Older women improve population fitness by reducing mortality at menarche: Evidence from Sweden

April M. Falconi, PhD*[1]
Ralph Catalano, PhD[2]

*Corresponding Author
April Falconi
1070 Arastradero Road
Palo Alto, CA 94304
afalconi@stanford.edu

1. Stanford University School of Medicine, 1070 Arastradero Road, Palo Alto, CA 94304
2. School of Public Health, University of California, Berkeley, 50 University Hall, Berkeley, CA 94720

ABSTRACT

The “Grandmother Hypothesis” posits that natural selection conserved longevity in females because older women increase their daughters’ reproductive fitness by helping reduce mortality among grandchildren. Although this theory has been previously discredited because that reduction appears small, we argue that collective breeding, when combined with the theory of disposable soma, suggests that the presence of older women may improve the fitness of populations through mechanisms beyond reduced child mortality. We use Swedish data, considered the most reliable pre-19th through 20th-century vital statistics, to test whether birth cohorts reaching menarcheal age exhibited lower mortality than expected from trends and other forms of autocorrelation when the population included relatively many older women. Results support our argument and suggest that the presence of older women in a population may enhance the fitness of younger women not only by reducing child mortality but also by reducing mortality attendant to depletion of the soma at or near menarche.

Keywords: Grandmother Hypothesis; Alloparenting; Menarche; Longevity; Time series analysis

1. Introduction

Evolutionary explanations for why women live longer than men include the argument that post-reproductive women help care for their grandchildren, thereby reducing infant and child mortality among descendants [1]. Natural selection would, therefore, conserve mutations that favor longevity in women. This "Grandmother Hypothesis" has received much scholarly attention with studies that typically compare mortality levels among infants and children with and without grandmothers present. A recent review reports that 69% of these comparisons find that the presence of a maternal grandmother reduces the probability of death among children [2].

Other research has focused on the types of support grandmothers offer that reduces mortality of infants and children. Older women provide not only instrumental support to younger members of their social networks (e.g., child rearing, intergenerational transfers of wealth), but also transmit knowledge and expertise. Researchers speculate that although humans store information over the lifespan, they acquire a disproportionate share during the period of juvenile dependency. Selection, therefore, may have favored juveniles whose social networks included older, more experienced persons able and willing to provide knowledge and advice [3]. Empirical findings also suggest that post-reproductive women increase their descendants' chances of survival by promoting peace and stability within kin circles and other social groups [3].

The grandmother hypothesis appears a special case of the "cooperative breeding hypothesis," which posits that helping others care for their young enhances inclusive fitness. This "alloparenting" is thought to have benefited both related and unrelated

individuals by allowing ancestral populations to expand into areas where climate and resources required relatively more adult work to provide food and shelter for infants and children [4].

Work by Lee (2014) similarly suggests that the benefits provided by intergenerational resource transfers (gifts of food or assistance from one generation to another) are large enough to benefit all members of a network. Analysis of intergenerational transfers has shown that sharing among kin and others raises fitness through smoothing of dependency burdens in the group. Social arrangements with broader sharing (i.e., pooling of resources within and outside a kin group) appear to have higher fitness relative to population groups with less sharing. Such groups reproduce and grow with greater success in the face of resource constraints and evolutionary competition [5].

Critics of the grandmother hypothesis contend that the fitness benefits accrued from grandmothering are not large enough to have natural selection conserve female longevity [6,7]. Associations between grandparental and child survival appear, moreover, too dependent on culture or society, as well as on the age and sex of children and the age and lineage of the grandmother to justify the claim of a general fitness benefit [8,9]

We argue that the grandmother hypothesis has been too narrowly tested to appreciate the full salutary effects of older, post-reproductive women in a population. Prior tests have neglected to account for the positive externalities that such women may impart to the population beyond their direct descendants. Indeed, mechanisms assumed or implied by the grandmother hypothesis (e.g., intergenerational transfers,

cooperative breeding, alloparenting) predict that birth cohorts whose critical developmental periods coincide with a population of relatively many older women will enjoy lower mortality than expected from trends and other forms of autocorrelation.

We further hypothesize that the salutary benefits conferred by older women extend beyond infants and young children who have been the focus of earlier work. Although "critical" by any definition, the first year—or even the first five years—of life do not exhaust critical developmental periods. Much literature argues, for example, that experiences during the emergence of reproductive biology affect health and longevity [10-12]. Theoretical underpinnings for such work (e.g., disposable soma theory) assume that normal human development sublimates the needs of the soma, or biology not directly involved in reproduction, to the emergence of reproductive systems [13,14]. The emergence of reproductive ability therefore induces a "critical period" during which exogenous stressors, or a lack of physical or psychological resources to cope with stressors, can disrupt somatic functioning. The sequelae of these disruptions could manifest in greater morbidity and, particularly before the emergence of modern medicine, greater mortality among those sublimating the soma to reproductive biology.

Combining the grandmother hypothesis with the theory of disposable soma leads to the suspicion that having relatively many older women in a population could reduce mortality among those transitioning to reproductive age. We test this suspicion using data from Sweden because its institutions provide the longest time-series of dependable vital statistics currently available. Our test begins with the earliest of these data (i.e., 1751) and ends in 1860. We end our test at 1860 when age at menarche began a continuing downward trend that makes estimates of association less efficient and when

the introduction of public health programming and medical advances begin to decrease mortality sufficiently to potentially mask signals of the effect of cooperative breeding [15,16].

2. Methods

2.1 Data

We obtained age- and sex-specific cohort mortality and life expectancy data from the Human Mortality Database [17]. This database includes life table and mortality data from countries with dependable and virtually complete vital statistics. Among countries with vital statistics that meet the standards for inclusion in the HMD, Sweden's start earliest at 1751. These early and continuous data provide an opportunity to test our hypothesis in a population like that assumed by the evolutionary argument at the base of the grandmother hypothesis (i.e., a population with a stable age at menarche over time and with mortality relatively unaffected, before 1860, by modern medicine and public health programming.)

2.2. Variables

We constructed our independent variable from the annual number of women aged 55 and older measured in 1,000's. Research indicates that the average age of menopause historically occurred between the ages of 49 and 52 in western societies and has not changed significantly over time [18,19]. We, therefore, reasoned that by age 55 most women had ceased allocating energetic resources toward their reproduction and their own young children.

We focus on mortality among females because the disposable soma literature uses, with relatively little controversy, menarche to define the emergence of reproductive capability among them but offers no equivalently objective, non-controversial definition for males. We, therefore, used the annual odds of deaths among females 15 to 19 years old as our dependent variable, given evidence suggesting an average age of menarche at 17 in western Europe through most of the 19th Century [20,21]. We transformed this variable to natural logarithms to reduce the effect of outliers on our estimations and to allow interpretation of our results in the familiar effect-on-odds metric. For reasons described below, we also included period life expectancy among women greater than 54 years old as a covariate.

2.3 Analyses

Dating to the seminal work of Galton [22], tests of association essentially measure the extent to which two variables move away from their statistically expected values in the same cases. "Cases" in our test include the 110 years (i.e., 1751 through 1860) characterized by the odds of death among 15 to 19-year-old Swedish females and the number of Swedish women aged 55 or older. Our test determines whether the observed likelihood of death differs from statistically expected values in the same years that the number of older women differs from its expected values. The grandmother hypothesis would predict an inverse association.

Statistical tests of association typically assume that the expected value of any variable is its mean. Variables measured over time, however, often violate this assumption because they exhibit “autocorrelation” in the form of secular trends, cycles,

or the tendency to remain elevated or depressed, or to oscillate, after high or low values. The statistically expected value of an observation in such series is not the mean of all observations but rather the value predicted by autocorrelation. Researchers dating to Fisher [23] have solved this problem by using time-series modeling to arrive at the expected values of autocorrelated series. We used the most mature and widely disseminated type of such modeling to arrive at the expected values of our data. The method, devised by Box and Jenkins [24], identifies which of a large family of mathematical expressions best describes measurements made serially in time or space. Metaphorically, the modeling procedure assumes that the measurements passed through an unobserved "filter" that imposed autocorrelation upon them. The procedure uses mathematical "signatures" to narrow the likely filters to a few and then applies estimates of "fit" to identify the most likely candidate. The differences between the values predicted by the model and the observed series approximate the values that passed through the filter. They meet the assumptions of traditional tests of association because they are independent of each other (i.e., exhibit no autocorrelation), their expected value equals their mean (i.e., 0), and they exhibit constant variability over time.

Like all observational tests, ours cannot rule out the possibility that a discovered association arises from other than our assumed mechanism. We structured our test to reduce the threat of the most intuitive rival hypothesis—that an association arises between our variables because exogenous shocks affect mortality among older and younger women. A pathogenic shock, unusually bad weather for example, could cause higher than expected death rates among females aged 15 to 19 and reduce the

population of older women below expected levels by causing higher than expected mortality among them. Unusually benign weather could, obversely, lower the death rate among both older and younger females and induce a spurious association. We reduce the likelihood of such “general mortality” effects by including period life expectancy among women aged 55 and older as a covariate in our test. Period life expectancy approximates the average age at death for persons at or above the criterion age, 55 in our case. Its variation over time gauges, among other circumstances, the dose of pathogens encountered by the population.

Our test proceeded through the following steps.

1. We regressed the natural logarithm of the odds of death among Swedish females aged 15 through 19 on period life expectancy of Swedish women age 55 and older over the 110 years beginning 1751 and ending 1860.
2. We used Box and Jenkins (1976) methods to detect and model autocorrelation in the residuals of the regression estimated in Step 1.
3. We used Box-Jenkins methods to detect and model autocorrelation in the annual number of Swedish women aged 55 and older for the same 110 years used in step 1. As expected, the number of older women trends upward over the test period reflecting, among other possible phenomena, increases in nativity over the test years and general improvements in health. The Box-Jenkins approach removes such trends by taking the first differences (i.e., value at time t subtracted from value at t+1) of the series and then detects and models remaining autocorrelation in the differenced series. The residuals of the model estimate the degree to which annual differences in the

population of older women exceeded or fell below values expected from autocorrelation.

4. We estimated the following test equation formed by adding, as a predictor variable, the residuals of the model estimated in Step 3 to the model developed in Step 2.

$$(1 - \emptyset B^{\mathrm{p}})\left({}^{P}/_{1-P}\right)_t^e = C + \omega_0 X_{1t} + \omega_1 X_{2t} + (1 - \theta B^{\mathrm{q}})\, a_t \quad [1]$$

$\left({}^{P}/_{1-P}\right)_t^e$ is the natural logarithm of the odds of death among 15 to 19-year-old females in year t. ∅ is the autoregressive parameter of the Box-Jenkins model. B^{p} is the "backshift operator" or value of $\left({}^{p}/_{1-p}\right)_t^e$ at year t-p or of a at year t-q. X_{1t} is the period life expectancy of Swedish women 55 and older in year t. X_{2t} is the residual of the best fitting Box-Jenkins model, estimated in step 3 above, of the number of Swedish women aged 55 years and older (in 1000's) in year t. θ is the moving average parameter of the Box-Jenkins model. a_t is the residual of the model at year t. The Grandmother Hypothesis predicts that ω_1 will be significantly less than 0 ($p<.05$; 2-tailed test).

3. Results

Table 1 shows the mean, standard deviation, and range of the time series used in our test. Figure 1, the results of the first two steps in the test, shows the observed and expected (i.e., from period life expectancy of women 55 and old and from autocorrelation) values of the natural logarithm of odds of death among a thousand 15

to 19-year-old females during the test period. The parameters of the model that yielded the predicted values were as follows with the same notation as in equation 1 above. All the estimated parameters exceeded twice their standard errors.

$$(1 - .3802B)\left({}^{P}/_{1-P}\right)_{t}^{e} = -1.6902 - .0915X_{1t} + (1 + .2549B^{4})\, a_t \quad [2]$$

Step 3 in our test, or estimating the expected (i.e., from autocorrelation) values of the number of women (in 1000's) aged 55 or older in Sweden during the test period, yielded the following Box-Jenkins model.

$$(1 - .6819B)\nabla Z_t = 1.2240 + (1 + .7214B)\, a_t \quad [3]$$

Z_t is the number of women 55 and older measured in 1000's. ∇ is the difference operator that indicates the series has been transformed to its first differences (i.e., values at time t subtracted from those at t-1) to model trend. All parameters exceeded twice their standard errors. Figure 2 shows the residuals from model 3 above (i.e., a_t), that we add to model 2 to form the test equation estimated in step 4.

As shown in Table 2, the result of step 4 in our test supports our theory. The negatively signed parameter (i.e., -0.0565; SE = 0.0296; $\underline{p} < 0.05$, single-tailed test) for the residuals of model 3 imply that deviations from the expected odds of death among women transitioning to reproductive age moved opposite deviations from the expected number of older women. The association survived adjustment for period life expectancy among women 55 and older. The finding cannot, therefore, arise spuriously from

exogenous shocks to health that affected mortality among both older women and those transitioning to reproductive age. Nor can the association arise from shared trends, cycles, or other forms of autocorrelation (e.g., regression to the mean).

Figure 3 plots the unexpected odds of death (i.e., the residuals of the model estimated in steps 1 and 2 above) among females transitioning to reproductive age over the unexpected number of women over 55 years of age (i.e., residuals of the model estimated in step 3 above). This scatter plot suggests that the inverse association we find in our test arises mostly from years when the number of older women available to invest in younger women fell well below expected. We, therefore, applied the methods of Chang, Tiao and Chen [25] to identify years that exhibited counts of older women below the lower bound of the 95% confidence interval of expected values derived in step 3, above. This method iteratively adjusts the Box-Jenkins model parameters as well as the confidence interval of the residuals to reflect inclusion of each outlier. The exercise identified eight years (i.e., 1763, 1772, 1773, 1774, 1800, 1809, 1827, and 1830) below the confidence interval. We specified a variable in which we scored each of these years with the difference between their observed and expected value and assigned 0 to other years (i.e., those with observed values within the 95% confidence interval of the model adjusted for outliers). We substituted this “low-outliers” variable for X_1 in our test equation and estimated the parameters again. As expected, the coefficient for this variable (-.1090, SE = 0.0510; $p < 0.01$, single-tailed test) was smaller than that for X_1 (-0.0565; SE = 0.0296). This finding suggests that, consistent with cooperative breeding, and, more specifically, the grandmother hypothesis, more

females transitioning to reproductive age died than expected when the number of older women available to invest in them fell significantly below expected levels.

We note that our specification of period life expectancy among women age 55 and older as a covariate makes our test conservative in that the association we found can arise only from the aging of unexpectedly large numbers of women into the population aged 55 and older. Any increase in that population due to unexpectedly reduced death rates could not have contributed to our discovered association.

We put the "dose response" of our discovered association in context in two ways. First, because we used the natural logarithm of the odds of death among a thousand females aged 15 to 19 as our dependent variable, the coefficient for the low-outliers variable (i.e., -0.1090) implies an approximate 10% increase in the odds when the number of older women unexpectedly declined by 1000 persons. Outlying unexpected declines ranged from -2.118 to -0.536 suggesting a range of increased odds of death of about 5 to 21% attributable to reduced investment by older women.

Second, we estimated change in R^2 attributable to significant unexpected declines in the number of older women. The "base" model estimated in the first two steps above accounted for 57% of the variance in the odds of death among women aged 15 to 19. Adding the low-outliers variable to the equation increased R^2 to 59.3%.

Because our interest in these phenomena arose from literature reporting an association between the presence of grandmothers and infant mortality, and because that and other work argues women invest more in parenting than do men, we specified an independent variable derived from the number of older women. We, however, know of no empirical reason to rule out that older men could also have invested in the

wellbeing of women transitioning to reproductive age. We, therefore, repeated our test but substituted low outliers of older men for low outliers of older women as the independent variable. Although we found a negatively signed coefficient (i.e., -0.0797), it did not exceed its standard error (i.e., 0.0867).

4. Discussion

Relatively small populations of older, post-reproductive women predict an increased odds of mortality among cohorts of females at age of menarche in historic Sweden. This inverse relationship suggests that older women contribute to the health of young women entering their reproductive lives. Although historical life table data do not allow identifying mechanisms that account for the observed relationship, these findings appear to support the "disposable soma" argument that the emergence of reproductive capability induces a window of vulnerability in which increased alloparental investment manifests in lower mortality.

Increases in life expectancy in post-demographic transition societies suggests that older and younger individuals enjoy more of a shared lifespan than they did historically [26]. The proportion of women 55 years or older in mid-18th Century Sweden, for example, represented approximately 15 percent of the female population (and belonged to a particularly select group of particularly hardy women), compared to 33 percent of the female population in 2015 [17]. Healthier and longer-lived women, combined with lower fertility rates, means that older women contribute more now than historically to intergenerational investments in children [27,28]. Evolution toward an increasingly “vertical family structure,” in Sweden implies that families more often are

comprised of multiple living generations, with a significant percentage of older relatives who directly invest the wellbeing of grandchildren [28]. Although modern medicine and public health programming may have reduced the effect of such investments on mortality among the young, grandparental investment has been shown to benefit children's mental health and academic achievement in modern populations [27]. Such investments may, therefore, improve the Darwinian fitness of those receiving them by improving success in mating markets.

This study makes three contributions to the existing literature. First, we offer a test over a time-scale and at a level of population aggregation implied by the fitness argument at the base of the cooperative breeding hypothesis in general and the grandmother hypothesis in particular. Second, this study expands prior work on the benefit of older women in a population to children's health by testing for associations with mortality among females transitioning to reproductive life. This period in the life course has been ignored in prior tests of the grandmother hypothesis, despite the rapid and dramatic developmental changes that occur in this narrow window of time. Third, our results suggest that earlier work (e.g., Kachel et al., 2011; Peccei, 2001) may underestimate the fitness benefits to youth of having older women in their communities [26].

The results of our test support, but hardly confirm, the suspicion that alloparenting by older women may have contributed to the conservation of longevity among females. Although repeating our approach elsewhere would seem a reasonable agendum in any research program aimed at further testing of the "grandmother hypothesis," we note that the lack of longitudinal life table data from pre-demographic

transition societies would likely deter such efforts. Gaining further leverage on the grandmother hypothesis from historical data will require applying different approaches, fixed effect panel designs for example, to relatively short time series from several societies.

References

[1] K. Hawkes, J. O'Connell, N. Blurton Jones, H. Alvarez, & E. Charnov, Grandmothering, menopause, and the evolution of human life histories, Proceedings of the National Academy of Sciences of the United States of America, 95 (1998) 1336-1339.

[2] R. Sear, & R. Mace, Who keeps children alive? A review of the effects of kin on child survival, Evolution and Human Behavior, 29 (2008) 1-18.

[3] L. Carstensen, & E. Lockenhoff, Aging, emotion, and evolution, Annals of the New York Academy of Sciences, 1000 (2003) 152-179.

[4] S. Hrdy, Evolutionary context of human development: The cooperative breeding model, in: C. Salmon, & T. Shackelford (Eds.), Family Relationships: An Evolutionary Perspective, Oxford University Press, Oxford, UK, 2007, pp. 39-67.

[5] R. Lee, Intergenerational transfers, social arrangements, life histories, and the elderly, in: M. Weinstein, & M. Lane (Eds.), Sociality, Hierarchy, Health: Comparative Biodemography: Papers from a Workshop, National Academies Press, Washington, DC, 2014, pp. 223-246.

[6] J. Peccei, A critique of the grandmother hypotheses: Old and new, American Journal of Human Biology, 13 (2001) 434-452.

[7] A. Kachel, L. Premo, & J.-J. Hublin, Grandmothering and natural selection, Proceedings of the Royal Society B, 278 (2011) 384-391.

[8] H. Euler, & B. Weitzel, Discriminative grandparental solicitude as reproductive strategy, Human Nature, 7 (1995) 39-59.

[9] B. Strassmann, & N. Kurapati, Are humans cooperative breeders? Most studies of natural fertility populations do not support the grandmother hypothesis, Behavioral and Brain Sciences, 33 (2010) 35-39.

[10] L. Hoyt, & A. Falconi, Puberty and perimenopause: Reproductive transitions and their implications for women's health, Social Science & Medicine, 132 (2015) 103-112.

[11] R. Romeo, & B. McEwen, Stress and the adolescent brain, Annals of the New York Academy of Sciences, 1094 (2006) 202-214.

[12] A. Falconi, A. Gemmill, R. Dahl, & R. Catalano, Adolescent experience predicts longevity: Evidence from historical epidemiology, Journal of Developmental Origins of Health and Disease, 5 (2014) 171-177.

[13] L. Harshman, & A. Zera, The cost of reproduction: The devil in the details, TRENDS in Endocrinology and Metabolism, 22 (2006) 80-86.

[14] T. Kirkwood, Evolution of ageing, Nature, 270 (1977) 301-304.

[15] K. Lynch, & J. Greenhouse, Risk factors for infant mortality in nineteenth-century Sweden, Population Studies, 48 (1994) 117-133.
[16] P. Ellison, Morbidity, mortality, and menarche, Human Biology, 53 (1981) 635-643.
[17] B.U. University of California, & M.P.I.f.D.R. (Germany), Human Mortality Database.
[18] C. Diers, Historical trends in the age at menarche and menopause, Psychological Reports, 34 (1974) 931-937.
[19] S.M. McKinlay, The Normal Menopause Transition: An Overview, Maturitas, 23 (1996) 137-145.
[20] A.-S. Parent, G. Teilmann, A. Juul, N. Skakkebaek, J. Toppari, & J.-P. Bourguinon, The timing of normal puberty and the age limits of secual precocity: Variations around the world, secular trends, and changes after migration, Endocrine Reviews, 24 (2000) 668-693.
[21] G. Backman, Die beschleunigte entwicklung der jugend, Cells Tissues Organs, 4 (1948) 421-480.
[22] F. Galton, Natural Inheritance, Macmillan and Co., London, 1899.
[23] R. Fisher, Studies in crop variation: An examination of the yield of dressed grain from Broadbalk, Journal of Agricultural Sciences, 11 (1921) 107-135.
[24] G. Box, & G. Jenkins, Time Series Analysis: Forecasting and Control, Holden-Day, San Francisco, 1976.
[25] I. Chang, G. Tiao, & C. Chen, Estimation of time series parameters in the presence of outliers, Technometrics, 30 (1988) 193-204.
[26] D. Coall, & R. Hertwig, Grandparental investment: Past, present, and future, Behavioral and Brain Sciences, 33 (2010) 1-59.
[27] D. Coall, & R. Hertwig, Grandparental investmnet: A relic of the past or a resource for the future?, Current DIrections in Psychological Science, 20 (2011) 93-98.
[28] T. Bengtsson, & R. Ohlsson, The demographic transition revised, in: T. Bengtsson (Ed.), Population, Economy, and Welfare in Sweden, Springer-Verlag, Berlin, 1994, pp. 13-36.

Figure 1. Observed and Expected (from autocorrelation and female period life expectancy at age 55) natural logs of the odds of death among Swedish women 15 through 19 for 110 years starting 1751 and ending 1860 (first year of expected values lost to modeling).

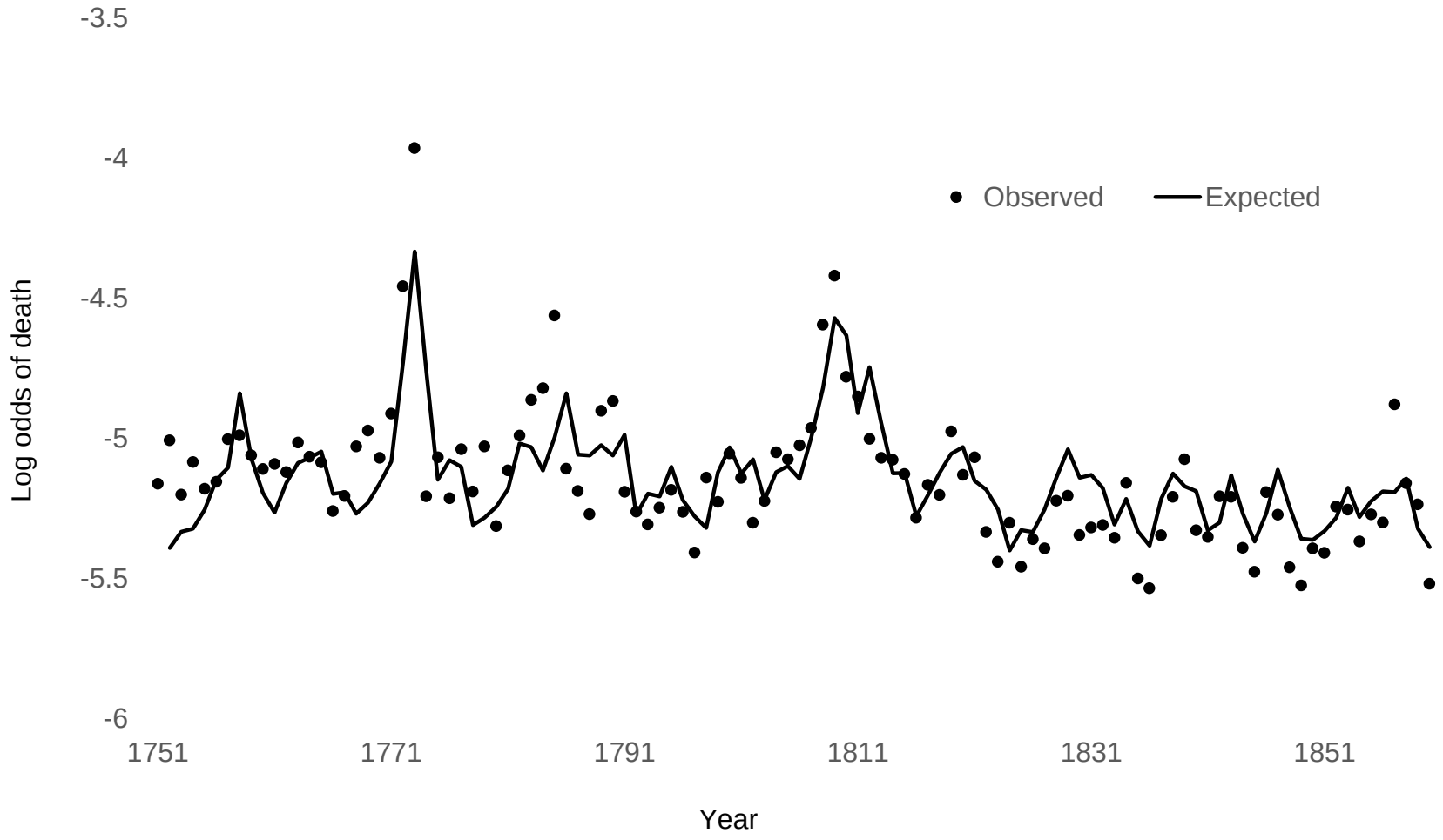

Figure 2. Residuals of best fitting Box-Jenkins model of the number of Swedish women aged 55 and over (in 1000's) for the years 1751 through 1860 (first 2 years lost to modeling).

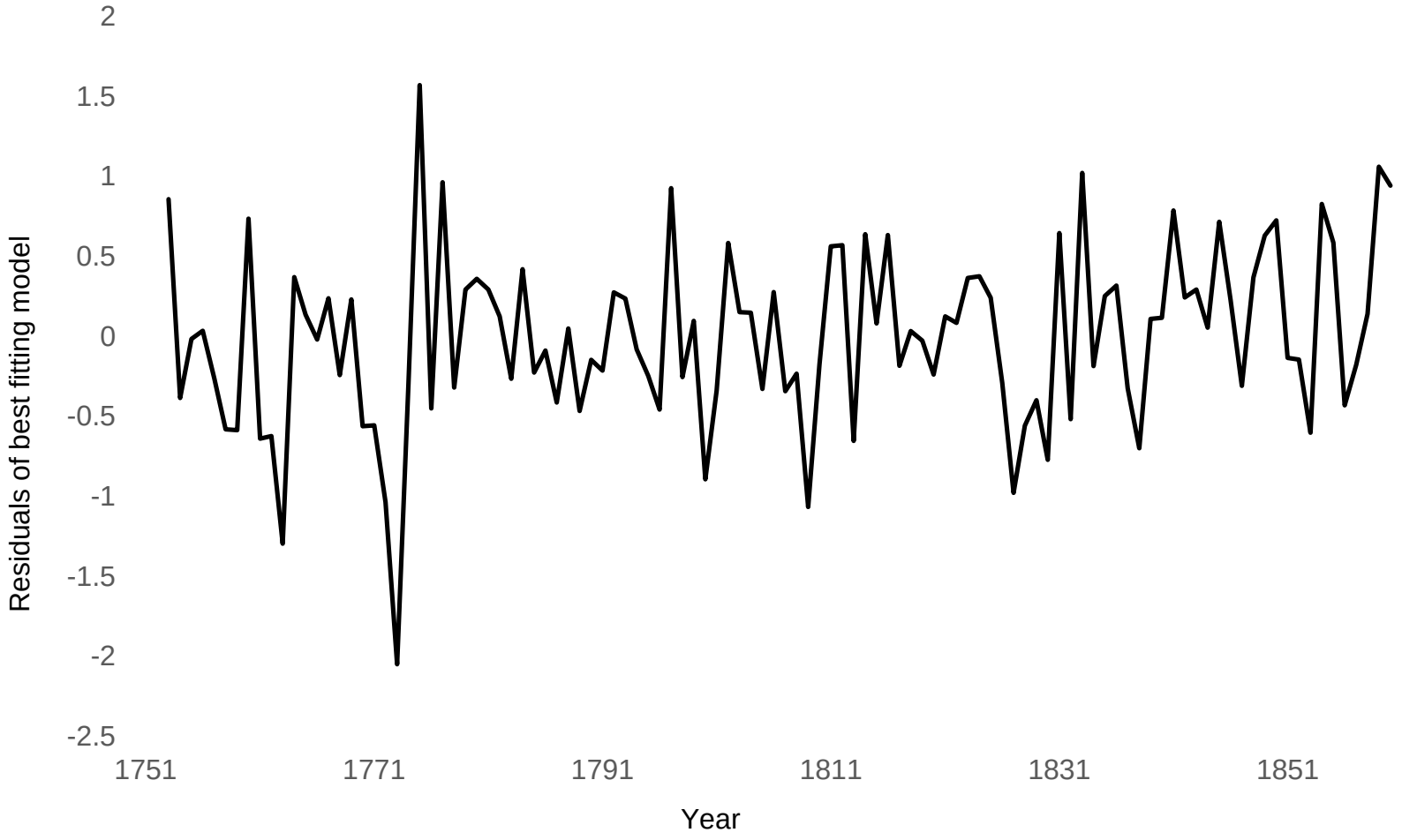

Figure 3. Natural logs of the odds (adjusted for autocorrelation and female period life expectancy at age 55) of death among women aged 15 through 19 plotted, with best fitting line, over number of women over 55 years old (in thousands and adjusted for autocorrelation).

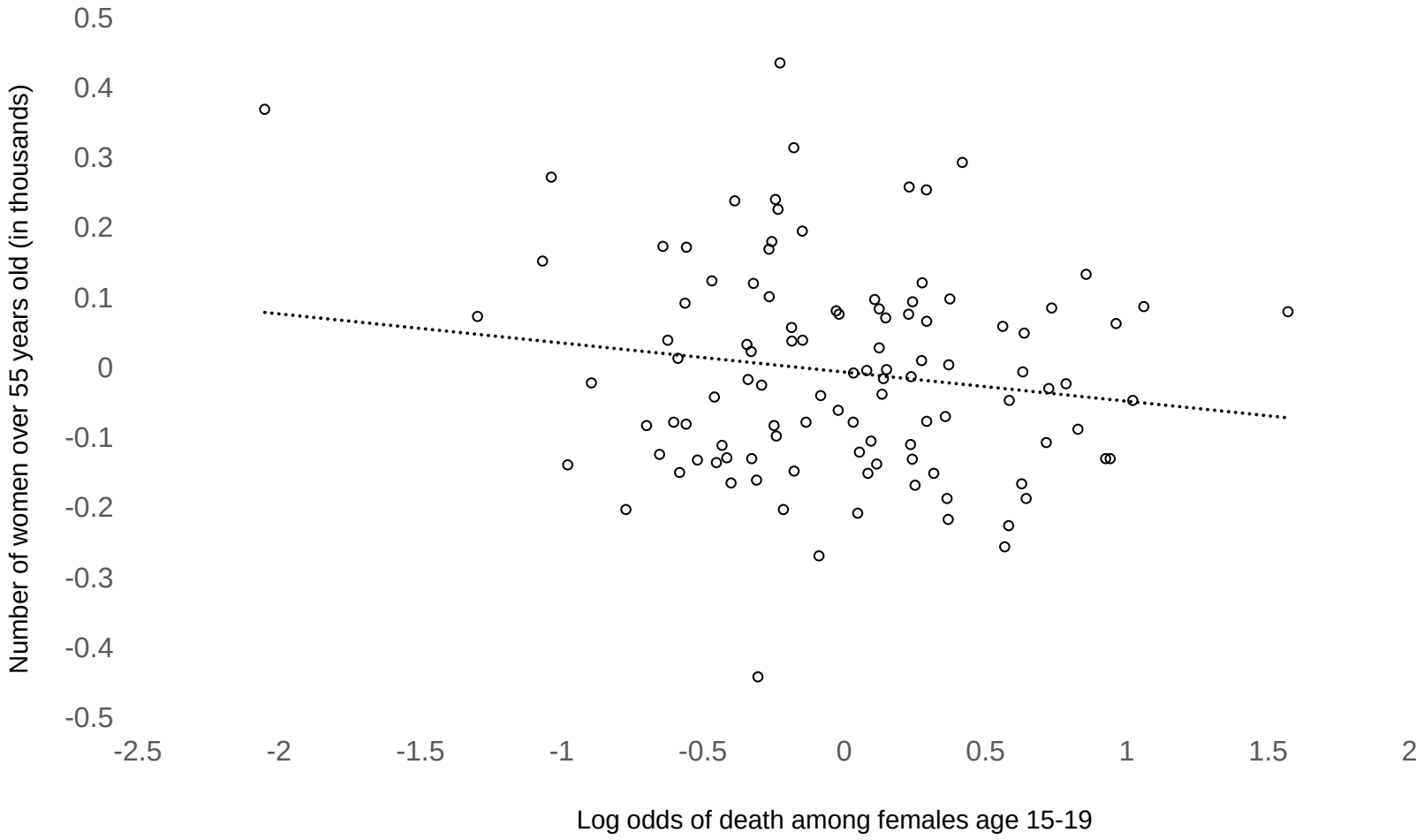

Table 1. Means, standard deviations, and range of Swedish time series (annual, 1751 through 1860) used in the analyses.

	Mean	Standard Deviation	Range
Odds of death females aged 15 to 19 years	0.0060	0.0019	0.0039 – 0.0190
Number of women aged at least 55 years	176,216	33,673	134,310 – 253,578
Number of men aged at least 55 years	107,684	20,111	74,621 – 153,552
Period life expectancy of women aged at least 55 years	16.3687	1.2111	11.0900 – 19.0100

Table 2. Estimated coefficients for equation predicting the natural logarithms of the annual (1751 – 1860) odds of death among Swedish women aged 15 through 19 years.

	Estimate	Standard Error
Constant	-1.9566**	0.3766
Period life expectance for women aged at least 55 years	-0.0875**	0.0144
Observed less expected (from autocorrelation) number of women aged at least 55 years	-0.0565*	0.0296
Moving average parameter at t-4	-0.2721**	0.0984
Autoregressive parameter at t-1	0.3422**	0.0697

*p<.05 (single-tailed test)

**p<.01 (single-tailed test)