

Title:

The role of inherited factors for longevity: southern Sweden 1813-2009.

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Short abstract.

We estimate a model of overall mortality among married persons aged 50 years and above taking genetic as well as socioeconomic and environmental factors in succeeding generations into account. We consider whether these factors have direct or indirect effects on health. The demographic and economic individual level data come from the Scania Economic Demographic Database linked with central register data after 1968. To these, local grain prices, as an indicator of food costs, and the local infant mortality rate, as an indicator of the disease load, have been added. Preliminary, we find that age of death of the mother and the father have persistent impacts on their adult children's overall mortality regardless of sex, even after controlling for socioeconomic and environmental factors over the life course. In addition, we find strong birth cohort effects and effects of the disease load in the first year of life.

Extended abstract

The idea that inheritance played a part in human longevity dates at least back to the mid-nineteenth century (Smith and Griscom 1869; see Cohen 1964). Since then, scientists have tried to disentangle to what extent longevity is determined by genetic and environmental factors respectively (Pearl 1931; Cohen 1964; Finch and Tanzi 1997). Genetic factors, transferred from one generation to the next and traceable over many generations, have been linked to a variety of specific diseases and also to overall longevity. The latter is, however, more difficult to evaluate. First, it is difficult to identify genes that give its bearers long life. Second, relatives often share not only genetic but also environmental factors. For this reason, we expect that the life span of family members would be more strongly correlated than if genes were the only common denominator.

Family studies show, however, that the correlation in longevity between parents and children is rather low (Pearl 1931; Cohen 1964; Wyshak 1978; Finch and Tanzi 1997). In other words, transfer of longevity from parents to their children, even with genetic and environmental factors taken together, is weak. The correlation of longevity among siblings is somewhat stronger (Cohen 1964; Wyshak 1978). Some family studies suggest a stronger maternal than paternal link (Abbott et al. 1974), others do not (Wyshak 1978). Twin studies based on Nordic registers, undertaken to estimate the genetic component separately from shared environmental components as well as non-shared components, show that up to one third of the variation in life-span is caused by shared factors, and 25 per cent by genetic factors alone (Ljungquist, Berg, and Steen 1995; Christensen 2006).

The methodological approach used in most studies in trying to disentangle genetic from inherited environmental factors has been foremost to compare parent-offspring, siblings, and twins. Some twin studies also allow for differentiation between monozygotic and dizygotic twins. The reason for comparing twins, siblings, and parents with children, is to control for environmental, cultural, and social factors (see Wyshak 1978, 319–20). The studies are often restricted to persons who married at least once, which means that factors causing infant and child deaths are eliminated. The results from such studies do not, however, answer the question of how important family-shared factors are to total lifespan, but rather shows the effect on a certain period of life, i.e., in old age. The question is to what extent the comparisons of twins, siblings, and parents-children actually serve the purpose of controlling for non-hereditary factors. Even in a society with a considerable degree of social mobility, family members might still share the same environment throughout their lives, regardless of whether their mobility is stable or changing. The assumption that environmental factors to a large extent can be controlled for by comparing family members is basically an adaptation to the data. Few datasets on multiple generations contain information about occupation, landholding, or other indicators of income and wealth. What problems might then occur from the assumption that environmental factors largely are controlled for by comparing family members if individual and shared environmental factors are not explicitly included in the analyses?

In a society with a high degree of social mobility, not only in general but with large differences in social mobility between family members, it is easier to identify the role of inherited factors. It is likely that the achievements of individuals, relatively speaking, are more important for longevity than in a setting where social mobility is low and where family-shared factors consequently are less important. Thus, the effect of family-shared factors on longevity is partly determined by the extent to which family members share the same situation in adult life. Differences in results between various studies may therefore be a result of differences in social mobility. The problem is that the concept of family-shared factors in some studies de facto only includes genetic and childhood factors, whereas in others they include the entire life of the offspring. Our approach is more precise in defining family-shared factors and takes environmental factors explicitly into account.

Environmental factors, whether shared with other family members or not, influence the health of a person from the fetal stage throughout life. Factors that influence the development of cells and organs are called early-life factors. The speed of cellular development is fast during gestation, infancy, and childhood and then gradually declines until ages around 20 to 30 years when it almost stops. Later-life factors consequently affect us after our cells and organs are fully developed. Some factors cause only a temporary retardation of health, while others cause life-long health problems whether manifested immediately or with delay. It is therefore useful to distinguish between family factors shared early in life and those that potentially are shared later in life depending on differences in social mobility between siblings.

Epidemiologists and demographers showed interest in early-life factors, cohort factors, already in the 1920s and 30s when trying to explain the great mortality decline (Kermack et al. 1934). While period factors came into focus from the 1950s onwards, cohort factors have in recent years gained renewed interest (Barker 1994; Bygren, Edvinsson, and Broström 2000; Elo and Preston 1992; Finch and Crimmins 2004; Fogel 1994; Fridlitzius 1989; Kuh and Ben-Schlomo 1997; Preston, Hill, and Drenstedt 1998). Fogel (1994) has proposed several plausible causal mechanisms that connect malnutrition in utero and during early life to chronic diseases in later life. These propositions have

also been supported by the work of Barker (1994, 1995), who suggested that the preconditions for coronary heart disease, hypertension, stroke, diabetes, and chronic thyroiditis are initiated in utero without becoming clinically manifest until much later in life. Bygren et al. (2000) found that changes in food availability of mothers during pregnancy affected sudden death from cerebro- and cardiovascular disease in adult offspring.

The first years after birth are also important for mortality in later life. In two recent essays of the same areas as we are analyzing here, Bengtsson and Lindström (2000, 2003) show that the disease load experienced during the birth year, measured as infant mortality rate in the local area, had a significant influence on old-age mortality, particularly in airborne infectious diseases. Years with very high infant mortality, due to outbreaks of smallpox or whooping cough, had a strong impact, while modest changes had almost no impact at all. The causal relationship between cellular development during early childhood and mortality in old age has been supported by medical research (Liuba 2003; see Bengtsson and Lindström 2003, for further references).

Later-life factors, such as diet and working conditions, and in particular life-style factors (smoking, etc), have a strong impact on the lifespan. Several of these factors are correlated with socio-economic position. The importance of these factors seems to increase in importance over time (Bengtsson and Dribe 2011).

The first question is whether there is an association between age of death of parents and offspring. The second is whether potential impacts remain after controlling for environmental factors throughout the life-course. The third question is whether potentially transferred factors influencing longevity stem from shared genetic or shared environmental components. Here it is important to distinguish between environmental factors present early in life and during adulthood. The fourth and final question is whether factors affecting the father or the mother in their childhood will have an impact in health of their offspring. We will focus on the parent-offspring link and comparisons between siblings.

We analyze mortality among all married or previously married persons above the age of 50 years that lived in any of five parishes in rural Sweden between 1813 and 2009 by taking a life-course approach. Longitudinal demographic data on individuals and household socioeconomic data for parents and their offspring have been combined with community data on food costs and disease load. The data used come from the Scania Economic Demographic Database, Statistics Sweden, the National Board of Health and Welfare, and the War Archive and covers five rural parishes in the southernmost part of Sweden (from 1968 onwards the population is traced all over Sweden).

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