The transmission of health across 7 generations in China, 1789-1906

Jean-François Maystadt* and Giuseppe Migali†

June 7, 2021

Abstract

We study the intergenerational transmission of health using registered data from China between 1789 and 1906. We first document the intergenerational correlations in lifespans, and we find much higher correlations for mothers, compared to fathers. We then compare children born from brother and twin fathers, and the intergenerational transmission from fathers becomes weaker and is likely to be mostly driven by genetic factors. On the contrary, our results suggest a strong role of women in affecting their children's health outcomes across generations in developing countries.

JEL Classification: I14, I29, I3

Keywords: Long-term health outcomes, Intergenerational correlations.

*IRES/LIDAM, UCLouvain; FNRS - Fonds de la Recherche Scientifique, Belgium, and Department of Economics, Lancaster University.

†corresponding author: g.migali@lancaster.ac.uk, Department of Economics, Lancaster University Management School, Bailrigg Lancaster LA1 4YX, UK. Dipartimento G.E.S., Universita' Magna Graecia, Catanzaro, Italy.

1 Introduction

Equality of opportunities is often declared as a societal objective. However the extent to which individuals have the opportunity to fulfil their aspirations largely depends on the ability to overcome intergenerational constraints. Economists have long been interested in measuring the intergenerational transmission (IGT) of socio-economic outcomes (Solon, 1999; Black and Devereux, 2010). A key policy interest is to distinguish the role of the environment a child is growing in ("nurture") from the genetic transmission of parents' characteristics ("nature"). Nurture calls for targeted actions aiming at strengthening the initial endowments of individuals in terms of physical and human capital.

In this paper, we estimate the intergenerational elasticity of lifespan between parents and children, using linked registered data from rural China between 1789 and 1906. We find strong intergenerational transmission in health between parents and their sons, in particular for mothers. Controlling for assortative mating, the intergenerational correlations for fathers and mothers stand at about 0.27 and 0.58, respectively. We then compare the outcomes of children born from fathers who are brothers or twins. Interestingly, when comparing children from twin brothers, the intergenerational transmission of health between fathers' and children's lifespans is null, suggesting that it is mostly driven by genetic factors.

Our contribution lies at the crossroad of two strands of literature: first, studies using health outcomes to shed light on intergenerational mobility and, second, studies that highlight the relevance of such mobility in developing countries. While the economic literature in the IGT has focused on earnings, education or welfare dependence (Solon, 1992; Holmlund et al., 2011; Chetty et al., 2014), there is still limited evidence on the intergenerational

transmission of health outcomes. Existing studies establishing a positive intergenerational association in a variety of health outcomes are Currie and Moretti (2007), Classen (2009), Royer (2009), Bhalotra and Rawlings (2011), and Parman (2012). Most of them focus on weight, a relatively short-term and more volatile health outcome. An exception is Bhalotra and Rawlings (2011) who investigate the association between mothers' health and children's anthropometric measurements, together with neonatal, infant and under-five mortality, for 38 developing countries. Beyond the scope of their analysis, a major difference with our study is that they do not attempt to disentangle nurture from nature and focus on the associations between two generations. That is also the case for most of the other studies using weight as a health outcome.[1]

We consider a long-term health outcome, the lifespan, i.e. the approximated number of years between birth and death. As pointed by Parman (2012), lifespan has the major advantage to receive a common interpretation across contexts, time and gender. Parman (2012) is certainly the closest to our work. Using data on North Carolina, he finds very strong intergenerational correlations of lifespans between daughters and mothers and between sons and fathers. In particular with sons, the intergenerational elasticities for fathers and mothers stand at about 0.36 and 0.16, respectively. In our study, we find strong elasticities for mothers, whereas the association with fathers' lifespan is always weaker, especially when the specification is augmented with grand-father or father twin fixed effects, which are more likely to control for unobserved heritable traits ("nature"). Our results also contrast with Lindahl et al. (2016). By exploiting a sample of adoptees and non-adoptees, they find largely

[1]We exploit a parent-twin approach similar to Currie and Moretti (2007) and Royer (2009) among those studies looking at the intergenerational transmission of health.

similar intergenerational transmissions of health between biological mothers and fathers in Sweden. However, they also provide strong evidence of a nurturing role for mothers since the level of education of the adopting mothers has a significant effect on the health of the adoptive children.[2]

Furthermore, the study of intergenerational associations between parents and children is very relevant in developing countries, where imperfect credit and labor markets limit the ability to escape poverty traps across generations. While the literature has reached a relative consensus of an income intergenerational elasticity between 0.3 and 0.45 (Solon, 1999; Chetty et al., 2014), little is known about the magnitude of such correlation in developing countries. The gender dimension is particularly interesting to study, given the potential role of mothers in nurturing children (Duflo 2012). As far as we know, only few recent papers focus on the intergenerational transmission of health in developing countries (Bhalotra and Rawlings, 2011, 2013; Eriksson et al., 2014). As explained above with Bhalotra and Rawlings (2011), one major difference with the existing literature is that we seek to distinguish between nurture and nature, using grand-father and father-twin approaches. Eriksson et al. (2014) use age and gender adjusted average health measures in the parent's province as an instrumental variable, to assess the transmission of health across two generations in China. Compared to their approach, the use of grand-father and father-twin fixed effects does not require exclu-

[2]Our results also echo the biodemographic literature that has exploited historical data on lifespan to assess the inheritance of human longevity (Gavrilov and Gavrilova, 2001). Evidence is mixed with respect to the relative importance of the maternal or paternal lines of inheritance of human longevity (You et al., 2010). Furthermore, most of these studies do not seek to identify the intergenerational transmission of lifespan for a representative sample since they focus on a specific population (e.g. the Landed Gentry, some aristocratic families, centenarians in New England, or Moormons in Utah) or a relatively small area.(e.g. the Connecticut Valley, the French Jura, a Flemish village in Belgium, or a village in Sardigna). With the use of grand-father and father twin fixed effects, we offer a more credible identification strategy to distinguish nurture from nature and shed light on the paternal components of the nurturing effect for sons.

sion restrictions and the results are not driven by local average treatment effects (Black and Devereux, 2010). One of the strengths of our analysis is to consider successive generations, to assess the relevance of the AR(1) model and the stability of the IGT across generations. Furthermore, our paper does not only contribute to the more established literature on intergenerational transmission but also to the more recent advancements on multigenerational transmission (Solon, 2018; Lindahl et al., 2015; Anderson et al., 2018; Barone and Mocetti, 2020).

2 Background and data

We investigate the intergenerational transmission of health in the Liaoning province between 1789 and 1906. This province is located in North-East China (Figure B.1) and was the original home of the Manchu Qing dynasty emperors (1644-1912), the last dynasty of imperial China. Most of the eighteen century was seen as a period of political stability and economic expansion (Wong, 1997; Pomeranz, 2000; Meng Xue and Koyama, 2016) but signs of economic decline emerged toward its end under the Qianlong Emperor (1735-1796). However, the Qing ruling was hardly challenged during the nineteenth century, which is the focus of the present study. Following the First Opium War and the Treaty of Nanking (1842), political instability exacerbated and materialized in a series of popular uprisings including the Taiping rebellion (1850-1864) and the Dungan Revolt (1862-1872). Such revolts were largely driven by opposition to an autocratic and state-controlled regime, or what Chesneaux (1973) names as "bureaucratic feudalism". "In China the state was all-powerful and the peasant was as much exploited by the public demands of the state and bureaucracy as he was by the

individual greed of the landlord" (Chesneaux, 1973, 11). Such bureaucracy provided a wealth of administrative data to be exploited in social sciences. We, indeed, use the China Multi-Generational Panel Dataset-Liaoning (CMGPD-LN) that relies on population records that have been directly transcribed from the so-called Eight Banner population registers preserved in the Liaoning Provincial Archives (see Lee et al. (2010) which is the official user guide for this data). The "Eight Banner" – originally the military arm of the Manchus – was a civil and military administrative system organized by the Qing dynasty.

Being an extremely important feature of late imperial China, such registers document in great detail the demographic, economic and social life of the population during that period (Lee et al., 2010). The data comprise 29 sets of household registers from the Qing Imperial Household Office and there are 1.5 million records. Missing data are reported between 1888 and 1903 because the corresponding registers were destroyed by fire (Lee et al., 2010). Since we are interested in the lifespan, we transform the original data into a cross-section of precisely 266,164 unique individuals that belong to 1,063 distinct descents (family trees). They are followed through the registers every three years up to seven generations, between 1749 and 1909, in 13 districts of the Liaoning province (Lee et al., 2010; Lee and Campbell, 2011; Song et al., 2015). We construct the lifespan of each individual as the difference between the last year observed in the register and the year of birth. Lee et al. (2010) indicate that the year of birth is sometimes missing or badly recorded. We drop 4,791 individuals for which the year of birth and sex is not recorded. The major drawback of our lifespan definition is the omission of children dying in infancy and in early childhood (potentially before 3 years old since registry takes place every 3 years) and the possibility of unrealistic high lifespans on the other end of the distribution. To reduce the second problem, we first exclude 2,730

individuals registered as unauthorised migrants (so called 'Tao'), since their records seem too poor to be included in mortality analysis (Lee et al., 2010).

We further restrict the sample by excluding 46,004 individuals that entered into the registers prior to 1789. Before this year, the registers did not identify properly residential households and did not uniformly distinguish villages (Lee et al., 2010). We also exclude 11,579 individuals who were alive in 1909, since lifespan cannot be approximated for them. We then top code the lifespan to 75, since mortality record has been recognized as problematic for age above 75 (Dong and Lee, 2014). The resulting data consists of information on about 201,060 individuals covering up to seven generations of the same families. We assess the importance of our sample restrictions in Section 4.

The data offer other information on individual or household characteristics, such as sex, relationships within the household in each registry, district in which the village of residence is located, migration experiences. Little information is given on socio-economic characteristics of the individuals. However, as explained by Lee et al. (2010, p.7), the "overwhelming majority of the populations were hereditary peasants who provided labor and fixed rents in kind in return for land rights and other privileges".

The patrilineality in Chinese social organization is well documented by Song et al. (2015) and Campbell and Lee (2004) where sons were seen as more valuable than daughters. Understanding the role of women in the intergenerational transmission of health, in this highly patriarchal society, is of key interest for contemporaneous economic development. Furthermore, little role was played by the state in providing public goods, with the exception of enforcing order and some form of justice (Rankin, 1990). Due to a systematic policy of literary inquisitions from 1600 to 1788 (Xue and Koyama, 2021), human capital formation was

mainly a private or, at best, a communal matter during the period of our investigation. Clans were mainly providing financial support for informal and private tutoring to prepare their members for imperial examination (Hao and Xue, 2017). Based on the voluntary support of local elites, local charities also multiplied in imperial China, providing aid towards the most in need, such as the impoverished widows and orphans (Smith, 1987; Rankin, 1990; Xue and Koyama, 2021). However, prior to 1905, individual households or extended families were responsible for basic education and the provision of other public goods (Rankin, 1990). The imperial exam system was abolished only in 1905 and modern education began to expand when the Ministry of Education, the Offices of Provincial education and the county-level agencies, known as Education Exhorting Offices, were established (Xu et al., 2013; Hao and Xue, 2017).

Our sample

We use the obtained sample of 201,060 individuals to generate the lifespan and characteristics of parents (including mothers) and grandparents (including grand-mothers). Only sons can be linked to their fathers, mothers and paternal ancestors because daughters were expected to leave the family after marriage and become member of their husband's family (Wakefield, 1998). As illustrated in Figure B.2, only never married daughters or daughters who get married but do not have children are linked to their natural parents, however, since there are very few observations in those 2 categories we have to omit daughters from our analysis. This restriction reduces the analytical sample to 110,844 males (sons). Furthermore, in our data, the percentage of re-marriages is very low and there is no polygyny, which overall is known to be almost inexistent (Lee et al. 2010). Consequently, we are confident that the

wives are almost always the natural mothers of their children.

The top two panels of Figure 2 report the average lifespan over time by year of last appearance, for males and their parents. The bottom two panels show the average lifespan computed using a restricted definition, obtained as the difference between the year of the register in which the individual is reported to have died during the three years covered by that register and the year of birth.[3] The restricted definition will be also used for robustness purposes in Section 4.

Table 1 and Figure 1 illustrate the structure of a descent (family tree), showing how 110,844 individuals can be linked to 16,025 mothers and 11,889 fathers, to 7,907 grand-mothers and 5,445 grand-fathers, to 1,721 great-grand-mothers and 2,885 great-grand-fathers, to 1,627 great-great-grand-fathers, to 918 great-great-great-grand-fathers and, finally, to 566 great-great-great-great-grand-fathers. These numbers do not add up to the total number of individuals since the same individual represented as a child at one point in time may become father, grand-father, or have another family position in subsequent years. Contrary to Lindahl et al. (2008), the family status (father, grand-father, ...) does not necessarily coincide with a particular generation. For instance, an individual recorded as a father can be in any generation of the family tree. Furthermore, we only consider individuals for whom we can construct the lifespan. Among fathers we have more missing observations on their year of birth, thus mothers are over-represented despite the structure of the data. Unfortunately, for the same reason we are forced to omit daughters, it is impossible to identify mothers' parents. This implies that for mothers we can only know their parents in law.[4]

[3]We only have this information in the GMGPD-LN for a few individuals. That is why, in our main analysis, we approximate the lifespan using the difference between the last year observed in the register and the year of birth. We discuss the risk of measurement errors in Section 4.

[4]For interpretative purposes, we should bear in mind that the (great-) grand-mothers are (grand-) mothers

We should also note that the gain in lifespan between the individuals and their parents is an artefact of the data structure (by construction, an individual cannot die in childhood and become a parent). As indicated below Table 1, restricting the sons to future fathers would give an average lifespan of 50 years. The same explanation applies for the differences in lifespan with subsequent ancestors. However, this trend may also be partly explained by the decreasing lifespan observed during the period of investigation (Figure 2).[5] A decline in the standard of living during the Qing dynasty has been well documented (Allen et al., 2005; Campbell and Lee, 2004; Broadberry et al., 2017).

3 Methodology

3.1 Intergenerational correlations

To motivate our analysis beyond two generations, we will first adopt various specifications of the following model (Lindahl et al., 2015; Clark and Cummins, 2015):

$$LS_{it-l} = \alpha + \theta_t + \mu_d + \beta_j LS^{m,f}_{it-j} + \epsilon_i \tag{1}$$

where $l = 0 \dots 4$ and $j = 1 \dots 5$. With $l = 0$ and $j = 1$, the main coefficients of interest β_1 captures the elasticity between the (log) lifespan of individual i, LS_{it}, and the one of his

of the fathers and not of the mothers.

[5] Another explanation is that we may oversample short-lived people in more recent registers by excluding individuals still alive in 1909. In Section 4.4, we will assess the robustness of our results to the exclusion of those born from 1830 to give all individuals the opportunity to reach 75 by 1906. We can already note that the bottom part of Figure 2 shows a similar decline when using a stricter definition of lifespan, based on the few individuals in the CMGPD-LN indicated to die between two registers. We will further use that stricter definition of lifespan in Section 4.3. Such definition is also used in mortality studies by Campbell and Lee (2004), Campbell and Lee (2009) and Dong and Lee (2014).

parent (mother or father), $LS^{m,f}_{it-1}$. With $l = 0$ and $j > 1$, we can assess the direct effects of grand-fathers (LS^{f}_{it-2}), up to great-great-grand-fathers (LS^{f}_{it-4}), and grand-mothers (LS^{m}_{it-2}) up to great-great-grand-mothers (LS^{m}_{it-4}). In all our specifications, standard errors are clustered at the descent level (family tree). In the less restrictive specification, we are exploiting 803 descent groups for clustering the standard errors. We introduce a time indicator to capture registry-specific effects, θ_t. This corresponds to the year of first appearance of the individual in the registry. We also control for unobserved heterogeneity at district level with district fixed effects, μ_d.

To assess the relevance of the AR(1) model, we follow Lindahl et al. (2015) by comparing the direct effects obtained in equation (1) to the estimates of the intergenerational correlation across two consecutive generations (i.e $j - l = 1$). We therefore compute the predictions of the intergenerational transmission of health between a child and his ancestors, up to five generations apart based on the AR(1) estimates from consecutive generations. Similar to Lindahl et al. (2015), the associated standard errors are obtained using the Delta method. The difference between the direct effects and the respective predictions will give a sense of the direct impact of ancestors on the lifespan of the child. Such comparison cannot distinguish between the nurture and nature channels of transmission, but will help us to explore the extent to which we may underestimate the intergenerational elasticity by focusing on only two generations. The recent literature has indeed shown that estimates obtained from two generations severely underestimate the long-run intergenerational persistence in socio-economic outcomes across generations (Long and Ferrie, 2013; Lindahl et al., 2015; Clark and Cummins, 2015).

We will further investigate the AR(1) stability of the parent-son transmission of health

across 7 generations within the same family tree. More specifically, we use a specification similar to equation (1):

$$LS_i^g = \alpha + \theta_t + \mu_d + \beta_j LS_i^{m,f,g-1} + \epsilon_i \tag{2}$$

In short, we assess the association between the lifespan of parents and sons by pairs of generation. The index g denotes the generation of the sons, from 2 to 7.[6] One strength of our analysis is to be able to distinguish the effect of time (captured by register-specific year fixed effects) and the effect of generations, since all family trees do not start from the same register.

Based on this first empirical exploration of the AR(1) model, we will further compare it with more complex models, from AR(2) up to AR(5), of the following form:

$$LS_{it} = \alpha + \theta_t + \mu_d + \sum_j^4 \gamma_{1,j} LS_{it-j}^m + \sum_j^5 \gamma_{2,j} LS_{it-j}^f + \epsilon_i \tag{3}$$

The same description of fixed effects apply, and standard errors are also clustered at the descent level. In equation (3), since data are only available up to great-great-grand-mothers, we are able to control for assortative mating up to that level. The direct effects for great-great-great-grand-fathers are likely to include the indirect effect of their partners.

[6]The first generation is defined by the first household head appearing in each family tree. On average, the first generation was born in 1764. His descendants are then forming the successive generations within each family tree. We should nonetheless be cautious in comparing our results across generations since the sample of extended families with multiple generations (e.g. those with a seventh generation) may differ from other households for unobserved reasons.

3.2 The sibling and twin father approaches applied to registry data

Equation 3 can be simplified to $j = 1$ to allow for the identification of the nurture channel of the intergenerational transmission of health between parents and their children. The IGT may be largely driven by inherited genetic differences across families. In the absence of observed genomic information, the literature has largely relied on parental sibling and parental twin approaches, which consist in eliminating bias inherent to the presence of unobserved fixed family characteristics. In other words, using the parental sibling approach, we compare the intergenerational transmission of health among children who have the same grand-father. With the twin approach, by comparing children of identical twin parents we aim to both isolate the IGT from fixed family characteristics, and also remove (to a large extent) the variation in IGT due to genetic differences. More precisely, the various specifications take the following form:

$$LS_i = \alpha + \theta_t + \mu_d + \varphi_k + \beta_1 LS_i^m + \beta_2 LS_i^f + \gamma \mathbf{X_i} + \delta \mathbf{Z_i} + \epsilon_i \tag{4}$$

LS_i still denotes the (log) lifespan of the individual i, while LS_i^m and LS_i^f denote the lifespans of his mother and father, respectively. All our specifications also include time (θ_t) and district fixed effects (μ_d). Standard errors are clustered at the descent level (family tree). The coefficients β_1 and β_2 should measure the effect of the IGT mother-son and father-son, respectively.

Compared to the basic AR(1) specification ($j = 1$ in equation 3), in equation (4) we add

individual characteristics X_i to control for observed heterogeneity. Specifically, we include variables such as being disabled, being a migrant during the course of the individual's life, birth order, size of the household, number of brothers and sisters at the approximate time of birth, and the occurrence of natural disasters in the year before birth and during childhood (first 10 years of life). Within-household composition at the time of birth is likely to matter, since the position within the household has been found to be highly correlated with the risk of mortality in Campbell and Lee (2004) and Campbell and Lee (2009), based on our same data. These variables are further described in the Appendix A, with descriptive statistics in Table B1. Our results are always presented with and without control variables to limit the risk of bad controls (Angrist and Pischke, 2013). To draw causal inference, we include in equation (4) additional fixed effects, φ_k, and we follow two approaches.

We first use grand-father fixed effects to remove unobserved time-invariant family characteristics. With the second approach, we include father-twin fixed effects, to better control for nature. Although we also take into account of assortative mating, we should acknowledge that twin approach does not net out all genetic endowments.[7] We can only control for the genetic influence arising from the Y chromosomes, only transmitted from fathers to sons. Indeed, the patrilineal nature of our data does not allow us to identify mother twins, and the genetic component coming from mothers (through X chromosomes) cannot be controlled for (Holmlund et al., 2008; Amin et al., 2015).

Our twin approach relies on two main identifying assumptions. First, siblings born

[7]Inversely, we may also net out common nurturing experiences among twins and their consequences on their sons. However, if we restrict our analysis to non-twin fathers who are close in age (incrementally from 2 to 5 years difference), thus assuming they share the same childhood environment, we find a stable, positive and significant IGT effect. Hence, we are confident that the shared environment effect is not cancelling out the effect of genes.

the same year and of the same sex are considered as twins. In our analysis, we exploit 736 father-twin fixed effects. The interpretation of our results depends on the assumption that twin fathers are identical in their endowments. This is only true with monozygotic twins, compared to dizygotic twins who share on average 50 percent of the genes. A major drawback of our analysis is that we cannot be certain whether those same-year newborns are also monozygotic twins (or in particular circumstances even twins). Thus, our twin fixed effects may not completely remove the genetic component of the IGT between fathers and sons. We will therefore apply a bounding exercise proposed by Holmlund et al. (2008, 58-61). They indeed demonstrate that we can obtain proper identification without having information about monozygotic (MZ) and dizygotic (DZ) twins. The adjusted estimate is defined as:

$$\widehat{\beta_{TS}} = \frac{\widehat{\beta_{TW}} - \lambda\theta\widehat{\beta_{SIB}}}{1 - \lambda\theta} \tag{5}$$

where $\widehat{\beta_{TW}}$ and $\widehat{\beta_{SIB}}$ denote the estimates obtained using the father twins fixed effects and the father siblings fixed effects specifications, respectively. θ represents the share of DZ twins among all twin pairs. Similar to Holmlund et al. (2008), θ is approximately 0.5 in samples, like ours, that do not separate MZ from DZ twins. λ is an indicator for possible treatment differentials between twin and non-twin sibling parents. Under the assumption of absent treatment differentials ($\lambda = 1$), $\widehat{\beta_{TS}}$ provides a lower-bound estimate of the causal intergenerational transmission of health between parents and children. If twins are treated more similar than non-twin siblings, ($\lambda < 1$), the intergenerational transmission of health between fathers and sons should increase. In such a case, the estimate converges towards

the father twin fixed effects.

The second key identifying assumption is that (father) twins are treated similarly within their family after birth. This assumption would not be valid if (grand-) parents make compensating or reinforcing investments or if children's outcomes are affected by both the twin father and his partner. We cannot exclude such unobserved heterogeneity between father twins. However, we reduce that threat in auxiliary specifications controlling for father characteristics, Z_i, notably fathers' disability, age at approximated birth (and its square), and being a migrant during the course of his life.[8]

4 Results

4.1 Intergenerational correlations across generations

Table 2 provides the AR(1) estimates of the intergenerational elasticity between two consecutive generations, together with the estimates of the direct impacts of ancestors on the child lifespan (equation 1). The intergenerational correlations for father and mother stand at about 0.51 and 0.70, respectively (columns 1 and 2). These broad correlations suggest a low level of mobility in China during the Qing dynasty and a higher transmission of health between mothers and sons. The rest of Table 2 reveals a few interesting results. We find a small direct effect from grand-father (column 3) and we do not find any direct effect from great-grand-father (column 5), and great-great-grand-father (column 7). We are therefore

[8]We can dismiss differential treatment based on inheritance laws and customs since land was equally divided among the sons (Wakefield, 1998). Compensating mechanisms based on birth weights have been also found in other contexts by Oreopoulos et al. (2008). On the contrary, Behrman et al. (1994) point to reinforcing mechanisms. Unfortunately, our dataset does not allow us to control for individual and parental birth weight.

unlikely to underestimate the intergenerational transmission of health between fathers and sons in AR(1) models. On the contrary, column (4) reveals that there is a statistically significant direct effect (0.672) between grand mothers' lifespan and grandchild. The corresponding predicted association is lower. However its value, 0.409 (0.696×0.586), is based on the correlations between mothers and sons (0.696) and the AR(1) estimate (0.586) between mother and her mother in law. Hence, we might underestimate the true IGT between a daughter and her natural mother. A direct effect – larger than the predicted associations of 0.233 and 0.061 – is also found for great-grand-mother and great-great-grand-mother (columns 6 and 8). Overall, given the significant direct effect of female ancestors, and due to the limitations in computing precise predictions for them, the AR(1) model seems to be more appropriate for fathers.

Investigating the stability of the parent-child transmission of health across seven generations within the same family tree, Figure 3 suggests that the estimated coefficients for mothers and their relative differences with fathers remain stable overtime. For fathers, the elasticity is never significantly different from zero up to generation four, and afterwards we observe an increase. This implies a slightly higher IGT between the generation of sons born on average in 1865 and their fathers born on average in 1836.[9]

The direct effect played by grand-mothers is further confirmed when we extend the analysis by implementing AR(2) and AR(3) models in Table 3, where the estimated coefficients stand in a range between 0.29 (column 2) and 0.32 (column 3). Such result is consistent with Lindahl et al. (2015) who find that grandparents have an independent generational ef-

[9]We cannot exclude that our results may capture the non-monotonic effect of a distant event (Nybom and Stuhler, 2014), such as the Taipings wars (1850-1864) that took place in Southern China and led to the loss of 25 million lives (Yu, 2012).

fect on grandchildren, although they do not find significantly different correlations between grandfathers and grandmothers. The fact that almost all coefficients are not statistically significant in the AR(4) and AR(5) models is certainly due to the small sample size, i.e. 134 and 83 observations, respectively.[10]

Overall, Table 2 and 3 provide suggestive evidence that women matter much more than men for the intergenerational transmission of health in rural China between 1789 and 1906. In particular, mothers and grand-mothers seem to bear an important direct role on children's well-being. Indeed, grandmothers (precisely, paternal grandmothers) are more likely to live in the household with their grandchildren compared to great-grandmothers. Such results echo Zeng and Xie (2014) who find that the educational level of co-resident grandparents directly affects the educational level of their grandchildren. A similar effect is not found for non-resident grandparents. Nonetheless, this evidence does not allow us to claim any causal relationships since inherited genetic differences may inflate the intergenerational transmission of health. However, it is informative in shedding light on the possible cost associated with limiting our analysis to an AR(1) model. Even with this upward endogeneity bias, we know that the AR(1) model is not underestimating the intergenerational elasticity between fathers and sons, however it may underestimate that between mothers and sons.

4.2 The nurturing role of fathers

Results. To distinguish nurture from nature in the intergenerational transmission of health between fathers and sons, we restrict our analysis to two generations, the so-called AR(1)

[10]To make our results comparable across specifications, Table B2 shows similar results when the sample is restricted to the AR(3) model.

model. In Table 4, we report the estimated coefficients β_1 and β_2 of equation (4) using several specifications augmented with a large set of fixed effects. In Panel A, the estimation is conducted using only lifespan of fathers. In Panel B, we also include the lifespan of mothers to account for assortative mating.[11]

In column (1), Panels A and B, of Table 4, we reproduce the AR(1) models similar to Table 2 (column 1) and Table 3 (column 1), respectively. The intergenerational transmission of health for fathers is 0.51, and then reduces to 0.27. Assortative mating clearly matters. The magnitude of the estimates for fathers (0.27) provided in Panel B by the AR(1) model is in the same range of what has been found for earnings in the US, the UK or Nordic countries in more contemporaneous times (Solon, 1999; Holmlund et al., 2011; Parman, 2012; Chetty et al., 2014).

The intergenerational association between mothers and sons stands at about 0.58. The higher elasticities between sons and mothers, compared to those between sons and fathers contrast very much with the existing literature. For instance, Holmlund et al. (2011) find a higher association between fathers' and children's education (0.25), than the one for mothers (0.20). Compared to a closer literature on the IGT of health, Parman (2012) also finds higher elasticities between sons and fathers (0.359) than between sons and mothers (0.157). Our results are nonetheless largely consistent with the strong correlation between mothers' and children's health found in developing countries (Bhalotra and Rawlings, 2011, 2013; Classen, 2009). However, those papers do not provide estimates for the patrilineal linkage, with the one exception of Eriksson et al. (2014). The latter exploit health information for

[11] We provide the detailed results of specifications with control variables (columns 3, 5, and 6) of Panel B of Table 4 in Table B3 in the Appendix. In Table B4, we harmonize the samples across specifications shown in Table 4. Results are qualitatively unaltered.

both mothers and fathers in rural China between 1991 and 2009, and find a slightly higher intergenerational correlations for fathers (0.298) than mothers (0.272).

Columns (2) and (3) of Table 4 refer to the grand-father fixed effect model, while the last three columns to the twin father fixed effect model. While assortative mating still matters, the addition of grand-father fixed effects almost halves the intergenerational transmission of health for fathers. Unobserved family characteristics have clearly a role to play in driving social mobility in this context. The gap between the elasticities for fathers and for mothers resists to the addition of individual characteristics (column 3). Comparing then children whose fathers share the same genetic background (column 4), the intergenerational elasticity between fathers and sons stands at 0.11, and is not statistically different from zero. In column (5), the introduction of individual characteristics does not only show a non-significant coefficient for the father's lifespan but its size is basically null. The lack of causal intergenerational effect between fathers and sons contrasts with the existing literature using a parent-twin approach. According to recent reviews (Black and Devereux, 2010; Holmlund et al., 2011; Amin et al., 2015), the corresponding intergenerational effects between fathers and sons range between 0.07 and 0.48 (average of 0.23), with systematically larger elasticities for fathers, compared to mothers.[12] In our study, nurture does not seem to explain the IGT of health between fathers and sons, which appears to be mostly driven by genetic factors.

Identifying assumptions. The interpretation of the estimated coefficients in columns (4), and (5) of Table 4 strongly relies on the assumption that twin fathers would be treated

[12]As far as we know, Currie and Moretti (2007) and Royer (2009) are the only papers applying a twin approach for health outcomes. However, the comparison is limited since they do not consider the father-son relationship.

in a similar way after birth. We cannot directly observe investment made by grand-parents into father twins' human capital. However, column (6) of Table 4 shows that our conclusions remain qualitatively unchanged when adding father control variables. The intergenerational association between fathers and sons remains null and far from being significant.

As described in Section 3, one remaining concern is that we cannot be completely certain that our twin fathers are effectively twins, and even less monozygotic. To deal with this issue, we implement the bounding exercise proposed by Holmlund et al. (2008). Similar to these authors, in Table B5, we only focus on the specification without controls.[13] The upper-bound estimate of the IGT of health between fathers and sons is obtained using equation (5). Assuming absence of treatment differentials between twin and sibling fathers ($\lambda = 1$), Table B5 indicates an IGT lower bound of about 0.09 (column 3). We can therefore conclude that had we been able to identify and use only MZ twins, our analysis would have confirmed a causal intergenerational elasticity not significantly different from zero. As expected, if we relax the assumption of absent treatment differentials ($\lambda < 1$) in columns (4) to (7), the IGT converges towards the father twin fixed effect estimate.

Finally, the interpretation of our results also relies on the random nature of parental twinning. Such assumption has been recently questioned by Bhalotra and Clarke (2016), based on individual data from 72 countries between 1972 and 2012. In our study, father twinning does not seem to be correlated with the lifespans of their mothers or their fathers (see Table B6).

[13] Results with controls are qualitatively similar.

4.3 Measurement errors

Measurement errors may be due to i) the way we approximate the lifespan of the individuals recorded in the population registers; or ii) missing observations for unrecorded deaths. To explore the role of measurement errors and to shed light on the mechanisms of our results, we estimate equation 4 with alternative samples. We implement these robustness checks replicating Table 4. The results are reported in Table B7.

First, we may wrongly infer that someone is dead when he is not observed anymore in a register. However, the coefficient of correlation between the approximated lifespan and the *restricted* lifespan, computed for those we know they die between two registers, stands at 0.98. This is a strong indication that attrition is likely to be a minor issue. The graphical representations in Figure 2 are also almost identical when comparing the 'broad' and the restricted lifespan. When using the son's restricted lifespan to estimate equation 4, the gap between the elasticities for mothers and fathers is confirmed (compare Panel A of Table B7 and Panel B of Table 4). We observe some variations, upwards for mothers and downwards for fathers, likely driven by the sharp reduction in sample sizes.[14]

One additional concern may also be related to higher errors in assigning paternity than maternity. That would artificially generate a smaller intergenerational coefficient for fathers than for mothers. Nonetheless, Panel B of Table B7 downplays that concern. We replicate our main results restricting the sample to first and second sons, for which the paternity is more certain than for later offspring. Such a sample restriction barely makes any difference to our main results.

[14]In the CMGPD-LN, the information on individuals who died during the three years covered by a register is only available for about 14% of the analytical sample. Given the small sample size, in Panel A of Table B7 we can only use the strict definition of lifespan for sons, and not for parents.

We also investigate the importance of migration. Although we control for *observed* migration and exclude unauthorized migrants whose records are recognized as being very poor, we cannot exclude that *unobserved* migration biases our results. To make an educated guess of the likely bias, we first use the characteristics at birth of the *observed* migrants to identify potential migrants (*unobserved*). Employing a simple nearest-neighbor matching technique (Heckman et al., 1997), about 7 percent of our analytical sample is matched to *observed* migrants.[15] We then replicate our main estimations in Panel C of Table B7, excluding these potential migrants. The results are largely unchanged and confirm the low level of physical mobility reported in the historical background of this study.

Second, bias may arise due to unrecorded early mortality. The difference with the approximated lifespan issue is that early dying children are never recorded in the registers. These missing observations are more likely to occur for children dying before the age of six, "since many parents did not register their children until after they had survived to around age 5" (Lee et al. 2010: 19). Since early mortality is more prevalent in poor households (van den Berg et al., 2006) — where parents' lifespan is likely to be low —, the resulting selection may create a downward bias in our estimates of the intergenerational transmission of health. We can make an educated guess of that potential bias, by excluding from our analytical sample those boys that we observe dying before 6. Panel D of Table B7 confirms that our main results may underestimate the intergenerational elasticities by the omission of children dying in early childhood.[16] Had we observed all children dying at early age, our

[15]The propensity score is based on the following co-variates: household size at birth, number of brothers and sisters at birth, birth order, and earlylife and antenatal shocks.

[16]Conclusions are unaltered when using a higher threshold at 10 or 15 years old, instead of 6 years old. Note that we cannot really deal with measurement errors arising when individuals died in-between registers and whose death year is attributed to the last year. The introduction of register year fixed effects would deal with the possibility that such measurement error would be systematic in some particular registers due

coefficients would be higher in magnitude.[17]

4.4 Truncation and censoring

Censored and truncated observations are well-known problems in biodemography (Gavrilov and Gavrilova, 2001). First, our results may be biased in favor of shorter-lived persons in more recent years. This may happen because, by excluding from our analysis those alive in 1909, for whom the lifespan is not yet available in the data, we may omit longer-lived people. Hence, we have conducted a survival analysis to evaluate whether our restrictions on lifespan may introduce bias. In particular, we have adopted semi-parametric, discrete time, hazard models (Jenkins, 2005) to estimate the probability to die for an individual who is (right) censored at year 1909. Table B9 presents the results with and without controls for the individual (columns 2 and 3) and parental characteristics (column 3). The latter include paternal and maternal characteristics, such as mother's disability, age at approximated birth (and its square), and the fact to be a migrant during her course of life. We find that longer parental lifespan reduces the probability to die, and the effect of the mother is higher. Focusing on the hazard ratio, one extra year of father's and mother's life would translate into a decrease of their son's probability to die by about 1.8% and 2.4%, respectively. Comparison with our main results is limited since the survival analysis cannot directly control for any genetic effect. However, these estimates are of similar magnitude, although on the upper hand, if compared with Lindahl et al. (2016). These authors find that one extra year of life

to common shocks. In our specifications, we do not see any obvious reason why such measurement errors could not be considered as random and would introduce any significant endogeneity bias.

[17]The logarithm transformation may give more weights to those dying early in life (down-weighting higher lifespans). To test this hypothesis we have, first, re-estimated our main models in Table 4 and then repeated the robustness check in Panel D of Table B7, without a logarithm transformation of the lifespan. The results, reported in Table B8 and Panel E of Table B7, respectively, are similar to those with a logarithm lifespan.

for one biological parent decreases the (non-adopted) child's risk of death by about 1%.[18] One difference with Lindahl et al. (2016)'s results for non-adopted children and biological parents is the fact our that findings confirm a stronger association for mothers compared to fathers.

Furthermore, we have re-estimated the father-twin fixed effects models, excluding in a backward fashion individuals born before 1906 up to 1830. In the most restricted case, when we exclude all those born after 1830, we give the same chance to all individuals to reach the maximum lifespan by 1906. Figure 4 shows that the intergenerational transmission of health between parents and sons remains fairly stable across the cohorts born between 1860 and 1906.[19] The causal elasticity between fathers and sons is confirmed to be null. When we exclude those born before 1860 up to 1830 and despite the sharp reduction in sample sizes, we witness a further widening of the coefficients obtained for mothers and fathers.

Second, censored observations result from several sample restrictions — explained in Section 2 — such as the exclusion of those entering the registers prior to 1789 and the top coding of the lifespan at 75. Such restrictions may result into selected samples, hence we proceed to several additional analyses to assess their importance. We first replicate our main results not imposing any restriction on our sample, i.e. keeping those entering the registers prior to 1789, keeping those alive in 1909, and not top coding lifespan above 75. The coefficients reported in Panel A of Table B10 are slightly higher than the corresponding in Panel B of Table 4, which might therefore be lower-bound estimates. The conclusions

[18]The magnitude of our effects for mothers are actually closer when Lindahl et al. (2016) use a predicted age at death for biological parents. However, we could not apply the same method since we do not have enough health-related information during the course of parents' life.

[19]Such stability also constitutes an indirect evidence that our results are not affected by the destruction by fire of registers between 1888 and 1903 or by the first sino-Japanese war (1894-1895).

are nonetheless unaltered. Panels B and C of Table B10 confirm the robustness of our main results, when imposing one or two of our sample restrictions. Panel D include all our sample restrictions and, therefore, duplicates our main results.

4.5 The role of mothers

Our results indicate that the intergenerational correlation between fathers and sons is mostly driven by unobserved genetic factors. The patrilineal structure of our data does not allow us to identify mother twins. The intergenerational transmission of health between mothers and sons can still be driven by genetic factors. Nonetheless, the difference in the intergenerational correlations obtained for mothers and fathers (net of family unobserved fixed effects) is puzzling enough to explore further the role of mothers. Our findings, indeed, contrast very much with the literature, in particular with the studies on the intergenerational transmission of education in developed countries (Behrman and Rosenzweig, 2002; Black and Devereux, 2010; Holmlund et al., 2011; Pronzato, 2012). One explanation may be that in developing countries mothers have been found to impact more on children's welfare, in particular on their health (Duflo, 2012).[20] However, the stronger intergenerational transmission for mothers may

[20]A legitimate concern relates to the external validity of our results. While the one-child policy implemented after 1979 has radically changed demographics in China, our results are relevant for other developing countries, above all for those societies with agricultural dominance. In fact, the population in our study is mostly composed by hereditary peasants, working mainly for the imperial estate. Although they received larger initial land allotments from the government, compared with peasants elsewhere (Dong and Lee 2014), they appear to be fairly representative of the Northeastern Banners Population administered by the Eight Banner. The latter is a civil and military administrative system organized by the Qing Dynasty (1644-1911) to govern the Manchurian and Mongolian provinces in the greater North and Northeast China as well as the Qing garnison population in China Proper. The Northeastern Banners lived in the area that today corresponds to the three provinces of Liaoning, Jilin and Heilongjiang (Lee et al, 2010: 5). It is difficult to know whether the validity of our results may be extrapolated beyond these three provinces, nevertheless our data document the demographic, economic, and social life at a greater level of detail and context than virtually any other late imperial local society (Lee et al. 2010). Similar to Lindahl et al. (2015), we compare the health distribution of the individuals of our sample to the one of the China Health and Nutrition Survey from 1991 and 2015, a survey representative of 15 Chinese provinces (including Liaoning). Figure B.2

receive different interpretations.

First, mother's lifespan may capture differences in initial endowments at times of marriage, because in our models we are estimating the difference in health transmission between a mother and the wife of her husband's brother. Unfortunately, we do not observe the resources transferred by women at marriage. Historical evidence points to the disconnection of married women with their original family (Wakefield, 1998; Lee et al., 2010), but no information is collected about dowry agreed at times of marriage. An omitted variable bias may arise if such initial endowments are correlated with both mothers' and sons' lifespans. Although imperfect as a test, we assess whether our results would change if we control for maternal characteristics. In particular, we re-estimate the regressions presented in Panel B of Table 4 augmenting column (6) with both paternal and maternal characteristics.[21] The results reported in column (6) of Panel A of Table B11 do not differ from our main findings.

Second, orphanhood may drive our results. It is plausible that mothers' death matters more for the child's survival at young age, compared to fathers' death. For instance, Beegle et al. (2010) find maternal orphanhood, and not paternal orphanhood, to have a permanent adverse effect on children's health and educational attainment in rural Tanzania. The same is true for education in South Africa (Case and Ardington, 2006) or in Kenya (Evans and Miguel, 2007). In Panel B of Table B11, we exclude children whose mothers died before they reached the age of 6. Our analysis indicates that the main coefficient of interest for mothers

shows estimates of the Lorenz curves based on both datasets. As expected, the results suggest the health distribution from our historical data is more unequal compared to the contemporaneous and national health distribution. Nonetheless, our results may gain relevance for contemporaneous China in the context of the relaxation of the one-child policy.

[21] The intergenerational correlations in health between mothers and sons are presented in column 1 of Table B.10. However, adding father-related fixed effects allows to assess the sensitivity of the mother coefficient to the control of her husband's unobserved family characteristics.

only slightly decreases. Little change is also observed when we exclude fathers dying before the child has reached the age of 6 (Panel C of Table B11). Hence, orphanhood does not seem to capture much of the role played by mothers.

Third, we may also capture a statistical artefact that women die at a younger age. Even assuming that the health transmission is equally shared between fathers and mothers, it could be artificially stronger for mothers than for fathers, simply because women (in particular in poor households) tend to die earlier. To explore this channel, we replicate our main estimations by splitting the sample between parents dying below or above the median lifespan. Panels D and E of Table B11 report the results for the case of mother's and father's lifespans below the median, respectively. Although we observe an increase in the magnitude of the intergenerational elasticity for both parents (columns 1-3), compared to Panel B of Table 4,[22] the transmission of health between mothers and sons remains much higher than the one between fathers and sons.

Fourth, common shocks and stresses may constitute an alternative mechanism. For instance, if mothers and children are more exposed to the same environment (e.g. house-related damages, air pollution), our estimates would capture the consequences of these experiences rather than the effect of mothers.[23] Controlling for the occurrence of natural disasters would not be localized enough to remove such unobserved heterogeneity between mothers and sons. We do not have information on the location of the sons when they died, but we can conjec-

[22] In columns (4) to (6) of Panel D, the transmission of health between mothers and sons is not higher for women dying earlier, on the contrary it becomes insignificant. However, that may be driven by the reduction in sample sizes.

[23] An alternative explanation for the strong association between mother's and son's health could be a more direct biological link between mothers and children (in the womb). Conditions in utero have indeed been found to have long lasting effects on later life health (e.g. the hypothesis formulated by Barker (1992)) (e.g. Bhalotra and Rawlings 2011, 2013).

ture that children dying at younger age are more likely to have been confronted to similar parental environmental factors than children dying at older age. We cannot even observe when parents and sons split in terms of household structure. However, household divisions have been documented to be most likely when sons get married (Wakefield, 1998). From our data, we know that, on average, sons aged 18 are likely to marry within the next 3 years. As an approximation, we therefore exclude those dying after the age of 20 and we re-estimate Panel B of Table 4. Looking at the results in Panel F of Table B11, contrary to our expectations, the magnitude of the IGT under the grand-father fixed effects models remains similar to the one in our benchmark model (Panel B of Table 4), and the difference between the IGT from fathers and mothers is still striking. Results obtained under the father-twin fixed effects models are unconclusive, since the lack of precision may be largely driven by the reduction of the sample sizes to 772 observations. Overall, these findings reject our initial conjecture that the effects would be much stronger for this sub-sample.

Overall, the contrast between mothers and fathers in terms of intergenerational transmission of health highlights a considerable and stable difference across alternative samples. This consistency in our findings only constitutes an indirect way to interpret the mechanisms behind the strength of the intergenerational transmission for mothers. Nonetheless, the evidence is sufficiently compiling to support a strong effect for mothers, in contrast to fathers.

5 Conclusions

In this paper, we study the intergenerational transmission of health using linked registered data from China between 1789 and 1906. Our preferred specifications rely on comparing children with the same grand-father and those from twin fathers. The intergenerational association from fathers is weak and seems to be entirely driven by genetic factors. The marginal nurturing effect of the father cannot be explained in our analysis by the limit of the two-generations model, since grand-fathers and further ancestors do not seem to have any major direct impact on the children's health. On the contrary, we find a strong and consistent (although not causal) intergenerational elasticity between mothers and their children, standing at about 0.4.

Our results contrast with the existing literature that seems to point to a stronger role played by fathers in the intergenerational transmission of socio-economic outcomes, such as earnings, education or health. Our analysis suggests that the existing literature cannot necessarily be generalized to developing countries, where women play a prominent role in affecting children's welfare, in particular health outcomes. However, we are aware that our study is not being able to control for genetic endowments on the mothers' side, and that more research, using quasi-experimental approaches, would be needed to be more affirmative on the subject. It would also be interesting to know whether the higher intergenerational elasticity between women and sons is confirmed in developing countries when a twin approach is applied on health or other outcomes, and when the approach is extended to the parents-daughters relationships.

Acknowledgement

We thank Cameron Campbell, Seema Jayachandran and participants in the International Workshop on Applied Economics of Education (Catanzaro, Italy), the Economics seminar at the University of Ferrara (Italy), the LICOS 25 years workshop at KU Leuven (Belgium), the first Dondena Workshop on Public Policy at Bocconi University (Italy), the EEA-ESEM Lisbon 2017, the Economics seminars at the University of Luxembourg and the University of Caen, the NEUDC 2017 (Boston), and the Christmas meeting for Belgian Economists (Leuven) for their comments and suggestions.

References

Allen, R. C., Bengtsson, T., and Dribe, M. (2005). *Living standards in the past: New perspectives on well-being in Asia and Europe*. Oxford University Press on Demand.

Amin, V., Lundborg, P., and Rooth, D.-O. (2015). The intergenerational transmission of schooling: Are mothers really less important than fathers? *Economics of Education Review*, 47:100–177.

Anderson, L., Sheppard, P., and Monden, C. (2018). Grandparent effects on educational outcomes: A systematic review. *Sociological sciences*, 5:114–142.

Angrist, J. D. and Pischke, J. S. (2013). *Mostly harmless econometrics: an empiricists companion*. Princeton: Princeton University Press.

Barker, D. J. P. (1992). Fetal and infant origins of adult disease. *BMJ*.

Barone, G. and Mocetti, S. (2020). Intergenerational mobility in the very long run: Florence 1427-2011. *The Review of Economic Studies*.

Beegle, K., de Weerdt, J., and Dercon, S. (2010). Orphanhood and Human Capital Destruction: Is There Persistence into Adulthood. *Demography*, 47(1):163–180.

Behrman, J. R. and Rosenzweig, M. R. (2002). Does Increasing Women's Schooling Raise the Schooling of the Next Generation? *American Economic Review*, 92(1):323–334.

Behrman, J. R., Rosenzweig, M. R., and Taubman, P. (1994). Endowments and the allocation of schooling in the family and in the marriage market: The twin experiement. *Journal of Political Economy*, 102(6):1131–1174.

Bhalotra, S. and Rawlings, S. (2011). Intergenerational persistence in health in developing countries: The penalty of gender inequality. *Journal of Public Economics*, 95:286–299.

Bhalotra, S. and Rawlings, S. (2013). Gradients of the intergenerational transmission of health in developing countries. *Review of Economics and Statistics*, 95(2):660–672.

Bhalotra, S. R. and Clarke, D. (2016). The twin instrument. *Centre for the study of African economies, WPS*, 38.

Black, S. and Devereux, P. J. (2010). Recent developments in intergenerational mobility. Working Paper 15889, NBER.

Broadberry, S., Guan, H., and Li, D. D. (2017). China, europe and the great divergence: A study in historical national accounting. *Warwick Working paper*, 324:1–61.

Campbell, C. D. and Lee, Z. J. (2004). Mortality and Household in Seven Liaodong Populations, 1749-1909. In Bengtsson, T., Campbell, C. D., and Lee, Z. J., editors, *Life under pressure: Mortality and living standards in europe and Asia, 1700-1900*, chapter 10, pages 293–324. Cambridge, MA: MIT Press.

Campbell, C. D. and Lee, Z. J. (2009). Long-term mortality consequences of childhood family context in Liaoning, China, 1749-1909. *Social Science and Medecine*, 68:1641–1648.

Case, A. and Ardington, C. (2006). The Impact of Parental Death on School Outcomes: Longitudinal Evidence from South Africa. *Demography*, 43:402–420.

Chesneaux, J. (1973). *Peasants revolts in China*. London: Thames and Hudson.

Chetty, R., Hendren, N., Kline, P., and Saez, E. (2014). Where is the land of Opportunity? The Geography of Intergenerational Mobility in the United States. *The Quarterly Journal of Economics*, 129(4):1553–1623.

Clark, G. and Cummins, N. (2015). Intergenerational Wealth Mobility in England 1858 − 2012: Surnames and Social Mobility. *The Economic Journal*, 125(582):61–85.

Classen, T. (2009). Measures of the intergenerational transmission of body mass index between mothers and their children in the United States, 1981 2004. *Economics and Human Biology*, 8:30–43.

Conley, D., Strully, K. W., and Bennett, N. G. (2006). Twin differences in birth weight: the effects of genotype and prenatal environment on neonatal and post-neonatal mortality. *Economics & Human Biology*, 4(2):151–183.

Currie, J. and Moretti, E. (2007). Biology as Destiny? Short- and Long-Run Determinants of Intergenerational Transmission of Birth Weight. *Journal of Labor Economics*, 25:231–264.

Dong, H. and Lee, J. Z. (2014). Kindship Matters: Long-term mortality consequences of childhood migration, historical evidence from northeast China, 1792-1909. *Social Science and Medecine*, 119:274–283.

Emran, M. S., William, G., and Shilpi, F. (2018). When Measure Matters. Coresidency, Truncation Bias, and Intergenerational Mobility in Developing Countries . *Journal of Human Resources*, 53(3):589–607.

Eriksson, T., Pan, J., and Qin, X. (2014). The intergenerational inequality in China. *China Economic Review*, 31:392–409.

Evans, D. and Miguel, E. (2007). Orphans and Schooling in Africa: A Longitudinal Analysis. *Demography*, 44:35–57.

Gavrilov, L. and Gavrilova, N. (2001). Biodemographic Study of Familial Determinants of Human Longevity. *Population: An English Selection*, 13(1):197–222.

Hao, Y. and Xue, M. M. (2017). Friends from Afar: The Taiping rebellion, Cultural proximity and primary School in the Lower Yangzi, 1850-1949. *Explorations in Economic History*, (63):44–69.

Heckman, J. J., Ichimura, H., and Todd, P. E. (1997). Matching as an Econometric Evaluation Estimator: Evidence from Evaluating a Job Training Programme. *The Review of Economic Studies*, 64(4):605–654.

Holmlund, H., Lindahl, M., and Plug, E. (2008). The Causal Effect of Parents' Schooling on Children's Schooling: A Comparison of Estimation Methods. Discussion Paper 3630, IZA.

Holmlund, H., Lindahl, M., and Plug, E. (2011). The Causal Effect of Parents' Schooling on Children's Schooling: A Comparison of Estimation Methods. *Journal of Economic Literature*, 49(3):615–651.

Jenkins, S. P. (2005). Survival analysis. Unpublished manuscript, Institute for Social and Economic Research, University of Essex, Colchester, UK.

Lee, Z. J. and Campbell, C. D. (2011). China multi-generational panel dataset, Liaoning (CMGPD), 1749-1909. Dataset 2011-09-02, Ann Arbor, MI: Inter-university Consortium for Political and Social Research.

Lee, Z. J., Campbell, C. D., and Chen, S. (2010). China multi-generational panel dataset, Liaoning (CMGPD), 1749-1909. User Guide. User guide, Ann Arbor, MI: Inter-university Consortium for Political and Social Research.

Lindahl, M., Lundberg, E., Palme, M., and Simeonova, E. (2016). Parental Influences on Health and Longevity: Lessons from a Large Sample of Adoptees. *NBER Working Paper*, 21946.

Lindahl, M., Palme, M., Massih, S. S., and Sjogren, A. (2015). Long-Term Intergenerational Persistence of Human Capital: An Empirical Analysis of Four Generations. *Journal of Human Resources*, 50(1):1–33.

Long, J. and Ferrie, J. (2013). Intergenerational Occupational Mobility in Great Britain and the United States since 1850. *American Economic Review*, 103(4):1109–1137.

Meng Xue, M. and Koyama, M. (2016). Autocratic rule and social capital: Evidence from Imperial China. Technical report, Mimeo.

Nybom, M. and Stuhler, J. (2014). Interpreting trends in intergenerational mobility. *Swedish Institute for Social Research working paper*.

Oreopoulos, P., Stabile, M., Walld, R., and Roos, L. L. (2008). Short-, Medium-, and Long-Term Consequences of Poor Infant Health: An Analysis Using Siblings and Twins. *The Journal of Human Resources*, 43(1):88–138.

Parman, J. (2012). Gender and Intergenerational Mobility: Using Health Outcomes to Compare Intergenerational Mobility Across Gender and Over Time. Working Paper 122, College of William and Mary.

Pomeranz, K. (2000). *The Great Divergence, China, Europe and the making of the modern world economy*. Princeton, New Jersey: Princeton UP.

Pronzato, C. (2012). An examination of paternal and maternal intergenerational transmission of schooling. *Journal of Population Economics*, 25(2):591–608.

Rankin, M. B. (1990). The Origins of a Chinese Public Sphere. Local Elites and Community Affairs in the Late Imperial Period. *Etudes chinoises*, 9(2):13–60.

Royer, H. (2009). Separated at Girth: US Twin Estimates of the Effects of Birth Weight. *American Economic Journal: Applied Economics*, 1(1):49–85.

Smith, J. F. H. (1987). Benevolent Societies: The Reshaping of Charity During the Late Ming and Early Ch'ing. *The Journal of Asian Studies*, 46(2):309–337.

Solon, G. (1992). Intergenerational Income Mobility in the United States. *American Economic Review*, 82(3):393–408.

Solon, G. (1999). Intergenerational mobility in the labor market. In Ashenfelter, O. and Card, D., editors, *Handbook of Labor Economics*, volume 3, Part A, chapter 29, pages 1761–1800. Elsevier, 1 edition.

Solon, G. (2018). What do we know so far about multigenerational mobility. *Economic Journal*, 128(612):F340–F352.

Song, X., Campbell, C. D., and Lee, J. Z. (2015). Ancestry Matters: Patrilineage Growth and Extenction. *American Sociological Review*, 80(3):574–602.

van den Berg, G. J., Lindeboom, M., and Portrait, F. (2006). Economic conditions early in life and individual mortality. *The American Economic Review*, 96(1):290–302.

Wakefield, D. (1998). *Fenjia : Household Division and Inheritance in Qing and Republican China*. University of Hawaii Press.

Wong, R. B. (1997). *China Transformed: Historical Change and the Limits of European Experience*. Ithaca, NY: Cornell University Press.

Xu, Y., Foldvari, P., and Van Leeuwen, B. (2013). Human capital in Qing China: economic determinism or a history of failed opportunities? *Mimeo*.

Xue, M. M. and Koyama, M. (2021). Autocratic rule and Social capital: Evidence from Imperial China. *Mimeo*.

You, D., Gu, D., and Yi, Z. (2010). Familial Transmission of Human Longevity Among the Oldest-Old in China. *Journal of Applied Gerontology*, 29(3):308–332.

Yu, M. (2012). *The Taiping Rebellion: A Military Assessment of Revolution and Counter-revolution*. University Press of Kentucky.

Zeng, Z. and Xie, Y. (2014). The effects of grandparents on children's schooling: Evidence from rural China. *Demography*, 51(2):599–617.

Figure 1: Tree representing the patrilineal structure of the data and observed generations

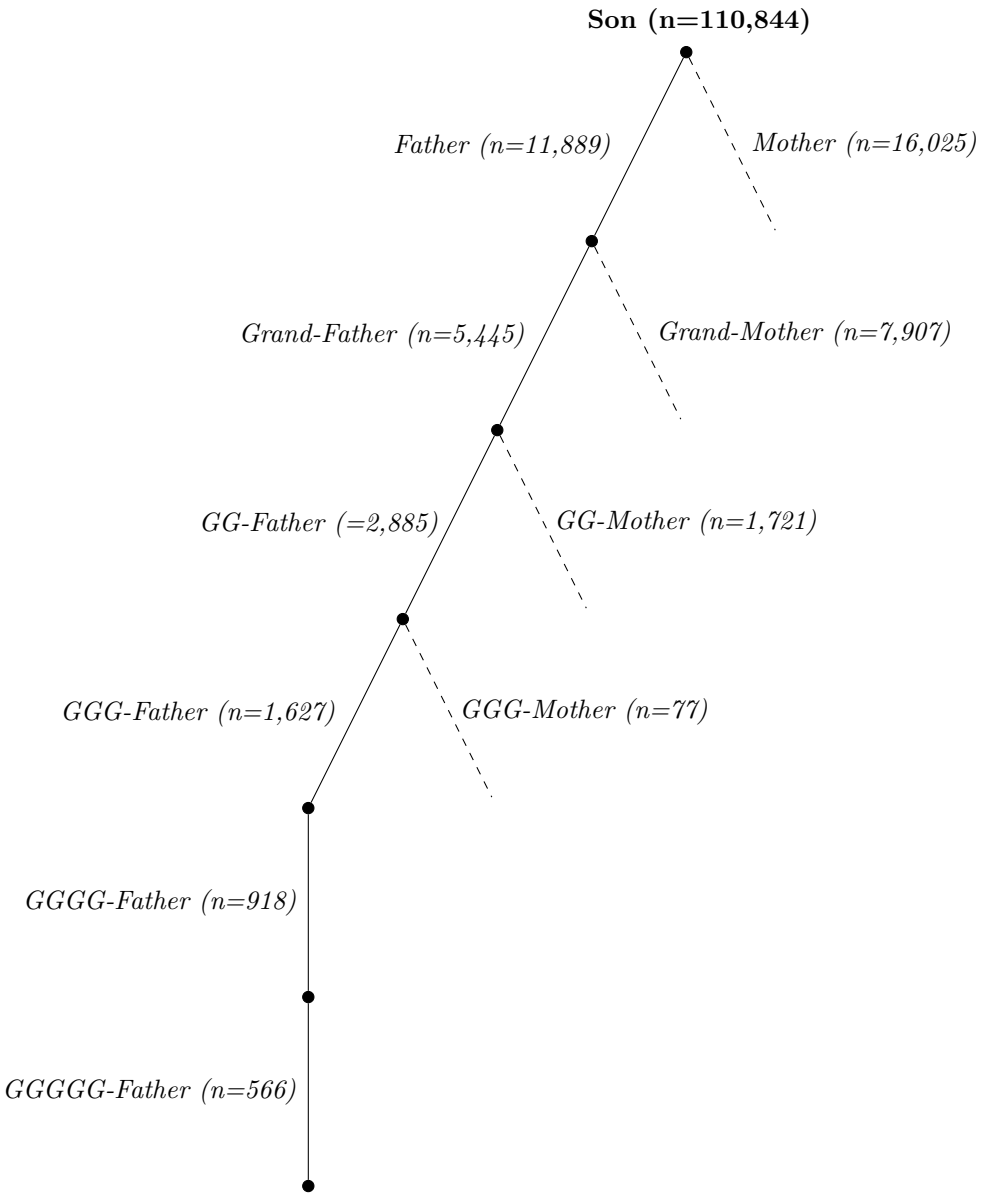
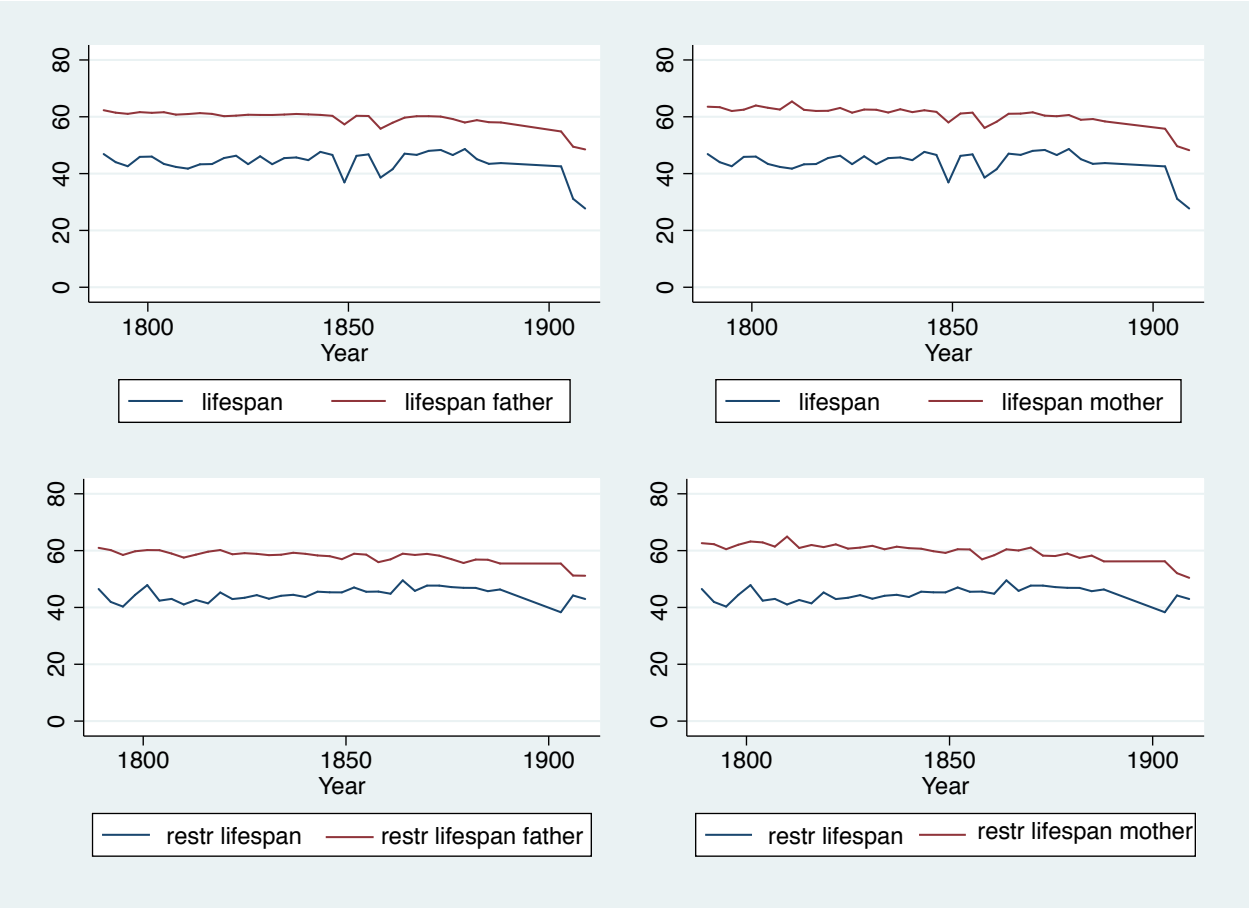
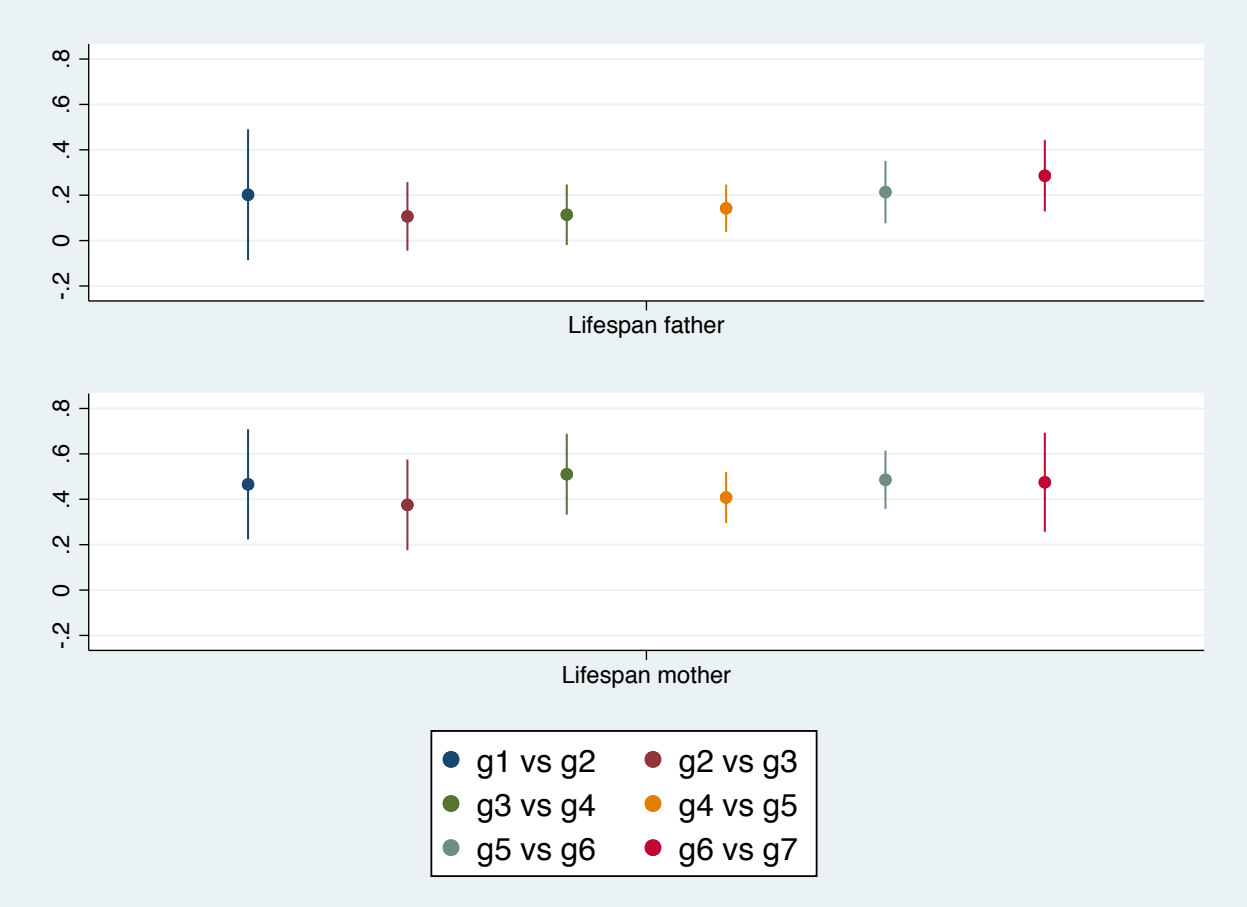


Figure 2: Lifespan over time - 1789-1906



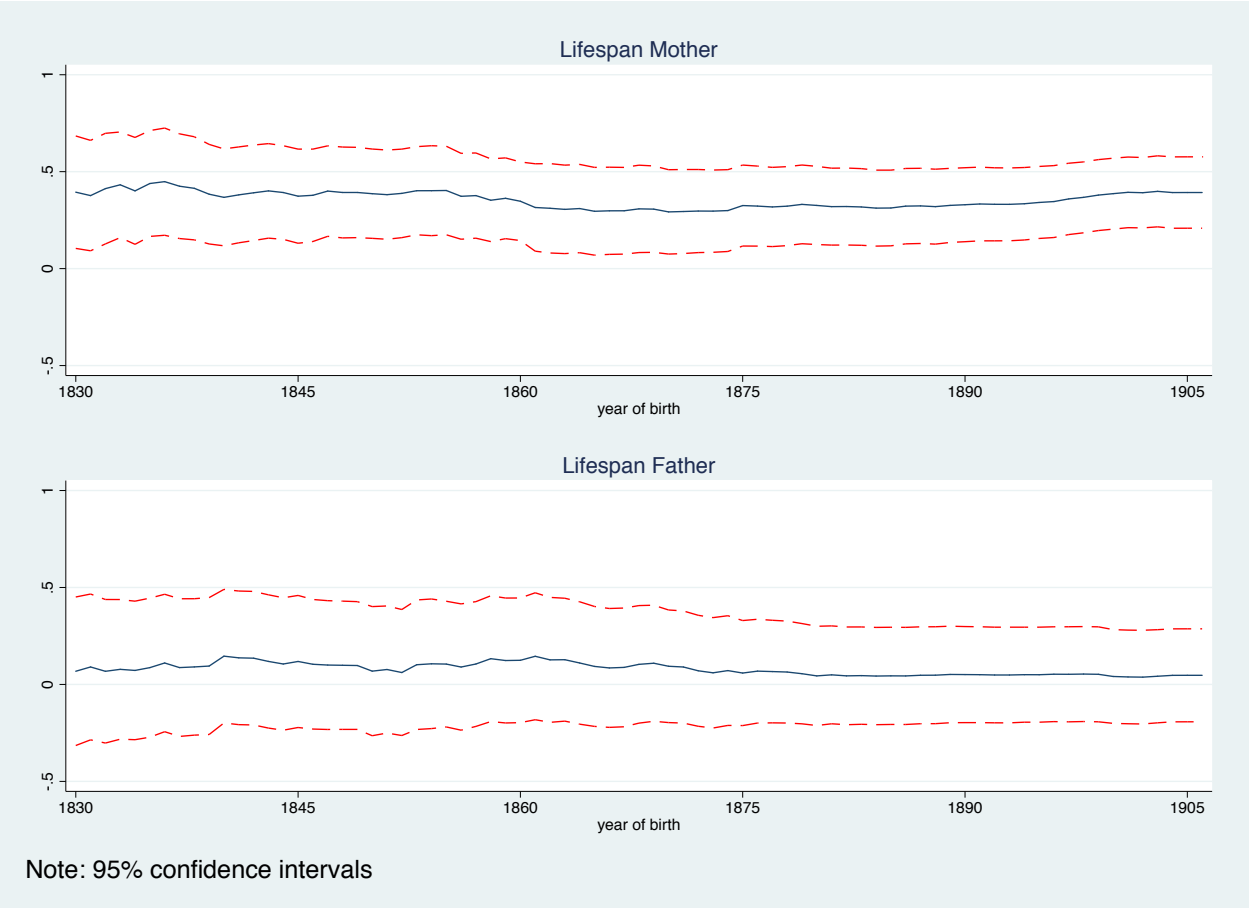
Note: The top figures present the lifespans of sons, fathers and mothers overtime while lifespan is constructed as the difference between the last year observed in the registers and the year of birth. The bottom figures show the same trends but when the lifespan is constructed (for a sub-sample) as the difference between the year of birth and the year of the register in which the individual is reported to have died within the next three years.

Figure 3: IGT across 7 generations



Note: We report the estimated coefficients and confidence intervals (95%) for father's lifespan (top figure) and mother's lifespan (bottom figure) obtained by estimating AR(1) models across 7 generations.

Figure 4: IGT across cohorts 1830-1906 - Twin fathers estimation



Note: We report the estimated coefficients and confidence intervals for mother's lifespan (top figure) and father's lifespan (bottom figure) obtained by estimating father-twin fixed effects models, excluding in a backward fashion those born before 1906 up to 1830. All models also include year and district fixed effects, individual characteristics together with a control for assortative mating (partner's lifespan).

Table 1: Descriptive statistics

Variable	*Mean*	*Std. Dev.*	*N*
Lifespan males	35.777	22.141	110844
Lifespan mother	41.126	13.903	16025
Lifespan father	43.384	15.446	11889
Restricted Lifespan			
Lifespan males	40.784	23.338	30020
Lifespan mother	40.354	15.251	4615
Lifespan father	46.214	17.541	2762
Lifespan ancestors			
Lifespan Gmother	62.035	10.489	7907
Lifespan Gfather	55.908	13.591	5445
Lifespan GGmother	71.472	6.028	1721
Lifespan GGfather	59.697	13.048	2885
Lifespan GGGmother	80.805	8.415	77
Lifespan GGGfather	61.310	12.773	1627
Lifespan GGGGfather	61.619	12.432	918
Lifespan GGGGGfather	64.610	13.327	566

Averages for parents and ancestors exclude duplicates.
Note: if we restrict the males sample only to those that will be fathers (49714) the average lifespan
increases to 50.225 (s.e 15.996).

Table 2: IGT across generations

	LogLifespan							
				Paternal ancestor				
	father	mother	Gfather	Gmother	GGfather	GGmother	GGGfather	GGGmother
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Dependent variables:								
LogLifespan child	0.508***	0.696***	0.103***	0.672***	0.009	0.468***	-0.005	0.643*
se	(0.025)	(0.027)	(0.013)	(0.043)	(0.015)	(0.131)	(0.017)	(0.318)
N	99969	97812	91043	52700	73659	7589	51791	297
LogLifespan father			0.155***					
se			(0.013)					
N			10709					
LogLifespan mother				0.586***				
se				(0.019)				
N				8881				
LogLifespan Gfather					0.061***			
se					(0.016)			
N					4648			
LogLifespan Gmother						0.571***		
se						(0.079)		
N						1061		
LogLifespan GGfather							0.045*	
se							(0.026)	
N							1580	
LogLifespan GGmother								0.260*
se								(0.132)
N								27
predictions			0.078***	0.409***	0.004	0.233***	0.000	0.061*
se			(0.008)	(0.021)	(0.004)	(0.017)	(0.000)	(0.034)

Each estimates is from a separate regression of the loglifespan of a family member on the lifespan of an older member.
All models include Year and District fixed effects.
Standard errors clustered at descent level. Significance levels: *** 1% ** 5% * 10%
Predictions are obtained by the product of the IGC estimates of consecutive generations.
Standard errors of prediction are computed using the Delta method.

Table 3: AR models of IGT across generations

Dependent variable:	LogLifespan				
	AR(1)	AR(2)	AR(3)	AR(4)	AR(5)
LogLifespan mother	0.580***	0.479***	0.407***	0.015	0.548
	(0.022)	(0.026)	(0.057)	(0.448)	(0.513)
LogLifespan father	0.265***	0.223***	0.216***	0.173	0.144
	(0.017)	(0.022)	(0.046)	(0.217)	(0.367)
LogLifespan Gmother		0.292***	0.324***	1.653**	1.328**
		(0.034)	(0.082)	(0.763)	(0.589)
LogLifespan Gfather		0.037**	0.073*	-0.101	-0.217
		(0.014)	(0.042)	(0.229)	(0.224)
LogLifespan GGmother			0.084	-0.570	0.488
			(0.141)	(0.667)	(0.422)
LogLifespan GGfather			0.005	0.490**	0.118
			(0.044)	(0.232)	(0.229)
LogLifespan GGGmother				0.040	-0.307
				(0.616)	(0.662)
LogLifespan GGGfather				0.018	-0.248
				(0.305)	(0.427)
LogLifespan GGGGfather					0.480
					(0.325)
N	96572	49762	5989	134	83

All models include Year and district FE.
Standard errors clustered at descent level.
Significance levels: *** 1% ** 5% * 10%.

Table 4: IGT estimates with grand-father and twin-father fixed effects

Dependent variable:	Log Lifespan					
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Father LS only						
LogLifespan father	0.508***	0.256***	0.270***	0.191	0.120	0.104
	(0.025)	(0.023)	(0.022)	(0.120)	(0.123)	(0.132)
N	99969	83719	83719	3236	3236	3236
Panel B: Father and mother LS						
LogLifespan mother	0.580***	0.408***	0.409***	0.405***	0.393***	0.388***
	(0.022)	(0.023)	(0.023)	(0.094)	(0.094)	(0.094)
LogLifespan father	0.265***	0.142***	0.166***	0.114	0.047	0.043
	(0.017)	(0.021)	(0.020)	(0.120)	(0.122)	(0.132)
N	96572	83719	83719	3236	3236	3236
Year and District FE	✓	✓	✓	✓	✓	✓
Individual characteristics			✓		✓	✓
Father characteristics						✓
GF FE (nbr 21282)		✓	✓			
Twin father FE (nbr 736)				✓	✓	✓

Standard errors clustered at descent level.
Significance levels: *** 1% ** 5% * 10%.
Models are restricted to equivalent sample sizes including individual characteristics.

For Online Publication

Separate Appendixes with Supplemental Material for:

The transmission of health across 7 generations in China, 1789-1906

Appendix A Description of control variables

Being disabled during the course of one individual's life is recorded in early registers for "health conditions such as consumption, paralysis, insanity, retardation and blindness to more exotic injuries such as tiger bites. Late registers do not provide such detail, but do identify males exempted from duty because of chronic illness" (Lee et al. 2010: 22).

Being a migrant during the course of one individual's life is defined for about 21,000 individuals in the CMGPD-LN dataset. One strength of the dataset is to be able to trace migrants since individuals in the CMGPD-LN dataset "are defined and organized by hereditary institutional affiliation, so that individuals retain their institutional affiliation and continue to be listed in the same register in the same order even when they move to another community." (Lee et al. 2010: 23). That is certainly an advantage given the coresidency bias in estimating the intergenerational regression coefficient in coresident samples, since the coresidency criterion usually used in standard household surveys generates a truncated sample (Emran et al., 2018).

Birth order is computed based on the calculated years of birth of all siblings sharing the same father.

Size of the household is based on the number of live individuals present in the household in the current register.

Number of brothers and sisters at the approximate time of birth is based on the number of siblings of a particular sex living in the household in the current register.

The occurrence of natural disasters in the year before birth and during childhood is constructed based on the list of events (floods, droughts, and earthquake) by year

provided in the CMGPD-LN dataset. Such a list is compiled from "Gazeteers published by the Liaoning Government Local History Office" (Lee et al. 2010: 94).

Appendix B Tables and Figures

Table B1: Descriptive Statistics of control variables

	Mean	**Std. Dev.**
disabled	0.046	0.209
hh size	15.962	13.742
N. brothers	0.954	1.229
N. sisters	0.114	0.413
migrant	0.091	0.287
early life shocks	2.216	2.187
antenatal shocks	0.379	0.764
birth order 1	0.458	0.498
birth order 2	0.267	0.442
birth order 3	0.138	0.345
birth order 4	0.068	0.252
birth order 5	0.032	0.177
birth order 6	0.016	0.125
father disabled	0.128	0.334
father migrant	0.127	0.333
age at son's birth	21.919	12.441
mother disabled	0	0.011
mother migrant	0.099	0.298
age at son's birth	26.855	9.903

Analytical Sample AR(1) model size 96,572.

Table B2: AR models of IGT across generations - AR(3) size

Dependent variable:	LogLifespan				
	AR(1)	AR(2)	AR(3)	AR(4)	AR(5)
LogLifespan mother	0.446***	0.408***	0.407***	0.015	0.548
	(0.056)	(0.056)	(0.057)	(0.448)	(0.513)
LogLifespan father	0.260***	0.217***	0.216***	0.173	0.144
	(0.045)	(0.046)	(0.046)	(0.217)	(0.367)
LogLifespan Gmother		0.332***	0.324***	1.653**	1.328**
		(0.081)	(0.082)	(0.763)	(0.589)
LogLifespan Gfather		0.075*	0.073*	-0.101	-0.217
		(0.042)	(0.042)	(0.229)	(0.224)
LogLifespan GGmother			0.084	-0.570	0.488
			(0.141)	(0.667)	(0.422)
LogLifespan GGfather			0.005	0.490**	0.118
			(0.044)	(0.232)	(0.229)
LogLifespan GGGmother				0.040	-0.307
				(0.616)	(0.662)
LogLifespan GGGfather				0.018	-0.248
				(0.305)	(0.427)
LogLifespan GGGGfather					0.480
					(0.325)
N	5989	5989	5989	134	83

All models include Year and district FE and restricted to the sample with AR(3).
Standard errors clustered at descent level.
Significance levels: *** 1% ** 5% * 10%.

Table B3: IGT estimates with GF and twin-parent fixed effects[a]

Dependent variable:	Log Lifespan					
	(1)	(2)	(3)	(4)	(5)	(6)
LogLifespan mother	0.580***	0.408***	0.409***	0.405***	0.393***	0.388***
	(0.022)	(0.023)	(0.023)	(0.094)	(0.094)	(0.094)
LogLifespan father	0.265***	0.142***	0.166***	0.114	0.047	0.043
	(0.017)	(0.021)	(0.020)	(0.120)	(0.122)	(0.132)
disabled			0.458***		0.329***	0.328***
			(0.018)		(0.064)	(0.063)
Log(household size)			-0.150***		0.023	0.024
			(0.014)		(0.040)	(0.040)
n. of brothers			0.027***		-0.004	-0.003
			(0.006)		(0.026)	(0.026)
n. of sisters			0.090***		0.072	0.071
			(0.016)		(0.064)	(0.065)
migrant			0.715***		0.480***	0.572***
			(0.034)		(0.068)	(0.072)
earlylife shock			0.015***		0.005	0.007
			(0.004)		(0.012)	(0.013)
antenatal schocks			-0.003		-0.033	-0.033
			(0.006)		(0.026)	(0.026)
birth order 1			0.347***		0.522***	0.499***
			(0.040)		(0.188)	(0.190)
birth order 2			0.245***		0.373**	0.354*
			(0.036)		(0.181)	(0.182)
birth order 3			0.191***		0.288*	0.269
			(0.033)		(0.168)	(0.168)
birth order 4			0.144***		0.263	0.249
			(0.032)		(0.165)	(0.166)
birth order 5			0.114***		0.288	0.278
			(0.032)		(0.177)	(0.177)
birth order 6			0.054*		0.213	0.210
			(0.031)		(0.144)	(0.144)
father disabled						0.002
						(0.069)
father migrant						-0.251***
						(0.083)
father's age at son's birth						-0.005
						(0.006)
father's age^2 at son's birth						0.000
						(0.000)
N	96572	83719	83719	3236	3236	3236
R2	.3471417	.5309148	.5564008	.5113415	.5390209	.5411287
Year and District FE	✓	✓	✓	✓	✓	✓
Individual characteristics			✓		✓	✓
Father characteristics						✓
GF FE (n. 21282)		✓	✓			
Twin father (n. 736)				✓	✓	✓

[a] Full table (panel B, Table 4).
Standard errors clustered at descent level.
Significance levels: *** 1% ** 5% * 10%.

Table B4: IGT estimates with grand-father and twin-father fixed effects - same size

Dependent variable:	Log Lifespan					
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Father LS only						
LogLifespan father	0.569***	0.408**	0.472***	0.126	0.004	-0.008
	(0.077)	(0.166)	(0.173)	(0.154)	(0.153)	(0.166)
N	2958	2958	2958	2958	2958	2958
Panel B: Father and mother LS						
LogLifespan mother	0.677***	0.419***	0.461***	0.394***	0.373***	0.372***
	(0.071)	(0.121)	(0.125)	(0.105)	(0.103)	(0.102)
LogLifespan father	0.342***	0.295*	0.355**	0.078	-0.040	-0.043
	(0.071)	(0.154)	(0.157)	(0.149)	(0.148)	(0.161)
N	2958	2958	2958	2958	2958	2958
Year and District FE	✓	✓	✓	✓	✓	✓
Individual characteristics			✓		✓	✓
Father characteristics						✓
GF FE (nbr 21282)		✓	✓			
Twin father FE (nbr 736)				✓	✓	✓

Standard errors clustered at descent level.
Significance levels: *** 1% ** 5% * 10%.
Models are restricted to equivalent sample sizes including GF and Twin FE.

Table B5: IGT estimates adjustment

Dependent variable:	Log Lifespan						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
			$\lambda = 1$	$\lambda = 0.75$	$\lambda = 0.50$	$\lambda = 0.25$	$\lambda = 0.1$
LogLifespan father	0.142***	0.114	0.086	0.097	0.105	0.110	0.113
	(0.046)	(0.120)	[0.240]	[0.192]	[0.160]	[0.137]	[0.126]
GF FE	✓						
Twin father		✓					
$\theta = 0.5$			✓	✓	✓	✓	✓

Standard errors in parentheses clustered at descent level. Standard errors in brackets are computed following Conley et al. (2006) using the pooled variance and taking the square root of $V(\widehat{\beta_{TS}} = \frac{V(\widehat{\beta_{TW}})(\frac{1}{1-\theta})^2(N_{TW}-1)+V(\widehat{\beta_{SIB}})(\frac{\theta}{1-\theta})^2(N_{SIB}-1)}{N_{TW}+N_{SIB}})$
θ is the share of dizygote over monozygote twins.
Year and District FE included. Control for assortative mating.
Significance levels: *** 1% ** 5% * 10%.

Table B6: Effect of parents lifespan on twins probability

Dependent variable:	Twin Father	
	(1)	(2)
LogLifespan mother	0.009	0.009
	(0.006)	(0.07)
LogLifespan father	0.003	0.003
	(0.004)	(0.004)
N	29163	29163
Parents characteristics		✓

All models include year, birth year and district FE.
Standard errors clustered at descent level.
Controls: father migrant and disabled, mother migrant.
Significance levels: *** 1% ** 5% * 10%.

Table B7: Measurement errors. IGT estimates with alternative samples

Dependent variable:	Log Lifespan					
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Using the strict definition of son's lifespan						
LogLifespan mother	0.516***	0.352***	0.332***	0.866***	0.719***	0.685***
	(0.030)	(0.062)	(0.060)	(0.249)	(0.227)	(0.228)
LogLifespan father	0.170***	0.108	0.104	-0.258	-0.237	-0.231
	(0.030)	(0.071)	(0.069)	(0.273)	(0.284)	(0.321)
N	25207	16400	16400	720	720	720
Panel B: Only with first and second birth sons						
LogLifespan mother	0.612***	0.459***	0.435***	0.416***	0.405***	0.400***
	(0.022)	(0.029)	(0.028)	(0.118)	(0.115)	(0.117)
LogLifespan father	0.283***	0.151***	0.150***	0.166	0.117	0.121
	(0.017)	(0.025)	(0.024)	(0.140)	(0.141)	(0.149)
N	74812	61700	61700	2413	2413	2413
Panel C: Excluding potential migrants						
LogLifespan mother	0.592***	0.410***	0.423***	0.414***	0.397***	0.391***
	(0.023)	(0.025)	(0.025)	(0.106)	(0.106)	(0.106)
LogLifespan father	0.272***	0.149***	0.168***	0.099	0.053	0.021
	(0.018)	(0.022)	(0.022)	(0.127)	(0.129)	(0.139)
N	87824	75327	75327	2890	2890	2890
Panel D: Excluding children dying before 6						
LogLifespan mother	0.403***	0.264***	0.276***	0.425***	0.418***	0.411***
	(0.015)	(0.016)	(0.016)	(0.076)	(0.075)	(0.076)
LogLifespan father	0.190***	0.095***	0.118***	0.069	0.013	-0.021
	(0.013)	(0.015)	(0.014)	(0.096)	(0.101)	(0.105)
N	89866	76954	76954	3020	3020	3020
Panel E: Excluding children dying before 6 - no logs						
LogLifespan mother	0.198***	0.126***	0.129***	0.251***	0.245***	0.238***
	(0.009)	(0.009)	(0.009)	(0.044)	(0.044)	(0.044)
LogLifespan father	0.095***	0.048***	0.059***	0.038	0.009	-0.018
	(0.008)	(0.009)	(0.009)	(0.057)	(0.059)	(0.062)
N	89866	76954	76954	3020	3020	3020
Year and District FE	✓	✓	✓	✓	✓	✓
Individual characteristics			✓		✓	✓
Father characteristics						✓
GF FE		✓	✓			
Twin father FE				✓	✓	✓

Standard errors clustered at descent level. Significance levels: *** 1% ** 5% * 10%.
GF FE: nbr 5587 Panel A. Twin father FE: nbr 223 Panel A.
GF FE: nbr 20533 Panel B. Twin father FE: nbr 709 Panel B.
GF FE: nbr 19380 Panel C. Twin father FE: nbr 669 Panel C.
GF FE: nbr 20056 Panel D. Twin father FE: nbr 695 Panel D.

Table B8: IGT estimates with grand-father and twin-father fixed effects - No logarithm transformation of lifespan

Dependent variable:	Lifespan					
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Father LS only						
Lifespan father	0.203***	0.096***	0.101***	0.096*	0.060	0.034
	(0.011)	(0.010)	(0.010)	(0.058)	(0.061)	(0.066)
N	100087	83840	83840	3241	3241	3241
Panel B: Father and mother LS						
Lifespan mother	0.234***	0.155***	0.153***	0.244***	0.234***	0.228***
	(0.010)	(0.010)	(0.010)	(0.043)	(0.043)	(0.043)
Lifespan father	0.110***	0.056***	0.065***	0.052	0.018	0.000
	(0.008)	(0.009)	(0.009)	(0.058)	(0.060)	(0.065)
N	96688	83840	83840	3241	3241	3241
Year and District FE	✓	✓	✓	✓	✓	✓
Individual characteristics			✓		✓	✓
Father characteristics						✓
GF FE (nbr 21282)		✓	✓			
Twin father FE (nbr 736)				✓	✓	✓

Standard errors clustered at descent level.
Significance levels: *** 1% ** 5% * 10%.
Models are restricted to equivalent sample sizes including individual characteristics.

Table B9: Survival Analysis of the Effect of Parental Lifespan

Dependent variable:	Lifespan		
	(1)	(2)	(3)
Lifespan father	-0.019***	-0.019***	-0.018***
	(0.000)	(0.000)	(0.000)
log odds ratio	0.981	0.981	0.982
Lifespan mother	-0.022***	-0.022***	-0.024***
	(0.000)	(0.000)	(0.000)
log odds ratio	0.978	0.978	0.976
N×m	725745	721117	721117
LogL	-290306.2	-280066.8	-278538.1
Year and District FE	✓	✓	✓
Individual characteristics		✓	✓
Parental characteristics			✓

Results from semi-parametric Cloglog models.
Standard errors clustered at descent level.
Significance levels: *** 1% ** 5% * 10%.

Table B10: IGT estimates with different sample restrictions

Dependent variable:	Log Lifespan					
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: no restrictions - keep year <1789 , keep 1909 and age over 75						
LogLifespan mother	0.595***	0.407***	0.414***	0.430***	0.430***	0.424***
	(0.019)	(0.021)	(0.021)	(0.085)	(0.084)	(0.083)
LogLifespan father	0.304***	0.161***	0.191***	0.118	0.060	0.038
	(0.016)	(0.020)	(0.020)	(0.107)	(0.108)	(0.121)
N	109984	88912	88912	3448	3448	3448
Panel B: keep year <1789 , keep 1909 and top coding over 75						
LogLifespan mother	0.628***	0.428***	0.436***	0.443***	0.442***	0.436***
	(0.021)	(0.022)	(0.023)	(0.095)	(0.094)	(0.094)
LogLifespan father	0.313***	0.163***	0.194***	0.105	0.045	0.018
	(0.017)	(0.021)	(0.021)	(0.120)	(0.121)	(0.132)
N	109984	88912	88912	3448	3448	3448
Panel C: drop year <1789 , keep 1909 and top coding over 75						
LogLifespan mother	0.608***	0.428***	0.436***	0.443***	0.442***	0.436***
	(0.021)	(0.022)	(0.023)	(0.095)	(0.094)	(0.094)
LogLifespan father	0.293***	0.163***	0.194***	0.105	0.045	0.018
	(0.017)	(0.021)	(0.021)	(0.120)	(0.121)	(0.132)
N	102371	88912	88912	3448	3448	3448
Panel D: drop year <1789 , drop 1909 and top coding over 75						
LogLifespan mother	0.580***	0.408***	0.409***	0.405***	0.393***	0.388***
	(0.022)	(0.023)	(0.023)	(0.094)	(0.094)	(0.094)
LogLifespan father	0.265***	0.142***	0.166***	0.114	0.047	0.043
	(0.017)	(0.021)	(0.020)	(0.120)	(0.122)	(0.132)
N	96572	83719	83719	3236	3236	3236
Year and District FE	✓	✓	✓	✓	✓	✓
Individual characteristics			✓		✓	✓
Father characteristics						✓
GF FE		✓	✓			
Twin father				✓	✓	✓

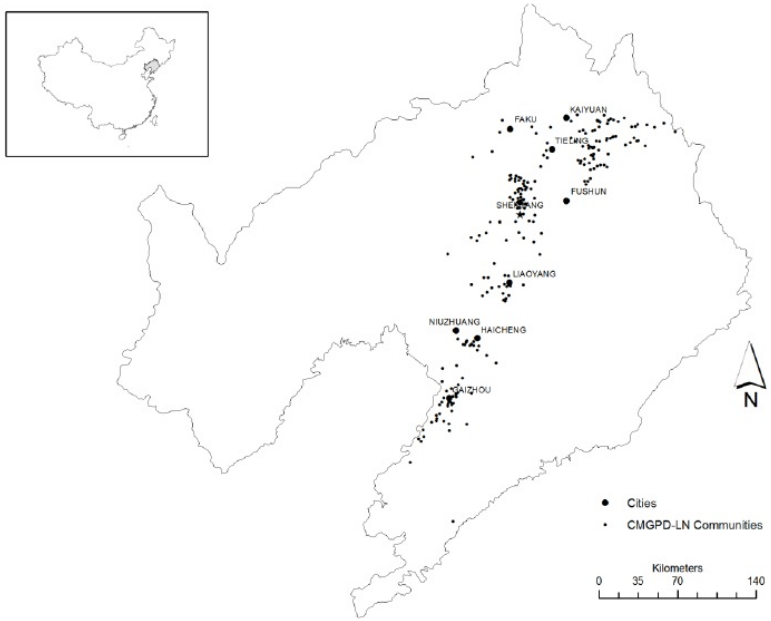
Standard errors clustered at descent level. Significance levels: *** 1% ** 5% * 10%.
GF FE: n. 21282 Panel A-C, n. 21282 Panel D. Twin father: n.779 Panel A-C, n. 736 Panel D.

Table B11: Interpretation. IGT estimates with alternative samples

Dependent variable:	Log Lifespan					
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Main models						
LogLifespan mother	0.580***	0.408***	0.409***	0.405***	0.393***	0.388***
	(0.022)	(0.023)	(0.023)	(0.094)	(0.094)	(0.094)
LogLifespan father	0.265***	0.142***	0.166***	0.114	0.047	0.043
	(0.017)	(0.021)	(0.020)	(0.120)	(0.122)	(0.132)
N	96572	83719	83719	3236	3236	3236
Panel B: Excl. children whose mothers died before 6						
LogLifespan mother	0.462***	0.303***	0.310***	0.388***	0.374***	0.368***
	(0.019)	(0.026)	(0.025)	(0.101)	(0.102)	(0.101)
LogLifespan father	0.202***	0.104***	0.125***	-0.006	-0.087	-0.092
	(0.015)	(0.021)	(0.020)	(0.116)	(0.121)	(0.132)
N	88568	76123	76123	3011	3011	3011
Panel C: Excl. children whose fathers died before 6						
LogLifespan mother	0.473***	0.365***	0.365***	0.380***	0.359***	0.355***
	(0.019)	(0.026)	(0.025)	(0.104)	(0.106)	(0.105)
LogLifespan father	0.272***	0.143***	0.177***	-0.018	-0.083	-0.058
	(0.020)	(0.026)	(0.026)	(0.145)	(0.146)	(0.163)
N	84619	72244	72244	2858	2858	2858
Panel D: Mother's LS below the median						
LogLifespan mother	0.659***	0.556***	0.568***	0.266	0.281	0.289
	(0.024)	(0.038)	(0.038)	(0.192)	(0.189)	(0.189)
LogLifespan father	0.305***	0.187***	0.230***	0.134	0.043	0.021
	(0.022)	(0.033)	(0.032)	(0.195)	(0.189)	(0.192)
N	57429	46854	46854	1633	1633	1633
Panel E: Father's LS below the median						
LogLifespan mother	0.710***	0.518***	0.547***	0.457**	0.505**	0.513**
	(0.028)	(0.036)	(0.036)	(0.216)	(0.224)	(0.227)
LogLifespan father	0.207***	0.137***	0.179***	-0.045	-0.064	0.020
	(0.018)	(0.037)	(0.037)	(0.233)	(0.230)	(0.244)
N	54114	44267	44267	1472	1472	1472
Panel F: Excluding children dying above 20						
LogLifespan mother	0.397***	0.413***	0.469***	0.074	0.133	0.136
	(0.026)	(0.052)	(0.057)	(0.174)	(0.181)	(0.184)
LogLifespan father	0.176***	0.132***	0.173***	-0.403	-0.470	-0.379
	(0.018)	(0.035)	(0.035)	(0.312)	(0.316)	(0.322)
N	32803	22769	22769	772	772	772
Year and District FE	✓	✓	✓	✓	✓	✓
Individual characteristics			✓		✓	✓
Parental characteristics						✓
GF FE		✓	✓			
Twin father FE				✓	✓	✓

Standard errors clustered at descent level. Significance levels: *** 1% ** 5% * 10%.
GF FE: n. 21282 Panel A. Twin father: n.736 Panel A.
GF FE: n. 19693 Panel B. Twin father: n.685 Panel B.
GF FE: n. 19140 Panel C. Twin father: n.665 Panel C.
GF FE: n. 13585 Panel D. Twin father: n. 460 Panel D.
GF FE: n. 12834 Panel E. Twin father: n. 433 Panel E.
GF FE: n. 7642 Panel F. Twin father: n. 270 Panel F.

Figure B.1. China Multi-Generational Panel dataset-Liaoning



Source: Lee et al. (2010)

Figure B.2. Tree representing information observed on daughters

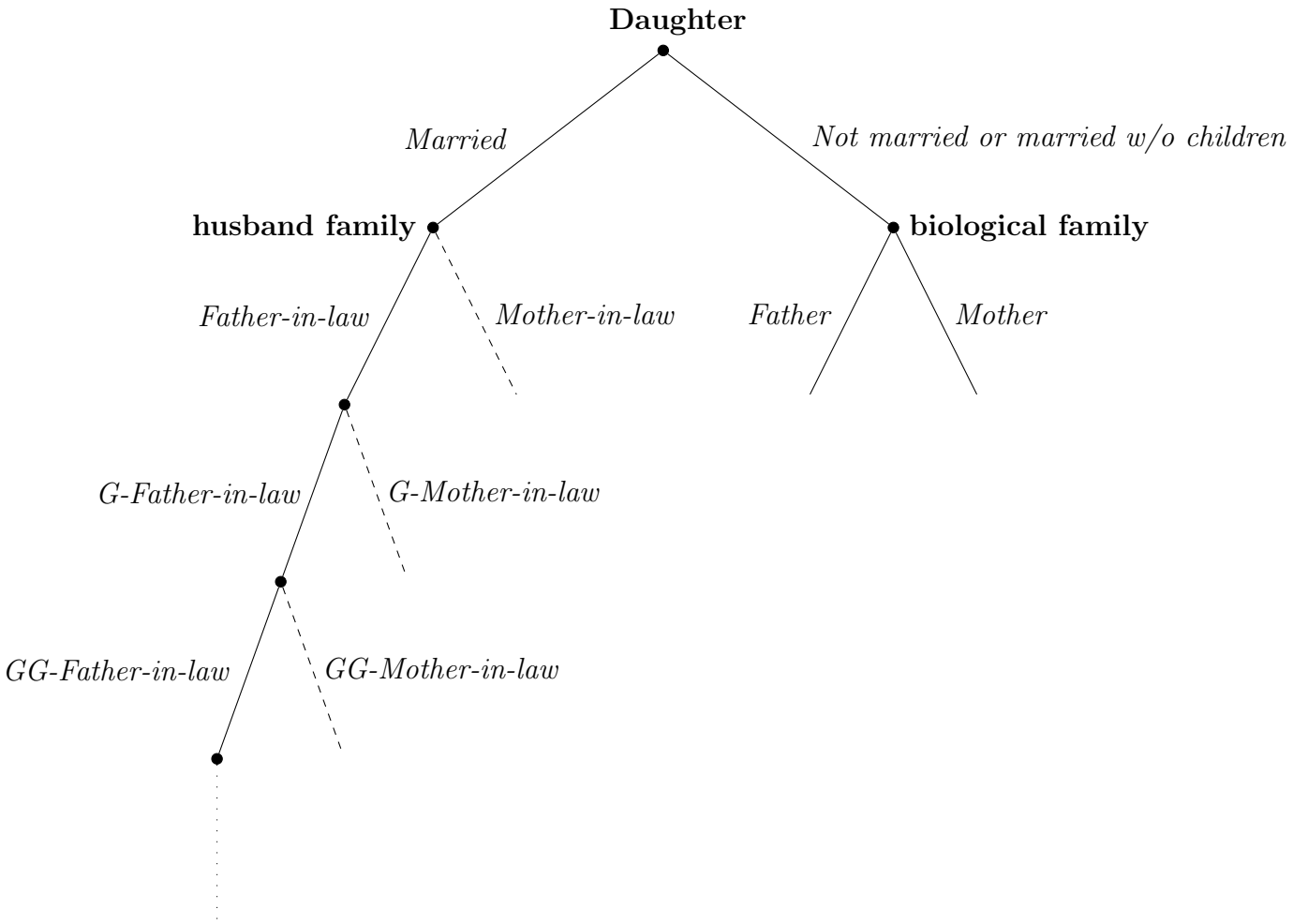
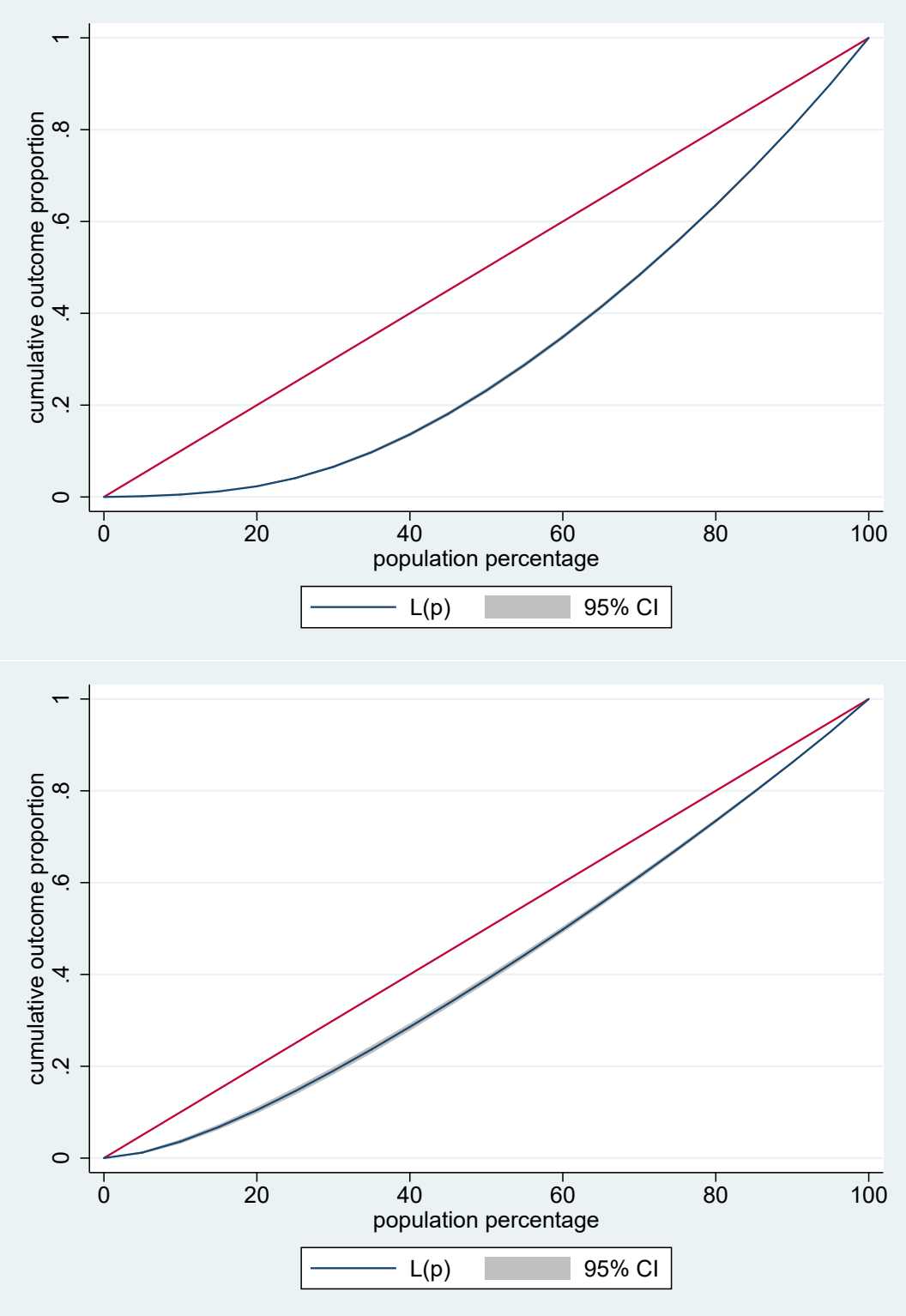


Figure B.3. Estimates of Lorenz curves (accumulated lifespan share by decile of the lifespan definition).



Note: The Figure on the top provides the Lorenz Curve estimated over the restricted lifespan of our main sample, while the one on the bottom is based on China Health and Nutrition Survey from 1991 and 2015. Only men are selected and, for comparability reasons, the strict definition of lifespan is constructed in both datasets.