Height and mortality in the Netherlands, 1850-1900: A sibling design study

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Introduction

Whether height is associated with mortality in adulthood is a debate that has interested economic historians for decades, beginning with Waaler (1984). On its face, it is logical that height is negatively related to mortality – in other words, that taller heights would be associated with living longer. This is because height and mortality are both influenced by health in early-life. However, research on the topic presents conflicting findings: while many studies find a relationship between height and mortality, a number of others do not (for a review of the literature, see Sear, 2010). To help better-understand this issue, I employ a sibling design. I am thus able to control for shared family inheritance (a mixture of genes and shared environmental factors in childhood). I do so with a unique dataset of Dutch men and their brothers, born in the latter half of the nineteenth century.

To start, I explore why height may be associated with mortality. As noted, height and mortality are both reflective of health in early-life, or, more accurately, of the share of health that is determined by early-life environmental conditions (Alter, 2004). However, height and mortality appear to be somewhat differently impacted by early-life environmental conditions.

For height, arguably the most important early-life environmental condition is nutrition. The environmental determinants of height are perhaps best-conceptualised as nutritional status, or the quality and quantity of food intake, minus the energy that the body expends (Floud et al., 2011). Most energy is expended simply by keeping the body functioning (basal metabolism), but also via physical labour, stress, toxins and pollutants, and, most relevantly for this study, disease (*ibid.*). If more energy is expended than consumed during development, individuals tend be shorter. What individuals eat is also important: diets are lacking in specific nutrients have also been found to be shorter (Bhutta et al., 2013). Evidence for the importance of early-life nutrition in determining adult height comes from the literature around exposure to famines in childhood and adult height (e.g. Portrait et al., 2017).

While nutrition appears to be the largest determinant of height, disease also likely plays a significant role. Bozzoli et al. (2009) found some evidence of scarring, whereby cohorts exposed to a worse disease environment (proxied by the infant mortality rate) in infancy were more likely to be shorter in adulthood. Moreover, malnutrition and disease exacerbate one another (Norman et al., 2008). However, Öberg (2014) argued that disease does not have a consistent impact on growth, with the severity and duration, as well as the disease's interactions with other environmental factors, likely influencing disease's relationship to height. Nutrition, in contrast, perhaps has a more uniform relationship to height (*ibid.*).

Next, I explore later-life mortality's relationship to early-life environmental conditions. While nutrition may be the largest determinant of height, Öberg (2014) argued that disease is the largest determinant of death in adulthood. In a historical Swedish cohort, Bengtsson & Lindstrom (2000) found that exposure to disease in childhood was more strongly correlated with earlier death in adulthood than other potential determinants, including food prices (a proxy for nutrition). In a twentieth-century American cohort, Blackwell et al. (2001) found that poor health in childhood was strongly correlated with adult morbidity. The authors also tested adult height's relationship to adult morbidity, but their findings were not significant.

Other early-life environmental conditions, particularly nutrition, appear to play a role in determining later-life mortality. The foetal origins of adult disease hypothesis links lower birthweight to a greater risk of heart disease in adulthood (Barker, 1990). He argued that this

occurs via 'foetal programming', whereby insufficient nutrition during the critical period *in utero* results in permanent changes to the foetus, although what changes more precisely occur have been debated (Barker, 1990; Sultan, 1994). While initial slower growth may be compensated-for, these changes to the foetus cannot be altered, and can negatively impact later-life health (Barker, 1990). However, studies examining food prices in early-life have not found a relationship to mortality in later-life (e.g. van den Berg et al., 2009), although this may be due to the difficulty in associating a population-level indicator with an individual-level outcome.

In these conceptualisations, height and mortality's drivers are similar, but appear to differ in their importance. The largest determinant of height may be early-life nutrition, while that of mortality may be early-life disease. Öberg (2015) argued that disease may negatively impact an individual's longevity without having an impact on their height. Further, factors that contribute to growth are not associated with being long-lived (e.g. dairy consumption) (Katz & Meller, 2014; Öberg, 2014). A cursory glance at the longest-lived countries indicates that height is not necessarily associated with longevity: the longest-lived nation are the Japanese, with men 170 centimetres (cm) tall, one cm shorter than the global average (NCD-RisC, 2016).

Perhaps in part because of these different drivers, height and mortality's relationship has been found to vary (e.g. Sear, 2010). One source of studies' heterogeneous findings may be the difficulty of adequately controlling for factors that confound the relationship between height and mortality, as they are numerous and varied (Öberg, 2014). To better-specify height's relationship to mortality, a sibling design may prove useful. This would allow all sibling-invariant early-life determinants of height and mortality to be controlled for. However, to my knowledge, no existing study on the topic has taken this approach. To address this gap, I pose the research question: to what extent is height related to mortality, taking into account shared family inheritance?

Methods

Setting

My research population consists of Dutch men who were born between 1841 and 1900. This is a fascinating context in which to study height's relationship to mortality. The Dutch were growing rapidly during this period: in the mid-nineteenth century, they were relatively short compared to other Europeans; by the century's end, they were on their way to being the tallest nation in the world (Baten & Blum, 2012). Similarly, the mortality environment was changing. Death from infectious disease in the late nineteenth century was becoming less common, with fewer epidemics, improved nutrition and sanitation, and, ultimately, a greater share of the population reaching old age (Alter, 2004).

Data and variables

The starting point of this dataset is the Historical Sample of the Netherlands (HSN), a representative sample of the Dutch population (Mandemakers, 2000). From 1850 onward, the HSN contains detailed household information, as well as birth, marriage and death certificates. The Heights and Life Courses dataset contains a sample of male HSN RPs who survived until the age of conscription (19 or 20 years) (Kok et al., 2016). These RPs are then linked to height and mortality information of their brothers, part of the Male Kin Height dataset. This dataset only contains reliable information prior to 1900. I thus have a sample of 3,396 HSN RPs, and 3,189 of their brothers, born between 1850 and1900.

In terms of variables, I use age at death (in years) as the dependent variable. I use height in cm as my key independent variable. Because height's relationship to later-life outcomes has been found to be non-linear (e.g. Thompson et al., 2021), I include a quadratic term, and test for joint significance with an F-test. In the full-sample adjusted model, I also control for

relevant household and population characteristics, namely birth cohort, father's occupation, religion, and infant mortality rate at birth, family size, and population size (for a full explanation of control variables, see Thompson et al., 2021, which employs the same dataset).

Analyses

To analyse height's association with mortality, I first generate a Kaplan-Meier curve. Next, I perform survival analyses: an unadjusted Cox regression (Model 1); a Cox regression adjusted with a quadratic term and relevant covariates (Model 2); an unadjusted partial likelihood model stratified brother groups (Model 3); and a partial likelihood model stratified by brother groups, sibling-variant covariates (e.g. birth year) (Model 4). I censor the 1,001 RPs without death information at age of last observation. Results are reported in hazard ratios, whereby a hazard greater than one is associated with a greater hazard of death.

Results

First, I examine the results of a Kaplan-Meier curve. Here, all five of the height groups have nearly identical probabilities of survival. The differences in survival among height groups are not significant, based on the results of a log-rank test. However, it appears that RPs in the first quintile have a marginally lower probability of survival than the other height quintiles between the ages of 40 and 70.

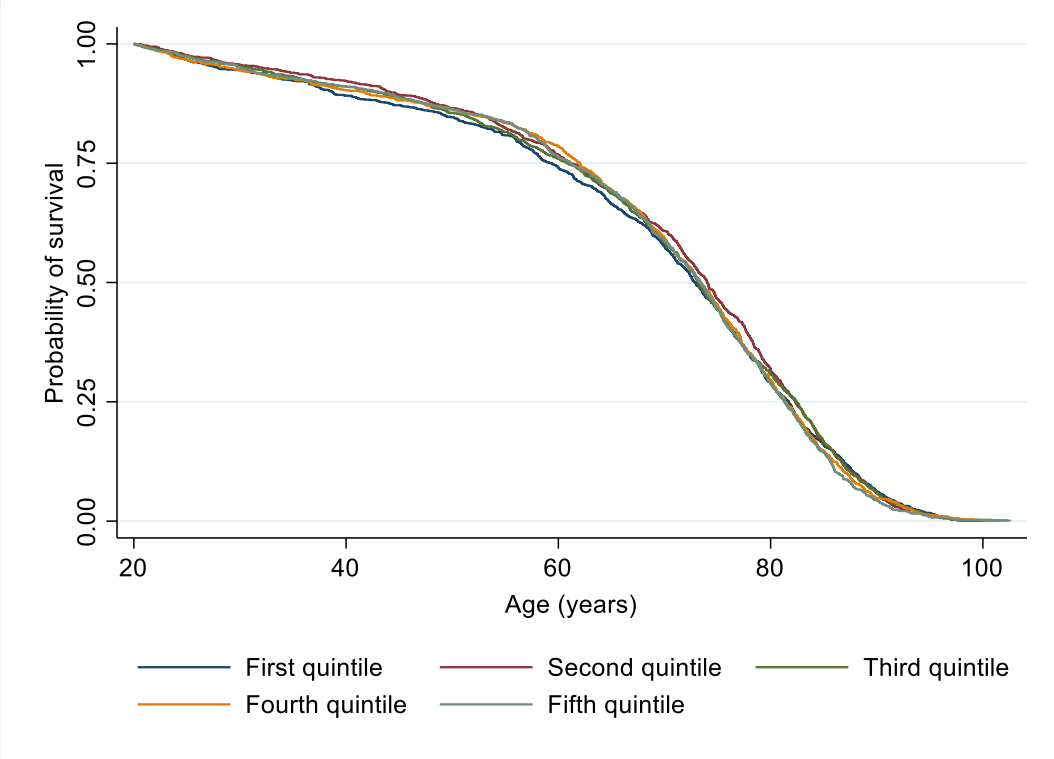

Figure 1. Kaplan-Meier curve.

Next, Table 1 presents the results of the Cox regressions. In Model 1, height has virtually no relationship to the hazard of death, with a hazard ratio close to one. However, in Model 2, once a quadratic term and confounders are included, height has a curvilinear relationship to the hazard of death. Here, those who are 165 cm, three cm below the mean height, have the lowest hazard of death, with those who are very short and somewhat tall having higher hazards of death. These terms are significant individually at α=0.05, and jointly at α=0.01. In Models 3 and 4, which are stratified partial likelihood estimates, height has no

significant relationship to the hazard of death, again with hazard ratios close to one. Overall, I find evidence that height is negatively associated with mortality outcomes, but not over and above shared family inheritance.

Table 1. Height's association with the hazard of death

	Model 1	Model 2	Model 3	Model 4
Height (cm)	1.001	0.889**	0.996	0.967
	(0.002)	(0.051)	(0.005)	(0.116)
Height $(cm)^2$		1.000**		1.000
		(0.000)		(0.000)
Restricted F-test (height terms)		10.56***		0.11

* $p \leq 0.10$, ** $p \leq 0.05$, *** $p \leq 0.01$; Robust standard errors are included in parentheses.

Discussion

This paper contributes to the literature on height's relationship to mortality. In particular, this is the first study to compare height's relationship to mortality among family members. Without controlling for shared family inheritance, my findings mirror other studies set in the nineteenth century (e.g. Alter et al., 2004; Costa, 1993), whereby height has a negative relationship to mortality.

However, this relationship does not persist once shared family inheritance is controlled-for. I find several plausible explanations for this. First, it may be that height's relationship to mortality has been over-estimated in previous studies, due to the difficulty in completely controlling for confounding factors.

Second, context may matter. In Sear (2010), the author found that height's relationship to mortality was generally insignificant in studies in high-mortality contexts, where death before old age is more common. However, in low-mortality contexts, height appears to be more consistently related to mortality (*ibid.*). In high-mortality contexts, death from infectious diseases and violence is much more common. Good health may not be as protective against deadly infectious diseases as it is against chronic diseases (Bengtsson & Alter, 2019). Death may simply be more democratic in high-mortality environments.

Height may also be less reflective of early-life health in high-mortality contexts. Growth tends to be more uniform in low-mortality contexts, in which men finish growing in adolescence. In contrast, there is evidence men grew well into their twenties in a high-mortality context, with some growing as much as 20 cm between late adolescence and adulthood (Thompson et al., 2020). This prolonged growth is thought to be due to worse environmental conditions. A man therefore may be tall, but have experienced deprivation in early-life.

Further, greater variability and extremity of environmental conditions in high-mortality contexts may explain why height's relationship to mortality appears to be weaker in these contexts than in low-mortality ones. If height and mortality are differently influenced by early-life environmental conditions, height and mortality perhaps may be less related in contexts when these determinants are more extreme (e.g. epidemics and/or food shortages). For example, surviving typhus in early-life may shorten a man's life, but not his height.

While this paper represents an important elaboration on the literature, it also has several shortcomings: height's association with mortality apparently varies by cause of death, which I do not yet incorporate (due delays resulting from Covid-19). In future drafts of this paper, I will do so.

I also focus only on those who survived until adulthood. It may be that this group is in some way systematically different from those who died at younger ages. These men may be a

select group of survivors, who are taller and healthier than their peers who died prior to conscription. If this were the case, the relationship between height and mortality would likely vary based on the share of individuals scarred or selected out (Alter, 2004). I find similar relationships between height and mortality across birth cohorts, so I do not anticipate that selection bias is clouding my results.

Overall, I find that height's relationship to mortality is explained by shared family inheritance. As of yet, this paper has not been able to offer more a more definitive understanding of these mechanisms. In future, conducting cause-specific mortality analyses will help to better-specify height's relationship to mortality.

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