distinction as one between determinants of economic outcomes that are due to circumstances beyond personal control and those due to effort "for which an individual can be held responsible."

1.3 Inequality and heterogeneity

Population biologists are also interested in the impact of inter-individual heterogeneity and stochasticity on population dynamics (e.g., Kendall et al. 2011), selection (Steiner and Tuljapurkar 2012), demographic stochasticity (Vindenes, Engen, and Saether 2008), extinction (Kendall and Fox 2003; Robert, Sarrazin, and Couvet 2003), epidemiology (Marescot et al. 2018), and more. The enormous and increasing concentration of wealth in the top 1% of the population is, to many people, fraught with moral implications. The extreme concentration of lifetime reproductive output among the top few individuals of the seabird the Kittiwake (*Rissa tridactyla*; Clutton-Brock 1988) does not inspire the same kind of concerns among population biologists. However, whether variation is due to inequality of opportunity generated by individual heterogeneity, or to inequality of outcome among individuals experiencing the same rates, has profound implications for natural selection and evolutionary demography.

Analyses of individual stochasticity have introduced a number of terms with the same meaning. Caswell (2009, 2011); van Daalen and Caswell (2015); van Daalen and Caswell (2017) use the term 'individual stochasticity' to describe the random variation in outcomes due to stochastic processes at the individual level. Tuljapurkar, Steiner, and collaborators used the term 'dynamic heterogeneity' to describe the same process (e.g., Tuljapurkar, Steiner, and Orzack 2009; Steiner, Tuljapurkar, and Orzack 2010; Steiner and Tuljapurkar 2012). Snyder and Ellner (2016, 2018) use the terms 'pluck' and 'luck' to refer to heterogeneity and individual stochasticity, respectively. Good discussions of terminology and challenges can be found in Cam et al. (2013), Cam, Aubry, and Authier (2016). It is recognized that the variance in any quantity reflects both heterogeneity and stochasticity.[4]

Regardless of the terminology used, these studies all recognize the important distinction between heterogeneity in the rates experienced by individuals and the random outcomes of those rates. This corresponds closely to inequality of opportunity and of outcome, and I will use them interchangeably. In the demographic context, heterogeneity in socioeconomic or environmental conditions, and the variance in longevity computed from those rates are so fundamental that it is useful to have a terminology that clearly separates them.

[4] It is natural to think that heterogeneity will increase variance, but this is not always the case. Consider tossing coins with a probability $p = 0.5$ of heads and recording the number of heads in n tosses. . The mean value is $\mu = np$ and the variance is $\sigma^2 = np(1-p)$. Now introduce heterogeneity; let each coin have its own probability, with the mean probability being 0.5. Then the mean number of heads is unchanged, but the variance is reduced, not increased, by an amount proportional to the variance in probability (i.e., the degree of heterogeneity) among the coins. The homogenous case is due to Bernoulli; the heterogeneous case to Poisson; the history is described in Heyde and Seneta (1977) and Hacking (1990). See Aitken (1949: pp. 50ff.) for the explanation of this counter-intuitive effect of heterogeneity, for another case due to Lexis, and for the relation of these kinds of heterogeneity to analysis of variance.

2. Inequality of lifespan

Socioeconomic inequality has major demographic consequences: "Inequality kills," in the words of Therborn (2014). Differences in income, wealth, education and other factors reduce the length of life, and of healthy life. Life expectancy is the most commonly reported demographic statistic, but as an expectation of longevity it obviously tells nothing about the variation among individuals in the length of life. In the last two decades or so, patterns of variation have received an increasing amount of attention. The spread of the distribution of age at death has variously been described as lifespan variation, disparity, or inequality. It has been measured by various indices of inequality from the economic literature, which turn out to be highly correlated (e.g., van Raalte, Sasson, and Martikainen 2018).

A major finding was the discovery that increasing life expectancy was often accompanied by a reduction in lifespan variation (Wilmoth and Horiuchi 1999). Vaupel and collaborators found this relationship over a wide range of human societies (Vaupel, Zhang, and van Raalte 2011; van Raalte et al. 2012) and, with slight modifications, among our primate relatives (Colchero et al. 2016). van Raalte, Sasson, and Martikainen (2018) used these results to argue that variation in lifespan should be monitored as well as life expectancy. From a closely related perspective, Edwards and Tuljapurkar (2005), Tuljapurkar and Edwards (2011), and Edwards (2011) examined global patterns of change in the variance in longevity and decomposed that variance into between-country and within-country components. Many of these studies refer to the variance in longevity computed from a life table as 'inequality':

> "Inequality in length of life is the most fundamental of all inequalities; every other type of inequality is conditional upon being alive" (van Raalte, Sasson, and Martikainen 2018)
>
> "Public interest in social and economic equality is burgeoning. We examine a related phenomenon, lifespan equality . . . " (Colchero et al. 2016)
>
> "Interest in inequality, including lifespan inequality, is growing." (Németh 2017)

This inequality is often described as due to heterogeneity among individuals. The distinction between variance due to social inequality and variance due to individual stochasticity has been missing. Those components of variance can be teased apart whenever the calculations are stratified into groups based on heterogeneity.

3. Variance and its components

We want to calculate and decompose the variance in some demographic outcome ξ into its components. The outcome could be any quantity for which means and variances can be calculated (longevity, healthy longevity, stage occupancy, lifetime reproductive output, lifetime income, etc.).

3.1 Means and variances of longevity

The moments of longevity are most easily calculated from a Markov chain model. They can be calculated from the life table as well; the life table is itself a Markov chain, but the life table formulation requires extra integrations or summations, which can be more easily computed with matrix multiplication.

Write the population projection matrix as

$$\mathbf{A} = \mathbf{U} + \mathbf{F}, \tag{3}$$

where $\mathbf{U}$ is a matrix of transition probabilities among transient (i.e., living) states and $\mathbf{F}$ a matrix of stage-specific fertilities. The states may be age classes, stages, or combinations of age and stage (Goodman 1969; Bernstein et al. 2018; Steiner, Tuljapurkar, and Coulson 2014; Caswell 2012; Caswell et al. 2018).

This formulation implies an absorbing Markov chain with $\mathbf{U}$ as the matrix of transition probabilities among transient (i.e., living) states. The fundamental matrix of the chain is

$$\mathbf{N} = (\mathbf{I} - \mathbf{U})^{-1}. \tag{4}$$

Let $\boldsymbol{\eta}_1$ and $\boldsymbol{\eta}_2$ be vectors containing the first and second moments of longevity, starting from each possible state. They are given by

$$\boldsymbol{\eta}_1^{\mathsf{T}} = \mathbf{1}_s^{\mathsf{T}}\mathbf{N} \tag{5}$$

$$\boldsymbol{\eta}_2^{\mathsf{T}} = \boldsymbol{\eta}_1^{\mathsf{T}}(2\mathbf{N} - \mathbf{I}_s) \tag{6}$$

from which the variance is

$$V(\boldsymbol{\eta}) = \boldsymbol{\eta}_2 - (\boldsymbol{\eta}_1 \circ \boldsymbol{\eta}_1)\,. \tag{7}$$

The random variable $\boldsymbol{\eta}$ is a vector whose entries are the times to absorption (i.e.,

death) from each of the transient states. More sophisticated analyses can be written using Markov chains with rewards to account for fractional years lived by those who die (Caswell and van Daalen 2021; Schneider, Myrskylä, and van Raalte 2023), but will not change any of the analyses reported here.

3.2 A matrix protocol for variance decomposition

The partition of variation into components between and within groups is the task for which variance was introduced and named by Fisher (1918). It became the basis for the the analysis of variance in statistics and for artificial selection in agriculture.

1. The first step is to define a set of individuals of interest. The variance of interest is the variance in ξ among the members of this set. Examples might be the set of male Americans at age 40, a cohort of newborn medflies, a set of women at age 55 in several European countries, etc.
2. The set of individuals is divided into groups, indexed by $\gamma = 1, \ldots, g$. These groups define the heterogeneity within the set of individuals.
3. For each group, calculate the mean and variance of ξ, conditional on group membership,

$$m_i = E(\xi|\gamma = i) \tag{8}$$
$$v_i = V(\xi|\gamma = i) \tag{9}$$

   for $i = 1, \ldots, g$. The calculations of these means and variances are obviously specific to the life cycle and on the choice of variable ξ.
4. Collect these conditional means and variances into a mean vector and a variance vector:

$$\mathbf{m} = \begin{pmatrix} m_1 \\ \vdots \\ m_g \end{pmatrix} \qquad \mathbf{v} = \begin{pmatrix} v_1 \\ \vdots \\ v_g \end{pmatrix}. \tag{10}$$

5. Specify the distribution of individuals among the groups. Because the set of interest is a mixture of the g groups, this is called the mixing distribution (e.g., McLachlan and Peel 2004). I denote it by $\boldsymbol{\pi}$, a probability vector of dimension $g \times 1$. The mixing distribution may be imposed by the choice of groups; for example, quantiles of some property (e.g., income) lead to a uniform or flat mixing distribution. In some cases, the mixing distribution may be defined by the relative sizes of groups (e.g., groups in a stable population structure as in van Daalen et al. 2022). A flat

mixing distribution is also appropriate, in analogy to an experiment with equal treatment sizes, to assess the importance of the heterogeneity groups giving each one an equal opportunity to contribute to the variance.

6. Calculate the mean of ξ in the entire set of individuals, including all the groups. This mean is the expectation, over the mixing distribution, of the conditional means

$$E(\xi) = E_\pi \left[E(\xi|\gamma) \right] \tag{11}$$
$$= \boldsymbol{\pi}^{\mathsf{T}} \mathbf{m}. \tag{12}$$

7. Calculate the variance of ξ in the entire set of individuals, including all the groups. This variance contains two terms,

$$V(\xi) = E_\pi \left[V(\xi|\gamma) \right] + V_\pi \left[E(\xi|\gamma) \right] \tag{13}$$
$$= \underbrace{V_{\mathrm{w}}(\xi)}_{\text{within}} + \underbrace{V_{\mathrm{b}}(\xi)}_{\text{between}} \tag{14}$$

where V_{w} is the within-group variance and V_{b} is the between-group variance (e.g., Rényi 1970; Frühwirth-Schnatter 2006). These components are given by

$$V_{\mathrm{w}}(\xi) = \boldsymbol{\pi}^{\mathsf{T}} \mathbf{v} \tag{15}$$
$$V_{\mathrm{b}}(\xi) = \boldsymbol{\pi}^{\mathsf{T}} (\mathbf{m} \circ \mathbf{m}) - (\boldsymbol{\pi}^{\mathsf{T}} \mathbf{m})^2. \tag{16}$$

8. Calculate the fraction of the total variance contributed by heterogeneity among groups as

$$\mathcal{K} = \frac{V_{\mathrm{b}}}{V_{\mathrm{b}} + V_{\mathrm{w}}}. \tag{17}$$

This ratio is also called the intraclass correlation coefficient in quantitative genetics (Falconer 1981), and $\sqrt{\mathcal{K}}$ is known as the correlation ratio in probability theory (Rényi 1970).

The within-group variance is the weighted mean of the conditional variances, with weights specified by $\boldsymbol{\pi}$. If the within-group variances are calculated from a life table or Markov chain, they are totally due to individual stochasticity, because within groups individuals have no more differences, no more heterogeneity, than the balls in a Galton box.

The between-group variance is the weighted variance of the conditional group means, again with weights specified by $\boldsymbol{\pi}$. This variance is due to heterogeneity; if the groups all had identical rates, there would be no heterogeneity, and V_{b} would be zero.

4. Heterogeneity and longevity

In this section, I explore cases in which genuine inequality exists between different groups, and how that inequality of opportunity contributes to the variance in longevity.

4.1 Longevity and inequality in income, education, and occupation

We begin with a study that examines longevity in relation to several dimensions of socioeconomic inequality: income, education, and employment. Luy et al. (2015) analyzed life expectancy in relation to these variables for men and women in Germany. They developed a sophisticated set of procedures to estimate age-specific mortality, from age 40 onwards, from the German Life Expectancy Survey (see their paper for details). The results are typical: For example, being in the first rather than the fourth quartile of income costs about five years of life expectancy for men and four years for women:

	Life exp. at 40	
Income	Men	Women
1st quartile income	32	40
2nd quartile income	35	41
3rd quartile income	36	44
4th quartile income	37	44

Following the procedure in Section 3, the group means, group variances, and the mixing distribution for men (Luy et al. 2015 give rates for both men and women), are

$$\mathbf{m} = \begin{pmatrix} 31.6 \\ 35.3 \\ 36.0 \\ 37.3 \end{pmatrix} \quad \mathbf{v} = \begin{pmatrix} 155.6 \\ 137.1 \\ 141.4 \\ 133.7 \end{pmatrix} \quad \boldsymbol{\pi} = \begin{pmatrix} 1/4 \\ 1/4 \\ 1/4 \\ 1/4 \end{pmatrix}. \tag{18}$$

The flat mixing distribution is appropriate here because by definition each quartile con-

tains 1/4 of the population.

$$V_w = \boldsymbol{\pi}^{\mathsf{T}}\mathbf{v} \tag{19}$$
$$= 142.0 \tag{20}$$

$$V_b = \boldsymbol{\pi}^{\mathsf{T}}(\mathbf{m}\circ\mathbf{m}) - (\boldsymbol{\pi}^{\mathsf{T}}\mathbf{m})^2 \tag{21}$$
$$= 4.4\,. \tag{22}$$

Partitioning the variance for both sexes yields

	Variance inlongevity	
Source	Men	Women
income inequality (between groups)	4	3
stochasticity (within groups)	142	120
variance ratio $\mathcal{K}$	0.028	0.024

Despite the non-trivial effect of income inequality on life expectancy, that inequality contributes only 2%–3% of the variance in longevity. If variance in longevity is thought of as inequality, it is a very weak signal of income disparity.

The data of Luy et al. (2015) permit the same analysis to be performed on education levels (low, medium, and high). High education adds about seven years to life expectancy for men, and about three years for women, relative to low education. To calculate the variance components, I use a flat mixing distribution, not because these categories are equally abundant, as quantiles are, but as if this was an experimental design to evaluate the effects of education on longevity. The results are

	Variance inlongevity	
Source	Men	Women
education inequality (between)	7	1
stochasticity (within)	138	122
variance ratio $\mathcal{K}$	0.05	0.01

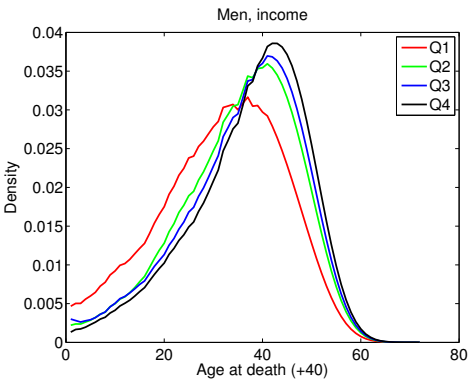
Luy et al. (2015) also measured mortality in groups defined by work status (self-employed, public servants, manual, and employees). The variance components for this source of inequality are

	Variance inlongevity	
Source	Men	Women
work status inequality (between)	3	7
stochasticity (within)	137	114
variance ratio $\mathcal{K}$	0.02	0.05

It becomes apparent from this example that at least some dimensions of social inequality, significant enough that social scientists pay a great deal of attention to them, are only weakly connected to variance in longevity. Only a few percent of the variance in longevity is explained by the variance between groups.

A graphical perspective is given in Figure 3, which shows the distribution of longevity for each of the four income quartiles for men. The variance in these distributions is due to individual stochasticity; in the calculation of these distributions individuals within an income group are as identical as the balls in the Galton box. It is clear that the differences among the means of the income groups, as important as those are to the rich and the poor, is dwarfed by the variance within each group.

Figure 3: **The distribution of longevity, from age 40, for four quartiles of household income. The variance within each of these distributions is due to individual stochasticity.**



Calculated from data of Luy et al. (2015)

4.2 Longevity and income inequality in the United States

A particularly rich investigation of the longevity consequences of income inequality is a study by Chetty et al. (2016). They analyze mortality rates and life expectancy in the United States for groups defined by percentiles of household income, based on 1.4 billion person-years of data. There is a strong income effect: Higher income is associated with lower mortality and greater life expectancy. The life expectancy gap, at age 40, between the richest and the poorest 1% was 14.6 years for men and 10.1 years for women. There can be no doubt that income inequality in the United States has substantial and important effects on mean longevity.

The effects on variance in longevity can be evaluated using the data provided by Chetty et al. (2016: Table 15) in the appendices to their paper. Following their methodology, observed mortality rates to age 76 were extrapolated to age 90 using a Gompertz mortality function.

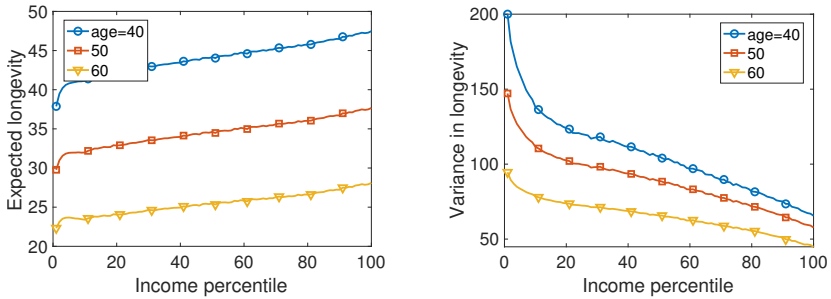
The mixing distribution is flat by definition, since each percentile of the distribution includes 1% of the probability mass. The variance decomposition for females starting at age 40 is

Source	Variance inlongevity
income inequality (between)	3.9
stochasticity (within)	106.1
variance ratio $\mathcal{K}$	0.036

The dramatic inequality in income is responsible for less than 4% of the variance in longevity.

This result is specific to individuals at age 40, but it is possible to apply the analysis to any age. Figure 4 shows the mean and variance of remaining longevity, as a function of household income percentile, at ages 40, 50, and 60. The mean longevity (life expectancy) increases with income at about the same rate for each of these ages. The variance in longevity decreases with income percentile, and decreases with age.

Figure 4: Means and variances of female longevity at ages 40, 50, and 60 years, as function of income percentile.

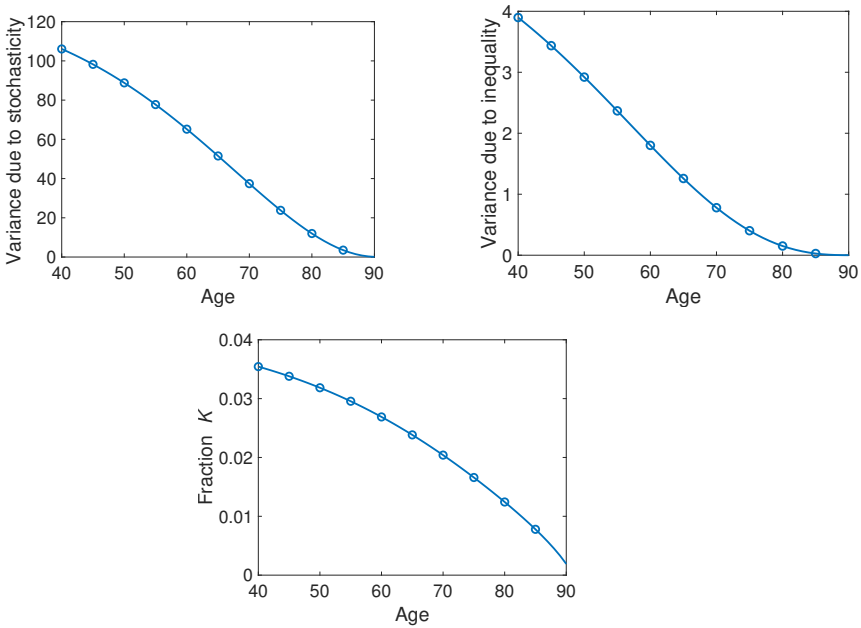


Calculated from data in Table 15 of Chetty et al. (2016).

Figure 5 shows the within-group variance and between-group variances, and the variance ratio $\mathcal{K}$ as a function of age. The variance ratio, already small (3.5%) at age 40, decreases monotonically with age. Thus, at older ages, the variance in longevity of a

group of (female in this case) individuals is less and less determined by the heterogeneity in income among individuals, and is more and more the result of stochasticity.

Figure 5: **Above: The within-group and between group variances in longevity remaining at various ages. Below: The fraction of variance in remaining longevity due to variance in household income.**



Calculated from data of Chetty et al. (2016).

4.3 Longevity and neighborhood deprivation

Income, education, and occupation are important socioeconomic measures. But perhaps they explain such a small amount of variance in longevity because they are so specific. Perhaps results would differ if more types of heterogeneity were included. Seaman, Riffe, and Caswell (2019) did so, carrying out an analysis using an integrative deprivation measure, applied to neighborhoods (part-postcode sectors), in Scotland.

The deprivation measure, the Carstairs score, is a standardized z-score based on four individual-level census variables: overcrowding, male unemployment, low social class,,

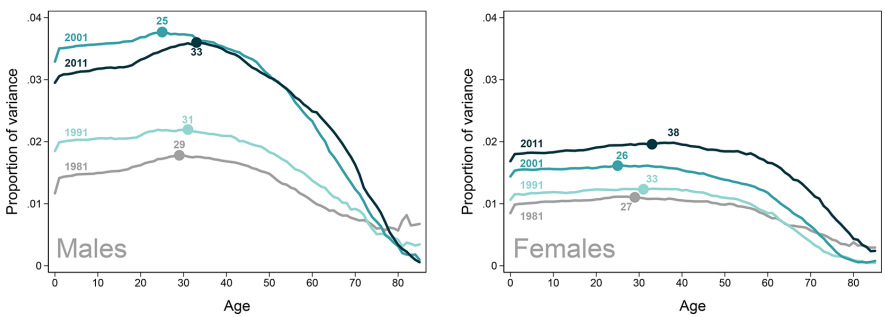
and car ownership. The score is taken to capture important aspects of the material resources needed to access goods, services, amenities, and the physical environment within each location.

Seaman, Riffe, and Caswell (2019) created population-weighted quintiles of deprivation scores, so each quintile contains 20% of the population, and analyzed data from 1981, 1991, 2001, and 2011. Deprivation has a definite effect on life expectancy. Life expectancy is reduced by one to two years for females and by two to three years for males with each quintile increase in deprivation.

However, the contribution of within-group individual stochasticity far outweighed that of the between-quintile inequality. For males, over the four time periods examined, only 1%–3% of the variance in life expectancy at birth is due to deprivation. For females, the percentages are even lower, from 0.9%–1.7%.

Figure 6 shows the variance ratio (the proportion of the variance in remaining life longevity due to inequality in deprivation) for all ages and all time periods. Nowhere is the ratio greater than 4%, similar to the values seen in Sections 4.1 and 4.2.

Figure 6: The variance ratio $K(x)$, showing the proportion of the variance in remaining life expectancy due to heterogeneity in deprivation scores, for males and females in years 1981, 1991, 2001, and 2011.



Source: Seaman, Riffe, and Caswell (2019) under a CC-BY-NC license.

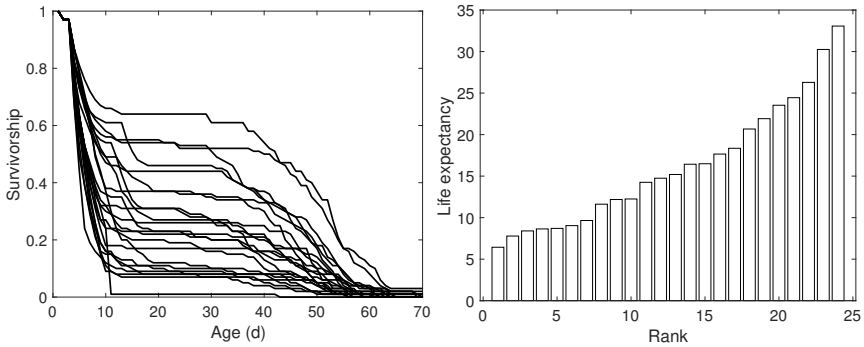
4.4 What if there is more heterogeneity?

Heterogeneities in income, education, occupation, and neighborhood deprivation make only a small contribution to the variance in longevity. Maybe a more extreme degree of inequality will create a larger signal and make a larger contribution to, the variance of longevity. To this end, Figure 7 shows survivorship curves for 24 populations differing in early lifetime nutritional conditions. The difference among these survivorship functions

exceeds anything likely to be found in any human population. This inequality in nutrition leads to a fivefold difference in life expectancy.

The data are not, of course, for a human population, but are the results of a laboratory experiment on the Mediterranean fruit fly, or medfly, *Ceratitis capitata*. Because these flies can be raised in the laboratory under controlled conditions, they have been used as a model system for the study of life history evolution and aging (e.g., Vaupel and Carey 1993; Carey, Liedo, and Vaupel 1995; Vaupel et al. 1998; Carey 2003; Carey et al. 2016).

Figure 7: Survivorship and life expectancy at birth for medflies raised, as larvae, on 24 different species of host plants.



Calculated from data of Krainacker (1986); Krainacker, Carey, and Vargas (1987).

Medfly larvae develop on fruits; some species of fruit are more nutritious than others. Figure 7 is based on an experiment raising larval medflies on 24 different fruits and recording survival and reproduction on each diet (Krainacker 1986; Krainacker, Carey, and Vargas 1987). In the experiment, all adult flies received the same diet, so the heterogeneity here reflects early life nutritional conditions. Using life table data from Krainacker (1986), projection matrices were constructed and the mean and variance of longevity calculated for each larval food treatment, using the methods in Section 3.

4.4.1 Medfly mixing distributions

The experiment of Krainacker, Carey, and Vargas (1987) was designed to evaluate the effects of the 24 host plants on various life history traits, including longevity. Thus a flat mixing distribution, with each entry in $\boldsymbol{\pi}$ equal to $1/24$, is appropriate for decomposing the variance in this experiment.

Suppose that, instead of a laboratory experiment, we wanted to explore a scenario involving medflies in nature. Imagine that medflies are smarter than we give them credit

for and allocate their eggs to different hosts in proportion to the fitness of the offspring raised on those hosts. Measuring fitness by the intrinsic rate of increase $r_i = \log \lambda_i$, where λ_i is the dominant eigenvalue of the projection matrix for individuals raised on host i, a mixing distribution biased towards high-quality hosts might be

$$\boldsymbol{\pi} = \frac{1}{\sum_i r_i} \begin{pmatrix} r_1 \\ \vdots \\ r_{24} \end{pmatrix}. \tag{23}$$

We can compare the results of this experiment with the laboratory experiment with the flat mixing distribution.

4.4.2 Variance components

The variance components using the flat mixing distribution are

Source	Variance inlongevity
diet inequality (between)	53
stochasticity (within)	296
variance ratio $\mathcal{K}$	0.15

Using the mixing distribution proportional to r yields a result only slightly different:

Source	Variance inlongevity
diet inequality (between)	52.2
stochasticity (within)	330.8
variance ratio $\mathcal{K}$	0.136

Similar as the results may be, it is important to recognize that they are the answers to two different questions: How does inequality in nutrition contribute to variance in longevity (1) in an experiment designed to evaluate each diet equally, or (2) in a population whose members experience diets differentially in proportion to a fitness measure? It is easy to imagine other mixing distributions designed to address other questions (e.g., based on the relative abundance of different diets in the environment).

Despite the much greater degree of heterogeneity in conditions in this experiment, inequality of opportunity contributes only about 15% of the variance in longevity. Perhaps we need more heterogeneity.

4.5 Even more heterogeneity

The heterogeneity in life histories among income percentiles, or deprivation classes, or early life nutritional statuses pales in comparison to the heterogeneity among species across the so-called Tree of Life. We don't have demographic data from the entire tree, but the COMPADRE (plants) and COMADRE (animals) matrix databases have compiled a large and increasing number of matrix population models for a wide variety of species (Salguero-Gómez et al. 2015, 2016).

In a comparative analysis of stochasticity in demographic outcomes, Varas Enríquez, Van Daalen, and Caswell (2022) analyze 332 populations of 141 species of plants and 83 populations of 47 species of animal. Their paper reports results of individual stochasticity (means, variances, and skewness) for both longevity and lifetime reproductive output; I return to the latter below.

Treating the populations as if this was an experiment, and thus using a flat mixing distribution, the resulting variance decompositions are

For animals:

Source	Variance inlongevity
between-species heterogeneity	60.3
within-species stochasticity	127.4
variance ratio $\mathcal{K}$	0.32

and for plants:

Source	Variance inlongevity
between-species heterogeneity	335.2
within-species stochasticity	1030.0
variance ratio $\mathcal{K}$	0.245

Even with these extreme differences among groups (i.e., species), inequality of opportunity accounts for only about 25%–30% of the variance in longevity. Having more groups, or more varied groups, seems unlikely to overcome the magnitude of individual stochasticity in the distribution of longevity.

5. Latent heterogeneity

We have seen that heterogeneity among groups can be imposed by the investigator in an experiment, by treating individuals differently, assigning them to diets, or medications and placebos, and so on. Or the groups may be defined by externally measurable properties (income, education, occupation, etc.). However, in some cases the heterogeneity is latent and unobserved and must be estimated as part of the analysis.

The longevity distribution for medflies in Figure 2 is an example. It is the distribution of ages at death of a set of individuals (a cohort of over one million medflies), in which each individual was followed until death and its age at death recorded. Unlike the identical balls in the Galton box or the individuals identically processed through the Markov chain calculation defined by equations (2) or (7), we have no way of knowing if these medflies are homogeneous or heterogeneous. It's like finding a completed Galton box in the woods, partially covered with dead leaves, not knowing whether the distribution you see upon uncovering it is the result of stochastic outcome of identical trials, or if some of the balls really did have an intrinsic tendency to move left or to move right.

If the cohort of medflies is a mixture of groups with different demographic properties, then the distribution of age at death is described by a mixture model. The probability distribution of a mixture model is a linear combination of a set of distributions for some number g of groups

$$P(x) = \pi_1 P_1(x) + \cdots + \pi_g P_g(x), \tag{24}$$

where $P_1, \ldots, P_g$ are the probability distributions for the g groups, and $\pi_1, \ldots, \pi_g$ are the probabilities of the various component distributions. The π_i are, as in the case of observed heterogeneity, the mixing distribution. Now, however, $\boldsymbol{\pi}$ must be estimated from the distribution of longevity, along with the demographic rates of each group.

5.1 Heterogeneous frailty

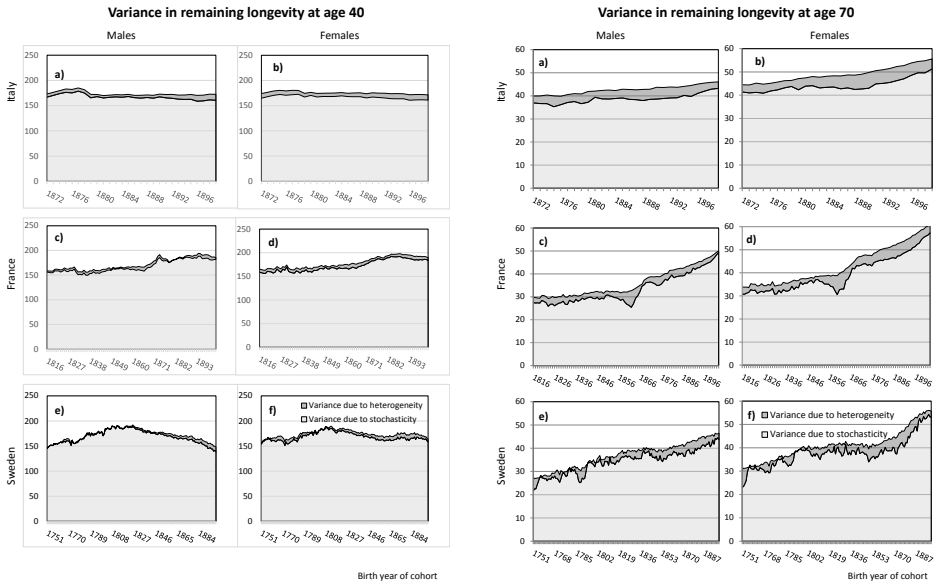
The mortality schedules of many populations, human and otherwise, often follow the Gompertz (mortality rate increasing exponentially with age) or the Gompertz-Makeham (Gompertz plus a baseline age-invariant hazard) model (Vaupel 2010).

Vaupel, Manton, and Stallard (1979) pointed out the importance of unobserved latent heterogeneity in the mortality rates experienced by individuals, referring to it as 'frailty' (see Vaupel and Missov 2014 for a review). In a popular version of the frailty model, each individual is assigned, at birth, a multiplicative factor, drawn from a gamma distribution, that increases or reduces the risk of death at every age. Figures 8 and 9 explore the consequences of this form of latent heterogeneity for the variance in longevity.

Because individuals with higher frailty are at greater risk of death, a mixed cohort becomes progressively dominated by low frailty individuals, distorting the shape of the mortality function when measured on a cohort. Instead of the exponential increase in mortality with age that is experienced by every individual, the mortality schedule of the cohort becomes sigmoid, slowing its increase at old ages. Vaupel and Yashin (1985) showed a variety of models, some much simpler than the gamma-Gompertz model, in

which heterogeneity's 'ruses' could qualitatively alter mortality patterns at the cohort or population level.

Figure 8: Variance in remaining longevity at ages 40 (left) and 70 (right), for male and female cohorts in Italy, France, and Sweden. The dark area is variance due to heterogeneity in frailty.



Source: Hartemink, Missov, and Caswell (2017) under a CC-BY license.

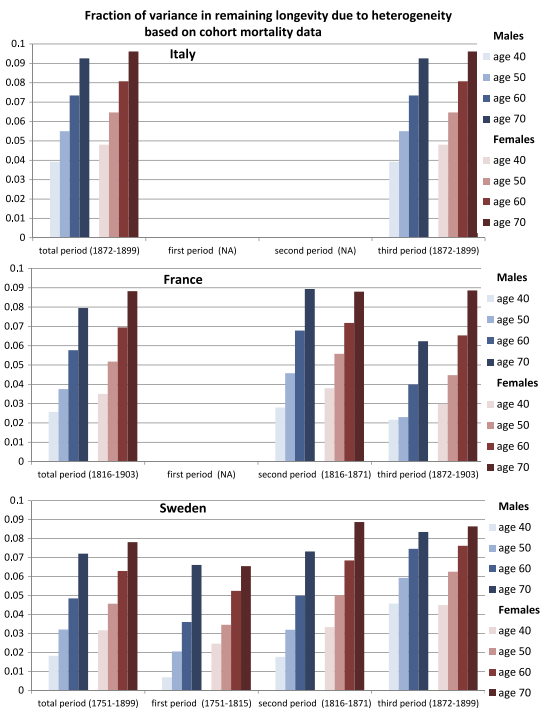
Although most attention has focused on the effect of frailty on the shape of mortality schedules, the heterogeneity also affects the statistics of longevity. Caswell (2014) presents a matrix formulation of the gamma-Gompertz model in which individuals are jointly classified by age and stage, with stages defined by a discrete gamma distribution of frailty. This model permits calculation of the components of variance in longevity due to between-frailty-group heterogeneity and within-group stochasticity. Hartemink, Missov, and Caswell (2017) use this model to analyze the variance in longevity in a gamma-Gompertz-Makeham model for mortality, in which the mortality rate for an individual aged x with frailty z is

$$\mu(x, z) = zae^{bx} + c, \tag{25}$$

where a is the starting mortality rate, b is the Gompertz aging rate, and c is the constant Makeham mortality rate. They applied the calculation to cohort and period life tables for Italy, France, and Sweden, examining the changes in the overall variance and its components over time. Figure 8 shows the variance in longevity for males and females for the three countries, with the variance due to inequality in frailty shown as the dark band at the top of each graph. It is clear that a great majority of the variance is due to within-group stochasticity, not between-group heterogeneity.

Figure 9 shows the mean values of the variance ratio $\mathcal{K}$ for the remaining longevity, of male and female cohorts, at ages 40, 50, 60, and 70. The variance ratio increases with age, but is always small, and at age 40 it is no more than a few percent. Using period rather than cohort life tables produced essentially the same results.

Figure 9: **The fraction $\mathcal{K}$ of the variance in remaining longevity, at ages 40, 50, 60, and 70, due to heterogeneity in frailty for male and female cohorts in Italy, France, and Sweden.**



Source: Hartemink, Missov, and Caswell (2017) under a CC-BY license.

These populations were chosen for analysis because long time series of rates, both cohort and period, were available in the Human Mortality and Human Fertility Databases. Over these periods, which saw significant changes in demographic rates, the contribution of inequality of opportunity due to latent frailty remains small.

5.2 Finite mixture models for laboratory insect populations

Hartemink and Caswell (2018) assembled age at death data for 32 populations from 10 laboratory studies of invertebrates, mostly insects, and fitted finite mixture models to the longevity distributions. They used the Weibull survival model as the basic distribution. The Weibull is a flexible survival model that can produce mortality schedules that are increasing, decreasing, or flat with respect to age (Pinder, Wiener, and Smith 1978). The statistical analysis used (the EM algorithm and BIC criteria for model selection) estimated the number g of groups in the mixture, the mixing distribution $\boldsymbol{\pi}$, and the Weibull parameters for each group (see Hartemink and Caswell 2018 for details).

Over the 32 populations, including males, females, and both sexes combined for species where such information was available, the estimated number of latent groups ranges from 1 (i.e., no heterogeneity) to 8. The median value of $\mathcal{K}$ is 0.35 with an interquartile range of 0.23 to 0.44. In other words, typically about 35% of the variance in longevity is explained by unobserved heterogeneity, subject to the hypothesis that the baseline mortality function is indeed Weibull.

Figure 10 shows one case: the distribution of longevity in a cohort of over one million medflies (Vaupel and Carey 1993; Carey 1993), perhaps the largest laboratory cohort ever measured. The empirical distribution of longevity is fit poorly by a single Weibull function. The mixture model in which eight latent groups were identified fits the empirical distribution extremely well, with an estimated mixing distribution

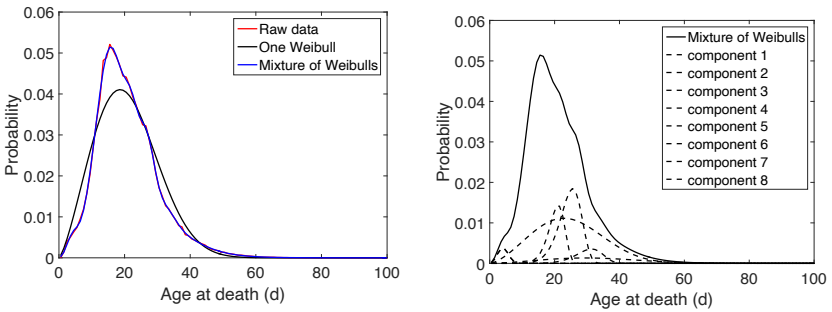
$$\boldsymbol{\pi} = \begin{pmatrix} 0.09 & 0.15 & 0.30 & 0.05 & 0.0004 & 0.04 & 0.04 & 0.02 & 0.36 \end{pmatrix}^{\mathsf{T}}. \qquad (26)$$

The estimated mixing distribution is far from flat in this case; two groups account for 66% of the population, while one of the groups accounts for only 0.04%. The distributions of each of the eight estimated groups are shown in Figure 10 (right panel). The distributions are very different, in both shape and in the modal age at death. In this analysis, heterogeneity among these latent groups accounted for 41.5% of the variance in longevity.

The larvae of medflies and some other insects in the laboratory are grown in group cultures and may experience strong competition during the larval stage. Longevity is measured from the age at adulthood (eclosion), and it is possible that larval competition may lead to skewed distributions of adult size at metamorphosis. If so, that may lead

to higher contributions of heterogeneity to variance (Hartemink and Caswell 2018). At least some studies have found that smaller adults resulting from crowded larval conditions have longer adult life expectancy (Peters and Barbosa 1977). In the medfly larval food experiment analyzed in Section 4.4, life expectancy was measured from birth, larvae were raised in relatively uncrowded conditions, and the variance ratio is about one-third of that in the million medfly study.

Figure 10: **The distribution of longevity in a cohort of a million medflies under laboratory conditions. Left: The distribution of raw, observed longevities, the result of fitting a single Weibull survival model, and the result of fitting a mixture of eight different Weibull models. Right: The distribution of observed longevities and the distributions of the longevities for each of the groups identified in the mixture model.**



Source: Hartemink and Caswell (2018) under a CC-BY license.

5.3 The Southern Fulmar

Parametric mixture models, as in the cases of the gamma-Gompertz-Makeham frailty model and the Weibull mixture model, are, of course, dependent on the choice of the component distributions. In the gamma-Gompertz case, Yashin, Vaupel, and Iachine (1994) showed early on that there exists a model with an entirely different structure that produces a distribution of longevity identical to that of the gamma-Gompertz model. The gamma-Gompertz is a fixed frailty model, in which individuals are assigned a frailty at birth that never changes. The model presented by Yashin, Vaupel, and Iachine (1994) is a dynamic frailty model in which all individuals start with the same frailty, which develops stochastically over age and yet produces the same mortality function. Results like those of Hartemink and Caswell (2018) need to evaluated in the context of this limited identifi-

ability. In general, it is valuable to try to identify observed causes of heterogeneity rather than hunt for latent heterogeneity.

A mixture model analysis based on data collected in the field and with fewer parametric assumptions is presented by Jenouvrier et al. (2018) in a study of the Southern Fulmar (*Fulmarus glacialoides*, a long-lived procellariiform seabird). The analysis is based on 45 years of capture-mark-recapture data. A stage-classified matrix model was constructed, and the estimation procedure (details are described in Jenouvrier et al. 2018) provided maximum likelihood estimates of the number of groups, the mixing distribution, and the parameters in the matrix population model for each group. Three heterogeneity groups were identified, differing in stage-specific survival, breeding probability, and breeding success, with a mixing distribution

$$\boldsymbol{\pi} = \begin{pmatrix} 0.14 & 0.67 & 0.19 \end{pmatrix}^{\mathsf{T}}. \tag{27}$$

Decomposing the variance in longevity leads to the results

Source	Variance inlongevity
between-group heterogeneity	11.7
within-group stochasticity	188.7
variance ratio $\mathcal{K}$	0.059

with only 5.9% of the variance due to between-group heterogeneity.[5]

6. Healthy longevity

Longevity is a special case of healthy longevity in which the two (admittedly extreme) health states are living and dead. While healthy longevity is almost always addressed in terms of expected values (e.g., health expectancy, disability-adjusted life years), methods now exist to easily calculate the variance in healthy longevity due to individual stochasticity. These methods use the concept of Markov chains with rewards (MCWR; introduced by Howard (1960), extended to random rewards for use in demography by Caswell (2011); for a recent text see Sheskin (2010)). In a MCWR, every transition in the Markov chain accumulates a reward, interpreted very broadly, which is a random variable with specified moments. The accumulation stops when the individual reaches an absorbing state (i.e., death).

MCWR calculations can be applied to both prevalence data (the fraction of the pop-

[5] This study also examined variance in lifetime reproductive output (LRO) and age at first reproduction. Between-group heterogeneity accounted for 22% of the variance in LRO and 3.7% of the variance in age at first reproduction.

ulation with a condition at every age) and incidence data (the rates of transition among health states at every age). They go beyond binary measures (disabled vs. non-disabled) to include quantitative measures and measures defined by groupings of ages and health states (Caswell and Zarulli 2018; Caswell and van Daalen 2021). Caswell and Zarulli (2018) apply the method to prevalence data, treating healthy longevity as years lived without disability, for nine European countries in the SHARE survey data (Belgium, Czech Republic, Denmark, Estonia, France, Germany, Portugal, Sweden, and Switzerland). Their results give the means and variances in healthy longevity for each country, for males and females at at age 55.

These nine countries have differing histories, social support systems, health care structures, and cultures, and can be treated as groups within a population. Suppose that we want to know whether these differences matter to healthy longevity; we could use a flat mixing distribution in which every entry of $\boldsymbol{\pi}$ is 1/9, corresponding to an imaginary experiment in which an equal number of citizens of each country were followed starting at age 55 and their healthy lives recorded.

In that case, the variance decomposition yields

	Variance in healthy longevity	
Source	Female	Male
country inequality (between countries)	3.94	3.36
stochasticity (within countries)	60.8	42.2
variance ratio $\mathcal{K}$	0.06	0.07

The heterogeneity among countries in the mortality rates and disability prevalences is responsible for only 6%–7% of the variance among individuals in disability-free longevity. The remaining 93%–94% is due to individual stochasticity.

Alternatively, we might use a mixing distribution in which the entries of $\boldsymbol{\pi}$ are proportional to the population, at age 55, in each country, in 2011 when the SHARE data were collected. This would correspond to an analysis of the variance in an equally imaginary study where a big net was used to collect a random sample of 55-year old citizens of Belgium,. . . ,Switzerland, in proportion to their relative abundances. The countries are of very different sizes, dominated by France and Germany, which together would account

for over 70% of the mixing distribution:

$$\boldsymbol{\pi}_{\text{female}} \begin{pmatrix} 0.054 \\ 0.055 \\ 0.026 \\ 0.007 \\ 0.310 \\ 0.413 \\ 0.054 \\ 0.042 \\ 0.037 \end{pmatrix} \qquad \boldsymbol{\pi}_{\text{male}} = \begin{pmatrix} 0.055 \\ 0.056 \\ 0.027 \\ 0.006 \\ 0.300 \\ 0.421 \\ 0.050 \\ 0.044 \\ 0.039 \end{pmatrix}. \tag{28}$$

In this case, the variance decomposition is

	Variance inhealthy longevity	
Source	Female	Male
country inequality (between countries)	1.05	2.02
stochasticity (within countries)	59.0	40.5
variance ratio $\mathcal{K}$	0.02	0.05

The between-country variance is reduced compared to the flat mixing distribution; this makes sense because in the limit in which the mixing distribution consisted of only one group (one country), there would be no between-group variance. The variance ratio results are not greatly changed; heterogeneity among countries now contributes only 2% (for females) and 5% (for males) of the variance in healthy longevity. The remaining 95% and 98% is due to individual stochasticity.

Although the choice of mixing distribution has, in this case, only a small effect on the variance decomposition, it is important to note that the two analyses are asking about different questions about different populations. This flexibility is an advantage of the approach.

7. Conclusions

The cases examined here are far from the only examples. Other studies report similar small contributions of heterogeneity among countries (Edwards 2011; Smits and Monden 2009; Permanyer and Scholl 2019) and between education levels (van Raalte et al. 2012) to the variability in longevity. Even earlier, Vaupel (1988) wrote

"...the variance in life expectancies among people with different frailties is

> small compared with the variance in life spans among people at the same level of frailty."

He used the formula (14) to relate the variance components to the inheritance of frailty and longevity between generations.

Here, I have tried to examine a wide range of degrees of heterogeneity and methods of identifying it. Some conclusions emerge:

- Variance in longevity (and other demographic outcomes) arises from heterogeneity and from stochasticity.
- Heterogeneity corresponds to the economic concept of inequality of opportunity. Individual stochasticity corresponds to inequality of outcome.
- The distribution of longevity calculated from a life table, a mortality schedule, or an absorbing Markov chain is entirely due to stochasticity, because such calculations treat every individual identically.
- The distribution of longevity observed by following a set of individuals from a starting state to death reflects both stochasticity and (latent, unobserved) heterogeneity.
- Identifying the latent heterogeneity is a difficult and model-dependent process.
- Given the means and variances of longevity in a number of groups, the variance can be decomposed into between-group (due to heterogeneity) and within-group (due to stochasticity) components.
- It is important to distinguish inequality of opportunity and of outcome, if only because inequality of opportunity (e.g., socioeconomic heterogeneity in terms of income, education, etc.) is more potentially subject to policy interventions.
- Longevity is subject to a great deal of individual stochasticity. As a result, socioeconomic heterogeneity (and some other kinds as well) accounts for only a small fraction of the variance:
  - Income, education, occupation account for 2%–5% of the variance
  - Latent frailty accounts for 5%–10% of the variance
  - Extreme heterogeneity in early life nutrition, in a laboratory population of medflies, accounts for about 15% of the variance
  - Latent heterogeneity in the life cycles of an Antarctic seabird accounts for 6% of the variance in longevity
  - Heterogeneity among the great range of life histories of plants and animals explains 25% (plants) to 32% (animals) of the variance in longevity
  - Differences among a set of European countries accounts for 2%–7% of the variance in healthy, disability-free longevity.
- The statistics of longevity due to individual stochasticity are a mathematical consequence of the mortality schedule. Improvements in survival at early ages reduce the variance due to stochasticity; improvements at later ages increase the variance.

Patterns of change in the mean and variance of calculated longevity reflect that mathematical relationship.

8. Discussion

8.1 Age as heterogeneity

Demography is the study of inequality. It recognizes that mortality, fertility, and other demographic rates vary with age, and that this heterogeneity must be accounted for and its consequences analyzed. The analytical machinery of life tables, projection matrices, or partial differential and integral equations provide the means and variance of the outcomes that result from these age differences.

It is revealing, then, to consider age itself as a kind of heterogeneity. Consider a set of individuals of different ages, treat those ages as groups, and calculate the mean and variance of remaining longevity for each age group. Suppose that the set of individuals of interest is drawn from the population at its stable age distribution, and use that distribution as the mixing distribution. The variance in longevity among this set of individuals will reflect the differences in life expectancy among the age groups and the stochasticity within each group. Age is a strong inequality of opportunity as far as longevity is concerned. What do the variance components look like? As an arbitrary example, using the period rates for Swedish females in 2007 yields the following:

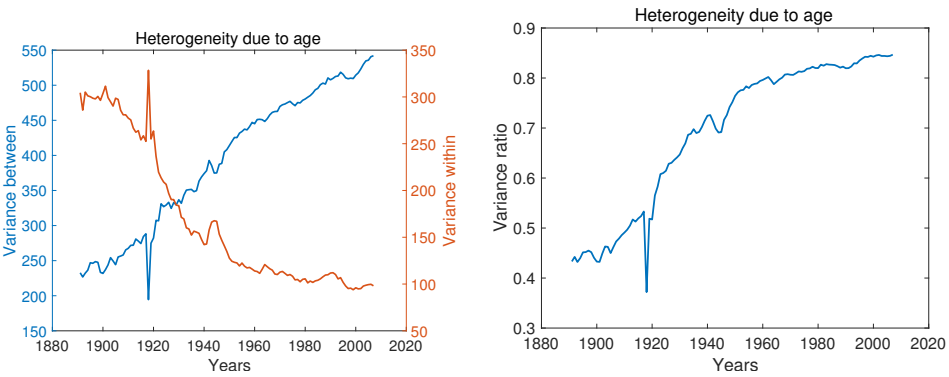
Source	Variance inlongevity
heterogeneity (between ages)	542.0
stochasticity (within ages)	98.1
variance ratio $\mathcal{K}$	0.85

The heterogeneity in age within the population at its stable age distribution accounts for 85% of the variance in remaining longevity among those individuals. This is the highest value of $\mathcal{K}$ seen so far, and is a measure of the impact of age variation relative to the variation due to income, education, or other socioeconomic factors.

Figure 11 repeats this calculation using data for Swedish females from 1891 to 2007 from the Human Mortality Database and the Human Fertility Database. Sweden, of course, went through significant demographic changes over this time. Life expectancy at birth increased from 53 years to 83 years. As this was happening, the between-age-group variance increased, and the within-age-group variance declined. The result was a quite smooth increase in $\mathcal{K}$ from explaining about 43% to about 85% of the variance. An interesting discontinuity appears in 1918, presumably reflecting the influenza pandemic. It increased the within-group variance and reduced the between-group variance, with a net negative effect on the variance ratio. Accounting for such changes is an important

research challenge; the sensitivity analyses presented by van Daalen and Caswell (2020) might usefully be applied here.

Figure 11: **Left: The variance in remaining longevity between and within age groups. Right: The variance ratio $\mathcal{K}$. The mixing distribution is proportional to the stable age distribution in each year.**



Source: Calculated from period life tables for Swedish females, 1891–2007 (Human Mortality Database 2019).

8.2 Equality of what?

Sen (1992) argues persuasively that the question 'Equality of what?' is central in studies of inequality. He notes the existence of different spaces in which equality can be evaluated and, importantly, that equality in one space can imply inequality in another. Equality of opportunity can lead to inequality of outcome; equality of outcome may require inequality of opportunity.

This is certainly true in the demography of longevity. Consider what complete equality of opportunity would look like: Every individual would be subject to exactly the same rates, regardless of anything, even age. Everyone would would be subject to the same mortality rate μ, leading to an exponential distribution of longevity with a mean $1/\mu$ and a variance $1/\mu^2$. The standard deviation of longevity would equal life expectancy, a value far greater than any observed for any human population. (This model describes the demography of the decay of radioisotopes).

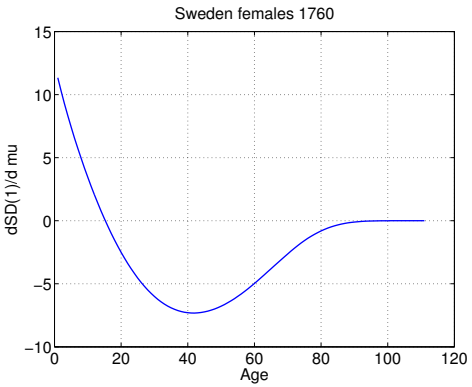
Complete equality of opportunity would thus generate a large inequality in outcome. On the other hand, equality of outcome in longevity, with everyone living to the same age, would lead to a rectangular survivorship function, which was suggested as an 'ideal' by Fries (1980). But it would require a great inequality of opportunity. For part of the

population, survival to the next age would be certain; for another part, death in the next year would certain.

8.3 The sensitivity of within-group variance: early and late mortality

The variance in longevity calculated from a life table is determined by the mortality schedule. The sensitivity of that variance (or of the other commonly used measures of variation) to mortality is positive for early ages, negative for later ages, and then converges to zero for latest ages (Van Raalte and Caswell 2013), as shown in Figure 12. Zhang and Vaupel (2009) showed that this is a general property of longevity measures. It implies that there is a critical age separating early from late deaths and that reducing early mortality reduces lifespan variation, but reducing late mortality has the opposite effect. It is important to recognize that historical reductions in early mortality lead inevitably to declining trends in the variance in longevity (Vaupel, Zhang, and van Raalte 2011; van Raalte, Sasson, and Martikainen 2018; Colchero et al. 2016). Such trends (again, when calculated from a life table) do not reflect any decrease in inequality of opportunity, because all individuals experience the same rates.

Figure 12: Sensitivity of the standard deviation of longevity to changes in age-specific mortality, for the Swedish female life table in 1760. In this case the critical age separating early and late mortality is approximately 18 years.



8.4 Imagining heterogeneity

Scientists, including demographers, love heterogeneity. We want to account for patterns of variation by finding differences among things that produce those patterns. This is the essence of the statistical analysis of experimental or observational data. Sometimes heterogeneity among individuals is visible, obvious, or even intentional. Some subjects receive the medicine, others receive the placebo. The heterogeneity is clear. The outcome is variable: some people get sick and some do not. As scientists, we have by and large agreed that one is allowed to invoke the heterogeneity in treatment as the cause of the variability in outcome only after showing that the heterogeneity can overcome the stochastic variability in the system. If you can't show that, you cannot reject the null hypothesis, and you don't get to invoke the heterogeneity — medicine versus placebo — as the cause of the differences in outcomes. You accept that the parsimonious explanation is stochasticity. This is the basis of statistical hypothesis testing; we are all trained to think and to analyze data this way.

But when a large component of demographic variability is found to be due to stochasticity, the attitude changes. It becomes tempting to invoke unknown sources of heterogeneity as the explanation. Instead of showing that heterogeneity overwhelms stochasticity, it seems that before one can invoke stochasticity, one must first show that it overcomes some unmeasured heterogeneity. Even when measured sources of heterogeneity are included and do not come close to explaining the variation, there is a tendency to cling to the idea that there must be some unknown heterogeneity at work. If only it were included, the variance would be explained. It is worth thinking about why this is so.

8.5 Inequality and stochasticity: how to think about them?

Socioeconomic signals that have undeniable impacts on the lives of individuals make only a small contribution to the variance in the length of those lives. What should we do?

One option is suggested by another case where heterogeneity was found to make only a small contribution to variance. In 1972, using the then new biochemical methods of measuring genetic diversity, Richard Lewontin surveyed studies that had used 'racial' groups in the measurement of biochemical genetic diversity (Lewontin 1972). He found that, on average, 94% of the variance was within groups, and only 6% between groups. He concluded, [6]

[6] Half a century has passed, and today, as I write this, the U.S. National Academies of Sciences, Engineering, and Medicine issued a major report on the use of racial categories. Their first recommendation is

> "Researchers should not use race as a proxy for human genetic variation. In particular, researchers should not assign genetic ancestry group labels to individuals or sets of individuals based on their race, whether self-identified or not." (National Academies of Sciences, Engineering, and Medicine 2023)

> "Human racial classification is of no social value and is positively destructive of social and human relations. Since racial classification is now seen to be of virtually no genetic or taxonomic significance either, no justification can be offered for its continuance." (Lewontin 1972)

The variance in life expectancy among income-based socioeconomic groups accounts for a similarly small fraction of the variance in longevity, but it is unlikely that the differences between the lives of the richest and the poorest will seem trivial to anyone experiencing them. The option of ignoring between-group variance and simply appreciating the large within-group variance due to stochasticity is not as attractive in the case of longevity as it is in the case of race.

The opposite tack, ignoring inequality of outcome and focusing only on inequality of opportunity, has also been suggested. In the context of health inequality, Gakidou, Murray, and Frenk (2000) argued for treating individual stochasticity as an epiphenomenon, and treating heterogeneity, despite its small contribution to variance, as the relevant aspect of health inequality:

> "We claim that we are not interested in the inequality that has arisen from chance (or luck) since at the outset all individuals had exactly the same risk." (Gakidou, Murray, and Frenk 2000)

Remarkably enough, shortly afterwards Gakidou and King (2002) presented an approach to evaluate both between-group and within-group variability in health, and emphasized the importance of individual-level variation. They analyzed the mortality of children under age 2, using what would be described here as a latent frailty model based on a beta-binomial model for survival. Their approach was used by the World Health Organization and led to a series of spirited arguments about the proper attention to pay to between-group and within-group variation (Braveman, Krieger, and Lynch 2000; Murray, Gakidou, and Frenk 2000).

These arguments skirt the question of genuinely stochastic variation; the demographic calculations of variance in longevity include such variation. Random outcomes pose a challenge to the usual concepts of equality and inequality. Anderson (1999) explores these philosophical issues in depth. She criticizes "the view that the fundamental aim of equality is to compensate people for bad luck" and argues that the goal of egalitarian justice "is not to eliminate the impact of brute luck from human affairs, but to end oppression, which by definition is socially imposed." Such arguments would lead to a focus on between-group variance, to the extent that inequality related to socioeconomic conditions is socially imposed.

But ignoring stochasticity would render many recent approaches to lifespan inequality meaningless because most of that inequality is stochastic. In the context of policy and planning in an aging society, it would eliminate some important demographic tools,

particularly in relation to risk. I believe that demographic tools are precisely the key; they make it possible to analyze the undeniably important differences among socioeconomic groups, simultaneously acknowledging the major impact of stochastic processes on demographic outcomes. They highlight the fact that calculated variances in longevity are a very different kind from of inequality than the inequalities that result from social, economic, and environmental heterogeneity among individuals. That appreciation can only improve the analysis of risk and uncertainty in public policy.

8.6 Individuals, groups, and decomposition; some ideas for the future

The variance decomposition approach is the basis for analysis of variance (ANOVA) in statistics. ANOVA developed into a dizzying array of experimental designs, designed to explore complicated interactions among multiple factors in manipulative experiments (e.g., Kirk 1968). To date, decompositions of variance in longevity have considered only the simplest one-way designs. Factorial designs, in which the set of individuals is partitioned into two or more cross-classified groups, can also be analyzed, partitioning variance into between-group main effects and interactions, and within-group stochasticity. Nested designs, in which a second source of inequality or heterogeneity exists within each group, also warrant examination. For example, male and female mortality schedules differ. A nested analysis could be developed that would decompose the variance into, say, between countries, between sexes within countries, and within sexes. These approaches will be explored elsewhere.

It would be interesting to also explore the standardized variance, $V(\xi)/E(\xi)^2$, which has some advantages for being dimensionless. It can be decomposed into between-group and within-group components (Rosenbluth 1951). The standardized variance plays a role in evolutionary genetics, where it is known as Crow's index of the opportunity for selection (see van Daalen and Caswell 2020 for an exploration in a demographic context).

Permanyer, Sasson, and Villavicencio (2023) have recently introduced a new approach that may shed light on different aspects of inequality. They claim that variance decomposition "tells us nothing about groups' relative performance." This is not strictly true, because the between-group component of variance is in fact an integrative measure of relative group performances. But variance decomposition does not reveal which groups perform better or worse than which others, and Permanyer's analysis does so. It remains to be seen how the differences between heterogeneity and stochasticity will play out in that context.

Seligman, Greenberg, and Tuljapurkar (2016) relate declines in variation in healthy longevity to different causes of death, especially their ages of action. A matrix formulation of competing causes of death in Markov chain models may lead to a welcome

connection between causes of death and individual stochasticity (Caswell and Ouellette 2016; Caswell, Verdery, and Margolis 2023).

Variance is ubiquitous, and many demographic outcomes are treated only as expectations when they also exhibit variances within and among groups. Longevity is not the only demographic outcome with interesting variance patterns. Lifetime reproductive output (LRO, also often called lifetime reproductive success, LRS) is one example. The net reproductive rate R_0 is the expectation of LRO; the total fertility rate, TFR, is the expectation of LRO under the additional assumption that all individuals live through their reproductive ages. But LRO is a random variable, and its statistics can now be calculated in a variety of demographic situations (Caswell 2011; van Daalen and Caswell 2015; van Daalen and Caswell 2017). In many cases, the entire distribution of LRO can be computed (Tuljapurkar et al. 2020). Patterns of variance in lifetime fertility and their decomposition into between-group and within-group components are only beginning to be explored. For example, in a laboratory study of a species of rotifer, heterogeneity due to maternal age contributed 26% of the variance in LRO under laboratory conditions that led to extreme population growth rates, but less than 1% under more ecologically relevant scenarios approximating stationarity (van Daalen et al. 2022). In the Southern Fulmar study described in Section 5.3, latent heterogeneity contributed 22% of the variance in LRO (Jenouvrier et al. 2018). Further exploration of the variances in LRO would be valuable and would bring fertility into the same focus as longevity.

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