Predicting old age life expectancy with conditions at perimenopause: A test of the sensitive periods model in adulthood

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Abstract
While studies using the sensitive periods framework have traditionally focused on the effects of early life exposures on later life health, characteristics of the menopausal transition (i.e., perimenopause) suggest this window may serve as a sensitive period in adulthood. The application of the sensitive periods model to perimenopause was tested through an exploratory analysis of the relationship between mortality rates at ages 45-49 and life expectancy at age 60. Cohort mortality data from two historic populations France (1814-1919) and England and Wales (1841-1919) were analyzed using time series methods. Results indicated a significant inverse association between mortality at ages 45-49 and life expectancy at age 60 among females in both countries. Study findings suggest a degree of plasticity associated with women’s aging, and in particular, the age group correlated with perimenopause.

Keywords: Perimenopause, Critical Periods, Longevity, Life expectancy, Mortality
Introduction

Life course epidemiology suggests that various biological and social factors occurring throughout the life span influences health in adulthood (1). Certain windows of time, however, represent critical or sensitive periods, during which environmental stimuli or threats can have particularly enduring effects on health and development (2). In critical periods, an exposure acting during a confined window of time has lasting effects on the structure or function of organs, tissues, and body systems, which cannot be modified in any significant way later in life (3). Sensitive periods are windows during which exposures have a stronger effect than they would have at other times. Outside these windows, excess disease risk associated with exposure is weaker (1, 4).

Studies testing the critical periods hypothesis have traditionally focused on the effects of early life exposures on later life health, which was pioneered by Barker’s work on the fetal programming of cardiovascular disease (1, 5). Much work since then has linked the fetal environment with various other morbidities, including diabetes mellitus, stroke, lung abnormalities, depression, and immune dysfunction (6). Such studies suggest that factors acting in prenatal and early postnatal life play an important role in the etiology of these diseases (2, 3). Researchers increasingly are finding that childhood and adolescence are critical periods for adult health outcomes as well. Studies show, for example, that environmental conditions and behaviors during childhood and adolescence can profoundly affect the risk for obesity and osteoporosis (7, 8).

A relatively scant body of evidence—in comparison to that on infancy and youth—also has investigated the role of critical and sensitive periods in adulthood. An example of such research is the clinical trials on the role of estrogen therapy during perimenopause, the transition period between regular pre-menopausal menstruation and the last menstrual period. This research has shown that hormone replacement therapy initiated during perimenopause—but not post-menopause—may prevent or delay cognitive decline in aging women (9). The significance of the sensitive periods model in adulthood is tested in this paper through an exploratory analysis of the relationship between women’s environmental conditions at mid-life and longevity.

Both individual-level and cohort-level studies have investigated the association between early life conditions and life expectancy, but the results of these studies implicate different age groups as sensitive periods. Evidence reporting the first year of life as a sensitive period shows that adverse circumstances experienced prior to age 1 are associated with significant decreases in cohort life expectancy (10, 11). Other research suggests the first five years of life are a sensitive period for life expectancy, indicating that deviations from trend in cohort mortality before age 5 is associated with shifts in later life cohort life expectancy (12, 13). Puberty also has been implicated as a sensitive period for longevity, with research showing an association between cohorts that experienced life-threatening stressors during adolescence and elevated mortality in adulthood, at least among males (14, 15).

No study, to my knowledge, has tested whether the presence of sensitive periods for longevity occur in adulthood. Individual level data, however, suggests that changes in risk for numerous diseases and health conditions—including cancer and cardiovascular disease—occurs during perimenopause (16). Adverse circumstances during this period of time, therefore, may have a more profound effect on health and life expectancy akin to the critical and sensitive windows occurring earlier in life. This paper used the sensitive
periods model as conceptual framework to test whether elevated mortality rates occurring at mid-life (i.e. ages 45-49, the age range at which perimenopause approximately occurs in populations), was predictive of longevity, defined as life expectancy at age 60.

Life course experiences distinguish the functional status and mortality levels of cohorts. Exposure to a particularly pernicious environment could, for example, translate to higher mortality levels for a given cohort or population (17). Described by Finch and Crimmins (2004) as the “cohort morbidity phenotype,” this phenomenon suggests that changes in a cohort’s epidemiological environment can affect mortality characteristics of surviving members of the cohort for the rest of their lives (18). Other studies have used a cohort’s own mortality as a proxy for stressful or threatening environmental conditions (12-14, 19), but none have examined the relation between conditions at midlife with later life longevity.

If women’s mortality rates were significantly associated with life expectancy at age 60, then these findings would suggest the occurrence of a sensitive period for women during perimenopause. An association in which higher mortality at age 45-49 was linked with lower life expectancy at age 60 would suggest the occurrence of cohort “scarring.” Under this scenario, adverse conditions or exposures at mid-life would permanently damage or impair survivors (20, 21). An association in which higher mortality at age 45-49 was linked with higher life expectancy at age 60 would suggest the occurrence of cohort selection. In this situation, conditions or exposures at mid-life would “cull” frail individuals from the population, “selecting” the more hardy individuals for survival to older ages (21). If, however, no sensitive periods existed over and above the general effect of a given cohort’s frailty, then no statistically significant associations would be expected between women’s mortality rates at ages 45-49 and life expectancy at 60.

Most existing life course studies are limited in that they examine exposures within one cohort at a single time, and therefore may have low external validity (3). This study examined 104 different cohorts in France between 1816 and 1919 and 79 cohorts in England and Wales between 1841 and 1919, testing whether perimenopause represented a sensitive period for women with respect to longevity. Implications about population health, therefore, can be drawn from the experience of a multitude of cohorts.

Data

Sex-specific cohort mortality and life expectancy data from the civilian populations in France (cohorts born between 1816-1919) and England and Wales (1841-1919) were drawn from the Human Mortality Database (HMD) (22). The HMD is a collection of high-quality death rates and life tables for countries where death registration and census data are virtually complete. Only 37 countries—spanning various lengths of time—provide data that meet the standards of the HMD. Because the longest spans of reliable cohort data belong to France and England and Wales, these countries were selected for analyses.

Dependent Variable

Longevity was assessed through female cohort life expectancy at age 60 because this age is commonly used to demarcate the beginning of old age in populations (23, 24). Because cohort life expectancy can only be calculated among birth cohorts whose
members have virtually all died, the time series for France and England and Wales end in 1919. Life expectancy at age 60 in both France and England and Wales gradually increased over the time series analyzed, although was interrupted by a few major events during the 19th and 20th centuries. In France, epidemics of cholera (1832, 1849, 1854) and the Franco-Prussian War (1870-1871) for example, temporarily decreased life expectancy. Likewise, in England and Wales, outbreaks of typhoid and flu (1846-1848) and cholera (1832, 1849, and 1865-1866) caused temporary spikes in mortality that affected life expectancy. General trends of increasing life expectancy, as well as the temporary interruptions caused by war and epidemics, were controlled in analysis.

Independent Variables

Female cohort mortality rates at ages 45-49 were used to indicate the conditions or circumstances to which a given cohort was exposed during perimenopause. Elevated mortality rates that occurred significantly above what would be statistically expected implied the presence of adverse conditions affecting the population. Cohort mortality rates at infancy (age 0) and five-year intervals which prior literature has indicated as critical or sensitive periods (i.e., early childhood (ages 1-4) and puberty (ages 10-14)) were tested for significance as potential predictors of female longevity. (Studies on menarcheal timing (i.e., first menses, which is a marker of puberty) and pubertal duration show the age range 10-14 likely encompasses the start and progression of pubertal events for most girls in France and England & Wales during the mid-19th Century (25-27.).) The mortality rate for ages 55-59 was also included in the models to control for recency effects on life expectancy at age 60.

The age classification designated to represent perimenopause is indicative of the average female in France and England and Wales during the mid to late 1800s. Research indicates the average age of menopause occurs between 49 and 52 years of age in westernized countries (e.g., Europe, United States of America) (28-30), and most studies find that menopausal age has not changed significantly over time (31-33). With research reporting that perimenopause lasts approximately four years and begins on average between the ages of 45 and 47.5 (31, 34), the age range of 45-49 would encompass the start and progression for most of perimenopause.

As with life expectancy, mortality rates improved over the 19th and 20th centuries. This general decreasing trend for all age groups is controlled for in analysis.

Methods

This test assessed whether life expectancy at age 60 changed from its statistically expected value given increases in mortality rates between the ages 45-49, controlling for cohort mortality rates in other presumed sensitive periods for female longevity, as well for endogenous cohort frailty. Several studies have provided evidence showing that a cohort’s life expectancy is most significantly influenced by the environmental conditions or exposures at certain ages. These sensitive windows for longevity include infancy (age 0), early childhood (ages 1-4), and puberty (ages 10-14) (12-14, 35). Cohort mortality rates from these age groups, therefore, were included as covariates. Because phenomena that would cause a cohort to die more frequently at midlife and at age 60 would be expected to manifest at other ages throughout the life course, cohort frailty was controlled
with the inclusion of cohort mortality rates for the presumed sensitive windows as well for cohort mortality at ages 55-59. Cohort mortality at ages 55-59, in particular, captured the closest measure of cohort frailty prior to the count of remaining life (i.e., life expectancy at age 60).

Significant (i.e., \( p < 0.05 \), two-tailed test) derivations from the expected value in the independent variable that were associated with deviations from the expected value in the dependent variable signaled the presence of a sensitive window. Mortality and life expectancy data exhibited autocorrelation (e.g., trends, cycles, tendencies to remain elevated or depressed, oscillations after higher or low values), which caused the expected value to deviate from the mean of the series. In order to estimate the expected values, autocorrelation was identified and removed from the dependent and independent variables using autoregressive integrated moving average (ARIMA) modeling, developed by Box and Jenkins (36). The ARIMA procedure involves applying numerous filters to a time series in order to predict values from past values and shocks in the series. This process yields a time series of residuals that are statistically independent of one another, exhibit no temporal patterns, and have an expected value of zero. ARIMA modeling provides significant benefits in the analysis of mortality and life expectancy data because it takes into account the temporal interdependence of the data.

Following the procedural strategy developed by Box and Jenkins for modeling time series, I identified and removed autocorrelation in the values for life expectancy and for cohort mortality for each year. This procedure identifies any non-stationarity or seasonality present in the data. The Box and Jenkins routines model these patterns as well as the tendency of a series to remain elevated or depressed after high or low values. Trends were then removed or differenced (i.e., the values of each year were subtracted from those of the following year). Other forms of autocorrelation were modeled with autoregressive or moving average parameters. I proceeded through the following analytical steps using software from the Scientific Computing Associates Statistical System.

Identification and Modeling of Autocorrelation in the Dependent and Independent Variables

1) I used Box-Jenkins routines to identify and model autocorrelation in the dependent variable (i.e., life expectancy at age 60) for France and England and Wales. In order to achieve stationarity, the series was differenced, and any other regularly occurring behavior the series shared with the original regression was removed. The residuals created from this process represent the degree to which life expectancy at 60 deviated from its expected value.

2) The same Box-Jenkins techniques were applied from the first step to identify and model autocorrelation in female cohort mortality rates at ages 45-49, as well as at infancy, ages 1 through 4, and the remaining five-year age groups (e.g., 10-14, 55-59) in France and England Wales.

Estimation

3) I ran a preliminary test of the bivariate association to examine first whether cohort mortality rates at ages 45-49 were significantly correlated with cohort life.
expectancy at age 60. This step involved adding the residuals of the age 45-49 mortality series to the equation formed from modeling female cohort life expectancy.

4) The parameters for the full Box-Jenkins models for both countries were estimated using maximum likelihood estimation. In this step, the residuals of the other age-specific mortality series generated from the second step were added to the model generated in step 3. The test equations that emerged from this process for France and England and Wales are as follows:

$$\nabla Y_{t_{\text{France}}} = C + \omega_1 \nabla X_{0t} + \omega_2 \nabla X_{1-4t} + \omega_3 \nabla X_{10-14t} + \omega_4 \nabla X_{45-49t} + \omega_5 \nabla X_{55-59t} + \left(1 - \theta_1 B - \theta_2 B^2 - \ldots - \theta_q B^q\right) a_t$$

$$\nabla Y_{t_{\text{England \& Wales}}} = C + \omega_1 X_{0t} + \omega_2 \nabla X_{1-4t} + \omega_3 \nabla X_{10-14t} + \omega_4 \nabla X_{45-49t} + \omega_5 \nabla X_{55-59t} + \left(1 - \phi_1 B - \phi_2 B^2 - \ldots - \phi_p B^p\right) a_t$$

$\nabla$ is the difference operator indicating that $Y$ has been differenced from $Y_{t-1}$ to remove trends and cycles from the series and render stationarity in its mean. $Y_t$ equals female cohort life expectancy at age 60 at year $t$. $C$ is a constant. $\omega_1$ through $\omega_5$ is the estimated parameters for the residuals of the age-specific cohort mortality rates. $X_{0t}$ through $X_{55-59t}$ are the residuals of the best fitting models of cohort mortality rate for each age group. $B^n$ is the backshift operator that either $\theta$ or $\phi$ acts on the value of the error term ‘a’ at year $t-q$ or $t-p$. $\theta$ is the moving average parameter. $\phi$ is the autoregressive parameter. $a_t$ is the error term at year $t$.

**Results**

Cohort life expectancy at age 60 increased for French females between 1816 and 1919, rising 11 years—from 14.2 (age 74.2) to 25.2 (age 85.2) remaining years of life (see Figure 1). Cohort life expectancy at age 60 also increased for females in England and Wales during the time series (1841-1919), rising 7.4 years, from 15.3 (age 75.3) to 22.7 (age 82.7) remaining years of life (see Figure 2). Table 1 shows the equations modeling cohort life expectancy at age 60 for these two countries. The difference operator, $\nabla$, in the equations for both France and England and Wales indicates there was a general increasing trend in life expectancy at age 60 throughout the series. The autoregressive parameters (i.e., 0.387, standard error = 0.095; and 0.536, standard error = 0.079) in the model of French life expectancy suggests that high or low values carried into succeeding years, with “echoes” of such values appearing 1 and 10 years later, but decreased geometrically. The positive, albeit small, coefficient (0.090, standard error = 0.018) in the model for England and Wales indicates that life expectancy slightly drifted above the general upward trend during many of the years between 1841 and 1919.
Table 2 shows the equations that modeled cohort mortality rates in France and England and Wales. A decreasing trend in female mortality rates in nearly all age groups was observed in both countries. This trend was removed by taking the first differences of the series. Each of the age groups also exhibited various moving average and autoregressive patterns, which were subtracted from the cohort mortality series. The negatively signed constant values for several of the equations indicate the rates drifted below the general downward trend for many of the years in the series.

The results in Table 3 show that cohort mortality at ages 45-49 was predictive of life expectancy at age 60 in the test of bivariate association in France (p<0.01) after autocorrelation was removed from both variables. In England and Wales, the association approached significance (p=0.053). Cohort mortality at ages 45-49 was significant (p<0.01) for both countries in the full models that controlled for cohort mortality in the other presumed sensitive windows. Results indicate that life expectancy at age 60 among females in France between 1816 and 1919 and in England and Wales between 1841 and 1919 varied inversely with female cohort mortality rates between the ages 45-49.

Cohort mortality rates for ages 55-59 were also significantly associated with life expectancy at age 60 in both countries, along with cohort mortality at infancy—but only in England and Wales. The coefficient for cohort mortality at ages 55-59 was the most significant variable in the models for both countries, suggesting that conditions immediately preceding the measure of life expectancy have the greatest predictive power on life expectancy at age 60. The coefficient for cohort mortality at ages 45-49 was the next most significant covariate for both countries, followed lastly by the covariate for infancy in England and Wales.

The coefficients shown in Table 3 indicate that life expectancy at age 60 among female cohorts in France and England and Wales decreased by 0.1292 years (1.55 months) and 0.2029 years (2.43 months) respectively, for each increase of 1 per thousand in the age 45-49 mortality rate. To translate into a more interpretable context, mortality rates for age 45-49 were transformed from a continuous variable to a binary variable equal to 1 for cohorts exhibiting greater than expected death rates during ages 45-49 and 0 otherwise. The test equations were run again, with results of the coefficient for the binary variable revealing that life expectancy at age 60 decreased 0.045 years (0.54 months) on average for French female cohorts that experienced higher than expected mortality at ages 45-49. Among similarly aged females in England and Wales, life expectancy at age 60 decreased 0.059 years (0.71 months). As a frame of reference, cohort life expectancy at age 60 for French females increased 11 years, or an average of 1.27 months per year, over the 104 years analyzed for this study. Female cohort life expectancy in England and Wales increased 7.4 years, or an average of 1.12 months, over the 79 years in this study. These findings suggest that “threatened” perimenopausal females lost approximately half (43% in France and 63% in England and Wales) of the gains in life expectancy that females in this age group acquired due to general improvements in health, modernization, etc. that occurred during the 19th Century.

As a robustness test for the female-specific findings, additional models were run in which cohort mortality for infancy and all five-year age groups leading up to 60 (e.g., age 0, 1-4, 5-9….55-59) were included as covariates. These analyses did not yield significantly different outcomes from the results of the original models, with the age 45-
49 category remaining the most statistically significant predictor of life expectancy at age 60 (after cohort mortality at age 55-59) for both countries.

The analyses described above for modeling cohort life expectancy and cohort mortality rates was repeated for males, for whom theory would predict no significant association, given that they do not experience an equivalent—and abrupt—shift in hormone concentrations at mid-life (37). The results in Table 4 show there was no significant bivariate association between male cohort mortality at ages 45-49 and male cohort life expectancy at age 60 for both France and England and Wales. When models were fitted that included the five age groups included in the equivalent female models, however, the age 45-49 category appeared significantly associated with life expectancy at age 60 for males in England and Wales only. Only the age 55-59 group was significantly associated with life expectancy at age 60 among males in France.

**Discussion**

Results of analyses showed a significant, inverse association between conditions at mid-life and life expectancy at age 60 among females. Mortality rates among females ages 45-49 were predictive of life expectancy at age 60, such that unexpected increases in mortality were related to concomitant decreases in life expectancy at age 60. Stressors or adverse exposures occurring during this window appear to have a greater association with life expectancy at age 60 than similar exposures in other age groups, including infancy. These results suggest the “scarring” or “damaged cohort” hypothesis, where adverse conditions appear to permanently damage or impair survivors of the cohort.

It is unlikely that these results occurred due to frailty associated with some cohorts that happened to die more frequently at ages 45-49 as well as in later life, given that endogenous cohort frailty was controlled with the inclusion of cohort mortality at infancy, childhood, adolescence, and ages 55-59. The remaining variance in mortality at ages 45-49 therefore, was unique and significant above and beyond any variance that mortality rates in this age group shared with mortality rates in other age groups.

The finding of a significant association between mortality rates at ages 55-59 and life expectancy was somewhat expected, given that conditions occurring in this window immediately precede the measure of the dependent variable. The significant association of the age 45-49 category with life expectancy at age 60 plausibly implicates perimenopause as the explanation for this finding. Perimenopause represents a time of physiological reorganization in a woman’s life, during which rapid changes occur in hormonal levels (38, 39). This physiological restructuring may leave women particularly vulnerable to both environmental threats and stimuli as they transition from a reproductive to non-reproductive state (16). These changes are consistent with the notion of critical and sensitive periods, during which intrinsic changes in the organization of an organism occur rapidly and when regulatory pathways are being constructed or modified (1, 4).

While the lack of an analogous relationship among French males lends further support to the proposed explanation of perimenopause as a significant period for longevity, the significant association among males in England and Wales weakens this inference. Additional research testing this study’s hypothesis in other countries and in contemporary populations would need to be conducted in order to determine whether the
significant finding among males in England and Wales was the result of statistical artefact.

Alternatively, the age at which perimenopause occurs in populations coincides with major social changes that could have increased women’s vulnerability and affected their longevity. In contemporary society, for example, women often experience increased stress at midlife due to their children leaving home, increased caregiving for elderly parents, relationship changes, or changes occurring in the workplace. It is possible, therefore, that some psychosocial mechanism (as opposed to the biological mechanism proposed by the onset of perimenopause) explains the significant association between ages 45-49 and life expectancy at age 60 among women in historical France and England and Wales.

According to other life course models for health and disease, such as the model for accumulation of risk, this study’s findings could also potentially be explained by the accumulation of adverse exposures over the life course that may have been “triggered” at ages 45-49. Circumstances occurring when women were 45-49, in other words, may have acted as the final link in a chain of events that subsequently affected life expectancy at age 60. Results supporting this model of causality would have been expected to show a direct relationship between cohort mortality and life expectancy, such that an increase in mortality at ages 45-49 would winnow or cull the relatively frail members from the cohort. The results of this study do not support this explanation, given the inverse association between cohort mortality and life expectancy. Elevated mortality at ages 45-49 appeared to damage or weaken cohort survivors, resulting in a lower life expectancy at age 60.

These findings corroborate other studies that have found that adverse conditions in adulthood are often more influential on later life health than biological programming that may have occurred earlier in youth. The association between early life socioeconomic position and adult mortality rates, for example, is significantly reduced after controlling for adult socioeconomic position (SEP). Adult SEP, moreover, significantly affects mortality rates independent of early life SEP. These findings have proven robust across multiple populations, including Sweden, England and Wales, and Japan.

An empirical challenge of this study—reflective of observational cohort analysis more generally—is the inability to definitively determine the causal mechanism(s) that led to the associations between cohort mortality at midlife and life expectancy at age 60. Significant findings found in aggregate, moreover, may not apply at the individual level. Some literature has, however, previously documented the importance of environmental exposures or circumstances during perimenopause to aging women’s health at the individual level. No study, to my knowledge, has tested the sensitive periods model among perimenopausal women at the population level. This study addressed this research gap by showing how conditions at midlife similarly impacted women across two historic populations in Europe, at least with respect life expectancy at age 60.

Study findings yield important insight for assessing population health risk. The results of this study indicate the importance of analyzing the relatively narrow—yet profound—window between the ages of 45 and 49 for women in terms of their later life health. Such information could be particularly relevant with respect to large cohorts of women, who by virtue of their number, can exert significant effects on the financing of
and demand for health and social services. For example, the height of the baby boom in the United States occurred in 1957. As the largest cohort of women in the U.S. history approaches 60 in the year 2017, it may be useful to factor into forecasts how their health status affects the demand for and costs of services. Assessing the health of these women during the not-too-distant past (i.e., between 2002-2006) could provide insight on what is to come for aging women.

Conclusion

In sum, results from this study suggest a degree of plasticity associated with women’s aging. The period of perimenopause may indicate a sensitive period for women in terms of the development or modification of health trajectories into old age. Although women’s reproductive health is integral to their overall health and wellbeing, little is known about the interrelationship between social and lifestyle factors and reproductive health and how these vary across populations and cultures (40). Assessing the health of women during this window could provide insight for the timing and targeting of preventive health interventions.
Works Cited

22. Human Mortality Database. University of California, Berkeley (USA) Max Planck Institute for Demographic Research (Germany); Available at www.mortality.org or www.humanmortality.de (data downloaded on March 1, 2015).
Figure 1: Observed and expected values of life expectancy at age 60 among women in France, 1816-1919

Note: The first 21 years of expected values were lost due to modeling
Figure 2: Observed and expected values of life expectancy at age 60 among women in England and Wales, 1816-1919

Note: The first 17 years of expected values were lost due to modeling
Table 1. Box-Jenkins equations for female cohort life expectancy at age 60 for France (1816 through 1919) and England and Wales (1841 through 1919).

<table>
<thead>
<tr>
<th>Country</th>
<th>Box-Jenkins Equation</th>
</tr>
</thead>
<tbody>
<tr>
<td>France</td>
<td>( \nabla Z_i = 0.1428 + \frac{1}{(1 - 0.3869B)(1 + 0.5362B^{1/3})} a_i )</td>
</tr>
<tr>
<td>England and Wales</td>
<td>( \nabla Z_i = 0.0899 + a_i )</td>
</tr>
</tbody>
</table>
Table 2. Box-Jenkins equations for female cohort mortality rates at ages 0, 1-4, 10-14, 45-49, and 55-59 for France (1816 through 1919) and England and Wales (1841 through 1919).

<table>
<thead>
<tr>
<th>Age</th>
<th>France</th>
<th>England and Wales</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age 0</td>
<td>$VZ_i = \frac{(1+0.3720B)(1+0.2352B^4)}{(1+0.2540B^4)} a_i$</td>
<td>$Z_r = 0.1318 + \frac{1}{(1+0.9097B)(1+0.3390B^6)} a_i$</td>
</tr>
<tr>
<td>Ages 1-4</td>
<td>$VZ_i = \frac{(1-0.2516B)(1+0.3206B^4)}{(1+0.3987B^4)} a_i$</td>
<td>$VZ_i = -0.0003 + \frac{1}{(1-0.3088B^2)} a_i$</td>
</tr>
<tr>
<td>Ages 10-14</td>
<td>$VZ_i = \frac{(1+0.3506B^6)}{(1+0.2596B)} a_i$</td>
<td>$VZ_i = -0.00005 + a_i$</td>
</tr>
<tr>
<td>Ages 45-49</td>
<td>$VZ_i = -0.0001 + \frac{(1+0.2859B^9)}{(1+0.2214B^9)} a_i$</td>
<td>$VZ_i = -0.0001 + \frac{1}{(1-0.2828B)} a_i$</td>
</tr>
<tr>
<td>Ages 55-59</td>
<td>$VZ_i = -0.0001 + \frac{(1+0.4230B)}{(1-0.3730B^3)} a_i$</td>
<td>$VZ_i = -0.0002 + \frac{1}{(1-0.0730B^2)(1+0.1615B^{26})} a_i$</td>
</tr>
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### Table 3: Coefficients and standard errors for predictors of female cohort life expectancy at age 60 in France (1816-1919) and England and Wales (1841-1919)

<table>
<thead>
<tr>
<th>Variable</th>
<th>France Model of bivariate association</th>
<th>France Model with covariates</th>
<th>England and Wales Model of bivariate association</th>
<th>England and Wales Model with covariates</th>
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</thead>
<tbody>
<tr>
<td>Constant</td>
<td>0.1408***</td>
<td>0.1347***</td>
<td>0.0967***</td>
<td>0.1054***</td>
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<tr>
<td>Cohort death rate age 0</td>
<td>-0.8920</td>
<td>1.1891</td>
<td>-5.4115**</td>
<td>1.8838</td>
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<td>Cohort death rate age 1-4</td>
<td>9.5157</td>
<td>11.3851</td>
<td>-14.2032</td>
<td>11.6961</td>
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<td>Cohort death rate age 10-14</td>
<td>-68.6985</td>
<td>64.9568</td>
<td>-147.4615</td>
<td>102.0756</td>
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<tr>
<td>Cohort death rate age 45-49</td>
<td>-141.0330**</td>
<td>47.3646</td>
<td>-202.8933***</td>
<td>58.6037</td>
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<td>Cohort death rate age 55-59</td>
<td>-133.2267***</td>
<td>25.6761</td>
<td>-191.3639***</td>
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<td>Box Jenkins parameters</td>
<td>φB = -0.3590***</td>
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<td>θB12 = 0.3564**</td>
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<td></td>
<td>φB10 = 0.4418***</td>
<td>0.0920</td>
<td>φB10 = 0.3385**</td>
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<tr>
<td></td>
<td>θB5 = 0.4051***</td>
<td>0.1018</td>
<td>θB5 = 0.4051***</td>
<td>0.1316</td>
</tr>
</tbody>
</table>

*p<0.05, 2-tailed test

**p<0.01, 2-tailed test

***p<0.001, 2-tailed test
<table>
<thead>
<tr>
<th>Variable</th>
<th>Model of bivariate association</th>
<th>Model with covariates</th>
<th>Model of bivariate association</th>
<th>Model with covariates</th>
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</thead>
<tbody>
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<td></td>
<td>Coefficient</td>
<td>Standard Error</td>
<td>Coefficient</td>
<td>Standard Error</td>
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<tr>
<td>Constant</td>
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<td>0.0464</td>
<td>0.1007*</td>
<td>0.0448</td>
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<td>Cohort death rate age 0</td>
<td>-0.0791</td>
<td>0.5957</td>
<td>-10.1492</td>
<td>6.7549</td>
</tr>
<tr>
<td>Cohort death rate age 1-4</td>
<td>14.0645</td>
<td>47.2611</td>
<td>2.3160</td>
<td>10.7743</td>
</tr>
<tr>
<td>Cohort death rate age 10-14</td>
<td>-7.6010</td>
<td>8.1314</td>
<td>-13.8859</td>
<td>8.9075</td>
</tr>
<tr>
<td>Box Jenkins parameters</td>
<td>( \phi B^2 = 0.4509^{***} )</td>
<td>0.0967</td>
<td>0.4600***</td>
<td>0.9866</td>
</tr>
</tbody>
</table>

* \( p<0.05 \), 2-tailed test  
** \( p<0.01 \), 2-tailed test  
*** \( p<0.001 \), 2-tailed test