

Chapter 7

Mortality Forecasts. Comments on How to Improve Existing Models – An Epidemiologist’s Perspective



Kaare Christensen

From an epidemiologist’s perspective, one way to improve mortality forecasts is to gain insight into the causes and predictors of mortality. If we know the “risk profile” of the current cohorts compared to the previous cohorts, then our forecasts may improve.

If we also include genetics in our perspective, the initial task will be to quantify the contribution of genes and environment to lifespan. Therefore, the first question will be, “Are the lifespans of relatives correlated?” and if so, “Is the correlation due to a shared environment or to shared genes?” It is important to pose these questions initially because the answers determine whether it is worthwhile to seek the causes and predictors of mortality in the environment as well as in the genetic make-up.

7.1 Are the Lifespans of Relatives Correlated?

Traditional family studies suggest a correlation in lifespan within families. However, studies have generally found only small correlations in lifespan between parents and offspring (0.01–0.15) (Pearl 1931; Cohen 1964; Wyshak 1978), whereas correlations between siblings tend to be higher (0.15–0.35) (Cohen 1964; Wyshak 1978). Heritability estimates based on regression analysis were in the range of 0.10–0.33 for parents-offspring and 0.33–0.41 for siblings, constantly over a period of 300 years (Meyer 1991), but these estimates include both genetic factors and shared environmental factors. Some family studies have found a stronger maternal than paternal effect (Abbott et al. 1974), but not all (Wyshak 1978). The lower correlation found for parents and offspring than for siblings, suggests that genetic non-additivity

K. Christensen (✉)

Institute of Public Health, University of Southern Denmark, Odense, Denmark
e-mail: KChristensen@health.sdu.dk

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(genetic effects due to gene interaction which are not passed from one generation to the next) is present. However, it may also reflect a higher degree of shared environment among siblings than among parents and offspring; the latter constitute two generations living under very different conditions.

7.2 The Relative Effects of Genetic and Environmental Factors on Lifespan

Twin studies are designed to separate the effects of additive and non-additive genetic factors, as well as shared and non-shared environmental factors. However, most of the early twin studies had methodological problems due to left-truncation of the cohorts included, selection bias, lack of zygosity diagnosis, or heavy right-censoring. Carmelli and Andersen (1981) included a sample of 2242 Mormon pairs of twins born 1800–1899 in which both twins had died, a criterion met by 60% of the original sample. Wyshak (1978) followed 972 Mormon twin-pairs (possibly included in the study of Carmelli and Andersen) until death. Unfortunately, since both studies lacked zygosity diagnosis, heritability estimates could not be provided. However, similarity in length of life was found. The similarity was more pronounced for twins of the same sex (including both MZ and DZ twins) than for twins of the opposite sex (always DZ twins), suggesting genetic influences on lifespan. Jarvik et al. (1960) followed a sample of 853 pairs of twins for 12 years; the sample included only pairs with at least one twin surviving to age 60. At the end of the follow-up period, both twins had died in only 35% of the pairs. The mean intra-pair difference in lifespan was found to be higher for DZ than in MZ twins, suggesting genetic influences on lifespan. Hrubec and Neel (1981) followed a sample of 31,848 male twin veterans born 1917–27 for 30 years to ages 51–61. Around 10% were deceased at the time of analysis. To avoid censoring problems, longevity was analyzed as a categorical variable (dead/alive). In this study, the heritability of “liability” to die was estimated to be 0.5.

The first non-censored and population-based twin study that could provide an estimate of the magnitude of genetic influences on lifespan was conducted by McGue et al. (1993). It covered 600 Danish pairs of twins born 1870–1880. Using path analysis, a heritability of 0.22 was found, with genetic influences being mainly non-additive. Later this study was expanded by Herskind et al. (1996) to include more than 2800 twin-pairs with known zygosity born 1870–1900. These cohorts were followed from age 15 to death. The study confirmed that approximately a quarter of the variation in lifespan in this population could be attributed to non-additive genetic factors, while the remaining three-quarters were due to non-shared environmental factors.

Ljungquist et al. (1998) studied the 1886–1900 Swedish cohorts of twins and concluded that around a third at most of the variance in longevity is attributable to genetic factors.

Hence, it seems to be a rather consistent finding in the Nordic countries that approximately 25% of the variation in lifespan is caused by genetic differences. It is interesting that animal studies have revealed similar estimates for a number of species not living in the wild (Curtsinger et al. 1995; Finch and Tanzi 1997).

Hence, the conclusion from these studies is that it is worthwhile to seek the causes and predictors of mortality in the environment as well as in the genetic make-up. However, the results from family studies with low correlations between family members suggest an absence of common genes with a substantial impact on lifespan.

7.3 Prediction of Mortality

From a forecast perspective, which focuses on reduction of mortality (rather than on sudden increases in mortality due to new diseases, war, etc.), there is little interest in estimating survival at younger ages, when the room for improvement is very limited. What is important for future mortality trajectories is mortality among elderly people (Vaupel et al. 1998).

A number of risk factors seem to lose their importance with age, probably because of heterogeneity and selection, *e.g.*, smoking, obesity, diseases and SES (socio-economic status). However, this does not mean that the survival rate of the elderly, including the very oldest, cannot be raised. In the latter category, the fraction dying every year is high (for example, 1/4 – 1/3 among nonagenarians), and there is both practical and theoretical evidence that intervention can have substantial positive effects on both quality of life and survival.

In relation to forecasting, it is important to note that certain predictors remain valid even at the highest ages, *e.g.*, self-rated health as well as physical and cognitive abilities (Nybo et al. 2001). This may provide an opportunity to improve forecasting if for example the physical abilities of new cohorts of elderly persons are assessed and compared to previous cohorts; *i.e.* “Are the new cohorts of the elderly healthier than the previous cohorts (and therefore expected to live longer)?” The first reports of this kind have been published based on US studies (Manton and Gu 2001; Manton et al. 1997). They indicate that the new cohorts of elderly are increasingly healthy.

7.4 Conclusion

The cohort differences in physical abilities among the elderly and the correlation between physical abilities and mortality may be the basis for improving the forecasting of mortality.

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