A Time-Series Test of Diminished Entelechy in Birth Cohorts

Introduction

Individual-level studies report that adverse conditions during the postnatal period[1-3] predispose infants to increased morbidity and mortality in adulthood and old age. This literature implies what we refer to as the "diminished entelechy" hypothesis that birth cohorts subjected to relatively virulent environmental insults early in life do not realize their otherwise expected life span.

We know of five empirical tests related to the diminished entelechy hypothesis.[3-7] None have objective measures of the insults experienced by birth cohorts and all use infant mortality as a surrogate. The tests assume that a relatively high fraction of a cohort born in stressful times will die in the first year of life. Several authors report an intra-cohort association between infant mortality and older age mortality.[3-6] Pearson, however, observes an inverse association between infant mortality and subsequent death rates from ages one to five.[7] He infers a culling effect in which a cohort remaining after suffering high infant mortality may be smaller but hardier than expected.

In sum, these studies do not offer compelling evidence for or against the diminished entelechy hypothesis. All use death rates at various points in the life span and, therefore, say nothing about the effect of early insults on the entelechy, or realized fraction, of life expectancy. Death rates in stressed birth cohorts could fall below those otherwise expected in early life and rise above expected levels later in life, but the "net effect" of early insults remains unknown.

We report a direct test of the diminished entelechy hypothesis. More specifically, we measure the association between infant mortality (i.e., mortality up to one year of age) and life expectancy at age one in Sweden for male and female cohorts born between 1751 and 1912. We analyze males and females separately because temporal variation in life expectancy differs by gender.

Methods

We acquired annual cohort infant mortality rates (deaths from 0 to 1 year per 100 live births) and cohort life expectancy at age one for males and females born in Sweden from the Human Mortality Database website.[8] We analyzed data from 1751 to 1912 because data from earlier years are not available and completed cohort life expectancy for later years is not yet determined. We use data from Sweden because few, if any, other societies have kept vital statistics for as long a time.

We derived our independent variable by removing trends, cycles, and other forms of autocorrelation from annual infant mortality rates.[9] This approach, known as Auto-Regressive Integrated Moving Average (i.e. ARIMA) modeling[10], removes patterns from the independent variable before testing its effect on the dependent variable and has the added benefit of avoiding spurious associations due to shared trends and cycles. Consistent with the literature described above, we assume that the remaining values measure the degree to which a birth cohort suffered more or fewer life-threatening stressors during its first year of life than expected from

history (see Figures 1a-b). We constructed our dependent variable by applying the same techniques to completed cohort life expectancy at age one.

As noted above, the empirical literature often cited in support of the diminished entelechy hypothesis reports elevated mortality at various ages among populations presumably stressed in the first year of life. We chose life expectancy at age one as our dependent variable because it gauges the net effect of any unusually pathogenic or salutary influences on a cohort that has survived infancy.

Results

Table 1 shows the results in which we added the statistically unexpected values of the infant mortality variables to the best fitting ARIMA models of life expectancy at age one. The diminished entelechy hypothesis is supported for both males and females. The coefficient for males, for example, implies that Swedish men realized 2.3 fewer months of potential life for each one per cent increase over expected values in their birth cohort's infant mortality rate. Based on the diminished entelechy hypothesis, a range of 10 gained to 9 lost months of life would be attributed to the forces that affected infant mortality among men in a birth cohort.

Individual-level research finds an association between infant morbidity and mortality in older adulthood, with fewer sequelae occurring at earlier ages in the life-course. Building on this work and our observed findings, we performed an exploratory analysis to examine which age-specific mortality rates are perturbed by infant mortality. We constructed age-specific mortality as the dependent variable,

for males and females, for the following categories consistent with the literature[3]: 1 to 4, 5 to 19, 20 to 54, and 55 to 80 years (e.g. ${}_4q_1$, ${}_{15}q_5$, etc.). Next, we employed the ARIMA modeling strategy for eight separate tests (four age groups for each sex), removing autocorrelation in all dependent variables before inserting the residual values of infant mortality into the test equations.

The results in Table 2 indicate that, for both males and females, a statistically unexpected increase in infant mortality is associated with a subsequent increase in that cohort's mortality for the 1 to 4 and 5 to 19 year age groups. No association was observed in the adult and older adult age groups.

Discussion

Our results support the diminished entelechy hypothesis in that life expectancy at age one fell below the values expected from history in cohorts in which the infant mortality rate increased over its expected value. These findings suggest that suffering relatively virulent environmental insults during infancy reduces the subsequent lifespan of birth cohorts. We found no support for the culling effect set forth by Pearson.

Additional exploratory analyses suggest that adverse conditions during infancy predispose children and young adults to increased mortality, but that these sequelae do not persist into older ages. This finding awaits replication in historical cohorts as well as further development of life-course theory.

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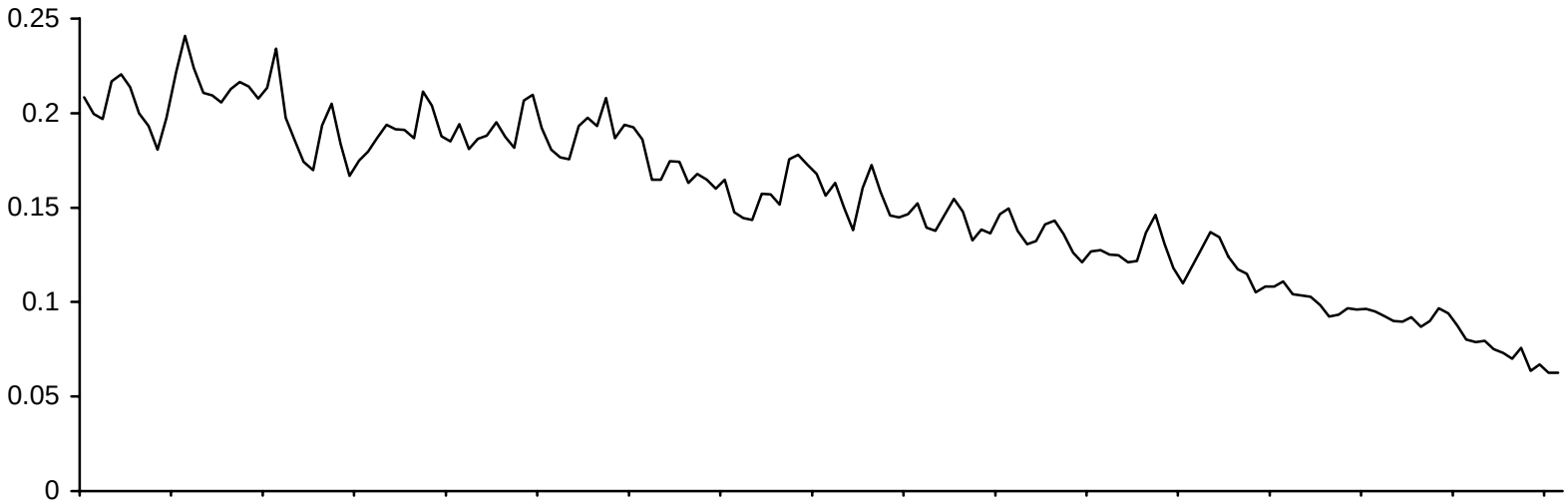
Figure 1a.



1b.

Figures 1(a) and (b). Illustration of ARIMA modeling, using female infant mortality in Sweden, 1751 to 1912, as an example. **(1a)** represents the unfiltered values of infant mortality (age 0 to 1) over the time period, taken directly from the Human Mortality Database. **(1b)** represents the residual, or unpatterned, values of infant mortality that were filtered by ARIMA modeling. The residuals are the statistically unexpected values of infant mortality not predicted from its own history. The unexpected component of infant mortality is then employed as an independent variable.

	Male Life Expectancy at age one	**Female Life Expectancy at age one**
Differencing	First Differences	First Differences
Constant	0.1543** (0.0382)	0.1671** (0.0461)
Residuals of Box Jenkins Models for Infant Mortality in Same Gender	-0.1906** (0.0458)	-0.1981** (0.0522)
Moving Average Parameters	B = 0.1057 (0.0784) B^{13} = 0.2081** (0.0789)	B = 0.0749 (0.0522) B^{13} = 0.2044* (0.0795)
Autoregressive Parameters	None	None

*p<.05; 2 sided test

**p<.01; 2 sided test

Table 1. Estimated equations for male and female birth cohort life expectancy at age one as a function of autocorrelation and infant mortality (n = 161 years beginning 1751).

	Age-Specific Cohort Mortality (q_x)			
	1 to 4 years	**5 to 19 years**	**20 to 54 years**	**55 to 80 years**
Residuals of Box Jenkins Models for Infant Mortality				
Males	0.3304** (0.0547)	0.2042** (0.0384)	0.0692 (0.0833)	0.0901 (0.0563)
Females	0.2813** (0.0617)	0.1374** (0.0372)	0.0420 (0.0364)	0.0932 (0.0642)

**p<.01; 2 sided test

Table 2. Estimation of the coefficients of the residuals of infant mortality (age 0 to 1) on cohort mortality over the life-course, by sex. Standard errors are in parentheses. N = 161 years beginning 1751. For clarity of presentation, fitted ARIMA parameters are omitted from the table.