Adolescent Experience Affects Longevity: Evidence from Historical Epidemiology

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ABSTRACT

Human development reportedly includes critical periods during which environmental stressors can affect traits that persist throughout life. However, controversy remains over which, if any, of these periods provides an opportunity for such stressors to affect health, as well as over whether these effects extend or shorten longevity in the population. The elaboration of reproductive biology and its behavioral sequelae in the age span 10 to 14 years suggests such a critical period, particularly among males. Consistent with that suggestion, we show an inverse association between cohort death rates at age 10 through 14 and cohort life expectancy at age 20 and 40 in Swedish and French male cohorts born in the 19th and early 20th centuries. This association survives controlling for cohort death rates during infancy, ages 1 through 4, 5 through 9, and 15 through 19. Our findings imply that better-informed and more strategic management of the stressors encountered by early adolescents may improve population health.

Much literature argues that stressful perinatal environments cause those who experience them to suffer excess morbidity later in life (i, ii, iii). The argument suggests that while the maternal and neonatal stress responses may increase the chances of surviving infancy, they also induce adaptations that increase risk of later life morbidity, such as cardiovascular disease (iv).

A smaller literature argues that adolescence may also present opportunities for threatening environments to affect developmental trajectories and, therefore, health later in life (v). During adolescence, significant psychobiological changes occur to accommodate maturation of reproductive biology and behavior (vi, vii). The period of early adolescence (approximately ages 10-14) encompasses a particularly important interval, when the onset of puberty results in a cascade of physical, emotional, cognitive, and social changes (7). Environmental threats during this time may, however, affect these changes and result in a tradeoff of longevity for reproductive success (viii, ix).

An evolutionary perspective suggests that a perceived threat among male adolescents may trigger physiological and behavioral responses that increase a male adolescent's likelihood of attaining reproductive success. Specifically, adolescent males may experience earlier onset of puberty, faster growth, and/or greater predisposition for high-risk behaviors (x). Changes in gonadal hormone levels during threatening situations affect adolescents' propensity to take risks, which appears to be motivated, at least in part, by adolescents' inclination to acquire social status (7). Such adaptations may have improved fitness over the course of human evolution, but may also come at

the expense of later life morbidity or premature mortality in contemporary populations (6).

The argument that a stressful adolescence will make fitness a higher priority than longevity has been supported by at least one empirical study. Falkstedt, Lundberg, Hemmingsson (xi) report that among males conscripted to military service in Sweden from 1949 to1951, socioeconomic position, as measured by parental occupation, at ages 9 to 11 elevated the risk of coronary heart disease (CHD) in middle age.

Limitations of the Falkstedt study include dependence on a highly select population (i.e., military conscripts) and the assumption that parental occupation measured environmental threats during adolescence. We contribute to the literature by testing the hypothesis that life span from age 20 among all males exposed to life-threatening stressors during early adolescence will fall below that among other males. We test the hypothesis among males ages 10-14 born in Sweden from 1861 to 1913 as well as among males born in France between 1816 and 1913. We control for trends, cycles, and other forms of autocorrelation in life span from age 20 as well as for exposure to life threatening stressors at infancy, childhood, and late adolescence.

RESULTS

Our findings support the suggestion that adaptation to threatening environments during adolescence predicts early mortality among males in countries representing the extremes in societal disruption by war and violence. More specifically, we find that cohort life expectancy at age 20 among French males born from 1816 through 1913, and among Swedish males born from 1861 through 1913, vary inversely with cohort

death rates at ages 10 through 14. The association remains significant when predictors in the equations include death rates in infancy, as well as in ages 1 through 4, 5 through 9, and 15 through 19. Indeed, among the death rate groupings, those from age 10 through 14 yield the largest dose response.

Table 1 shows the results our analyses. As described in the Methods section, we removed the effect of outlying values in the dependent variable on the estimated coefficients and their standard errors. We show standard errors to give a sense of the similarity of coefficients over the birth cohorts but remind readers that we have not sampled births for our study. Our data include all persons recorded as born in France from 1816 through 1913 and in Sweden from 1861 through 1913. Our coefficients, therefore, equal, rather than estimate, the associations we test.

Male cohort life expectancy at age 20 for both France and Sweden exhibited autocorrelation in the form of upward trends. In addition, the French series exhibited a one year "memory" of high and low values and the Swedish series exhibited an "echo" of such values at 5 years. The Swedish cohort life expectancy data exhibited no outliers. As anticipated (due to France's comparatively tumultuous history), the French civilian cohort life expectancy at age 20 exhibited outliers. These appeared for cohorts born in 1860, 1888, 1892, 1896, 1998, and 1899. We offer no explanation of the autocorrelation or outliers we detected because doing so would amount to *post hoc* speculation.

We repeated our test for the entire French population rather than just civilians. Results remained essentially the same although, as expected given military casualties, more outliers appeared.

We also repeated our tests for Sweden and France with cohort life expectancy at age 40 years substituted for that at age 20. We did so to determine if at least some of the excess later life mortality associated with stressful adolescence appears after peak fertility. We found life expectancy at age 40, like that at 20, inversely related with cohort death rates at age 10 through 14. The association remains significant when predictors in the equations include death rates in infancy, as well as in ages 1 through 4, 5 through 9, and 15 through 19.

To help put our findings in context, we transformed the continuous variable for adolescent death rates into a binary score of 1 for cohorts with greater than expected death rates during age 10 to 14 and 0 otherwise. We then performed steps 5 and 6 or our analyses again. The coefficient for the binary variable equals average life years lost after age 20 for male birth cohorts subjected to higher than expected threats to survival in adolescence. The coefficient for France (i.e., -.1028) suggests that males from cohorts highly threatened in adolescence lost about 1.23 months of life (i.e., .1028 X 12). Over the 98 years of this study (i.e., 1816 to 1913) cohort life expectancy at birth for French males at birth increased 10.49 years (i.e., from 39.21 to 49.70 years) or an average of 1.28 months a year. Males in our threatened adolescent cohorts therefore lost almost as much life as their gender gained each year from the modernization of French society. Applying this logic to Sweden yields the estimate that men from birth cohorts unusually threatened in adolescence lost more life after age 20 (i.e., 1.73 months) than modernization conferred on them each year (i.e., 1.7 months) over the test period.

DISCUSSION

Our findings support the argument that early adolescence is a critical developmental period among males in that male birth cohorts exposed to life threatening stressors during adolescence ultimately lived shorter lives compared to cohorts less stressed in adolescence. Population stressors experienced during ages 10 through 14 appear moreover, to decrease life span more than those experienced during infancy, ages 1 through 4, 5 through 9, and 15 through 19.

Adolescent plasticity, or the ability to change physiology and behavior in response to environmental stressors, allowed some French and Swedish males to survive and reproduce while others died (xii, xiii). Our findings demonstrate, however, that adaptations triggered during adolescence in stressed cohorts came at the expense of reduced life span both at age 20 and at age 40.

The literature concerned with the effects of early life stressors on later life health includes the argument that high levels of early life mortality may "cull" cohorts of less robust members and leave survivors who, on average, live longer than those in other cohorts (xiv). While research has provided support for such "culling" *in utero* (xv), our findings suggest that it does not apply to male adolescents.

Other studies suggest a narrower form of “culling” in which low socioecononomic status disposes children and adolescents to elevate risk for morbidity and premature mortality throughout their lives, independent of adult socioeconomic position (xvi, xvii, xviii, xix). While our data did not allow us to divide our birth cohorts into socioeconomic groups, theory would suggest an interaction effect in which males with low socioeconomic position would be more likely to die as adolescents during stressful

times than those with higher status males. The survivors would, therefore, presumably represent the hardier portion of the population (22). Our results suggest that such selection did not occur in Sweden or France, as stressed cohorts experienced higher levels of mortality relative to non-stressed cohorts even at age 40. This finding suggests that high doses of ambient threat during adolescence may diminish the health benefits conferred by high socioeconomic position.

We cannot know which mechanism or mechanisms induced premature mortality in our relatively stressed cohorts. Other studies, however, have demonstrated that stressful social circumstances during youth predict uptake of behavioral risk factors for disease, such as smoking and high BMI (11). These findings, combined with the theory noted earlier (7), suggest that the biology of risk taking induced by a stressed adolescence probably contributed to shortened life expectancy in our cohorts.

The shorter and temporally more variable cohort life span of males compared to females may have distracted the field from the fact that the underlying theory does not exclude effects on women (xx). Female adolescents also experience somatic adaptations in response to environmental threats, although their response appears more physiological than behavioral (10, xxi). Evidence suggests an association between environmental threats, in the form high extrinsic mortality (i.e., risk of death not conditional on an individual's behavior), and delayed puberty and timing of menarche (xxii). Theory predicts that this delay occurs during stressful times so that females may preserve somatic resources either for self-preservation or in expectation of better times in the future (28, xxiii). The implications of this theory for longevity remain unexplored.

The current study contributes to the existing literature in two ways. First, we use empirical evidence to demonstrate that the period of early adolescence, much like the perinatal period, is a critical period for male development. Second, our findings suggest that cohort effects can significantly impact sex-specific mortality trends well into adulthood. Population stressors that affect male adolescents should be taken into account when forecasting or explaining their adult mortality trends. Our results suggest, moreover, that current emphases on early life interventions may be less informed than intuition suggests. Greater resources and attention may need to be devoted to adolescent development given that it also presents as great an opportunity for population stressors to adversely affect human health.

METHODS

Variables and Data We used sex-specific cohort life expectancy at age 20 as our dependent variable. Cohort life expectancy measures remaining years of life at each age in annual birth cohorts. By axiom, demographers can calculate cohort life expectancy only among birth cohorts whose members have all, or nearly all, died. The time series described below, therefore, ends with cohorts born in 1913.

Few societies have kept reliable vital statistics from which we can calculate cohort life expectancy over long periods of time. Two countries that have kept reliable vital registration data, Sweden and France, differ greatly in the frequency of societal disruption by war—a phenomenon that could, by inducing high period mortality among young males, obscure other causes of differences in life span among birth cohorts. Sweden, which has kept reliable vital statistics since 1861, provides an opportunity to

test our hypothesis in society with relatively few wars. France, which has reliable data dating to 1816, allows a test in a society with a history of more hostilities. Convergent results from these extreme cases would offer strong, if not compelling, evidence for adolescence as a critical period.

The literature arguing for critical developmental periods assumes that populations exposed to unusual threats to health at ages specified *a priori* will suffer earlier mortality than other populations. Following this literature, we define the period of early adolescence as age 10 through 14 (10, xxiv, xxv). We use age- and sex-specific mortality rates as our independent and control variables. More specifically, we use the male infant mortality rate as well as male mortality rates from ages 1 through 4, 5 through 9, 10 through 14, and 14 through 19 as predictors of male cohort life expectancy at age 20. Results would support the argument that adversity in early adolescence affects later life health if mortality rates in the age group 10 through 14 inversely predict cohort life expectancy at age 20, controlling for mortality in the other four age groupings as well as for other confounders described below.

We obtained age- and sex-specific cohort life expectancy and death rates for Sweden and France from the Human Mortality Database (xxvi). This source archives life table data that meet quality standards agreed among demographers and other population researchers. The database includes data for the total Swedish population and both the civilian and total French population. We used the civilian data for our primary test although, as described below, we also replicated our results with the entire French population.

Analyses Our test turns on whether cohort life expectancy at age 20 falls below its statistically expected value in cohorts that exhibited mortality rates at ages 10 to 14 higher than their statistically expected value. Researchers typically assume that the statistically expected value of any variable is its mean. Cohort life expectancy, however, exhibits trends and the tendency to remain elevated or depressed or to oscillate after high or low values. These patterns, referred to as "autocorrelation," complicate observational tests because the expected value of an autocorrelated series is not its mean.

Researchers dating at least to Fisher's 1920 study of crop variation have solved the autocorrelation problem by the purely empirical approach of "decomposing" time series into temporally predictable and residual components (xxvii). This approach removes patterns from the dependent variable before measuring association with independent variables and has the added benefit of avoiding spurious associations due to shared autocorrelation including trends.

We implemented the Fisher approach through the following steps recommended in the literature (15).

1. Consistent with theory and our hypothesis, we make our test male-specific by regressing Swedish and French male cohort life expectancy at age 20 on female cohort life expectancy at age 20. This step controls for phenomena that affect life expectancy in both sexes (e.g., improved maternal nutrition, rising standards of living, better medical care).
2. We used the strategy developed by Box and Jenkins (xxviii) to model autocorrelation in the residuals of the regressions estimated in step 1 (i.e., male-

specific autocorrelation in cohort life expectancy at age 20). The strategy, Auto Regressive, Integrated, Moving Average (i.e., ARIMA) modeling, identifies which of a very 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" (e.g., residual mean square, Ljung-Box Q statistic) 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.

3. We applied the Box and Jenkins routines described above to the sex-specific infant mortality rates as well as to mortality rates at age 1 through 4, 5 through 9, 10 through 14, and 15 through 19.
4. We specified the following test equation by adding the residuals of the 5 age specific mortality series to the equation resulting from step 1.

$$\nabla Y_t = C + \omega_1 \nabla X_{1t} + \omega X_{2t} + \ldots \omega_6 X_{6t} + \frac{\left(1 - \theta_1 B - \theta_2 B^2 - \ldots - \theta B^q\right)}{\left(1 - \phi_1 B - \phi_2 B^2 - \ldots - \phi B^p\right)} a_t \quad [1]$$

∇ is the difference operator that indicates a series was differenced (i.e., values at t subtracted from values at t-1) to remove secular trends. Y_t is life expectancy at age 20 in Sweden or France for males in the cohort born in year t. C is a constant. X_{1t} is life expectancy at age 20 in for Swedish or French females in the cohort born in year t. X_{2t} through X_{6t} are the statistically unexpected components of male mortality rates at infancy, age 1 through 4, age 5 through 9, age 10 through 14, and age 15 through 19 for the cohort born in year t. ω_1 through ω_6 are the estimated coefficients. B^n is the "backshift operator" that yields the value of the life expectancy variable at year n. θ is the moving average parameter. ϕ is the autoregressive parameter. B^p and B^q are backshift operators that yield the value of a at year t-p for autoregressive and t-q for moving average patterns respectively. a_t is the error term for year t.

5. We estimated equation 1 and measured the association between the error terms of the equation and the infant mortality variable to ensure they were not related.

We used the methods of Chang et al. (xxix) to correct the estimations for outlying sequences of values in the cohort life expectancy variable that could reflect the effect of wars and other "shocks" to male longevity after age 20. The correction detects and adjusts for 4 types of outlying sequences. The first are simple single-year “spikes,” which suggest unusual single cohort effects. The second include temporary changes that begin with an outlying value and then regress back over several years to the level expected before the first outlier in the sequence. The third and fourth include sequences that begin with an outlying value but do not decay before the end of the

series or return to expected values abruptly without significant decay between the first and last outlier in the sequence.

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Table 1. Coefficients and standard errors for predictors of cohort life expectancy in France (1816 through 1913) and Sweden (1861 through 1913).

	France		Sweden	
	Coefficient	Standard Error	Coefficient	Standard Error
Constant	-.0421	.0301	-.0329	.0480
Cohort life expectancy for women at age 20	.6412*	.0391	.5906*	.1450
Cohort infant mortality rate	-2.8925*	1.2609	1.5006	2.9403
Cohort death rate age 1-4	-29.1378*	14.8784	-78.7937*	14.3814
Cohort death rate age 5-9	-23.6419	65.2271	-156.4667*	51.1328
Cohort death rate age 10-14	-343.5743*	100.6703	-536.9148*	124.6186
Cohort death rate at age 15-19	-.5177	48.4048	-179.0033*	68.6842
ARIMA parameters				
Moving average at lag 1	-.6697*	.0978		
Moving average at lag 5			-.5497*	.1736

p < .01; 1-tailed test

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