Genetic and Shared-Environment Effects on Stature and Lifespan. A Study of Dutch Birth Cohorts (1785–1920) Based on Genealogies

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Genetic and Shared-Environment Effects on Stature and Lifespan

A Study of Dutch Birth Cohorts (1785–1920) Based on Genealogies

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ABSTRACT

Historical demography is generally concerned with the changing economic, social and normative contexts of human behaviour and health outcomes. To most historical demographers, the "genetic" component of behaviour and health is either unknown or assumed to be constant. However, several studies point at the shift over time in the relative importance of environment and genes: in periods and social groups with strong normative or economic constraints on behaviour, the "genetic potential" is often not realized. Therefore, to some extent, the waning of environmental constraints on heritability plays a role in changes in demographic outcomes over time. Determining the relative importance of heritability versus shared environment in historical populations for which only genealogies are available poses a challenge. Kin may live in different periods, and in different cultural and social settings. This explorative paper analyses the association between heights of conscripted relatives, as well as their life span. I estimate how the associations are affected by respectively genetic relatedness, shared historical period and shared social and geographical environment. Furthermore, I make a distinction between kin related via the mother versus kin related via the father. All kinds of kin are involved in the analysis: (half, full and twin) brothers, fathers, grandfathers, uncles and cousins. The data consist of about 3,000 men culled from Texel island genealogies, which also include descendants of families who had left the island. Life span has a weak, but still discernible, genetic element. The heritability of height is much stronger, especially at age 19/20. The correlations of mother's kin with her son's heights are stronger than those of her husband's kin. The analysis does not yield a consistent effect of a protective environment on kin correlations in either height or life span.

Keywords: Anthropometrics, Life span, Heritability, Environment, Conscripts

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1 INTRODUCTION

In recent years, physical stature and life expectancy have become important markers of global developments and regional inequality in well-being (Harris, 2021; van Zanden et al., 2014). Indeed, these indicators offer a much richer perspective on living conditions than, for instance, Gross Domestic Product per capita. However, what is hardly ever addressed in this literature is the role of intergenerational transmission of health, which may play a role in persisting socio-economic inequality (Bhalotra & Rawlings, 2011). To understand processes of health transmission, we need to disentangle genetic and environmental factors and try to detect how their relative importance shifts across social groups or periods.

The systematic study of the heritability of human stature has a long history, going back to the early 1800s (Zimmer, 2018). About 80% of individual variation in heights can be ascribed to the genes inherited from father and mother. Yet, it has proven quite difficult to locate those genes and to understand the actual processes at work. According to Zimmer (2018), "the heredity of height can be as baffling as quantum physics" (p. 252). Collaborating teams of geneticists have studied the DNA of 700,000 persons and managed to identify 800 genes involved in height. Yet, by 2018 these genes still explained only 27% of height's heritability (Zimmer, 2018, p. 280). It is clear that many genes are involved in normal growth, whereas only some rare mutations cause exceptional growth or stunting. In contrast to height, human lifespan has a low heritability (Kemkes-Grottenthaler, 2004). A recent study, based on a very large genealogical dataset of eight million families, looked at life spans of kin surviving to at least age 25 and estimates the correlation between a parent's and child's lifespan as merely 0.07 to 0.10 (Black et al., 2023). This is much lower than correlations in socio-economic positions or other indicators of health than lifespan. They conclude that contingency plays a large role in determining life span. Van den Berg et al. (2017; 2019) argue that research should focus on people reaching very old age (relative to their birth cohort). This has a clear genetic component, although few specific loci have been determined.

Heritability studies tend to focus on genetic variance relative to the total variance. This approach has been criticized as too narrow. Indeed, traits (such as height) that occur persistently in each generation can do so because: a) the environment remains the same; b) they are (literally) copied in the form of DNA or c) they are reconstructed in each generation because of socialization and transmitted resources (Gamma & Liebrenz, 2019). In other words, shared geographic, social or cultural environment can also explain correlation of traits among kin. How to tease apart genetic and environmental effects? Determining the relative importance of heritability versus shared environment is the goal of twin studies, which have been applied to many psychological, behavioural and health outcomes. In historical population studies, focusing on the later lives of twins is hardly possible because of their relative rareness, the impossibility to distinguish monozygotic and dizygotic twins, and their high infant mortality rates. Therefore, pedigree designs are more commonly used than twin designs (van den Berg et al., 2017). However, assessing the role of "shared environment" of relatives other than twins is quite a challenge, as they may live in different periods, and in different cultural and social settings. This may explain why research in this direction tends to focus on relatively closed and non-dynamic groups, such as the Old Order Amish (Sorkin et al., 2005).

Using historical populations, especially over a longer period, is important because it is assumed that the relative contribution of genetic and environmental effects can change over time. Several studies suggest that in periods and social groups with strong normative or economic constraints on behaviour, the "genetic potential" of physical development is often not realized (Kohler et al., 2002; also Bras et al., 2013; Van Bavel & Kok, 2009). With respect to human growth, the environmental share in explaining growth outcomes is supposed to be higher in adverse situations, or periods or places in which people cannot reach their genetic potential because of insufficient nutrition, diseases or traumatic experiences during childhood or adolescence. However, recent research demonstrates that parental responses to crises can mitigate but also exacerbate the effects of a "bad start" and thus increase or decrease the health differences between their children. Examples are Japanese parents whose sons (but not daughters) born after an earthquake in 1923 benefited from disaster relief spending (Ogasawara, 2022). Also, American parents redirected their investments to their older children at the expense of their younger after the 1918 influenza pandemic (Parman, 2015; see also Almond & Mazumder, 2013).

A common approach to study the relative effect of the environment on reaching the genetic potential is to look at correlation of height among kin. Take, for example, the case of siblings. They share 50% of their genes. If one's height was solely defined by DNA, the variance in individual heights "explained" by the height of a sibling should be about 50%, or a (squared) correlation coefficient of 0.7. However,

because siblings are not alike in age, crises (e.g., epidemics or food shortages) can intervene and result in much lower correlations. Alter and Oris (2008) hypothesize that when parents can "control variation" in the conditions needed for children to reach their height potential, a higher correlation among siblings is found. They found this to be the case in higher socio-economic groups in 19th-century east Belgium. Lauderdale and Rathouz (1999) studying US Civil War veterans found higher correlations in boys from rural places — which were presumably more protected than urban areas.

In a similar vein, kin correlations in health in more recent, affluent, periods are expected to be higher. Studying several Finnish cohorts, Silventoinen et al. (2000) found that brothers growing up in economically favorable periods had higher correlations in height. However, a recent study based on pooled twin data from several countries could not replicate this finding (Jelenkovic et al., 2016b). They concluded that "heritability estimates did not present any clear pattern of secular changes across birth-year cohorts from 1886 to 1994. Thus, our findings do not support the hypothesis that the heritability of height increases along with increasing living standards and diminishing rate of absolute poverty within populations" (Jelenkovic et al., 2016b, p. 9). The recent findings of Black et al. (2023) on change over time in kin correlations in life span are rather ambiguous. They report no change in their major sample, covering the birth cohort 1880–1920. But the correlations of life span in fathers and sons increased (from 0.05 to 0.1) in a sample covering 1820–1880. However, they suspect this trend to be an artifact of "better measurement or selection" (p. 19). Black et al. (2023) also have a different interpretation of change over time in kin correlations. They expect lower correlations in life span when the younger generation benefits from economic improvement, but higher correlations when economic development increases socio-economic inequality.

This article explores the possibilities of genealogical data to shed light on the relative role of environmental and genetic factors in stature and life span. The data collection is mainly, but not exclusively, focused on Texel island in the north-western corner of the Netherlands. The first aim is to test whether kin correlations in height and life span are indeed higher in a) more favourable historical periods; b) in an isolated rural (island) environment and c) in farming families. In all these contexts I expect that variation in early-life experiences among siblings is relatively limited. I look at (male) heights in late adolescence (age 19/20) but also in adulthood (age 25). Life spans are studied of the same population of military recruits, thus after age 19. Obviously, I expect the correlations in height to be much higher than in life spans.

The second aim is to "decompose" the effect of "heritability" on height and life span, by trying to separate genetic and shared-environment effects. This can be done by expanding the conventional approach in twin studies. In such studies, outcomes of monozygotic (sharing 100% of genes) and dizygotic (50%) twins are compared, under the assumption of a shared environment. In this study, I use the genetic relationship between all kinds of kin, and try to control for shared environment as much as possible. In doing so, I aim to discover whether shared age is more important than shared social or geographic environment.

Finally, the data design also allows me to explore the contribution of mother's height and life span — proxied by the height of her kin. In historical studies, data on mothers in relation to sons (let alone daughters) are virtually non-existent. Yet, the mother's contribution to health and height of a child is probably more important than father's. This was because she was the prime caregiver, which made that her loss was felt more acutely than the loss of the father, as can be shown from effects on adolescent heights (Quanjer & Kok, 2019; also Quanjer et al., 2023). But mothers matter also more than fathers for a purely physical reason: the correlations between mothers' and children's stature is higher than between fathers' and children's (e.g., Silventoinen, 2003; Venkataramani, 2011). That taller mothers tend to have taller children may be related to the greater likelihood of taller women to have children with larger birthweight, which can be translated into larger stature and better health under favorable circumstances (Addo et al., 2013; Thomas et al., 1990). As to lifespan, there is some evidence that heritability of longevity is stronger among daughters than among sons (Cournil et al., 2000).

In the next section, I introduce the geographical setting and discuss in more detail the creation of the dataset for this article. Then, I compare correlations in height at age 19/20, at age 25 and in life span between "ego" (the military recruit) and his different types of male kin: (grand)fathers, brothers, uncles and cousins, where relevant distinguished by mother's or father's side. In the fourth section, I take a different approach by running regression models in which the independent variables are respectively the percentage of shared genes, shared socio-economic status, shared geographic environment and closeness in age.

2 SETTING AND SOURCES

The island population studied in this article cannot be considered as representative for the Dutch population, not even for its rural parts. This is because of the island's relatively favorable economy and health situation. Texel island was strategically located on the main roadstead of the Dutch East India Company, and ships anchored there were piloted and supplied (e.g., with fresh water). Thus, the island economy prospered in the 17th and a large part of the 18th centuries. Furthermore, the island was very suitable for farming, especially sheep grazing. The decline in shipping precipitated by the Anglo-Dutch wars was accelerated in the Napoleonic era. The early 19th century brought no recovery, as harbor activities shifted to the nearby town of Den Helder on the Dutch mainland. Many families dependent on naval activities left the island, which became culturally and socially isolated from the mainland. However, extensive land reclamation in the 1830s brought newcomers to the north of the island. In 1895, the municipality counted 4,950 inhabitants, in 1840 4,924 and in 1899 5,954. Sheep farming remained dominant in the 19th century, but in the 20th century the proceeds from cattle farming, horticulture and tourism became much larger (van der Vlis, 1975, pp. 335–336).

The increasing isolation probably shielded the inhabitants from infections. The combination of geographical isolation with good drinking water and relatively abundant supplies of fresh food (dairy products, fish, birds, eggs, rabbits) probably produced a rather healthy population. In the mid-1850s an observer wrote "The altitude and fresh sea breeze make Texel a healthy and fertile place [an expert has said] ... I do not believe ... there is a healthier region, people grow old here and except for the smallpox there are few infectious diseases" (Allan, 1856, p. 20, translation by Jan Kok). Indeed, infant and child mortality rates of the island contrasted favorably with the national averages, in particular around 1900 (Kok, 2023). The island also stood out with the stature of its recruits. Based on aggregate statistics, Bolk (1909) calculated their average height in the period 1898–1907 as 1.73 meters, which made Texel men the tallest in the country (also Kok, 2020). Finally, it should be noted that the island is not suited to test the impact of socio-economic inequality on health correlations (see previous section). The social structure of the island struck contemporary travelers as highly egalitarian (Kroes, 2019, p. 279). Furthermore, switching and combining occupations must have added to the lack of clear social distinctions. People tended to move between farming and seafaring jobs, sometimes combining this with trade or shopkeeping.

Although the island is not representative for the (rural) Netherlands, there are good reasons to study its population. The military records with height measurements at age 19/20, as well as at age 25, have been well-preserved, and can be linked to exceptionally detailed genealogical data. In particular, I use published genealogies listing all descendants of six 17th-century founder couples (Dijt & Dijt, 1970; Dijt & Dijt, 1973). The data collection starts with the third or fourth generation, that is persons born from 1785 onward, who were enlisted in 1813 (all adult unmarried men) and all those conscripted at age 19 (after 1862 age 20) from 1817 onwards. The published genealogy ends with the children of the sixth generation, most of them born in the last quarter of the 19th century. All male and female descendants were listed, often with dates of death, including those who had left the island. Several have been traced in North-America. However, most families who migrated remained in the province of North-Holland, especially in the nearby harbor town Den Helder. This town offered plenty of employment, e.g., on the large naval dockyards.

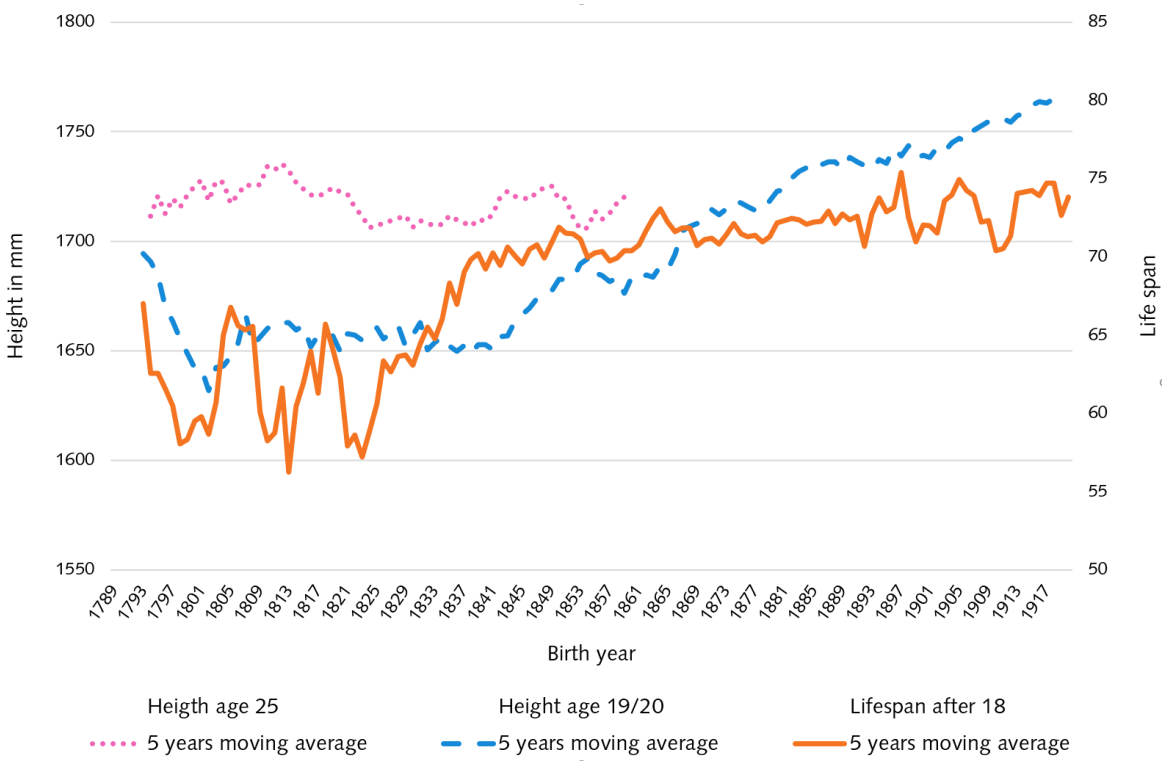
For the database I selected men who had reached at least the age of conscription. Using data on descendants implies I have relatively few observations in the early 19th century. Therefore, I added spouses and their brothers, and where possible their fathers, of descendants born before 1840. In order to include the first half of the 20th century, I also added the sons in the seventh generation, which could be found on an extensive genealogical website, also based on the published genealogies. The website, however, contains more information on people who had not left the island and/or were part of the site's manager's family tree (http://www.robgomes.nl/tng/, unfortunately out of order, but see https://www.genealogieonline.nl/genealogie-gomes/). My data collection ends with boys born in 1920 because it is not possible to collect individual heights after 1940.

This procedure resulted in a database of almost 4,000 boys who, in principle, could have been measured during the medical examination which was part of the draft. To find heights, I consulted the municipal archives of Texel and Den Helder, whereas boys measured after 1850 were traced online as the examination registers from 1870 onward have been digitized for the entire province of North-Holland (including Amsterdam) as well as some large cities (e.g., Rotterdam). This implies that I have

found more heights of offspring living elsewhere from birth cohorts after 1850. In total, heights for more than 1,000 boys are lacking for several reasons. First, they had emigrated or died before age 20 which has been missed by the genealogists. Second, the officials failed to note the recruits' heights in the records for Texel in entire periods (1824–1828, 1831–1832, 1837, 1847, 1859–1860). Third, boys could be absent from the examination, for instance because they were at sea. In principle, they would be enlisted when they failed to show up. In other research, it has been shown that boys from elite groups managed to avoid the examination altogether (Quanjer & Kok, 2020). Finally, after about 1900, boys who were to be exempted because a brother had been enlisted were no longer measured. In total I have 2,761 heights for descendants and in-laws in the database (of which 2,226 were island born). For the cohorts 1800–1855 it is also possible to include the heights at age 25 when men were included in the "civic guards" militia, but they refer only to men still living on the island. For Texel island the records of the civic guards have been preserved in good order — in contrast to many other places on the mainland (Beekink & Kok, 2017; Oppers, 1963). This yields another 860 heights.

In Figure 1, I plot the (five-year running averages of) median heights of all island conscripts as well as life spans of the recruits in the database. The conscripts born after about 1840 show an almost continuous growth in late-adolescent height, mirrored to some extent in adult height. For men born in the early 1850s, adolescent and adult height seem to have declined briefly. The gap between late adolescent and adult height is sizeable, suggesting that boys continued growing well into their twenties. Indeed, for those men for whom we have two measurements we witness an average growth after age 19 of seven centimeters. Life span as well increased rapidly, especially for the cohorts born between 1820 and 1840. For the later cohorts, the increase was more gradual.

Figure 1 *Heights (in mm) and life span (in years) of Texel island conscripts by birth year, 1785–1920*



Sources: Heights: Texel island municipality, Regional Archive Alkmaar, 1132 Inventaris van het archief van de gemeente Texel, 1440–1926, inventory numbers 1183–1185, 1193–1194 (conscription), 1166–1170 (civic guards); https://www.wiewaswie.nl/ for North-Holland recruits born after 1850. Life spans: own genealogical database constructed from Dijt and Dijt (1970; 1973) and http://www.robgomes.nl/tng. Total N with heights in this Figure is 6,172; N of civic guards registration heights is 2,306. N of life spans is 3,279.

Going back to the hypotheses informing this study, I expect that kin correlations in height and life spans, as well as the "genetic" contribution to total heritability, will be higher in relatively "protected" environments, thus environments less subject to epidemics, food or other economic crises or family disasters. I expect the island to offer such an environment in contrast to the mainland, the period after 1860 in contrast to the earlier period, and farming households in contrast to non-farming. As to the latter, families involved in shipping not only suffered from the decline in the island's involvement, but they were also often hit by the drowning of their men. The same is true for fishing families.

3 KIN CORRELATIONS IN HEIGHT AND LIFE SPAN

The genealogies linked to conscript and civic guard records yield the heights of 2,761 men who were related in many ways. I use these relations to construct pairs of different kin types. For instance, a father with four sons gives four father-son pairs, whereas the brothers provide six (3 + 2 + 1) unique pairs. Of course, this implies that individuals in large families appear more often than those from small families. Is there a difference in the heritability of father's or mother's health indicators? I cannot answer this question directly, because I lack mother's height. But I can make a distinction in family (grandfathers, uncles and cousins) from father's and mother's sides (see the diagram in Figure 2). Thus MB stands for mother's brother, FB for father's brother, MZS for mother's sister's son and so on. The percentages in the figure refer to shared DNA.

To be sure, the percentages of "shared DNA" in Figure 2 are a simplification of the genetic processes involved. In the formation of sex cells (*meiosis*) parts of the chromosomes from parents are "crossed over", as, for instance, mother's form of a gene is moved to father's chromosome. This means that one does not, by definition, always inherit a fixed percentage of genes from, e.g., the parental grandfather. Also, recombination events happen in females more often than in males, which implies that variation in genetic transmission among men is larger than among women (see https://gcbias.org/2013/10/20/how-much-of-your-genome-do-you-inherit-from-a-particular-grandparent/. Furthermore, a small part of total DNA is only transmitted from mothers to children. A recent study suggests that this "mitochondrial" DNA can also impact height and life span (Yonova-Doing et al., 2021).

Figure 2 *Schematic representation of family lines and percentage of shared DNA with ego*

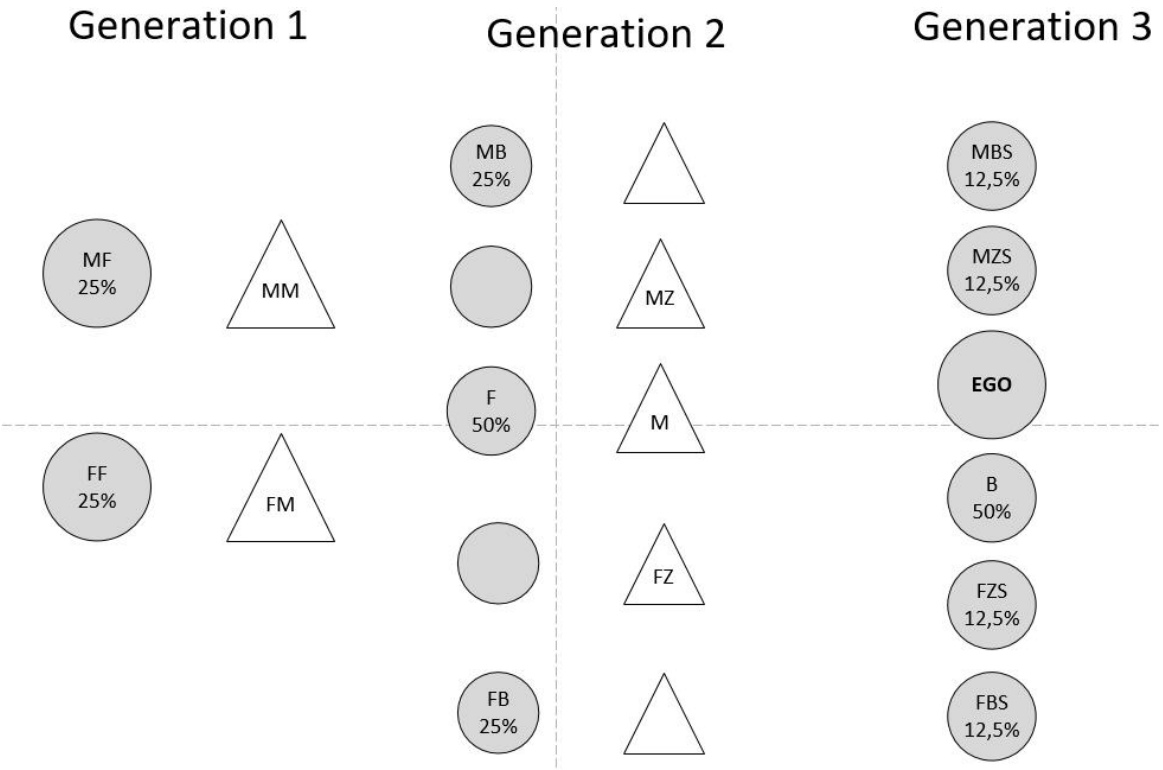


Table 1 *Pearson correlations in heights at conscription age 19/20 between kin, Texel family descendants 1785–1920*

Pairs	General	Born 1785–1859	Born 1860–1920	Both born Texel island	Both farming	Age difference < 4 years
Ego-Father	.384*** 1,594	.292*** 397	.398*** 1,197	.371*** 1,322	.364*** 527	
Ego-Brother	.502*** 2,124	.430*** 736	.453*** 1,388	.490*** 1,723	.467*** 831	.609*** 741
Ego-Father's father	.144*** 738	.341** 65	.138*** 673	.116** 592	.100 225	
Ego-mother's father	.152*** 616	.380*** 84	.125** 532	.119** 506	.145 148	
Ego-Father's brother	.213*** 2,862	.127** 517	.219*** 2,345	.213*** 2,423	.156*** 1,019	
Ego-Mother's brother	.299*** 2,300	.179*** 561	.290*** 1,739	.299*** 2,035	.312*** 747	
Ego-Father's brother's son	.241*** 2,230	.070 470	.247*** 1,760	.245*** 1,784	.198*** 831	.308*** 593
Ego-Mother's brother's son	.187*** 2,229	.077* 791	.185*** 1,438	.189*** 1,562	.305*** 616	.243*** 548
Ego-Father's sister's son	.199*** 1,557	.023 305	.185*** 1252	.198*** 1164	.267*** 507	.358*** 308
Ego-Mother's sister's son	.305*** 1,549	.340*** 256	.250*** 1,293	.322*** 1,128	.359*** 465	.339*** 374

*Notes: *** p < 0.001; ** p < 0.01; * p < 0.05. Integers are absolute number of pairs.*

Source: See Figure 1.

Table 1 presents the correlations in height at age 19/20. The first column gives the overall correlations for each type of pairs. Fathers' and sons' correlation is .384 which is actually quite high given the age difference and thus the different periods in which they grew up. An American study (1959–1969) focusing on ages 15–18 reports a coefficient of .27 (Silventoinen, 2003, p. 272). Another American study gives .31 for father-sons heights among white families and only .15 among black families (Malina et al., 1976). Our result for brothers (.50 based on 2,124 pairs) comes very close to Alter and Oris' (2008) figure of .48 (based on 1,938 pairs) in 19th-century eastern Belgium. Thompson et al. (2020) found .46 for the cohort 1834–1843 in the city of Maastricht. However, Alter and Oris report differences between rural and urban localities with higher correlations in the former. Silventoinen (2003) summarizes 22 (contemporary) studies in which brother-brother correlations range from .24 (India) to .79 (brothers 18+ in Great Britain). It is clear that there is a strong variation undoubtedly related to the presence or absence of a shared (protective) environment. The correlation with grandfathers (.144 and .152) is quite low, but then, of course, they grew up in different historical settings than the recruits. Heights of uncles (.213 and .299) show stronger associations and it is interesting that the correlation with mother's brother's height is the strongest.

Remarkably, height correlations among cousins are much higher than we would expect on the basis of the modest genetic resemblance of 12.5% even going up to .305 in the case of mother's sister's sons. In fact, high first cousin correlations in, for instance, health indicators have puzzled biometricians already for more than a century. Elderton, comparing physical and psychological characteristics of cousins, noted "We should conclude accordingly from the present results that for the purposes of eugenics cousins must be classed as equally important with uncles and aunts, and that they may eventually turn out to be as important as grandparents" (Elderton, 1907, p.20). Fisher (1918) also noted that, for instance in stature, cousin correlations were unexpectedly high, even exceeding "avuncular correlations" (cited in Rodgers et al., 2019). These "inflated" cousin correlations remained unexplained, until Rodger's et al. (2019) picked up "Fisher's challenge" in an analysis of height correlations using only cousins (through sisters) of different

removals. They find the same high correlations, but, in their view, the explanation is simple: Fisher had ignored the role of shared environment as, after all, the mothers of the first cousins had grown up together.

The table provides some clues as to mother's contribution to boys' heights. The association with the height of her father and brother is stronger than with father's father and respectively with father's brother, also after controlling for birthplace and SES. If indeed mother's health can be proxied by the heights of her father and brothers, then she seems to play a larger role than father. Perhaps the finding than the strongest cousin correlations are found with mother's sister's son also points in this direction. But what does it mean? Does this somehow reflect women's smaller variation than men's in how parental DNA is transmitted (see above)? Or do the findings show an effect of mitochondrial DNA? It is also possible that non-genetic factors are involved. Through "maternal constraint" (Gluckman & Hanson, 2004) the size of a fetus is adjusted to mother's birth canal. Such intergenerational health conditions (see also Wells, 2017) are passed on from the grandmother through mothers (as is mitochondrial DNA), thus should affect correlations with mother's brother and mother's sister's son only. Finally, we should consider the possibility that the joint socialization of sisters, e.g., though training in hygiene, has more lasting effects on their future children's health than the socialization of brothers.

Let's turn to the environmental hypotheses. The first is that kin correlations are stronger in more favorable periods which should result in less variation among kin, stemming from, e.g., food crises. In the period before 1860, epidemics and economic crises beset the Netherlands (de Meere, 1982), and the island suffered in particular from the decline in shipping. The Crimean War had detrimental effects on Dutch nutritional standards in the 1850s (Knibbe, 2007). Thus, I expect the statures of recruits born after 1860 to be stronger correlated with their kin, in particular with those of similar age. Overall, this seems to be the case: correlations with father, brothers, uncles and cousins are all higher (excepting with mother's sister's sons).

The second hypothesis relates to the (growing) isolation of the island, and the assumption that being reared in a similar environment leads to higher correlations in height. Overall, this is not corroborated by the results: correlations of kin pairs both born on the island do not differ from kin pairs in which at least one of them was born elsewhere.

What about the third hypothesis, stating that farming offering a protected environment? Table 1 shows that cousins both raised in a farming household had higher correlations than cousins in general (except for father's brother's sons). For the other kin categories correlations seem to be lower. This finding confirms the suggestion of Rodgers et al. (2019) that shared environment raises the correlation in height among first cousins.

Finally, we look at the effect of similarity in age. As expected, brothers and cousins of roughly similar age, probably experiencing the same environmental conditions at the same age, have higher height correlations than brothers and cousins in general.

Table 2 shows correlation in heights measured at age 25. As discussed in the previous section, many boys continued growing after age 19/20. Genetic factors not only affect the (adult) height potential, but also the speed and timing of growth during adolescence (Jelenkovic et al., 2016a, p. 8). Thus, individual delay of the adolescent "growth spurt", which has often been noted in the first part of the 19th century, could be caused by a combination of environmental and genetic effects. Table 2 shows that the correlations in adult height for early 19th-century birth cohorts were relatively low compared to correlations in conscript heights. For instance, the correlations of brother's heights at age 25 is .248 versus .430 at age 19 (see Table 1). This also implies that "catching-up" of stunted boys after age 19 depended more on specific individual constitution or circumstances and not on genetic predisposition. Indeed, recently a lack of brother-brother correlation in catch-up growth was described in a study of late 19th-century Maastricht (Thompson et al., 2020).

Finally, a similar exercise is done with life spans, simply defined as the number of years lived after age 19. We can expect strong individual variation because of differences in life style, but also because of accidents (as said, quite a few men drowned at sea) and war. The Second World War claimed many civilian lives on the island in 1945. In other words, effects of genetically inherited health factors associated with individual life span, and thus with kin correlations, are expected to be limited. Indeed, table 3 shows weak correlations, very much in correspondence with the findings of Black et al. (2023). They report a father-son correlation coefficient of 0.096, paternal grandfather-grandson coefficient of 0.027, maternal grandfather-grandson coefficient of 0.028 and brother-brother coefficient of 0.139.

Table 2 *Pearson correlations in heights at age 25 between kin, Texel island family descendants (all born and measured on Texel) 1785–1855*

Pairs	General	Both farming	Age difference < 4 years
Ego-Father	.247*** 306	.200** 98	
Ego-Brother	.248*** 594	.269*** 255	.285*** 194
Ego-Father's brother	.086 406	.126 181	
Ego-Mother's brother	.196*** 392	.302*** 122	
Ego-Father's brother's son	.067 441	.261** 261	.039 108
Ego-Mother's brother's son	.235*** 319	. 207 85	.226* 90
Ego-Father's sister's son	.131 159	.165 30	.322* 47
Ego-Mother's sister's son	.029 151	-.012* 41	-.002 39

*Notes: *** p < 0.001; ** p < 0.01; * p < 0.05. Integers are absolute number of pairs.*

Source: See Figure 1.

Table 3 *Pearson correlations in lifespan after age 19 between kin, Texel family descendants 1785–1920*

Pairs	General	Born 1785–1859	Born 1860–1920	Both born Texel island	Both farming	Age difference < 4 years
Ego-Father	.111*** 2,335	.080* 751	.095*** 1,584	.115*** 1901	.144*** 700	
Ego-Brother	.152*** 2,823	.132*** 1407	.123*** 1,416	.154*** 2,440	.160*** 1,007	.160*** 979
Ego-Father's father	.035 1,174	. 056 82	.030 1,092	.027 944	.026 332	
Ego-mother's father	.065* 9,96	.200* 116	.043 880	.041 847	-.059 237	
Ego-Father's brother	.019 4,005	. 030 941	.006 3,064	.027 3,419	.011 1,381	
Ego-Mother's brother	.053** 3,253	.043 1,036	.024 2,217	. 053** 2,878	.086** 1,022	
Ego-Father's brother's son	.055** 3,108	.038 1,072	.041 2,036	.052** 2,552	.040 1,151	.068 824
Ego-Mother's brother's son	.054** 2,609	. 034 1,177	.048 1,432	.048* 1,990	-.061 763	.074 544
Ego-Father's sister's son	.066** 2,062	-.011 634	.032 1,428	.031 1,696	.054 728	.074 583
Ego-Mother's sister's son	.022 3,236	.012 1,546	-.010 1,690	.002 2,447	.004 723	.015 621

*Notes: *** p < 0.001; ** p < 0.01; * p < 0.05. Integers are absolute number of pairs.*

Source: See Figure 1.

Contrary to expectations (see Introduction), the correlations in life span are not higher in the later period, and the "protective" island and farming effects are limited as well. The modest effect of shared environment is also evident from the correlations among brothers and cousins of similar age. In contrast to height at age 19, the coefficients are hardly higher. The life span of mother's father and mother's brother is stronger associated with ego's life span than that of his father's father and brother, suggesting once again that mother's health — possibly genetically transmitted — is more important to adult life expectancy than father's health.

4 GENETIC AND ENVIRONMENTAL COMPONENTS IN HEIGHT AND LIFE SPAN

The correlation tables have suggested that genetic relatedness is mirrored in correlation coefficients of height and life span in pairs of kin. But sibling correlations exceed intergenerational correlations, most likely because of shared environmental conditions. And the coefficients of first cousins (sharing 12.5% of their genes) tended to be higher than in pairs of grandsons-grandfathers (sharing 25%), again suggesting that shared environment adds strongly to the association. How to separate genetic effects from environmental effects? Clearly, this is core business of twin models, such as the ACE design, which estimates the effects of additive genes (A), the shared environment (C), and the unshared environment (E) using monozygotic and dizygotic twins. An example is the DeFries-Fulker model (DeFries and Fulker, 1985), which has also been applied in sibling studies, and even for extended kin (Rodgers & Kohler, 2005; Rodgers & McGue, 1994; Zieleniewski et al., 1987). The basic setup of a DeFries-Fulker model is

$$T_1 = \beta_0 + \beta_1 T_2 + \beta_2 R + \beta_3 R T_2 + e$$

Here, T_1 stands for the trait of interest, in our case height and life span. T_2 is the same trait in a relative. R gives the degree of genetic relatedness, thus, for instance, 0.5 for full brothers and 0.125 for cousins. The interaction of R with T_2 gives the heritability of the specific trait. Thus, β_1 is interpreted as the effect of shared environment, whereas β_3 is interpreted as the effect of heritability (Bras et al., 2013, p. 124). Extending the model beyond twins may overestimate the share of heritability as environmental influences on the trait in question are not equal, in contrast to twins (Bras et al., 2013, p. 124). Therefore, in their study of the heritability of fertility among siblings, Bras et al. (2013) propose an "extended" DeFries-Fulker model in which shared environment is specified with an index of age similarity, resulting in the following model:

$$T_1 = \beta_0 + \beta_1 A + \beta_2 R + \beta_3 A T_2 + \beta_4 R T_2 + e$$

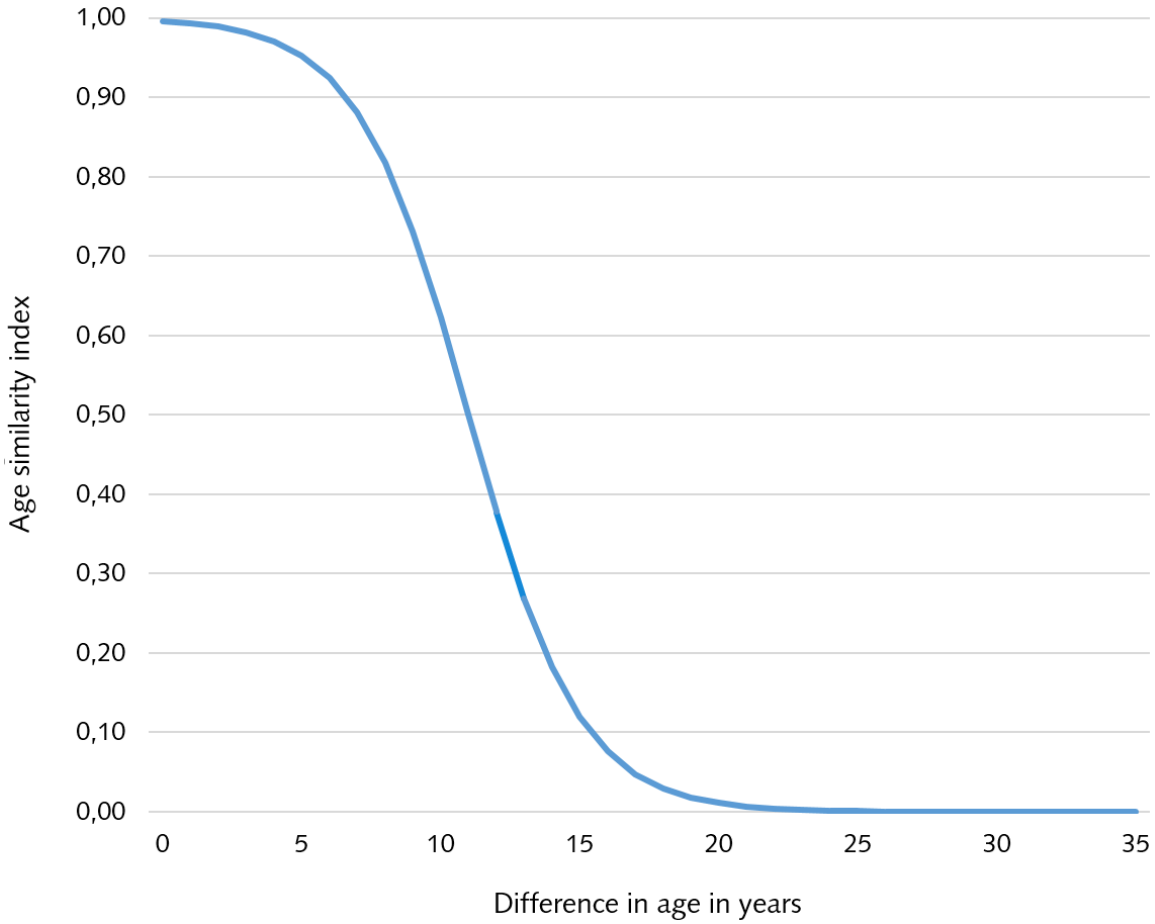
Here A stands for age similarity. Now, β_3 gives the estimation of the impact of shared environment and β_4 of heritability. In this section, I follow this approach by adopting a similar index for age similarity. The notion of "age similarity" refers to experiencing the same conditions at more or less the same age. This is not perceived as a linear process, the sharing effects should decline rapidly when the age difference becomes large. Thus, for this I use a polynomial function, as follows:

$$\text{Index of Age Similarity} = 1/(1 + \text{EXP}(0{,}5 * \text{Age difference} - 11))$$

Figure 3 shows the index by years of age difference.

However, as I extend Bras et al.'s approach beyond siblings, the assumption of a shared environment is still violated. As far as possible, I try to remedy that by adding additional parameters for shared geographic environment and shared socio-economic status of the family of origin, which are similarly interacted with height and life span of the relative. For SES, I selected the occupation of the father at the birth of the recruit, coded in HISCLASS category (van Leeuwen & Maas, 2011, adopted is the five-group classification in Mandemakers et al., 2018). Finally, I add a dummy indicating whether the relation is solely via the maternal line. This applies to correlations with mother's brother and mother's sister's son. Again, I interact this with the trait of interest.

Figure 3 *The age similarity index by years of difference in age*



In the next tables I apply this approach, focusing on the parameters of interest. Instead of using the absolute heights and lifespans, I use their z-scores by (30-year) birth period to neutralize the effect of the secular trend (see Figure 1). Also, z-scores allow for a better comparison between the outcome variables.

This model design implies that persons can appear multiple times, in different kin combinations (brothers, cousins, etc.). By definition, individuals from large families appear most frequent, which could affect results. For instance, family size can — through resource dilution — affect the height of children (Öberg, 2017) or even their adult life expectancy (cf. Baranowska-Rataj et al., 2017). Therefore, for each model, I add a separate one with weights ensuring that each individual is represented equally. The fweight function in Stata does not allow for non-integer values, which is why I have used the rounded value of (1/N observations) * 50. In addition, for all models I cluster the standard errors with robust clustering at the level of the individual.

As expected, the stronger the genetic relatedness, the stronger the association between ego's and kin's height (see Table 4). Closeness in age — an indication of shared environment — is also reflected in a positive effect. The assumption that a more favourable period leads to less variation among kin, and therefore a stronger effect of heritability is not affirmed: the effect of the degree of genetic relatedness is actually lower in 1860–1920 than in the earlier period. The outcomes in the model with weights do not differ significantly from the ones in the unweighted model. Interestingly, the interaction with a dummy for the maternal line shows a (weak) significant effect.

Table 4 *Effect of kin's height on ego's height (age 19/20) (z-scores) mediated by environmental and genetic factors, Texel island conscripts 1785–1920*

	Unweighted		Weighted	
	1785–1859	**1860–1920**	**1785–1859**	**1860–1920**
Kin's height	0.039	–0.088*	0.102	0.027
Kin's height* age similarity index	0.104*	0.116***	0.119*	0.084**
Kin's height* degree of genetic relatedness	0.813***	0.798***	0.808***	0.720***
Kin's height* both island born	-0.163**	0.005	–0.199**	-0.440
Kin's height* similar SES	0.018	0.037	–0.445	0.004
Kin's height* maternal line	0.110*	0.057*	0.109*	0.060
r^2	0.07	0.07	0.09	0.08
N observations	4,182	13,617	42,854	82,067
N clusters	837	1,568	837	1,568

*Notes: Robust cluster at the level of the individual conscript.*** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$. The age similarity index, the degree of genetic relatedness and dummies for island born, similar SES and maternal line are included, but not shown in the model.*

Source: See Figure 1.

Adult height is modeled in Table 5, which shows no specific effects of the maternal line, implying that mother's effect is strongest in youth and adolescence. Age similarity or shared environment seems to have a much stronger effect on late adolescent height (see Table 4) than on adult height. Thus, relatives who experienced the same adverse or beneficial conditions during their youth tended to have similar heights, but this did not extend to adult heights, suggesting strong variation in individual catch-up growth.

Table 5 *Effect on ego's adult height of kin's adult height (z-scores) mediated by environmental and genetic factors, Texel island conscripts 1785–1859*

	Unweighted	**Weighted**
Kin's height	.052	0.120
Kin's height* age similarity index	0.048	0.056
Kin's height* degree of genetic relatedness	0.379*	0.373
Kin's height* similar SES	-0.012	-0.046
Kin's height* maternal line	-0.003	-0.021
r^2	0.04	0.06
N observations	2,788	30,845
N clusters	602	602

*Notes: Robust cluster at the level of the individual conscript.*** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$. The age similarity index, the degree of genetic relatedness and dummies for similar SES and maternal line are included but not shown in the model.*

Source: See Figure 1.

Finally, I analyze life span after age 18. The explanatory power of the models is, predictably, weak. We find no shared environment effect. However, there is a clear effect of kin relatedness (for the total effect the main and interaction effects have to be added). The suggested effect of maternal line (see also Table 3) is not confirmed here. When we control for the frequency of individual observations, thus for a family size effect, the genetic factor in life span correlations becomes much stronger in the later period (final column), which agrees with the hypothesis that a more favorable period reduces the disturbing impact of environmental variation.

Table 6 *Effect of kin's lifespan on ego's life span (after age 18) (z-scores) mediated by environmental and genetic factors, Texel island conscripts 1785–1920*

	Unweighted		Weighted	
	1785–1859	**1860–1920**	**1785–1859**	**1860–1920**
Kin's lifespan	-0.070**	-0.056**	-0.043	-0.087***
Kin's lifespan* age similarity index	-0.005	0.005	-.0000	0.010
Kin's lifespan* degree of genetic relatedness	0.264**	0.220**	0.149	0.348***
Kin's lifespan* both island born	0.004	0.001	0.006	0.000
Kin's lifespan* similar SES	0.004*	0.000	0.007	0.000
Kin's lifespan* maternal line	0.024	0.009	0.006	0.027
r^2	0.01	0.01	0.01	0.01
N observations	8,869	16,864	60,958	88,779
N clusters	1,170	1,683		

*Notes: Robust cluster at the level of the individual conscript.*** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$. The age similarity index, the degree of genetic relatedness and dummies for both island born, similar SES and maternal line are included but not shown in the model.*

Source: See Figure 1.

5 DISCUSSION AND CONCLUSION

For historical demography, insights in health, its social variation and development over time, is crucial. Historical demography has much to gain from knowledge on the family and life course processes involved, as well as from knowledge on environmental influences on health. Thus, increasingly, topics such as bodily growth, fecundability, and morbidity appear on the historical demographers' to-do lists (Harris, 2016). But this also brings demographers into the complex debates of "nature versus nurture", or the relative roles of biological versus socio-cultural factors in human health and behavior. To what extent is health, reflected in height or life expectancy, inherited from the parents? And how fast can physical characteristics change? If we do not know this, how can we properly assess the historical impact of, for instance, increased real income or improved hygiene? Such questions inspire research into heritability and into the relative importance of biological factors and environment in explaining persistence of health across generations.

In the past century, the study of heritability had led the life sciences from developing statistical tools to study correlations in traits between kin (for instance by Galton and Pearson) to the ever-expanding search for genetic loci of specific physical or even psychological attributes (Zimmer, 2018). So far, social scientists have been focusing on the changing environmental conditions preventing or allowing people to reach their "genetic potential". In low-income countries, as well as in many historical settings, epidemics, food crises and sudden death of near kin can result in severe health hazards. In such circumstances, the experiences of siblings can differ strongly. Health shocks can therefore result in low inter- as well as low intragenerational correlations in, for instance, height. This is why kin correlations in for example height are expected to be higher in more favorable, "protected" settings and periods. However, fast improvements in socio-economic conditions can benefit especially the younger generation and thus decrease intergenerational correlations in health.

In what environmental conditions do we see an increase in the relative importance of heritability, and to what extent can we even detect the genetic component of this heritability? This article tried — tentatively and experimentally — to answer these questions by looking at an island population that experienced increasing "protection" — but not rapid social change — and by using the genetic variation offered by a variety of kinship ties. The studied health indicators are adolescent and adult height, and life span after age 18. I linked complete (descendant) genealogies of six island families to records of conscripts (age 19) and civic guards (age 25), beginning with the general conscription of 1813. The data allowed me to not only link brothers, but also fathers to sons, grandfathers to grandsons, uncles to nephews and cousins to each other. I investigated associations between heights and life spans of male kin, in order to detect the relative importance of heritability of these traits. By

making use of the degree of kin relatedness, I decomposed the kin effects in associations in genetic factors and shared environment. The latter was approached through similarity in age, socio-economic status of the family of origin, and birthplace.

The height and life span correlations between different types of kin already reveal the importance of shared environment. Brothers, especially when close in age, have much higher correlation coefficients than fathers and sons, even though, technically, the percentage of shared DNA is similar. Also, cousins have much higher coefficients than expected, although this "inflation" has already been noted a century ago. Again, shared environment (by their parents to begin with) seems to be the reason for this. Very interesting are the higher coefficients found in heights at age 19 and 25, as well as in life span correlations for mother's brother, relative to father's brother. Is mother's genetic transmission more stable, as has been asserted? Do we see an effect of mitochondrial DNA or other biological transmission processes which run only through mothers? The latter could be corroborated by the fact that the correlation with mother's sister's sons (in late adolescent height) is also remarkably high. The regression also confirms the impact of the maternal line on height at age 19/20.

Across all kin categories, correlations in late adolescent stature are consistently larger than at age 25. Probably this implies that speed and timing of children's and adolescent's growth have a stronger genetic component than catch-up growth after age 19.

In the second half of the 19th century, the island recovered from the economic stagnation caused by the decline of shipping, while becoming more isolated from contacts with the mainland. I hypothesized that kin correlations in height and life span would be larger for persons born after 1860, for those who were born on the island, and for those growing in farming households. I also expected the genetic component of heritability to be stronger in the more favorable period. Only the regression on life span with weights for the frequency of individual observations confirms this. But although the coefficients, especially of late adolescent height, tend to be somewhat higher, overall the "protected environment" hypothesis could not be confirmed, which is actually in line with recent findings (Black et al. 2023; Jelenkovic et al., 2016b). However, I do not consider this conclusive evidence that this discussion can be brought to a close. A more careful comparison of "environments" is called for, in terms of diseases, real income, family size and housing conditions.

This study proposed a rather experimental use of genealogies for historical demographic and anthropometric analysis. The approach is limited to men, because we only know male stature. In principle, data on life span, SES and birth places of female kin is available, but is not incorporated in the present analysis. A more serious caveat is the choice of this particular locality. This choice was based on the, rather unique, availability of both excellent genealogies and anthropometric data. However, the results should be contrasted with those from more socially diverse and economically deprived settings.

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