

The Quiescent Phase in Human Mortality: When Do Populations Start to Age?

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Abstract

Demographic studies of mortality hazards tend to emphasize the two ends of the lifespan, focusing on the declining pattern of child mortality or the increasing trajectory of deaths at older ages. We call attention to the relatively quiescent phase in between, representing the ages when we are least vulnerable to the force of mortality. Using nonlinear fitting methods and data for cohorts born 1800-1919, we estimate the parameters of the Siler model – which describes the mortality hazard across the full age spectrum. We then calculate inflection points in the Siler curve and characterize the start of the quiescent phase (representing the end of childhood vulnerability) and its end (the age when the hazard begins its exponential rise). As mortality hazards declined over time, the quiescent phase has gotten longer, with an earlier beginning in early childhood and a later end at the conclusion of the reproductive years. This analysis provides new insight into the dynamics of human development and implications for studies of individual and population aging.

Introduction: The Age Pattern of Human Mortality

Much of the demographic literature on mortality emphasizes deaths in early and mid-to-late life (Gomperz 1825; Medawar 1952; Wachter 2003; Levitis 2011). This is mortality that takes place on the far left and right sides of the hazard trajectories shown in **figure 1**. The sharp decline in mortality during the first few years of life, the exponential increase of mortality in adulthood, and mortality deceleration among the oldest-old have been noted and modeled by mathematical demographers for centuries (Thiele 1871; Lexis 1878; Siler 1979). However, with the exception of the “hump” of increased mortality (often attributed to injuries and death from external causes) in adolescence and young adulthood (Heligman and Pollard 1991), the phase between the exponential trajectories that characterize mortality hazards in infancy and adulthood has received relatively little attention and tends to be mentioned only in passing between the two more prominent segments. Nonetheless, this quiescent phase represents the ages when humans are least vulnerable to the force of mortality, and its features should be better characterized for a more complete understanding of the human mortality trajectory.

Questions about what causes the mortality hazard to decline after the first years of life or to begin rising again after a certain age are often considered to be more in the realm of biology, physiology, and gerontology than demography. While there are doubtlessly numerous genetic, environmental and behavioral factors that shape the hazard trajectory with age, an analysis of the quiescent phase may nonetheless provide insight into several questions: What explains the stability in mortality over the quiescent phase? Is the quiescent phase apparent in hazard trajectories across populations and over time? What defines the age boundaries of this remarkable (and yet unremarked upon) phase? And, considering the link between population hazard trajectories and individual-level senescence, what do the age boundaries of the quiescent phase tell us about human development, maturation, and senescence?

Methods

We use a Siler (1979) model to calculate the points of inflection that define the beginning and end of the quiescent phase for cohorts born 1800-1919 in Sweden, the nation with the longest available time series of cohort life tables. [In further research, we will apply a similar analysis to life tables from 14 additional high-longevity countries included in the Human Mortality

Database.]

We chose the Siler model, rather than the more-commonly used Gompertz (1825) because the latter includes information only on adult mortality risks, whereas the Siler model includes three competing but non-interacting components that characterize mortality across the age spectrum. Siler argued that living beings are exposed to three types of hazards throughout the lifespan: (1) a hazard that decreases from birth onward as the animal adjusts to its environment, likely as a result of maturation; (2) a constant hazard, reflecting a set of risks present in the “background” and to which the animal does not adjust over time; and (3) a hazard that increases with age, reflecting the growing risk of death as a result of senescence.

Combining these three components, Siler’s additive model produces a hazard trajectory that decreases in early life, remains relatively flat between later childhood and young adulthood, and then increases monotonically at older ages:

$$\mu(x) = e^{\alpha_1 - \beta_1 x} + e^{\alpha_2 + \beta_2 x} + e^{\alpha_3}, \quad (1)$$

where the α constants describe the hazard levels and the β parameters represent fixed rates of mortality decline and increase over age. The first term on the right hand side of the Siler model represents the exponentially declining mortality hazard during childhood, the second term (which is a conventional Gompertz trajectory) represents the exponentially increasing mortality hazard in adulthood, and the third term is the background mortality component, reflecting the overall (non age-dependent) level of mortality in a given time. The quiescent phase is the age interval (x_1, x_2) , such that $\mu(x) \approx \text{constant} = e^{\alpha_3}$ for all x in (x_1, x_2) . From a reliability theory perspective, a system is not considered to age during the quiescent phase.

Figure 2 demonstrates how the three components additively create a bathtub shaped hazard function for mortality across the lifespan. The Siler model fits human mortality data as well or better than most other models (Gage and Dyke 1986, Gage and Mode 1993). To estimate the five Siler parameters we used nonlinear optimization methods available through the `optimx` package in R (2011) and applied the method directly to the age-specific mortality hazard (q_x) trajectory for each successive cohort life table. The parameter estimates generated by the nonlinear least squares fitting procedure turned out to provide a superior fit as compared to maximum likelihood and other model-fitting methods, yielding a curve that closely approximated the empirical trajectory and more precise individual parameter estimates. We also obtained robust standard errors from sandwich variance estimators (White 1980; Zeileis 2006).

The Siler parameter estimates can be used, in turn, to calculate the two inflection points in the hazard curve. The first inflection point, x_1 , represents the end of infant mortality and the beginning of the quiescent phase. It occurs when the first (childhood) Siler component $e^{\alpha_1 - \beta_1 x_1}$ is equal to half of the third (background) hazard component, $1/2e^{\alpha_3}$. Thus, we calculate this quiescence start age as

$$x_1 = (\hat{\alpha}_1 - \hat{\alpha}_3 + \log 2) / \hat{\beta}_1. \quad (2)$$

The second inflection point, x_2 , represents the end of the quiescent phase and the beginning of senescence as defined by a sharp increase in the mortality hazard slope. It occurs when the second (adult) Siler component $e^{\alpha_2 + \beta_2 x_2}$ is equal to half of the third (background) component, $1/2e^{\alpha_3}$. Thus, the age when the quiescent phase gives way to the start of senescence is calculated as:

$$x_2 = (\hat{\alpha}_3 - \hat{\alpha}_2 - \log 2) / \hat{\beta}_2. \quad (3)$$

Below, we show trends in these inflection points as well as the calculated length of the quiescent phase for successive cohorts born 1800-1919. In addition to calculating the exact inflection points and their difference, we also provide lowess-smoothed curves of the trend trajectories. We conducted sensitivity analyses to examine how the inflection points and overall length of the quiescent phase (calculated by subtracting the starting age from the ending age) would change with alternative correction factors based on the calculated parameter values. While the specific inflection ages and the calculated length change somewhat, the qualitative trend in the characteristics of the quiescent phase across cohorts remains the same.

Results

Figure 3 shows how well the Siler curves with nonlinearly-estimated parameters fit the empirical mortality data for selected cohorts of females and males, respectively.

Figure 4 shows trends in the two inflection points for females and males in successive cohorts, including both the empirical calculation and a lowess-smoothed trend line. The start of the quiescent phase has fluctuated over time, showing a marked decline towards age 1 for cohorts born after 1870. The beginning of senescence, on the other hand, has shown a clear increase across cohorts experiencing improved survival, rising from the mid 30's to the mid 40s for females, and from the mid 20s to around age 40 for males. Finally, **Figure 5** tracks the changing length of the quiescent phase (in years) for Swedish male and female cohorts.

Discussion

This analysis provides some additional insight into the dynamics of human development and population aging. The minimum hazard at the beginning of the quiescent phase has been coming down, reaching its minimum point at earlier ages as infant survival improves. The hazard rate when senescence begins has also been decreasing, even as the age marking the transition between quiescent and senescent phases has risen. The length of the quiescent phase has increased over time, and for the most recent cohorts with available data, the force of mortality essentially remains quiescent from childhood until (roughly) the end of the reproductive years.

The quiescent phase itself has occurred at lower and lower levels of mortality over time. This is consistent with its interpretation as the "background" mortality element in the Siler model, representing deaths due to environmental and period conditions (e.g., accidents, wars, pestilence, etc.). Hence, the decreasing levels reflect a general improvement in survival.

In subsequent analyses for this paper, we will compare the characteristics of the quiescent phase across countries, and also examine the quiescent phase's historical evolution relative to other population features that have changed systematically over the course of the demographic transition. We will also consider the biological implications of the changes in the starting and ending ages of the quiescent phase and the apparent stabilization in its length over time.

In reliability theory (e.g. Gavrilov and Gavrilova 1991, 2001), a system is considered to age if its failure intensity (or hazard) increases over time (i.e with age). In many studies of aging, 65 is used as a common cut-off distinguishing the old from the middle-aged and young. However, our population-level analysis suggests that senescence (as defined by the inflection point in the hazard curve) in fact begins around age 40 (arguably around the time when the reproductive program of evolution has been played out in the individual). While demographers going back to Gompertz have recognized age 30-40 as the starting point for analyses of adult mortality, epidemiological and social studies of aging often chose the higher age cutoff in deference to convention or administratively constructed categories. To more fully understand health trajectories in later life, we need to build-in more leeway for understanding the factors that define the direction and path of those trajectories in midlife.

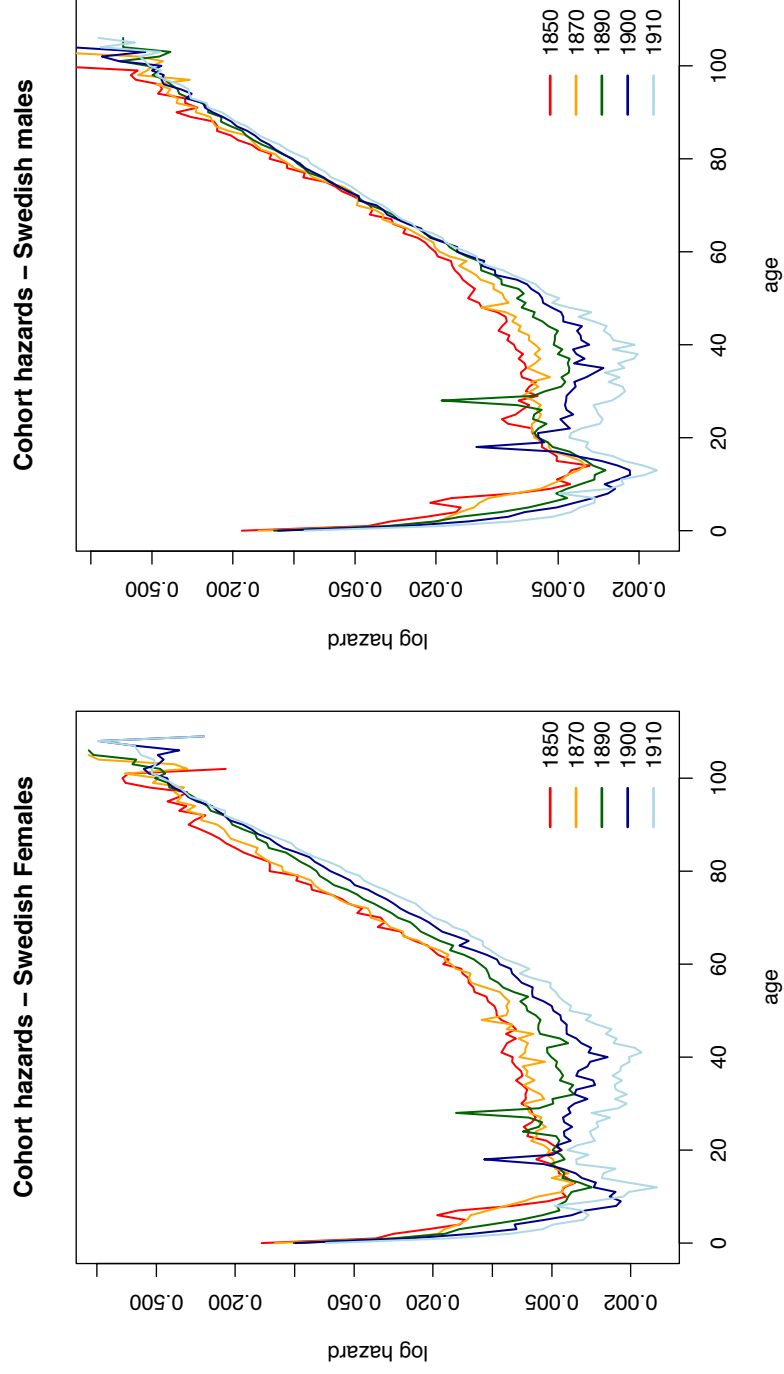


Figure 1: Age trajectories of the mortality hazards for selected female and male cohorts in Sweden.

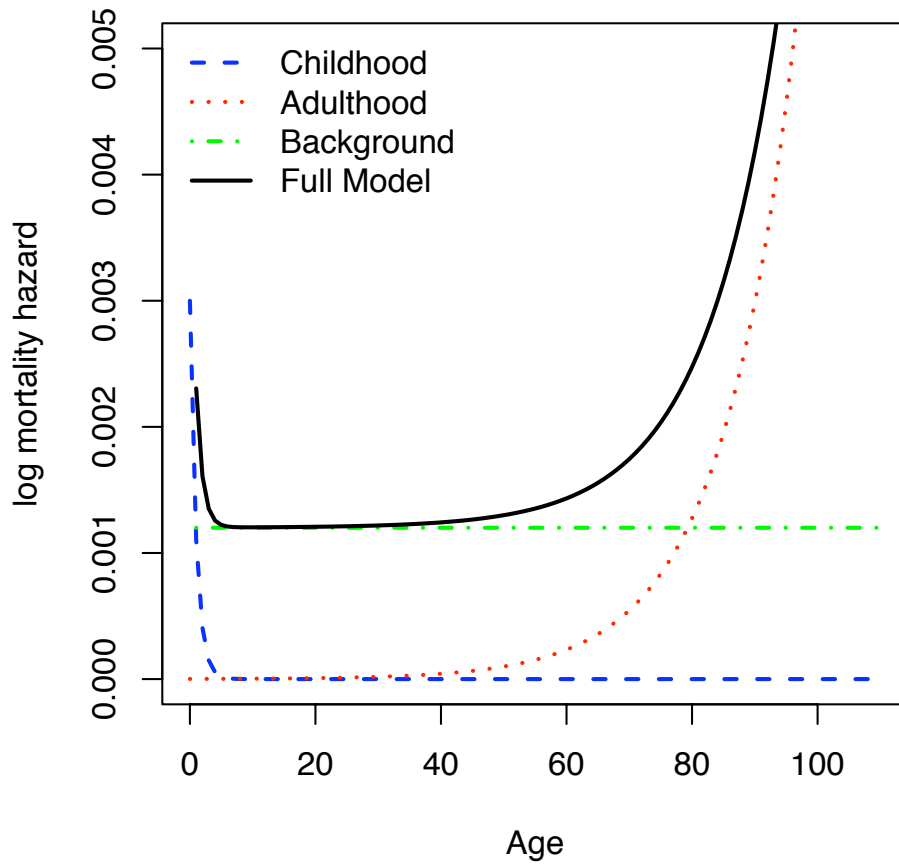


Figure 2: **The three-component Siler model:** $\mu(x) = e^{\alpha_1 - \beta_1 x} + e^{\alpha_2 + \beta_2 x} + e^{\alpha_3}$, where the first term on the right represents the mortality pattern dominant in childhood, the second term represents the mortality pattern dominant in adulthood, and the third term represents a background mortality level.

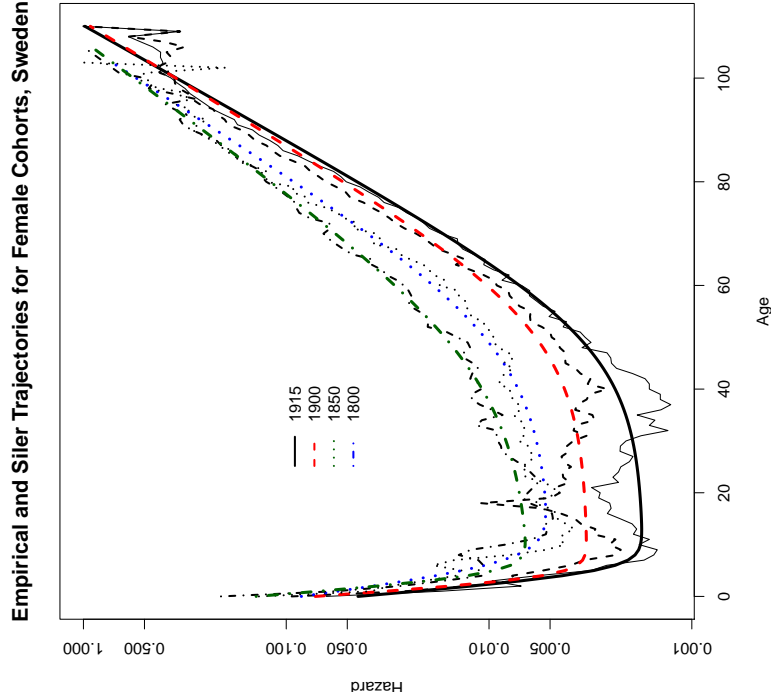
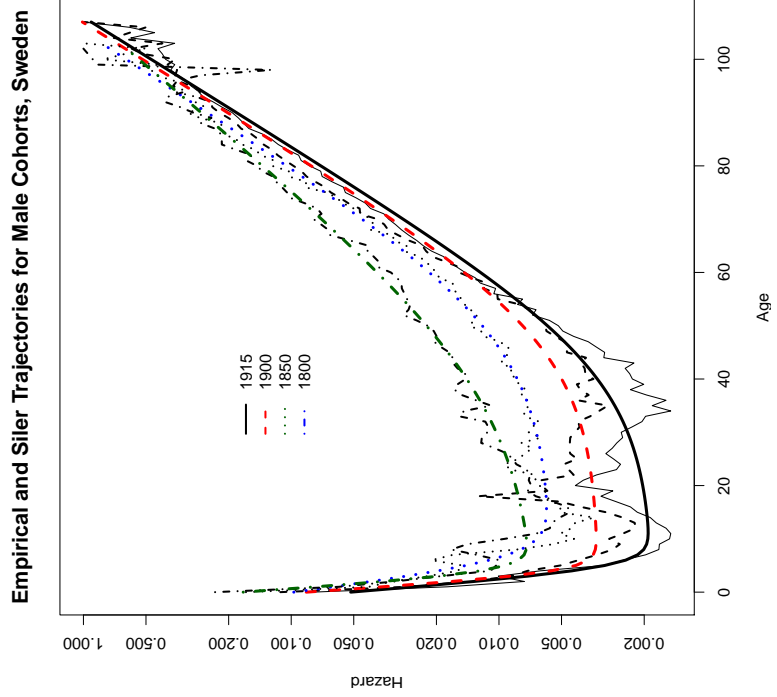


Figure 3: Empirical hazard and Siler trajectory for Swedish Cohorts born 1800-1919.

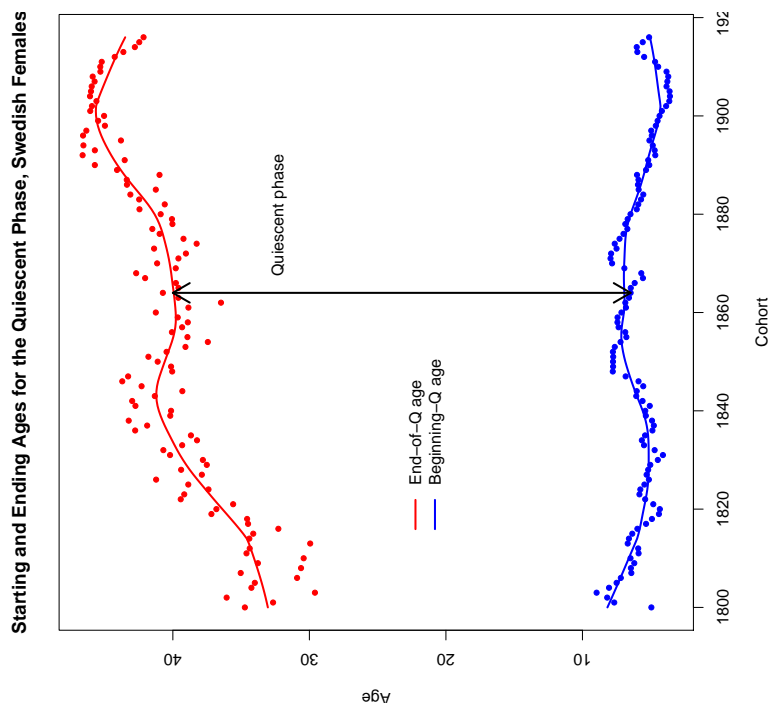
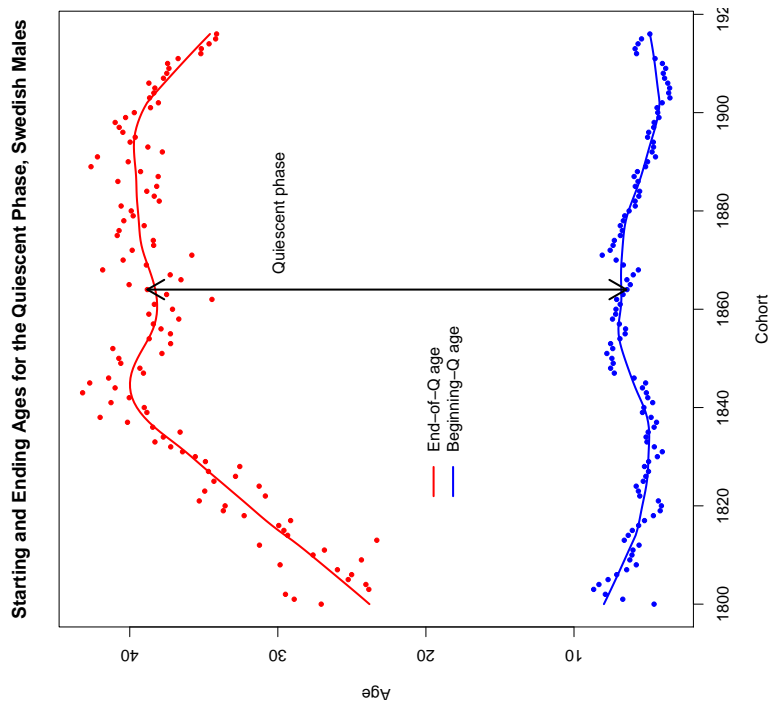


Figure 4: Trends in the beginning and ending ages of the quiescent phase for Swedish Cohorts born 1800-1919.

Length of the Quiescent Phase

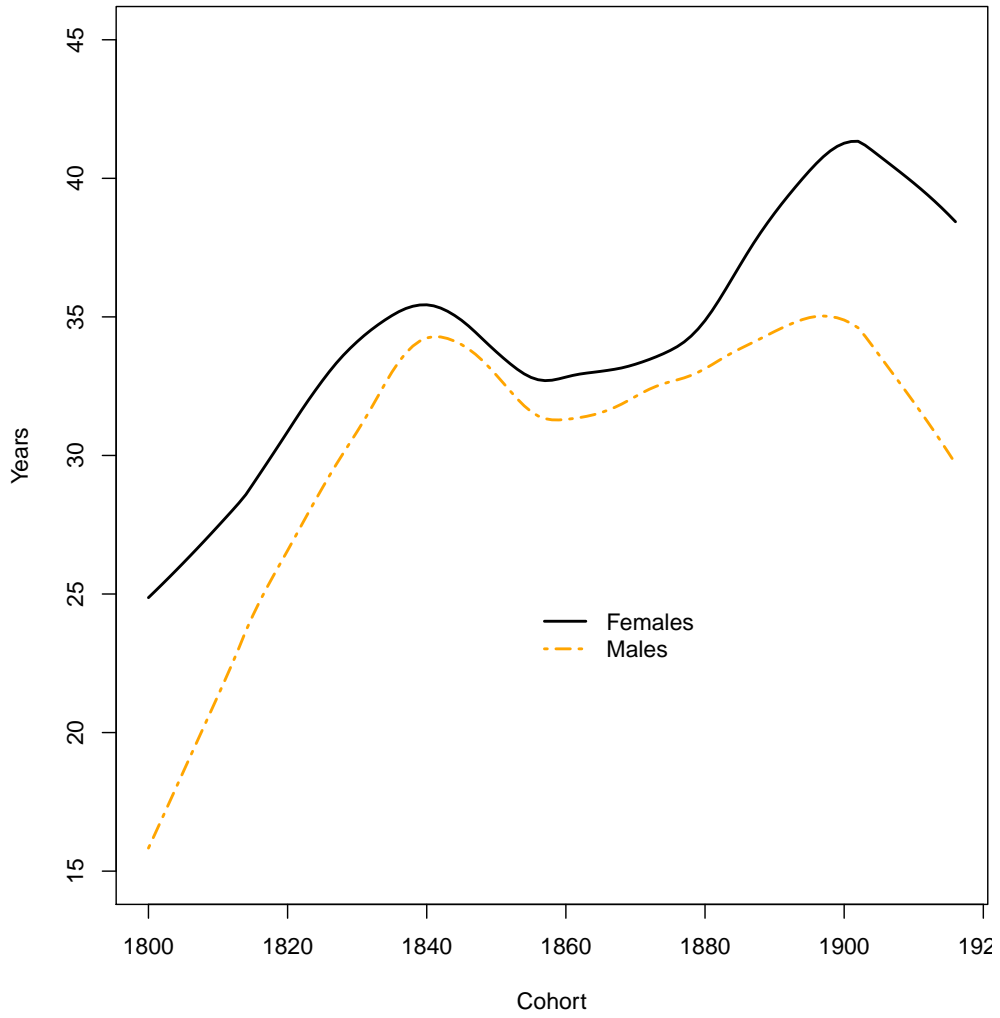


Figure 5: Length of the quiescent phase (in years) for Swedish cohorts.