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

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Abstract

We study the intergenerational transmission of health using registered data from China between 1789 and 1906. We first document the intergenerational correlations across 7 generations. We then identify causal associations comparing children born from twin parents. We find a strong and persistent intergenerational elasticity of about 0.54 between mothers and sons. Such elasticity should decrease by almost half when we consider the role of orphanhood. The intergenerational association from fathers

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is much weaker and seems to be driven by genetic factors. Our results highlight the nurturing role of women in affecting their children's health outcomes across generations in developing countries.

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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. Nature may render such interventions ineffective and potentially pave the way for policies (e.g. enhancing mobility, ...) aiming at increasing genetic diversity in the population.[1]

In this paper, we estimate the intergenerational elasticity of life expectancy between parents and children, using linked registered data from rural China between 1789 and 1906. We do find strong intergenerational transmission in health between parents and their sons, in particular for mothers. However, such elasticities are likely to be driven by unobserved genetic characteristics. To draw causal inference, we compare the outcomes of children born from parents who are same-sex twins. Interestingly, with individual controls, the intergenerational transmission of health between mothers' and children's lifespans stands at about 0.54. The equivalent association between fathers' and children's lifespans is null, suggesting that the IGT is driven by genetic factors. Correcting for potential orphanhood, the intergenerational elasticity should decrease to around 0.3. In other words, nurture matters

[1]The role of genetic diversity in economic development has been documented by Ashraf and Galor (2013) and is subject to a fierce debate in social sciences (Rosenberg and Kang, 2015).

much more to explain the IGT between mothers and their sons, not for fathers. Although less relevant for contemporaneous China, our analysis helps us to understand the nature of the intergenerational elasticity in health in a highly patrilineal society, as could be found in many developing countries today. The role of mothers in explaining the nurture component of the IGT is consistent with the important role of women in within-household allocation and its consequences for children's long-term health (Duflo, 2012).

Our contribution lies at the crossroad of three strands of literature. First, 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)[2], 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 (2003), Classen (2009), Royer (2009), Bhalotra and Rawlings (2011), and Parman (2012). Most of these studies 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.[3]

We focus on a long-term health outcome, life expectancy, proxied by the lifespan, i.e. the approximated number of years between birth and death. As pointed by Parman (2012),

[2]There is a large literature in other social sciences assessing the association between parents' and children's IQ scores using the so-called Behavioral Genetics Model (Herrnstein and Murray, 1994).

[3]We exploit a parent-twin approach similar to Currie and Moretti (2003) and Royer (2009) among those studies looking at the intergenerational transmission of health.

such a proxy for long-term health outcomes has the major advantage to receive a common interpretation across contexts, time and gender. Moreover, the use of lifespan avoids the standard estimation problems of "lifecycle bias", zero income, and non-linearity encountered in the literature on income mobility across generations (Black and Devereux, 2010). Historical studies using earnings data are often restricted to investigate the IGT between fathers and sons. Mothers have long been neglected. Chadwick and Solon (2002) is a noticeable exception for the U.S. but the use of earnings overlooks the importance of within-household allocation and assortative mating in affecting women's welfare (Duflo 2012). Investigating the intergenerational correlation of education in the U.S., Sweden and Norway, Behrman and Rosenzweig (2002), Holmlund et al. (2011), and Pronzato (2012) find stronger correlations between fathers' education and children's education than the ones between mothers and children. Given our focus on life expectancy, Parman (2012) is certainly the closest to our work. He estimates the intergenerational correlation in lifespans in North Carolina and finds very strong correlations between daughters and mothers and between sons and fathers. In particular for sons, the intergenerational elasticities for fathers and mothers stand at about 0.36 and 0.16, respectively. In our study, we do find stronger elasticities for mothers. Furthermore, we do find that the association with fathers' lifespan is weaker 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 echo the biodemographic literature that has exploited historical data on lifespan to assess the inheritance of human longevity. Gavrilov and Gavrilova (2001) provide an extensive review. The estimates of heritability in historical studies vary widely between zero and 0.89 but the majority points to an estimate of less than 0.3 (You et al., 2010).

However, 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.[4] A few exceptions exist but they tend to investigate the issue for very small areas.[5] Gudmundsson et al. (2000) and Mazan and Gagnon (2007) propose more general studies by focusing on the majority of the population in Iceland and Quebec, respectively. Some of these studies also assess the correlations between siblings (Swedlund et al., 1983; Perls et al., 2002; Kerber et al., 2001; Mazan and Gagnon, 2007; Salaris et al., 2013) or twins (McGue et al., 1993; Herskind et al., 2006; Yashin and Iachine, 1997). Evidence is also mixed with respect to the relative importance of the maternal or paternal lines of inheritance of human longevity (You et al., 2010). Therefore, we also contribute to the biodemographic literature. With the use of twin-parent fixed effects, we offer a more credible identification strategy to distinguish nurture from nature and shed light on the maternal and paternal components of the nurturing effect.

Second, 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). There is an emerging literature investigating economic mobility in India, Malaysia, Mexico, Nepal and Vietnam (Lillard and Willis, 1997; Binder

[4]e.g. the Landed Gentry (Beeton and Pearson, 1899), some aristocratic families (Gavrilov and Gavrilova, 2001), centenarians in New England (Perls et al., 2002), Moormons in Utah (Kerber et al., 2001).

[5]e.g. the Connecticut Valley (Swedlund et al., 1983), the French Jura (Cournil et al., 2000), a Flemish village in Belgium (Matthijs et al., 2002), or a village in Sardigna (Salaris et al., 2013).

and Woodruff, 2002; Emran and Shilpi, 2015; Hnatkovska et al., 2013; Azam, 2015, e.g.). 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 draw causal inference and to distinguish between nurture and nature, using a parent-twin approach. Eriksson et al. (2014) use the 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 work, we prefer the use of parent-twin fixed effects less requiring in terms of identifying assumptions. Related studies on developed countries have also focused on the IGT between two generations, the so-called AR(1) model (Lindahl et al., 2015; Clark and Cummins, 2015). Other studies have shown that estimating the IGT between generations may underestimate the persistence of socio-economic outcomes across generations (Long and Ferrie, 2013; Lindahl et al., 2015; Clark and Cummins, 2015). One of the strengths of our analysis is to consider up to 7 successive generations to assess the relevance of the AR(1) model and the stability of the IGT across generations. We therefore also contribute to the literature seeking to exploit more than 2 generations since the three aforementioned historical studies on the US, the UK, and Sweden are restricted to 3 or 4 generations. Furthermore, they overlook the role of women given the use of earnings (or education) data. 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, 2014, 2015; Lindahl et al., 2015).

Third, we contribute to a booming literature on the use of parent twins to distinguish nature from nurture. Behrman (2016) proposes an extensive review of the use of twins

in economics. Behrman and Rozensweig (2002) seminally investigate the intergenerational transmission of education using twin mother fixed effects. They find a negative association between mother's and children's education, contrasting with the positive association for father's schooling. The interpretation of the authors is that educated women are more likely to enter the labour market, with detrimental impact on their children's schooling. These results have been widely discussed, questioning the negative association for mothers (Antonovics and Goldberger, 2005). So far, there is a relative consensus in existing twin studies on a much larger impact in the intergenerational transmission of education from fathers compared to mothers (Black and Devereux, 2010). However, little is known about the generalization of these results to developing countries, in contexts where women empowerment has been associated with improved education among children and economic development in other contexts (Behrman et al., 1999; Behrman and Rosenzweig, 2002; Duflo, 2012).

2 Background and data

We investigate the intergenerational transmission of health in the Liaoning province between 1789 and 1906. The Liaoning province is located in North-East China (Figure 1) and was the original home of the Manchu Qing dynasty emperors (1644-1912). The Qing dynasty was 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 already emerged at the end of the eighteenth century under the Qianlong Emperor (1735-1796). The Qing ruling was then 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 a "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). Paradoxically, such a 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 directly 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 the demographic, economic, and social life of the population during that period in great details (Lee et al., 2010). The data comprises triennial data from 29 sets of household registers with 1.5 million records of approximately 260,000 unique individuals from the Qing Imperial Household Office, between 1749 and 1909 in the Liaoning province (Lee et al., 2010; Lee and Campbell, 2011; Song et al., 2015). Missing data are reported between 1888 and 1903 since the corresponding registers were destroyed by fire (Lee et al., 2010). Furthermore, 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".

Originally, the data follow individuals through the registers every three years. However, since our main dependent variable is the lifespan, we transform such a dataset into a cross-section of 266,164 individuals that belong to 1063 distinct descents (family trees). The data originate from 13 districts. We further restrict the sample by excluding 48,029 individuals that entered into the registers prior to 1789 and 82,856 individuals who were alive in 1909. The reason is that households registers before 1789 do not identify residential households and do not uniformly distinguish villages (Lee et al., 2010), while lifespan cannot be approximated for those still alive in 1909. The resulting data consists of information on about 135,000 individuals covering up to seven generations of the same families.

Our main information of interest consists in life expectancy, proxied by the individual lifespan. We construct the lifespan of each individual as the difference between the last year observed in the register and the year of birth. Note that Lee et al. (2010) indicate that the year of birth is sometimes missing or badly recorded. We drop 4,713 individuals for which the year of birth 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 exclude 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 also restrict the lifespan below 76, since mortality record has been recognized as problematic for age above 75 (Dong and Lee, 2014). Such restrictions result in a sample of 114,342 individuals. In Section 4, we will discuss several robustness checks, including using a much stricter definition of lifespan, obtained as 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. We also deal with possible migration bias (without being formally recorded as migrants), and relaxing sample restrictions.

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. 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 intergenerational transmission of health in this highly patriarchal society is of key interest for contemporaneous economic development.

We use the sample of 114,342 individuals to generate the lifespan and characteristics of parents (including mothers) and grandparents (including grand-mothers). However, in our analysis, we only use as units of observations the sample of sons linked to their male and female ancestors. Daughters were expected to leave the family after marriage and becomes member of their husband's family (Wakefield, 1998). Sons would remain within the original family. Omitting daughters reduces the analytical sample to 57,636 sons of which 35,310 have non-missing information for both parents.[6] 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.

Table 1 shows how 35,310 individuals can be linked to 6,916 mothers and 5,020 fathers, to 2,123 grand-fathers and 2,652 grand-mothers, to 1,095 great-grand-fathers and 328 great-grand-mothers, to 541 great-great-grand-fathers, and finally, to 289 great-great-great-grand-

[6] A large number of those with missing parental information appear in the first generation.

fathers.[7] For interpretative purposes, we should bear in mind that the (great-) grand-mothers are (grand-) mothers of the fathers and not of the mothers. Note that the gain in life expectancy of about 10 years between the individuals and the parents is an artefact of the data structure (by construction, an individual cannot die in childhood and becomes a parent). The same is true with subsequent ancestors. However, such a trend may also be partly explained by the decreasing life expectancy observed during the period of investigation.[8] Such a decline during the Qing dynasty in the standard of living has been well documented (Allen et al., 2005; Campbell and Lee, 2004; Broadberry et al., 2017). Figure 2 further shows the mid-1800 as a turning point. Section 4.3 will further illustrate whether such a decline in life expectancy is associated with instability in the intergenerational transmission of health.

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):

[7] 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 well become father, grand-father, or had another family position in subsequent years. Contrary to Lindhal 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. Such a distinction is a major advantage to study the stability of the intergenerational transmission of health across generations (see Section 4.3).

[8] Another explanation is that we may oversample short-lived people in more recent registers by excluding individuals still alive in 1909. In Section 4.2, 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 4.2. Such definition is also used in mortality studies by Campbell and Lee (2004), Campbell and Lee (2009) and Dong and Lee (2014).

$$LS_{it-l} = \alpha + \theta_t + \mu_d + \beta_{1,j} LS^m_{it-j} + \beta_{2,j} LS^f_{it-j} + \gamma \mathbf{X_i} + \epsilon_i \quad (1)$$

where $l = 0 \ldots 4$ and $j = 1 \ldots 5$. With $l = 0$ and $j = 1$, the main coefficients of interest $\beta_{1,1}$ and $\beta_{2,1}$ capture the elasticity between the (log) lifespan of individual i, LS_{it} and the one of his mother, LS^m_{it-1} and father, LS^f_{it-1}, respectively. With $l = 0$ and $j > 1$, we can assess the direct effects of grand-fathers (LS^f_{it-2}), up to great-great-great-grand-fathers (LS^f_{it-5}). Due to data constraints on linked female members of the family, data are only available up to great-grand-mothers (LS^m_{it-3}). That basically means that we will only be able to control for assortative mating up to that level. The direct effects for great-great-grand-fathers and great-great-great-grand-fathers are likely to include the indirect effect of their partners. In all our specifications, standard errors are clustered at the descent level (family tree). In the less restrictive specification, we are exploiting 541 descent groups for clustering the standard errors. We also introduce a time indicator to capture registry-specific effects, θ_t.[9] We control for unobserved heterogeneity at district level with district fixed effects, μ_d, and observed heterogeneity at the individual level with control variables, X_i. We include individual characteristics such as the fact to be disabled, to be 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). These variables are further described in Appendix A. 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

[9]This corresponds to the year of first appearance of the individual in the registry. Results are commented in Section 4.2.

mortality in Campbell and Lee (2004) and Campbell and Lee (2009) based on the same dataset. To render our estimated coefficients comparable across specifications, we restrict the sample to those individuals for which the lifespan is available for mothers, fathers, grand-mothers and grand-fathers. Such a restriction is relaxed when further generations are added to preserve the size of our analytical sample.

To assess the relevance of the AR(1) model, we follow Lindhal et al. (2015) by comparing the direct effects obtained in equation (1) to estimates of the intergenerational correlation across two consecutive generations (i.e $j - l = 1$). We therefore compute the predictions of 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 Lindhal 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). Based on this first empirical exploration, we will then further compare the AR(1) specification with more complex models, from AR(2) up to AR(5) of the following form:

$$LS_{it} = \alpha + \theta_t + \mu_d + \sum_{j}^{3} \gamma_{1,j} LS^{m}_{it-j} + \sum_{j}^{5} \gamma_{2,j} LS^{f}_{it-j} + \gamma \mathbf{X_i} + \epsilon_i \quad (2)$$

The same description of variables and fixed effects apply, while standard errors are also clustered at the descent level. The same sample restrictions apply.

3.2 The twin approach applied to registry data

Equation (1) can be simplified to $j = 1$ to allow for a causal identification of the nurture channel of the intergenerational transmission of health between parents and their children. The main challenge in identifying a causal relationship indeed consists in distinguishing nurture from nature. The IGT may be largely driven by inherited genetic differences across families. In absence of observed genomic information, the literature has largely relied on the twin approach, i.e. the comparison of individuals whose fathers or mothers are twins.[10] The idea is that by comparing the IGT among children of identical twin parents helps to isolate the IGT from fixed family characteristics, and removes (to a large extent) the variation in IGT due to genetic differences.[11] The twin strategy will be contrasted with a more standard approach of controlling for grandfather fixed effects, which similarly to parent-twin fixed effects also remove some unobserved differences in initial endowments. More specifically, the

[10] Other approaches would consist in controlling for directly observed genetic differences (Beauchamp et al., 2011) or the use of adoptees (Bjorklund et al., 2006; Sacerdote, 2007). Another approach has used instrumental variables such as great-grandfathers' outcomes of interest (Clark and Cummins, 2015) or unexpected events or reforms. However, the exclusion restriction that needs to be met and the generalisability of results, often driven by local average treatment effects, constitute important drawbacks of such an approach (Black and Devereux, 2010).

[11] Although we control for assortative mating, we should acknowledge that this approach does not net out all genetic endowments. The genetic component originating from the twin's partners cannot be controlled for (Holmlund et al., 2008; Amin et al., 2015). That is why we give a causal interpretation of the intergenerational transmission of health between mothers and sons based on the mother-twin approach and between fathers and sons based on the father-twin approach.

various specifications take the following form:

$$LS_i = \alpha + \theta_t + \mu_d + \varphi_k + \beta_1 LS_i^m + \beta_2 LS_i^f + \gamma \mathbf{X_i} + \delta \mathbf{Z_i} + \epsilon_i \tag{3}$$

LS_i still denotes the (log) lifespan of the individual i, while LS_i^m and LS_i^f, denote the ones of his mother and father, respectively. Standard errors are clustered at the descent level (family tree). In the less restrictive specification, we are exploiting 230 and 202 descent groups for clustering the standard errors with father-twin and mother-twin fixed effects, respectively. All our specifications also include time (θ_t) and district fixed effects (μ_d), together with individual control variables. The main difference with equation (1) with $j = 1$ is that we introduce additional fixed effects, φ_k. Our preferred specifications will compare children from plausibly same-sex twin parents, through the introduction of mother-twin (for β_1) or father-twin fixed effects (for β_2). The same individual controls, X_i, used in our previous models are also introduced. As mentioned above, we will also compare children sharing the same grand-father, through the introduction of grand-father fixed effects.

Our approach relies on two main identifying assumptions. First, siblings born the same year and of the same sex are considered as twins. In our analysis, we exploit 320 and 435 pairs of father and mother same-sex twins, respectively.[12] A major drawback is that we cannot be sure whether those same-year newborns are monozygotic twins. The causal

[12]Father twins are identified based on a common father. However, mother twins cannot be identified based on common fathers since identification based on fathers may wrongly infer that one sister is a twin with her sister-in-law if they are born the same year. Mother twins are based on the identification of a common mother. One possible caveat of using common mothers to identify twins is that the registers do not specify the identity of an individual's mother. The software used to create the dataset links children to the wife of the household head, giving priority to the link made between a child and a wife in the earliest available register. In cases of remarriage or polygyny, children may be incorrectly assigned to the wife who was observed in the register. However, Lee et al. (2010) indicate that it should be a minor issue since widower remarriage was very uncommon and polygyny was almost non-existent. We actually do not observe any polygynous household in our sample.

interpretation of our results depends on the assumption that twin parents are identical in their endowments. Such assumption is naturally more likely with monozygotic twins, compared to dizygotic twins who share on average 50 percent of the genes. We cannot be certain to remove completely the genetic component of the IGT with our parent-twin fixed effects. We will therefore apply the 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 in such a way:

$$\widehat{\beta_{TS}} = \frac{\widehat{\beta_{TW}} - \lambda\theta\widehat{\beta_{SIB}}}{1 - \lambda\theta} \tag{4}$$

where $\widehat{\beta_{TW}}$ and $\widehat{\beta_{SIB}}$ denote the estimates obtained under the parent same-sex twin fixed effects specification and under the parent same-sex siblings fixed effects specification, respectively. θ represents the share of DZ twins among all same-sex 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 ($\lambda = 1$) and when the sibling estimate is lower (larger) than the twin estimates, $\widehat{\beta_{TS}}$ provides a upper-bound (lower-bound) estimate of the causal intergenerational transmission of health between parents and children. When the sibling estimate is lower than the twin estimate, relaxing the assumption of absent treatment differentials ($\lambda < 1$) should decrease the intergenerational transmission of health between parents and sons. In such a case, the estimate converges towards the parent same-sex twin fixed effects.

The second key identifying assumption is that (parent) twins are treated similarly within their family after birth. Such assumption would not be valid if (grand-)parents make compensating or reinforcing investments or if children's outcomes are affected by both the twin parent and his or her partner. We cannot exclude such unobserved heterogeneity between parent twins. However, we reduce the threat by controlling for parents' exposure to shocks, parental controls and the partner's lifespan.[13] Parental control variables, Z_i, include fathers and mothers disabled, age at approximated birth (and its square), and the fact to be a migrant during his or her course of life.

3.3 The stability of the intergenerational transmission of health

We will investigate the stability of the parent-child causal transmission of health, across 7 generations within the same family tree. The first generation is defined by the first household head appearing in each family tree. His descendants are then forming the successive generations within each family tree. The first generation was on average born in 1764. However, the 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. We use a similar specification than in equation (3):

$$LS_i^g = \alpha + \theta_t + \mu_d + \beta_1 LS_i^{m,g-1} + \beta_2 LS_i^{f,g-1} + \gamma \mathbf{X_i} + \delta \mathbf{Z_i} + \epsilon_i \tag{5}$$

The main differences are that we cannot include parent-twin or grand-father fixed effects,

[13] 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). Unfortunately, our dataset does not allow us to control for individual and parental birth weight.

and estimate the association between the lifespan of parents and sons by pairs of generations. The index g denotes the generation of the sons, from 2 to 7. In such a way, we can assess the stability of the IGT across generations. We should nonetheless be cautious in comparing our results across generations since the sample of our extended families with multiple generations (e.g. those with a seventh generation) may differ from other households for unobserved reasons. As seminally shown in biodemography by Swedlund et al. (1983), it is likely the population of interest will become increasingly homogeneous across generations. We discuss that issue in more details in Section 4.3.

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). Controlling for assortative mating, the intergenerational correlation for father and mother stands at about 0.33 and 0.56, respectively (columns 1 and 2). The estimates provided by the AR(1) model are 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). However, 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). Com-

pared to a closer literature on the intergenerational transmission 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, the comparison is limited since those papers do not provide estimates for the patrilineal linkage. One exception is Eriksson et al. (2014) who find higher intergenerational correlations for fathers (0.298) in rural China between 1991 and 2009, although the difference is relatively small with the one for mothers (0.272).

The rest of Table 2 reveals a few interesting results. First, we find a small and sligthly significant 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 unlikely to underestimate the intergenerational transmission of health between fathers and sons in case we use the AR(1) model. On the contrary, column (4) reveals that there is a statistically significant estimate of the association between grand mothers' lifespan and that of grandchild. The associated elasticity of 0.711 is much larger than the predicted association of 0.259 (0.560×0.462) based on the correlations between mothers and sons (0.560) and mothers and grand-mothers (0.462). A direct effect – larger than the predicted association of 0.079 – is also found for great-grand-mother (column 6). Overall, the AR(1) model seems to be appropriate for fathers, whereas, given the direct role of grand-mothers and great-grand-mothers, it is likely to underestimate the intergenerational correlation of health for mothers across generations. Although we cannot control for assortative mating due to sample size constraints in columns (5) to (7), that should not alter our main conclusions since neglecting

assortative mating usually tends to inflate the estimated coefficients.[14]

The direct effect played by grand-mothers is further confirmed when we extend the analysis by implementing AR(2), AR(3), and AR(4) models in Table 3.[15] Such result is consistent with Lindahl et al. (2015) who find that grandparents have an independent generational effect on grandchidlren, although they do not find significantly different correlations between grandfathers and grandmothers. In Table 3, the estimated coefficients for grand-mothers stand in a range between 0.39 (column 3) and 0.64 (column 5). The fact that the estimated coefficient is not significant at any reasonable level of confidence in the AR(5) is certainly due to the reduction of sample size to 139 observations.

Overall, Table 2 and Table 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. Of particular interest is the fact that grand-mothers are not the mothers of the mothers but their mothers-in-law. Such distinction suggests that women may be decisive in explaining the intergenerational transmission of health through a nurture channel. Indeed, grandmothers (in particular, 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. No such effect is found for non-resident

[14]Introducing great-grand-mothers in the estimation corresponding to column (5) and great-great-grand-mothers in the one corresponding to column (7) would reduce the sample size to 293 and 22 observations, respectively.

[15]Similar to Table 2, we restrict the samples in columns (2) to (6) of Table 3 to individuals with available lifespan for mother, father, grandfather, and grand-mother. We relax such a sample restriction in column (1) of Table 3. Our results are not radically altered. Based on column (1) of Table 3, we provide the descriptive statistics of our control variables in Table B1 in the Appendix.

grandparents. Nonetheless, such evidence does not allow us to claim any causal relationships since inherited genetic differences may inflate the intergenerational transmission of health. However, it is very 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 but may well underestimate that between mothers and sons.

4.2 Nurture versus nature in the intergenerational transmission of health

Results. To distinguish nurture from nature, we restrict our analysis to the association in health between children and their parents. In Table 4, we report the estimated coefficients β_1 and β_2 of equation (3) using several specifications augmented with a large set of fixed effects, more likely to isolate the role of nurture from the role of nature. In Panels A and B, we introduce the lifespans of mothers and fathers, separately. To account for assortative mating, Panel C includes both variables of interest.[16] Column (1) of Panels A and B only include a grand-faher fixed effect. Comparing individuals sharing the same grand-father indicates an intergenerational transmission of health of 0.42 and 0.29 for mothers and fathers, respectively. When we turn to the equivalent estimations in Panel C, the coefficient of the lifespan of the father is further reduced to 0.19, while the intergenerational association between mothers and sons stands at about 0.38.[17]

Assortative mating clearly matters. We further interpret the results in Panel C of Ta-

[16]We provide the detailed results of Panel C of Table 4 in Table B2 in the Appendix.

[17]The coefficients equivalent to the same regressions with the full set of individual and parental controls stand at about 0.36 (0.047) and 0.18 (0.048) for mothers and fathers, respectively.

ble 4. In columns (2) and (3), we show the results obtained implementing our preferred identification strategy of the intergenerational causal effects. We estimate more requiring models introducing mothers and fathers' twin fixed effects. Such estimations are more likely to isolate the effect of nurture. Comparing children whose mothers share the same genetic background (column 2), the intergenerational elasticity between mothers and sons stands at 0.58. Comparing children of twin fathers (column 3), the coefficient of 0.16 obtained for the father lifespan is not statistically different from zero, and its magnitude is less than one third of the one of mothers. The intergenerational correlation between fathers and sons seems to be entirely driven by genetic factors. Mothers seem to matter much more in the nurturing transmission of health. In columns (4) and (5), such interpretation receives further support when we control for individual characteristics, including our proxy for pre-natal shock. As expected, the estimate of the intergenerational transmission of health between mothers and sons is reduced to around 0.54.[18] In theory, such estimation should capture the nurture effect, net of the pre-natal environmental conditions. On the contrary, the introduction of individual characteristics does not only show again a non-significant coefficient for the father's lifespan but the size of the coefficient is basically null. A ten percent increase in the mother's lifespan (equivalent to about 3.4 years of life at the mean) would translate into a rise of more than 5 percent in life expectancy for her child (about 1.8 years at mean value). Instead, the association between fathers and sons seems largely explained by unobserved genetic factors. Nurture matters much more for the intergenerational transmission of health between mothers and sons, than between fathers and sons.

[18]Similar results are found when we re-estimate the parent-twin fixed effects models, excluding in a backward fashion those born before 1906 up to 1830 (Figure 3). As discussed in more details when we assess the role of measurement errors, our results do not seem to be driven by the exclusion of those alive in 1909.

Identifying assumptions. The causal interpretation of the estimated coefficients in columns (4) and (5) of Table 4 strongly relies on the assumption that twin parents would be treated in a similar way after birth. We cannot directly observe investment made by grand-parents into parent twins' human capital. However, columns (6) and (7) of Table 4 show that our conclusions remain qualitatively unchanged when adding parental control variables. The intergenerational association between sons and mothers stands at 0.64 in our preferred specifications with mother-twin fixed effects. The coefficient for fathers' lifespan remains null and far from being significant.

As described in Section 3, one remaining concern is that we cannot be certain that our twin parents are effectively twins, and even less monozygotic twins. To deal with this issue, we implement the bounding exercise proposed by Holmlund et al. (2008). Similar to these authors, in Table B3, we only focus on the specification without controls. Results with controls are qualitatively similar. In Panel A of Table B3, the upper-bound estimate of the intergenerational transmission of health between mothers and sons is obtained using equation (4), whereas the lower-bound corresponds to the siblings estimate using grand-mother fixed effects.[19] In Panel B of Table B3, the sibling estimates of the IGT between fathers and sons are obtained using paternal grand-father fixed effects.

Assuming absence of treatment differentials between twin and sibling parents ($\lambda = 1$), Panel A of Table B3 indicates a IGT of about 0.72 (column 3). We can therefore conclude

[19]The link between mother (father) and sons are based on the mother (father) IDs. Mother (Father) twins are identified based on a common mother (father). For grandparents, we can only exploit the grandparent IDs that correspond to the parents of the fathers. Similarly, for higher generations, we can only exploit the paternal lineage. In the twinning analysis, the parents (in-law) of father-twins (mother-twins) are therefore based on the paternal grandparents IDs. It makes the identification of the maternal grand-parents impossible. Therefore, this sibling estimate is equivalent to a paternal grand-mother fixed effects.

that had we been able to identify and use only MZ twins, our analysis would have produced a statistically significant causal intergenerational elasticity of even higher magnitude. As expected, if we relax the assumption of absent treatment differentials ($\lambda < 1$) in columns (4) to (7), the IGT converges towards the mother twin fixed effect estimate. A similar bounding exercise provides an estimated IGT between fathers and sons bounded between 0.14 and 0.16 (Panel B of Table B3).

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). Using individual data from 72 countries between 1972 and 2012, they find that the mother's health is positively correlated with the probability of a twin birth. In our study, father twinning does not seem to be correlated with the lifespans of their mothers or their fathers (Panel A of Table B4). We do find that the lifespan of mothers in law is positively associated with the probability of being a twin among mothers (Panel B of Table B4). However, such results point to the importance of controlling for assortative mating, rather than shedding strong doubt on the validity of our identifying assumptions.[20]

Measurement errors may be due to i) the way we approximate the lifespan of the individuals recorded in the population registers; ii) missing observations for unrecorded deaths; or iii) the exclusion of individuals alive in 1909, for which we cannot approximate the lifespan.[21]

[20] With the means of a matching exercise, we can also show that the two-subsamples of boys twins and girls twins are balanced on parental and grand-parental lifespan and pre-determined characteristics. Results are available upon request.

[21] To explore the role of measurement errors and to shed light on the mechanisms of our results, we estimate equation (3) with alternative samples. To preserve a sample size sufficiently large size to draw inference, we implement these robustness checks on the grand-father fixed effects estimations.

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 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 representation in Figure 2 is also almost identical when using that stricter definition. When such information is used to construct the son's lifespan, we obtain similar results (Panel B of Table 5).[22] One 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 C of Table 5 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 offsprings. Such a sample restriction barely makes any difference to our main results. We also investigate the importance of migration. While we control for *observed* migration at the individual and parental levels and exclude unauthorised 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*). Using a simple nearest-neighbor matching technique (Heckman et al., 1997), about 7 percent of our analytical sample is matched to *observed* migrants.[23] We then replicate our main results in Panel D of 5, excluding these potential migrants. Our results are largely unchanged and confirm the low level of mobility reported in the historical background of this study.

[22]In the CMGPD-LN, we can use the information according to which the observed individual was annotated in the next available register as having died during the three years covered by that register. The information is only available for about 14% of the analytical sample. Given the small sample size, Panel B of Table 5 uses the strict definition of lifespan for sons.

[23]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.

Second, bias may arise due to unrecorded early mortality. The difference with the above issue is that such children will never be recorded in the registers. Such 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, e.g.) —whose parents' lifespan is likely to be low —, the resulting selection is likely to create a downward bias in our estimates of the intergenerational transmission of health. We can make an educated guess of the likely bias resulting from missing boys dying before 6, by dropping those we observe to do so. Panel E of Table 5 confirms that our main results may underestimate the intergenerational elasticity in particular between mothers and sons by the omission of children dying in early childhood.[24] The magnitude of the coefficient decreases by about 40% and 55% for mother's and father's lifespans, respectively. Had we observed all children dying at early age, our coefficients would be higher in magnitude.

Third, our results may be biased in favour of shorter-lived persons in more recent years. Since we exclude those who were alive in 1909, the lifespan data for longer-lived people were not yet available by the time of data collection, a well-known problem in biodemography (Gavrilov and Gavrilova, 2001). Our main results do not seem to be a consequence of the exclusion of the individuals still alive in 1909 and the subsequent risk of oversampling shorter-lived individuals for more recent years. Nonetheless, we re-estimate our parent-twin fixed effects models, excluding in a backward fashion those born before 1906 up to 1830. In

[24]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 who 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 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.

the most extreme case, when we exclude 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 mothers and sons remains fairly stable when we exclude cohorts born between 1850 and 1906.[25] When we also exclude those born before 1850 up to 1830, we witness a reduction in the coefficient but still standing above the level of 0.2 in the most extreme case. Such reduction may be partly explained by the reduction by about half in sample size, but also by some changes occurring around 1850 in the intergenerational transmission of health. We investigate further that issue in Section 4.3. The intergenerational transmission of health between fathers and sons remains stable at around 0.2 between 1850 and 1906 following the same exercise.

Interpretation. Our results point to the stronger nurturing role played by mothers, compared to the one by fathers. Such evidence contrasts very much with the literature, in particular regarding the intergenerational transmission of education in developed countries (Behrman and Rosenzweig, 2002; Black and Devereux, 2010; Holmlund et al., 2011; Pronzato, 2012). One explanation might be that in developing countries mothers have been found to impact greatly children's welfare, in particular their health, compared to educational outcomes (Duflo, 2012).[26] But the stronger intergenerational transmission for mothers

[25]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).

[26]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. The agricultural dominance of the society makes the study relevant for many developing countries. The population under study was largely hereditary peasants working mainly for the emperial estates. Although they received larger initial land allotments from the government at that time, compared with peasants elsewhere (Dong and Lee 2014), they appear to be fairly representative of the Northeastern Banners Population administered by Eight Banner, a civil and military administrative system organized by the Qing Dynasty (1644-1911) to govern the Manchurian and Mongolian provinces in greater North and Northeast China as well as the Qing garnison population in China Proper. The Northeastern banners lived in the

may receive different interpretations. First, children orphanhood may drive our results. It is plausible that mothers' death matters more for the child's survival at young age, compared to fathers' deaths. For instance, Beegle et al. (2010) find that maternal orphanhood has a permanent adverse effect on children's health and educational attainment in rural Tanzania, not paternal orphanhood. The same is true for education in South Africa (Case and Ardington, 2006) or in Kenya (Evans and Miguel, 2007). Excluding children whose mothers died before the child has reached the age of 6, Panel A of Table 6 indicates that the main coefficient of interest for mothers remains above 0.2, while the one for fathers is decreased to an elasticity below 0.1. Such a contrast is even strengthened while we exclude fathers dying before the child has reached the age of 6 (Panel B of Table 6). So, clearly orphanhood explains a large share of the IGT but does not seem to capture the whole nurturing role played by mothers. Interpolating the role of orphanhood on the intergenerational elasticity of health obtained through the mother-twin approach would give an elasticity of about 0.3. The same conclusions hold when orphanhood is defined with a more standard threshold of 15 years old.

Second, we may also capture a statistical artefact that women die at a younger age. Even assuming that the nurturing role is equally shared between fathers and mothers, the intergenerational transmission of health could be artificially stronger for mothers than for

three contemporary provinces of Liaoning, Jilin and Heilongjiang (Lee et al, 2010: 5). It is difficult to know whether our results may be extrapolated beyond these three provinces but these data document the demographic, economic, and social life of these populations at a greater level of detail and context than virtually any other late imperial local society (Lee et al. 2010). Similar to Lindhal et al. (2015), we also compare the health distribution of the individuals of our sample compared to the one of the China Health and Nutrition Survey from 1991 and 2015, a survey representative of 15 Chinese provinces (including Liaoning). Figure B1 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.

fathers, simply because women (in particular in poor households) tend to die earlier. To explore such an alternative mechanism, we replicate the main results, splitting the sample between parents dying below or above the median lifespan. Panels C and D of Table 6 summarize the results for the former. Although the intergenerational transmission becomes higher between parents and sons when one of the parents dies at a younger age — lifespan below the median value — the magnitude of the IGT between mothers and sons remains much higher than the one between fathers and sons.

Third, 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), we would not really capture the nurturing effect of mothers but rather the experiences of being in a similar environment. The control for the occurrence of natural disasters would not be localized enough to capture such unobserved heterogeneity between mothers and fathers. To shed light on this alternative channel, we could conjecture that children dying at younger age are more likely to have been confronted to similar parental environmental factors than children dying at older age. To explore such a mechanism, we exclude in Panel E of Table 6 those dying after the age of 20. Contrary to our expectations, the magnitude of the coefficient remains largely unaltered, while the contrast between fathers and mothers remains striking. Our results refute the expectation than sons dying at a younger age —likely to experience a similar environment compared to their fathers —may have a higher intergenerational transmission of health with their fathers.

Overall, the contrast between mothers and fathers in terms of intergenerational transmission of health features considerable stability across alternative samples. Such stability only constitutes an indirect way to shed light on the mechanisms behind the strength of the in-

tergenerational transmission for mothers. Nonetheless, the evidence is sufficiently compiling to support a strong nurturing effect for mothers, in contrast to fathers.

4.3 Is the intergenerational transmission of health stable overtime?

After establishing the nurturing role of mothers in the intergenerational transmission of health, we investigate the stability of such estimates across generations. To that purpose, we estimate equation 5 by pairs of generations. Table 7 suggests that the estimated coefficients remain relatively stable up to generation 5, although with an increasing trend between generations 3 and 5. We should already acknowledge that such an increasing trend cannot be explained with the likely homogenisation of the population that records several successive generations in our sample. However, a strong jump is observed for the association between generations 5 and 6. An increase by about one quarter to one third suggests that the society has witnessed greater equality of opportunities for the generation born on average in 1865 and whose mothers were born on average in 1836. Combined with the decline in lifespan (Figure 2) and the confirmation of our main results when cohorts born after 1830 are sequentially excluded (Figure 3), our results point to a possible structural break in the second half of the nineteenth century.

The social conditions of the latest period of the Qing dynasty could partly explain such a structural break. Society was organized along distinct social groups. The upper gentry ("Shen-Shih" or "Shen-Chin") derived great power, not only through the ownership of land, but also by the organization of local corps, the administration of justice, the control of

the economy (salt monopoly), and the imposition of taxes (Chang, 1967; Chesneaux, 1973). Access to the priviledged elite was highly regulated through a system of examinations and degrees controlled by the government (Chang, 1967). An exception to that regulation is the access to the lower gentry, with the "shen-yuan" and the "chien-sheng" whose titles were acquired by military education. The title of "chien-sheng" could be bought by men of military education (Chang, 1967). Since access to these groups were not hereditary, the levels of social mobility were high for preindustrial standards (Jiang and Kung 2015). As a result, the share of the upper and lower gentry was relatively stable during most of the Qing dynasty. However, such a relative stability was greatly disturbed during the Taiping war (1850-1864). Chang (1967, p.83) indicates that "Regulations were established providing for the contribution of money to the military fund by local people who were, in turn, to be rewarded with increases in the shen-yuan quota of their native places. Such a regulation first appeared in 1853 when there was an urgent need to increase the public revenue in order to meet the ever increasing expenses of war against the Taipings." As a result, the number of people with a lower gentry title increases by about 50 percent, from an estimated 1,094,734 to about 1,443,900 with a strongly reduced total population. The Taiping rebellions mainly took place in South China and led to the loss of at least 25 million lives (Yu, 2012). Such a change in the size of the gentry seems to have been associated with a strengthening in the intergenerational transmission of health.[27] Our data are too limited to shed further light on

[27]In Hao and Xue (2016), the Taiping rebellion is also associated with structural changes such as the implementation of an education reform, the creation of new schools, or the resulting formalization of local self-governance. However, such changes took place in early century. It was indeed "only with the abolishment of the exam system in 1905 that modern education began to expand" (Hao and Xue, 2016, 6). However, we cannot exclude that the dismantlement of kindship networks described by Hao and Xue (2016) may be associated with a change in the intergenerational transmission of health, even if the channel and the resulting bias are far from obvious to conjecture.

the main channels underlying the structural change in the intergenerational transmission of health around 1850. We cannot exclude that our results capture the non-monotonic effect of a distant event (Nybom and Stuhler, 2014). Understanding in depth the nature of such a structural break is certainly a path for further research.

5 Conclusions

Identifying the nurturing effect in the intergenerational transmission of welfare is key to guide policies aiming at promoting equality of opportunity. 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 from the same twin mothers or fathers. We find a strong intergenerational elasticity between mothers and their children, standing at about 0.54. Adjusting for potential orphanhoods, such elasticity should stabilise at about 0.3. Based on the evaluation of the direct impacts of further ancestors, we know that the related two-generations model may underestimate the true intergenerational elasticity between women and their descendants. The estimated elasticity for mothers is therefore likely to be a lower-bound estimate. On the contrary, the intergenerational association from fathers is much weaker and seems to be entirely driven by genetic factors. The weak nurturing effect on the father side 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 direct impact on the children's health.

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. A contrast which 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. More research, using a twin-parent approach, 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 to other outcomes than health, and when the approach is extended to the parents-daughters relationships.

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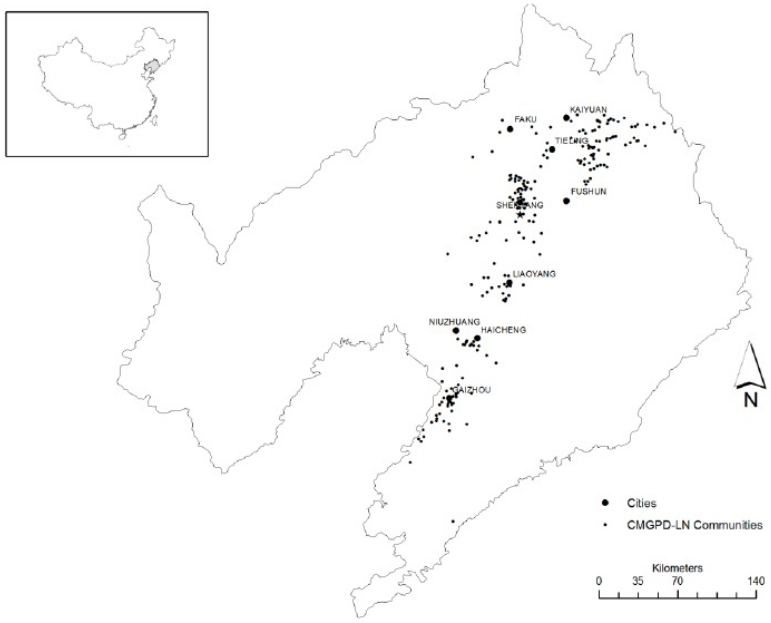
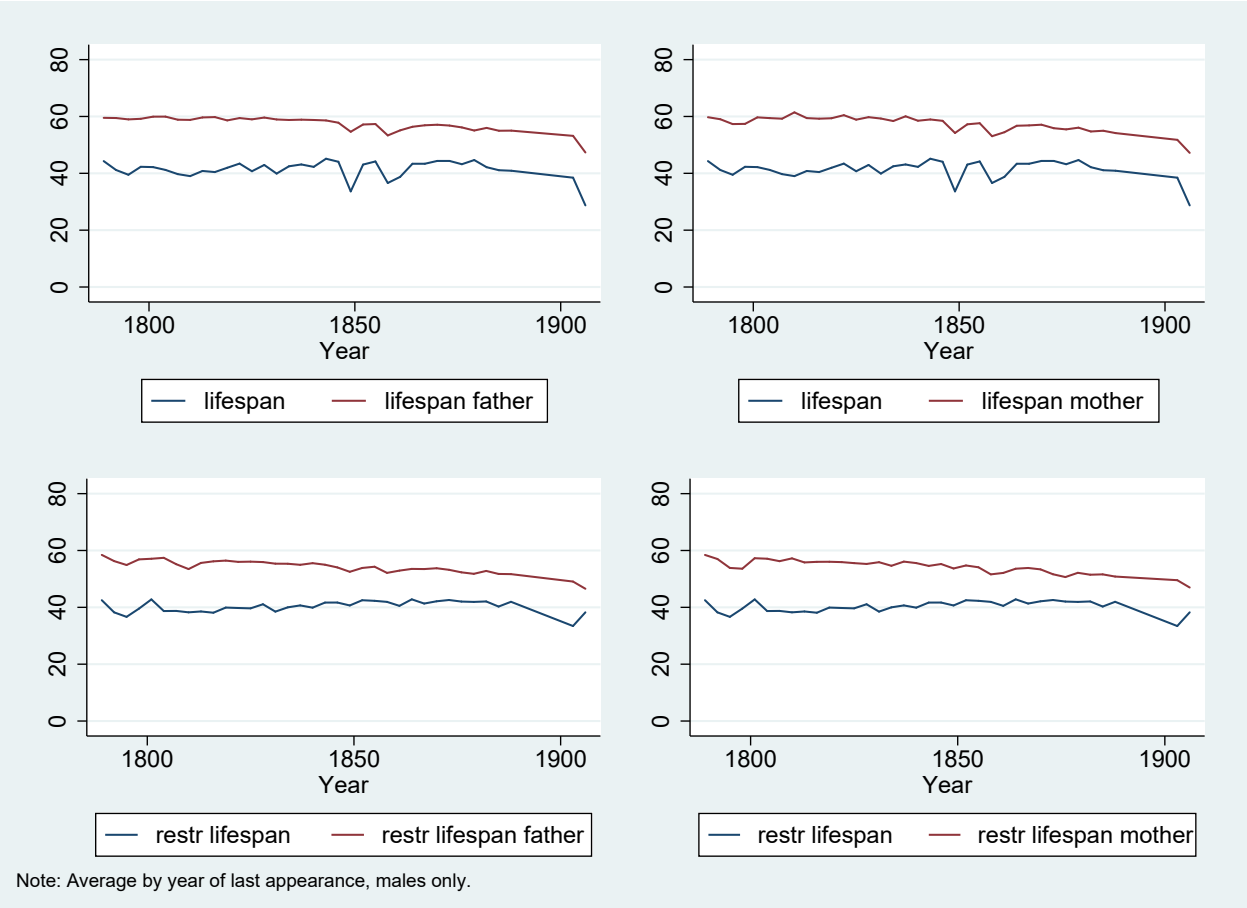
Figure 1: China Multi-Generational Panel dataset-Lioning



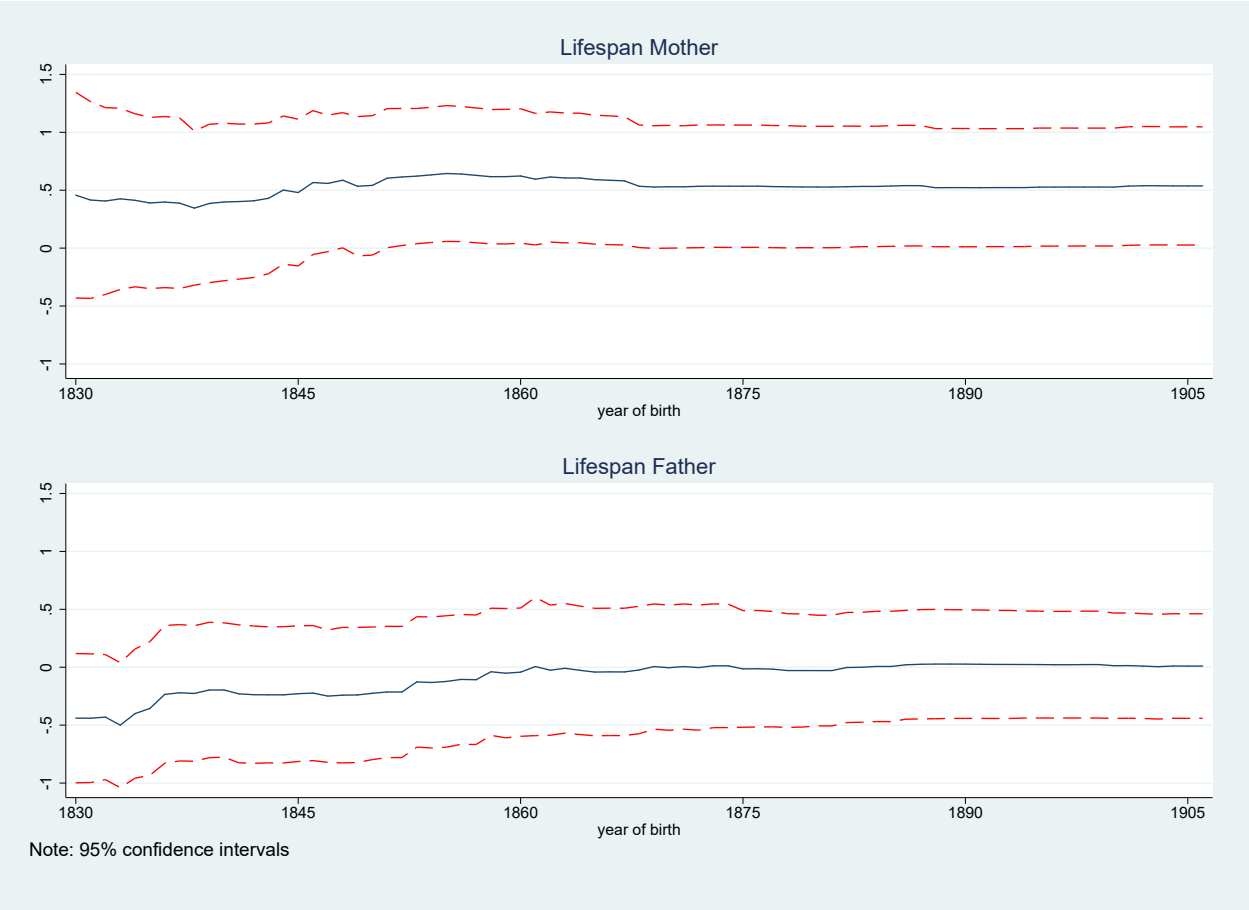
Source: Lee et al. (2010)

Figure 2: Lifespan over time - 1789-1910



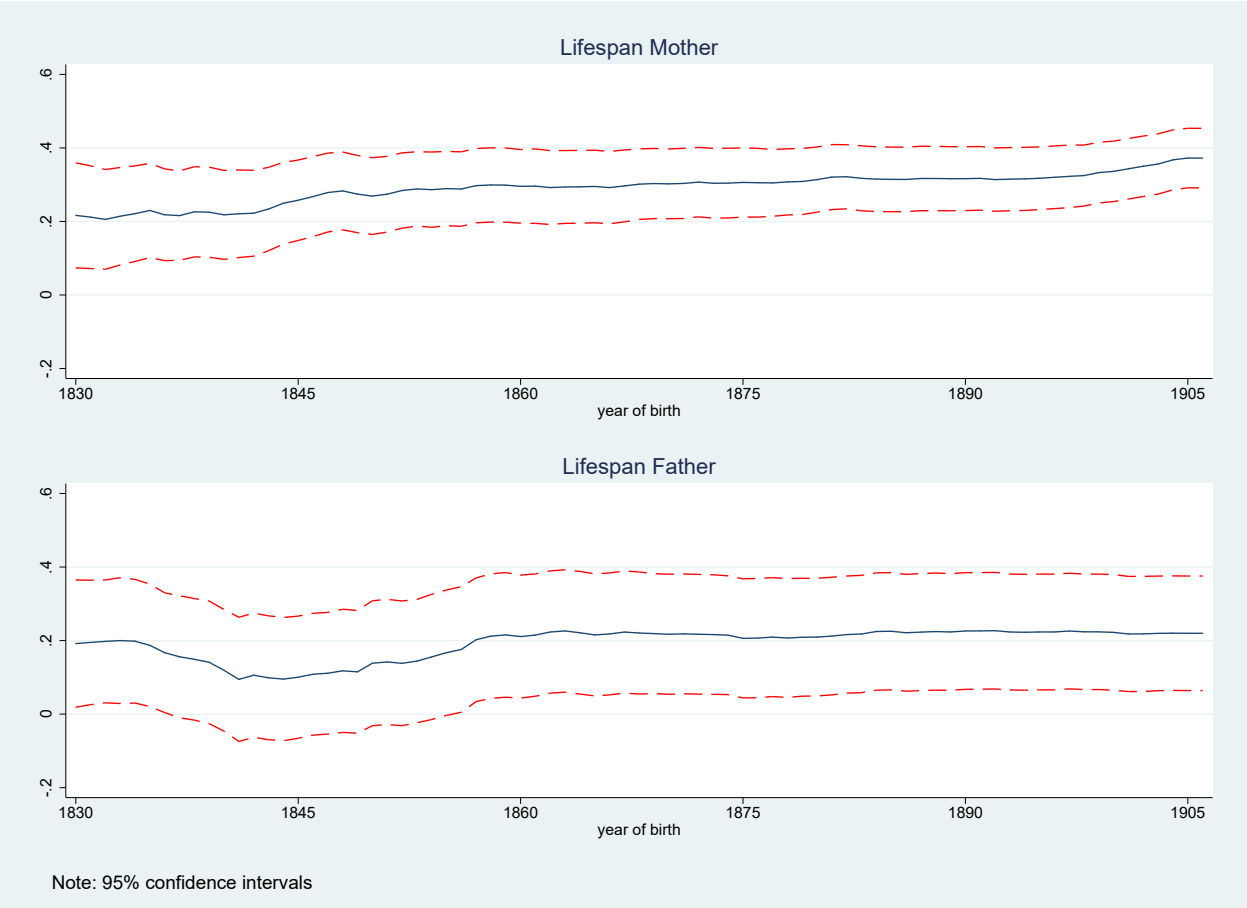
Note: The top figures present the lifespans of sons, fathers and mothers overtime while lifespan is constructed as the difference between the last year observed in the registers and the year of birth. The bottom figures show the same trends but when the lifespan is constructed (for a sub-sample) as the 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 cohorts 1830-1906 - Twins parent estimation



Note: We report the estimated coefficients and confidence intervals for mother's lifespan (top figure) and father's lifespan (low figure) obtained by estimating parent-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).

Figure 4: IGT across cohorts 1830-1906 - Siblings parent estimation



Note: We report the estimated coefficients and confidence intervals for mother's lifespan (top figure) and father's lifespan (low figure) obtained by estimating grand-father 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	33.824	21.548	35310
Lifespan mother	43.279	14.281	6916
Lifespan father	46.285	15.443	5020
Restricted Lifespan			
Lifespan males	34.598	21.418	13228
Lifespan mother	41.558	14.099	2733
Lifespan father	46.125	14.991	1900
Lifespan ancestors			
Lifespan Gfather	55.171	12.879	2123
Lifespan Gmother	60.849	9.559	2652
Lifespan GGfather	57.522	12.245	1095
Lifespan GGmother	67.89	6.948	328
Lifespan GGGfather	59.02	11.643	541
Lifespan GGGGfather	59.664	10.863	289

Sample size of analytical sample used.
Averages for parents and ancestors exclude duplicates.

Table 2: IGT across generations

	LogLifespan						
	father	mother	Gfather	Gmother	GGfather	GGmother	GGGfather
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Dependent variables:							
LogLifespan child	0.329***	0.560***	0.069*	0.711***	0.042	0.696*	-0.066
se	(0.040)	(0.049)	(0.038)	(0.078)	(0.049)	(0.387)	(0.066)
N	10658	10658	10658	10658	6906	625	4265
LogLifespan father			0.003				
se			(0.026)				
N			1861				
LogLifespan mother				0.462***			
se				(0.048)			
N				2489			
LogLifespan Gfather					0.119**		
se					(0.056)		
N					774		
LogLifespan Gmother						0.304*	
se						(0.171)	
N						145	
LogLifespan GGfather							-0.021
se							(0.060)
N							206
predictions			0.001	0.259***	0.000	0.079***	0.000
se			(0.009)	(0.037)	(0.003)	(0.023)	(0.000)
Year and District FE	✓	✓	✓	✓	✓	✓	✓
Individual characteristics	✓	✓	✓	✓	✓	✓	✓
Assortative mating	✓	✓	✓	✓			

Each estimates is from a separate regression of the loglifespan of a family member on the lifespan of an older member.
Predictions are obtained by the product of the IGC estimates of consecutive generations.
Standard errors of prediction are computed using the Delta method.
Sample size restricted to available lifespan for M, F, GF and GM.
Coefficients presented in columns (1) and (2) are generated from the same regression.
Standard errors clustered at descent level. Significance levels: *** 1% ** 5% * 10%

Table 3: AR models of IGT across generations

Dependent variable:	LogLifespan					
	AR(1)	AR(1)	AR(2)	AR(3)	AR(4)	AR(5)
	(1)	(2)	(3)	(4)	(5)	(6)
LogLifespan mother	0.584***	0.560***	0.537***	0.649***	0.467*	0.775*
	(0.032)	(0.048)	(0.048)	(0.176)	(0.238)	(0.439)
LogLifespan father	0.281***	0.329***	0.298***	0.355**	0.395*	0.507
	(0.026)	(0.040)	(0.041)	(0.151)	(0.200)	(0.338)
LogLifespan Gmother			0.393***	0.403	0.639*	0.747
			(0.071)	(0.307)	(0.334)	(0.795)
LogLifespan Gfather			0.059	-0.003	-0.073	0.286
			(0.037)	(0.112)	(0.127)	(0.376)
LogLifespan GGmother				0.127	-0.045	0.394
				(0.483)	(0.498)	(0.771)
LogLifespan GGfather				0.345**	-0.002	-0.171
				(0.172)	(0.208)	(0.374)
LogLifespan GGfather					-0.121	-0.248
					(0.238)	(0.313)
LogLifespan GGGGfather						-0.194
						(0.232)
N	35310	10658	10658	444	330	139

All models include Year and district FE and individual characteristics.
In Columns (2) to (6), sample size restricted to available lifespan for M, F, GF and GM.
Standard errors clustered at descent level.
Significance levels: *** 1% ** 5% * 10%.

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

Dependent variable:	Log Lifespan						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Panel A: mother LS only							
LogLifespan mother	0.425***	0.591**		0.551**		0.641**	
	(0.042)	(0.261)		(0.258)		(0.291)	
N	27898	1265		1265		1265	
Panel B: father LS only							
LogLifespan father	0.295***		0.221		0.074		-0.050
	(0.047)		(0.233)		(0.229)		(0.257)
N	27898		1150		1150		1150
Panel C: father and mother LS							
LogLifespan mother	0.384***	0.576**		0.537**		0.640**	
	(0.042)	(0.262)		(0.260)		(0.290)	
N	27898	1265		1265		1265	
LogLifespan father	0.186***		0.162		0.010		-0.072
	(0.046)		(0.232)		(0.230)		(0.255)
N	27898		1150		1150		1150
Year and District FE	✓	✓	✓	✓	✓	✓	✓
Individual characteristics				✓	✓	✓	✓
Parental characteristics						✓	✓
GF FE	✓						
Twin mother		✓		✓		✓	
Twin father			✓		✓		✓

Standard errors clustered at descent level.
Significance levels: *** 1% ** 5% * 10%.
Mother and father twins identified using mother and father ID, respectively.
For comparability reasons, models with mother and father twins are restricted to the sample size of col. 6 and col 7, respectively.
For presentation purposes, the coefficients and standard errors of the partner's lifespan is omitted in panel C. They should not receive much interpretation but are provided in table B2 of Appendix B.

Table 5: Measurement errors. IGT estimates with alternative samples

Dependent variable:	Log Lifespan		
	(1)	(2)	(3)
Panel A: Main results			
LogLifespan mother	0.384***	0.373***	0.359***
	(0.042)	(0.041)	(0.047)
LogLifespan father	0.186***	0.201***	0.181***
	(0.046)	(0.043)	(0.048)
N	27898	27898	27898
Panel B: Using the strict definition of son's lifespan			
LogLifespan mother	0.291***	0.251**	0.226*
	(0.102)	(0.099)	(0.115)
LogLifespan father	0.143	0.144	0.070
	(0.123)	(0.122)	(0.140)
N	7835	7835	7835
Panel C: Only with first and second birth sons			
LogLifespan mother	0.405***	0.374***	0.373***
	(0.047)	(0.046)	(0.053)
LogLifespan father	0.207***	0.207***	0.186***
	(0.050)	(0.047)	(0.050)
N	21827	21827	21827
Panel D: Excluding potential migrants			
LogLifespan mother	0.378***	0.381***	0.352***
	(0.044)	(0.044)	(0.050)
LogLifespan father	0.204***	0.211***	0.188***
	(0.047)	(0.046)	(0.050)
N	25074	25074	25074
Panel E: Excluding children dying before 6			
LogLifespan mother	0.216***	0.215***	0.226***
	(0.032)	(0.031)	(0.036)
LogLifespan father	0.083***	0.094***	0.078**
	(0.031)	(0.031)	(0.033)
N	24129	24129	24129
Year, District and GF FE	✓	✓	✓
Individual characteristics		✓	✓
Parental characteristics			✓

Standard errors clustered at descent level.
Significance levels: *** 1% ** 5% * 10%.

Table 6: Interpretation. IGT estimates with alternative samples

Dependent variable:	Log Lifespan		
	(1)	(2)	(3)
Panel A: Excl children whose mothers died before 6			
LogLifespan mother	0.242***	0.234***	0.204***
	(0.048)	(0.046)	(0.051)
LogLifespan father	0.108**	0.116**	0.081*
	(0.047)	(0.045)	(0.048)
N	24681	24681	24681
Panel B: Excl children whose fathers died before 6			
LogLifespan mother	0.345***	0.322***	0.302***
	(0.050)	(0.048)	(0.055)
LogLifespan father	0.110*	0.124**	0.079
	(0.057)	(0.055)	(0.058)
N	23270	23270	23270
Panel C: mother's LS below the median			
LogLifespan mother	0.473***	0.493***	0.489***
	(0.071)	(0.073)	(0.075)
LogLifespan father	0.248***	0.281***	0.272***
	(0.074)	(0.072)	(0.077)
N	14423	14423	14423
Panel D: father's LS below the median			
LogLifespan mother	0.353***	0.387***	0.379***
	(0.065)	(0.067)	(0.076)
LogLifespan father	0.274***	0.316***	0.320***
	(0.079)	(0.075)	(0.077)
N	13984	13984	13984
Panel E: Excluding children dying above 20			
LogLifespan mother	0.324***	0.395***	0.396***
	(0.069)	(0.073)	(0.085)
LogLifespan father	0.091	0.133**	0.150**
	(0.067)	(0.068)	(0.068)
N	7081	7081	7081
Year, District and GF FE	✓	✓	✓
Individual characteristics		✓	✓
Parental characteristics			✓

Standard errors clustered at descent level.
Significance levels: *** 1% ** 5% * 10%.
Partner's lifespan is included in all models but not reported here for presentation purposes.

Table 7: IGT across 7 generations

Dependent variable:	LogLifespan son					
	g2	g3	g4	g5	g6	g7
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: no controls						
LogLifespan mother (g1)	0.464***					
	(0.188)					
LogLifespan mother (g2)		0.600**				
		(0.171)				
LogLifespan mother (g3)			0.559***			
			(0.125)			
LogLifespan mother (g4)				0.321***		
				(0.109)		
LogLifespan mother (g5)					0.618***	
					(0.173)	
LogLifespan mother (g6)						0.331*
						(0.178)
Panel B: including individual and parental characteristics						
LogLifespan mother (g1)	0.435***					
	(0.204)					
LogLifespan mother (g2)		0.514*				
		(0.174)				
LogLifespan mother (g3)			0.525***			
			(0.130)			
LogLifespan mother (g4)				0.326***		
				(0.103)		
LogLifespan mother (g5)					0.670***	
					(0.174)	
LogLifespan mother (g6)						0.286*
						(0.148)
Average birth year						
	g2	g3	g4	g5	g6	g7
son	1794	1804	1823	1842	1865	1885
	g1	g2	g3	g4	g5	g6
mother	1764	1774	1793	1813	1836	1858
N	392	726	1,288	1,128	516	200

Each estimates is from a separate regression of loglifespan of son on lifespan of mother
All models include Year and District FE and control for Assortative mating
Standard errors clustered at descent level.
Significance levels: *** 1% ** 5% * 10%.

For Online Publication

Separate Appendixes with Supplemental Material for:

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

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 and Shilpi, 2016).

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).

B Tables and Figures

Table B1: Descriptive Statistics of control variables

	Mean	**Std. Dev.**
disabled	0.056	0.23
hh size	14.873	13.803
N. brothers	0.848	1.17
N. sisters	0.157	0.472
migrant	0.087	0.282
early life shocks	2.272	2.264
antenatal shocks	0.36	0.752
birth order 1	0.454	0.498
birth order 2	0.267	0.442
birth order 3	0.137	0.344
birth order 4	0.069	0.253
birth order 5	0.034	0.180
birth order 6	0.017	0.13
father disabled	0.179	0.383
father migrant	0.144	0.352
age at son's birth	21.603	11.49
mother disabled	0	0.009
mother migrant	0.106	0.308
age at son's birth	25.938	8.945

Analytical Sample size 35310.

Table B2: IGT estimates with twin-parent fixed effects[a]

Dependent variable:	Log Lifespan			
	(1)	(2)	(3)	(4)
LogLifespan mother	0.537**	0.318*	0.640**	0.183
	(0.260)	(0.176)	(0.290)	(0.191)
LogLifespan father	0.187	0.010	0.305	-0.072
	(0.231)	(0.230)	(0.237)	(0.255)
disabled	0.581***	0.372***	0.560***	0.385***
	(0.198)	(0.136)	(0.201)	(0.143)
Log(household size)	-0.241	-0.033	-0.194	-0.031
	(0.159)	(0.076)	(0.165)	(0.076)
n. of brothers	-0.098*	-0.037	-0.101*	-0.034
	(0.052)	(0.053)	(0.054)	(0.054)
n. of sisterst	0.044	0.084	0.047	0.080
	(0.116)	(0.096)	(0.116)	(0.100)
migrant	0.922***	0.686***	0.916***	0.716***
	(0.273)	(0.142)	(0.278)	(0.145)
earlylife shock	0.022	-0.024	0.017	-0.024
	(0.030)	(0.025)	(0.031)	(0.025)
antenatal schocks	0.019	0.006	0.012	0.000
	(0.047)	(0.055)	(0.047)	(0.056)
birth order 1	0.531	0.575	0.562	0.543
	(0.347)	(0.373)	(0.352)	(0.374)
birth order 2	0.419	0.349	0.449	0.320
	(0.335)	(0.365)	(0.342)	(0.365)
birth order 3	0.452	0.363	0.479	0.330
	(0.324)	(0.325)	(0.330)	(0.327)
birth order 4	0.399	0.444	0.416	0.431
	(0.320)	(0.322)	(0.326)	(0.320)
birth order 5	0.458	0.387	0.472	0.358
	(0.348)	(0.344)	(0.356)	(0.347)
birth order 6	0.249	0.239	0.260	0.236
	(0.335)	(0.324)	(0.338)	(0.326)
father disabled			0.021	0.006
			(0.176)	(0.138)
father migrant			0.412	0.328
			(0.281)	(0.247)
father's age at son's birth			-0.026	0.019
			(0.019)	(0.022)
father's age^2 at son's birth			0.000	-0.000
			(0.000)	(0.000)
mother disabled			0.000	-0.104
			(.)	(0.476)
mother migrant			-0.199	-0.403*
			(0.366)	(0.243)
mother's age at son's birth			0.022	0.039
			(0.038)	(0.025)
mother's age^2 at son's birth			-0.000	-0.001
			(0.001)	(0.000)
N	1265	1150	1265	1150
Year and District FE	✓	✓	✓	✓
Twin mother	✓		✓	
Twin father		✓		✓

Mother and father twins identified using mother and father id, respectively.
Full table (panel C, col.4-7, Table 4)[a].
Standard errors clustered at descent level.
Significance levels: *** 1% ** 5% * 10%.

Table B3: IGT estimates adjustment

Dependent variable:	Log Lifespan						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
			$\lambda = 1$	$\lambda = 0.75$	$\lambda = 0.50$	$\lambda = 0.25$	$\lambda = 0.1$
Panel A: mother twin FE							
LogLifespan mother	0.437***	0.576**	0.716**	0.660	0.623	0.596	0.584
	(0.068)	(0.262)	[0.374]	[0.500]	[0.755]	[1.520]	[3.816]
GM FE	✓						
Twin mother		✓					
$\theta = 0.5$			✓	✓	✓	✓	✓
Panel B: father twin FE							
LogLifespan father	0.186***	0.161	0.137	0.147	0.153	0.158	0.160
	(0.046)	(0.232)	[0.242]	[0.325]	[0.492]	[0.993]	[2.500]
GF FE	✓						
Twin father		✓					
$\theta = 0.5$			✓	✓	✓	✓	✓

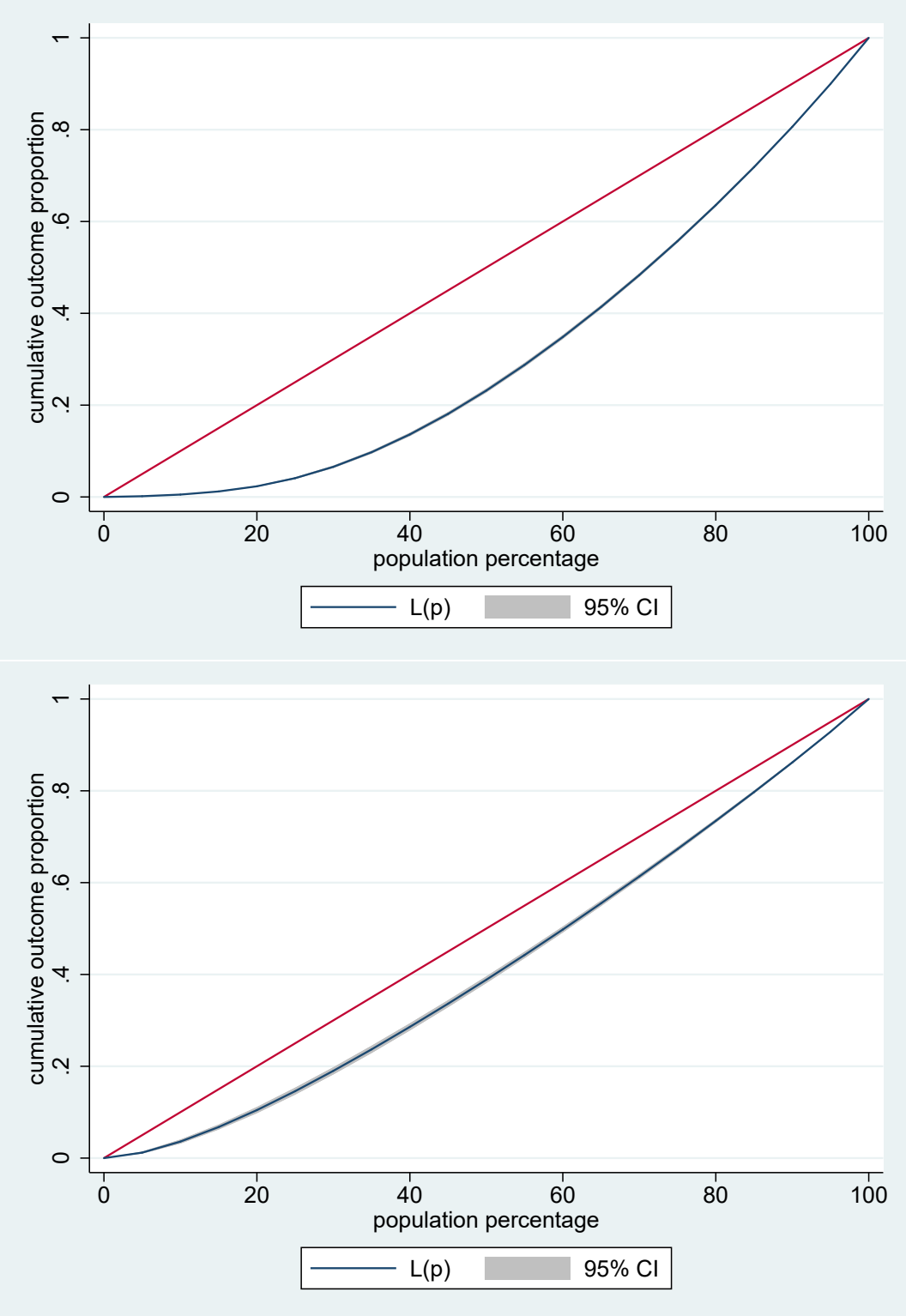
Standard errors in parentheses clustered at descent level. Standard errors in brackets are computed following Conley et al. (2006) using the pooled variance and taking the square root of $V(\widehat{\beta_{TS}} = \frac{V(\widehat{\beta_{TW}})(\frac{1}{1-\theta})^2(N_{TW}-1)+V(\widehat{\beta_{SIB}})(\frac{\theta}{1-\theta})^2(N_{SIB}-1)}{N_{TW}+N_{SIB}})$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

Table B4: Effect of parents lifespan on twins probability

Dependent variable:	Twin			
	Father		Mother	
	(1)	(2)	(3)	(4)
Panel A				
LogLifespan mother	-0.009	-0.006		
	(0.035)	(0.035)		
LogLifespan father	0.014	0.011		
	(0.018)	(0.018)		
N	8126	8126		
Panel B				
LogLifespan mother in law			0.083**	0.083**
			(0.035)	(0.036)
LogLifespan father in law			-0.019	-0.019
			(0.019)	(0.019)
N			8135	8135
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%.

Figure B1: 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.