

Intergenerational Persistence of Health in Indonesia: The Importance of Using Biomarkers

by

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Intergenerational Persistence of Health in Indonesia: the Importance of Using Biomarkers*

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Abstract

We examine health persistence between parents and their adult children in Indonesia using both subjective and objective health measures including biomarkers. Using Principal Components Analysis, we estimate the intergenerational persistence of the combination of these measures to be 0.30, providing some of the first estimates of the transmission of latent health for a middle income country. We also detect a highly significant second principal component suggesting that health has multiple dimensions. We find especially strong associations for biomarkers such as hemoglobin, the pulse rate and hypertension which have typically not been studied in prior intergenerational studies. Transmission is stronger from mothers, and to daughters. We find relatively little variation in intergenerational health transmission by family income or SES. However, we do find strong positive gradients between family SES and the pulse rate and obesity suggesting potential health pitfalls as low and middle income countries further develop. Our findings suggest a potentially important role for policies focused on maternal health in reducing the intergenerational transmission of health.

Keywords: Intergenerational persistence; health; biomarkers; Indonesia

JEL Classification Codes: D63; J62; I14.

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1 Introduction

The idea that all individuals should have the same chances to succeed in life regardless of their socioeconomic background in childhood is central to policy makers around the world. In recent decades, the concept of equality of opportunity has gained prominence due in part to concerns that rising inequality has reduced social mobility. Most notably, the “Great Gatsby” curve, has highlighted the strong association between income inequality and social immobility across countries (Corak, 2013; Durlauf et al., 2022; Song, 2022).

Although the literature on intergenerational mobility has focused mainly on income, education, or occupation, there is increasing recognition that health is an important dimension of socioeconomic well-being (Jones and Klenow, 2016). However, one notable challenge for studying health is that it is a *latent* concept that is not easily measured. Researchers typically have to resort to using blunt proxies for health such as birth weight, height, or longevity. Nevertheless, a growing number of studies in many countries using both survey and administrative data, and utilizing innovative approaches to better approximate latent health, have begun to estimate intergenerational persistence in health (Halliday, 2023; Mazumder, 2024).

This literature, however, has primarily centered on more advanced economies such as the US (Halliday et al., 2020, 2021; Fletcher and Jajtner, 2021), the UK (Bencsik et al., 2023), Denmark (Andersen, 2021), Germany (Graeber, 2023), Norway (Bütkofer et al., 2024), and Australia (Vera-Toscano and Brown, 2022), and has paid far less attention to lower and middle income countries (LMICs).¹ This is unfortunate, as the vast majority of the world’s population lives in LMICs, where rising inequality has been a persistent trend. These conditions likely contribute to declining mobility, making it all the more important to understand intergenerational processes in these settings. Moreover, poor health is arguably an even greater impediment to economic success in LMICs since health systems are not as developed and insurance is generally less accessible than in wealthier countries.

One reason for the absence of studies on LMICs is data limitations. In advanced economies, there are excellent data sources including long-running panels such as the Panel Study of Income Dynamics (PSID) in the US, or rich administrative data in Scandinavian countries. While data of this quality are not common in LMICs, an important exception is Indonesia, which has the one of the highest quality panel surveys in the Indonesian Family Life Survey (IFLS). In fact, the rate of attrition in the IFLS is lower than in many other panels in more advanced economies (Thomas et al., 2001). Moreover, the IFLS contains unique biomarker data that has not yet been fully exploited in intergenerational analyses.

We use the IFLS to build on this literature by examining intergenerational health mobility in Indonesia - a fast growing, middle income country. Indonesia exhibits strikingly high levels of inequality. In fact, it is now more unequal than the US, making it one of the most unequal countries globally (Zhang, 2021). The country also has a substantial burden of nutrition-related chronic disease. For example, the prevalence of anemia is high with 48.9% of pregnant women and 38.5% of children under five reported as anemic in 2018 (Sungkar et al., 2022). Adiposity is similarly widespread. Indonesia has the highest prevalence of overweight children under five among Southeast Asian countries (Rachmi et al., 2017). These patterns are increasingly seen in

¹Some exceptions include Chang et al. (2024) who study Taiwan, and Kumar and Nahlen (2023) and Kumar et al. (2025) who study India.

LMICs as economic development has led to greater consumption of calorie-dense but nutrient-poor foods. As LMICs develop, they undergo the so-called “epidemiological transition”, shifting from a disease burden dominated by infectious conditions to one increasingly characterized by chronic diseases (Omran, 1971).

The IFLS is an ideal data source for examining intergenerational health mobility in an LMIC. Not only does the IFLS have detailed health information, including biomarkers, but it has also been running sufficiently long that data is available on two generations of individuals from the same families. The length of the panel also allows us to create time averages of health outcomes over long periods of the life course resulting in less error prone measures of latent health (Mazumder, 2005; Halliday et al., 2021).

We utilize several measures of health in our analysis. First, we use a Quality Adjusted Life Year (QALY) or the fraction of a year that a person spends in good health. This is created by converting reports on self-reported health status (SRHS), taken over multiple surveys, into a QALY following the methodology used by Halliday et al. (2021). This also allows us to compare our estimates to those of studies undertaken in the US (Halliday and Mazumder, 2017), the UK (Bencsik et al., 2023) and Australia (Vera-Toscano and Brown, 2022). Since self-reported health is often criticized for its subjectivity, particularly in LMICs, it helps to also use more objective measures that may provide a broader reading of latent health.² We utilize high quality biomarkers including: hemoglobin (Hb), blood pressure, and the pulse rate, as well as anthropomorphic measures based on height and weight such as the body mass index (BMI). Biomarkers are especially useful for gaining insight into latent health as they provide measurable indicators of disease before symptoms may appear and be captured by clinical data or surveys.³ Using these measures, we create additional indicators for the presence of anemia, hypertension, obesity, and being underweight.

We begin by estimating intergenerational persistence across eight different measures of health. To do so, we employ two complementary metrics. The first is the Intergenerational Health Association (IHA), which captures the extent to which gaps in health levels persist across generations; conversely, one minus the IHA provides a natural measure of mobility. The second metric is the rank–rank slope, a measure of positional mobility constructed by converting health levels into ranks and then estimating the IHA on these ranked outcomes. Each measure illuminates different conceptual dimensions of intergenerational mobility (Deutscher and Mazumder, 2023). We estimate these parameters separately for each parent–child gender pairing, although our baseline estimates combine both parents and pool sons and daughters to reduce the influence of measurement error.

First, we estimate an IHA of 0.18 when using the QALY, while the corresponding rank–rank slope is 0.12. These estimates are noteworthy, as they are among the first from an LMIC that are directly comparable to those from higher-income countries. The IHA estimate aligns closely with estimates from richer settings,

²For example Sen (2002) argues that there can be meaningful differences between individual perceptions of health and actual objective health readings that could depend on the surrounding health environment. On the other hand, some empirical evidence suggests that this criticism may be overblown (Subramanian SV, 2009). Both Halliday et al. (2021) and Bencsik et al. (2023) find very similar levels of intergenerational persistence when using subjective or objective health measures, though both studies are in advanced economies.

³For example, Blanchflower and Bryson (2022) show that the pulse rate is particularly useful predictor of health status and well being.

whereas the rank–rank slope is somewhat lower.⁴

A broader look across subgroups reveals several systematic patterns in intergenerational health transmission. When we estimate persistence separately by parent–child gender combinations, we consistently find stronger maternal than paternal transmission, as well as greater parental influence on daughters than on sons. We also examine how children’s health correlates with parental SES and find that pulse rate and weight-related measures such as BMI and overweight exhibit significant positive associations. This suggests a *worsening* of these health indicators as SES improves. This pattern implies that LMICs may face important emerging health challenges, particularly related to chronic disease, as they continue to develop.

Importantly, our estimates of intergenerational persistence are substantially higher when using biomarkers. In the baseline sample, we estimate an IHA of 0.23 for hemoglobin, 0.27 for hypertension, and 0.32 for pulse rate. Consistent with previous studies ([Akbulut-Yuksel and Kugler, 2016](#); [Classen, 2010](#)), we find especially strong persistence in adiposity outcomes, with an IHA of 0.49 for BMI and 0.38 for overweight. These relatively large IHA estimates based on biomarkers suggest that SRHS-based measures may not fully capture unobserved components of health or underlying latent health in the Indonesian context.

Given our rich set of both subjective and objective health measures, we next use principal components analysis (PCA) to construct proxies for latent health. Interestingly, and in contrast to previous studies employing PCA in intergenerational health research ([Andersen, 2021](#); [Chang et al., 2023](#); [Kumar et al., 2025](#)), we find *two* significant and meaningful principal components. The IHA for both the first and second components (PC1 and PC2) is 0.27. When we use the sum of all components with eigenvalues exceeding unity, the estimated IHA rises to 0.303. These results suggest that Indonesia may exhibit greater intergenerational persistence—and correspondingly lower mobility—than the countries examined to date in this literature. A caveat, however, is that prior studies have not incorporated biomarkers such as the ones available in the IFLS into their analyses. We also estimate the PCA using our indicators for health conditions. Using this approach, and focusing on maternal transmission, we can interpret up to three components of health: one that is nutrition related, a second that is cardiovascular in nature, and a third that picks up self reported health.

We then examine heterogeneity in health mobility by first looking at rank persistence by parent income, socioeconomic advantage, ethnicity, and region. We do not find significant differences across any of these groups. However, when we analyze *absolute* mobility, by using conditional expected ranks ([Chetty et al., 2014](#); [Deutscher and Mazumder, 2023](#)) we detect several interesting patterns. In particular, there is a clear gradient in upward mobility in overweight, which is defined as a BMI exceeding 25, by education. Upward mobility in overweight declines as education increases. For example, an unschooled child whose parents were at the 25th percentile of the national overweight distribution could expect to reach the 46th percentile of the same distribution. On the other hand, a child with at least a high school education would be expected to reach only the 39th percentile. This suggests that the least educated Indonesians are catching up to their better-educated counterparts in terms of their overweight status.

⁴For example, the IHA estimates for the QALY are 0.23 for the US ([Halliday et al., 2020](#)), 0.19 for the UK ([Bencsik et al., 2023](#)), and 0.20 for Australia ([Vera-Toscano and Brown, 2022](#)). The corresponding rank–rank slope estimates are 0.26 for the US and 0.17 for the UK. A rank–rank slope was not estimated for Australia by [Vera-Toscano and Brown \(2022\)](#).

We make several contributions to the literature on intergenerational health mobility. First, we are among the earliest studies to examine health mobility in an LMIC. Second, owing to the high quality of the IFLS and its similarity to leading panel datasets such as the Panel Study of Income Dynamics (PSID), UK Household Longitudinal Study (UKHLS), and Household, Income and Labour Dynamics in Australia (HILDA), we are able to employ state-of-the-art methods and generate estimates that are directly comparable to those in [Halliday et al. \(2021\)](#), [Bencsik et al. \(2023\)](#), and [Vera-Toscano and Brown \(2022\)](#). Third—and perhaps most importantly—we are among the first to use biomarker data to study intergenerational health transmission. This is a particularly promising direction for future research, as certain measures, such as pulse rate, do not require invasive or expensive technology. Most previous studies, including those using panel or administrative data in high-income countries, lack longitudinal biomarker information of comparable quality. Moreover, relative to other LMIC studies, we examine a broader set of health domains beyond nutrition, which has been the primary focus of recent work on India ([Kumar and Nahlen, 2023](#); [Kumar et al., 2025](#)).

In addition, we make a couple of other important contributions. The first is that, following [Andersen \(2021\)](#), [Chang et al. \(2024\)](#), and [Kumar et al. \(2025\)](#), we construct measures of latent health by applying PCA to a battery of health indicators, yielding a comprehensive proxy for an individual's underlying health status. However, ours is the first study to identify a meaningfully important second principal component of health that is itself intergenerationally transmitted. We also employ the sum of all relevant components. Finally, we uncover salient intergenerational socioeconomic gradients. In particular, children of higher-SES parents exhibit worse adiposity-related outcomes, suggesting that economic development in LMICs may be accompanied by a rising burden of chronic disease.

The remainder of the paper is organized as follows. Section 2 describes the data. Section 3 outlines the empirical methods. Section 4 presents the main findings. We explore heterogeneity in Section 5. Finally, Section 6 concludes.

2 Data

We use the Indonesia Family Life Survey (IFLS) which is a longitudinal survey conducted over five waves between 1993 and 2015.⁵ The IFLS is representative of approximately 83% of the Indonesian population and includes over 30,000 individuals residing in 13 of the country's 27 provinces. In particular, the IFLS has been hailed for its particularly low attrition rate ([Thomas et al., 2001](#)). In addition to exhibiting low attrition across panel waves, the IFLS is especially well-suited to intergenerational health research because it measures a wide range of health outcomes, including detailed biometrics and anthropometrics. We are also able to track individuals for three years on average which allows us to mitigate the effects of measurement errors on our mobility estimates ([Mazumder, 2005](#); [Halliday et al., 2021](#)).

⁵These include waves in 1993-4, 1997, 2000, 2007-8 and 2014-15. We make use of all five waves

2.1 Health Measures

We use a variety of health measures including self-reported health status (SRHS) along with several biomarkers and anthropometric measures. In the IFLS, SRHS is a four point categorical variables in which respondents report their health as either: very healthy, somewhat healthy, somewhat unhealthy, or very unhealthy. Following [Johnson and Schoeni \(2011\)](#) and [Halliday et al. \(2021\)](#), we map these categories into a continuous measure of a Quality Adjusted Life Year (QALY).⁶ The QALY captures the portion of a year an individual spends in good health. Self-reported poor health was coded as 1 if an individual's self-rated health was somewhat unhealthy, or very unhealthy, and 0 otherwise. Our biomarkers include: hemoglobin, blood pressure (systolic and diastolic) and the pulse rate. These measures are also used to create indicators for anemia and hypertension. Anemia was defined as a hemoglobin level of 12 g/dL or below for women and 13 g/dL or below for men. Hypertension was coded as 1 if an individual had a diastolic blood pressure of at least 90 mmHg or a systolic blood pressure of at least 140 mmHg. Abnormal pulse was defined as a resting pulse rate of 60 beats per minute or lower, or 100 beats per minute or higher. Finally, we use the following anthropometric measures: Body Mass Index (BMI), an indicator for being underweight ($BMI < 18.5$), and an indicator for being overweight ($BMI \geq 25$).

Like [Halliday et al. \(2021\)](#), we follow the intergenerational mobility literature practice of using time averages of child and parent outcomes. Specifically, we have observations for each outcome (e.g. SRHS, hemoglobin, BMI, etc.) for a number of survey years for each individual. We average these outcomes across survey years to mitigate attenuation bias from measurement errors ([Mazumder, 2005](#)).

2.2 Descriptive statistics

We present descriptive statistics in Tables [1](#) and [2](#). We display statistics for time averages of each health measure as well as for a set of basic demographic and socioeconomic variables. When these other variables vary over time, we also report time averages as is the case with age. Because the reporting rates for each health outcome differ somewhat, we show descriptive statistics for each sample separately. In practice, the patterns in the demographic covariates are quite similar, so we only discuss these patterns for the QALY sample. In this sample, the average age is 56 for fathers, 53 for mothers, and 35 for children. We note that the age composition is very similar to [Halliday et al. \(2021\)](#) who used the PSID.⁷ As expected, we see that fathers tend to be slightly more educated than mothers. Furthermore, the children in the sample have significantly more years of education than their parents. For example, 40% of children have a high school degree or more compared to just 13% of fathers and 5% of mothers.

⁶We record very healthy as 95, somewhat healthy as 80, somewhat unhealthy as 50, and very unhealthy as 15.

⁷The average age was just over 56 for both parents and just over 38 for all children in [Halliday et al. \(2021\)](#).

Table 1: Summary Statistics

	Parents		Children		
	Father	Mother	Sons	Daughters	All
	(1)	(2)	(3)	(4)	(5)
Panel A: QALY					
Age	56.11	52.72	35.20	35.41	35.31
	(9.33)	(9.73)	(3.92)	(4.39)	(4.16)
Unschooled	0.12	0.25	0.02	0.04	0.03
Less than JHS	0.63	0.6	0.31	0.34	0.33
Junior High School	0.12	0.09	0.25	0.25	0.25
High School or above	0.13	0.05	0.42	0.37	0.40
QALY	74.72	74.48	77.86	76.42	77.14
	(9.53)	(8.94)	(12.28)	(13.06)	(12.70)
Very Healthy	0.01	0.01	0.12	0.10	0.11
Somewhat Healthy	0.73	0.73	0.71	0.68	0.70
Somewhat Unhealthy	0.26	0.26	0.17	0.21	0.19
Unhealthy	0.00	0.00	0.01	0.01	0.01
Avg. times observed	3.3	3.2			3.2
Number of obs	18,673	22,704	11,292	11,984	23,276
Panel B: Anemia/Hemoglobin					
	Father	Mother	Sons	Daughters	All
	(1)	(2)	(3)	(4)	(5)
Age	57.93	52.53	35.71	35.99	35.85
	(9.43)	(8.79)	(4.74)	(5.19)	(4.97)
Unschooled	0.12	0.21	0.02	0.04	0.03
Less than JHS	0.64	0.63	0.31	0.34	0.32
Junior High School	0.12	0.1	0.25	0.25	0.25
High School or above	0.13	0.05	0.42	0.37	0.39
HB Reading	13.52	12.30	14.71	12.51	13.58
	(1.56)	(1.17)	(1.55)	(1.34)	(1.82)
Anemia	0.33	0.73	0.11	0.64	0.38
Avg. times observed	2.9	2.9			2.9
Number of obs	14,292	18,226	8,952	9,948	18,900

Table 2: Summary Statistics

	Parents		Children		
	Father	Mother	Sons	Daughters	All
	(1)	(2)	(3)	(4)	(5)
Age	58.27 (9.40)	55.02 (10.12)	35.94 (4.72)	36.25 (5.21)	36.19 (4.98)
Unschooled	0.12	0.26	0.02	0.04	0.03
Less than JHS	0.63	0.6	0.31	0.35	0.33
Junior High School	0.12	0.09	0.25	0.25	0.25
High School or above	0.13	0.05	0.42	0.37	0.39
Systolic	123.31 (20.07)	125.74 (22.03)	118.84 (14.07)	113.11 (14.43)	115.91 (14.54)
Diastolic	98.21 (18.71)	98.01 (18.35)	84.68 (12.57)	83.69 (12.65)	84.17 (12.62)
Hypertension	0.62 (0.32)	0.65 (0.31)	0.21 (0.37)	0.22 (0.37)	0.22 (0.37)
Avg. times observed	2.8	2.7			2.7
Number of obs	13,743	17,686	8,727	9,611	18,338
Panel D: Pulse	Father	Mother	Sons	Daughters	All
	(1)	(2)	(3)	(4)	(5)
Age	57.14 (9.39)	53.83 (10.08)	36.35 (5.22)	36.54 (5.43)	36.45 (4.94)
Unschooled	0.12	0.26	0.02	0.04	0.03
Less than JHS	0.63	0.6	0.32	0.35	0.33
Junior High School	0.12	0.09	0.25	0.25	0.25
High School or above	0.13	0.05	0.42	0.37	0.39
Pulse	75.12 (8.62)	78.67 (8.89)	74.63 (11.10)	79.13 (9.89)	76.94 (10.77)
Normal Pulse	0.99	0.98	0.98	0.97	0.98
Avg. times observed	2.4	2.4			2.3
Number of obs	11,694	14,954	7,337	8,205	15,542
Panel E: BMI	Father	Mother	Sons	Daughters	All
	(1)	(2)	(3)	(4)	(5)
Age	55.18 (9.51)	52.33 (10.22)	35.62 (5.58)	35.84 (5.63)	35.73 (5.46)
Unschooled	0.11	0.25	0.02	0.04	0.03
Less than JHS	0.63	0.6	0.31	0.33	0.32
Junior High School	0.13	0.1	0.26	0.26	0.26
High School or above	0.13	0.05	0.42	0.37	0.40
BMI	21.3 (3.15)	22.66 (4.16)	22.50 (3.82)	24.55 (4.66)	23.55 (4.21)
Overweight	0.13	0.27	0.23	0.41	0.32
Underweight	0.17	0.16	0.11	0.06	0.08
Avg. times observed	3.3	3.2			3.1
Number of obs	15,692	19,309	9,391	10,406	19,797

Next, we look at our health outcomes. We see that children report slightly better QALYs with an average of 77 when compared to their parents who average between 74 and 75. This is likely due in part to the age at which health is reported. Next, we find notable differences in *Hb* and anemia across genders with males having higher levels of *Hb* and vastly lower rates of anemia in both generations. This reflects the well-known greater susceptibility of women to anemia. We also find that children have a higher average BMI of 23.6 than their parents with fathers averaging 21.3 and mothers averaging 22.7. Daughters have the highest rate of being overweight with a rate of 41% while fathers have the lowest rate at 13%. Similarly, underweight rates are relatively higher among sons who have a rate of 11%, while they are lower among daughters who have a rate of 6%. Thus, it appears that daughters are facing emerging health challenges related to both overweight and anemia. In Figure A6 we show the lifecycle patterns of each of our health variables from the ages of 30 to 85 using a sample of adults from both generations. As expected, most variables depict a marked decline in health as individuals age.

3 Methods

We begin by outlining the econometric framework used to estimate intergenerational persistence in health, highlighting how we link parental and child health measures in a consistent manner. We then describe our approach to measuring latent health, using Principal Components Analysis (PCA) to extract the underlying common component that summarizes the broad set of observed health indicators.

3.1 Quantifying Intergenerational Health Mobility

We estimate intergenerational persistence using a standard regression of child health on parent health:

$$y_{1i} = \alpha + \beta y_{0i} + \gamma X_i + \epsilon_i \quad (1)$$

where y_{1i} is the health of a child in family i and y_{0i} is the health of the parent. As discussed in the previous section, all of our health outcomes are time averages for a given individual. Therefore, these are cross-sectional regressions. We also note that because we have averaged a number of discrete variables (e.g. QALYs or an indicator for anemia) across a number of survey years, these time averages effectively become continuous variables. Next, the vector X_i is a parsimonious set of controls that includes quadratic age term for both parents and the child and gender indicators when needed. The parameter β is the IHA. It measures health *persistence* whereas $1 - \beta$ measures *mobility*. The dependent variables are either long time-averages of health outcomes or principal components from a PCA of a battery of health outcomes. We describe the PCA in the next section.

To estimate the rank-rank correlation, we replace the health variables in equation (1) with the ranks of these outcomes calculated in the *full* population. The slope estimate from this regression is the rank-rank slope. This provides a measure of positional mobility rather than mobility in health *levels* and was popularized by Chetty et al. (2014). It is especially useful for comparing population subgroups relative to a common

distribution (Deutscher and Mazumder, 2023).⁸

In addition, the rank-based framework allows us to characterize absolute upward and downward mobility by using the expected ranks of children at particular points in the health distribution. Following the literature (e.g. [Halliday et al. \(2021\)](#)), we evaluate the expected rank of the child conditional on their parents being at either the 25th or 75th percentile of the full distribution. We call the former measure p_{25} and the latter measure p_{75} . These parameters measure upward and downward mobility, respectively. They are especially powerful when applied to disparate demographic groups such as black and white individuals in the United States when compared to a common reference distribution.

3.2 Measuring latent health PCA analysis

We use PCA to combine our health proxies into a measure of latent health. PCA is used to reduce the dimensionality of the data while preserving important underlying patterns that are shared across variables. We apply PCA to two different sets of variables. The first is set of continuous variables including QALY, BMI, diastolic and systolic blood pressure, hemoglobin, and the pulse rate. The second is a set of indicator variables based on the continuous variables and include: poor health, anemia, hypertension, abnormal pulse, underweight, and overweight. These measures are based on established diagnostic thresholds in the medical literature. Per convention, we standardize all variables prior to application of the PCA.

To determine the number of principal components to retain, we employed the Kaiser criterion as well as visual inspection of the scree plot. The Kaiser criterion stipulates that we should retain all components with eigenvalues greater than one while the scree plot method looks for a clear “elbow” in the decline of eigenvalues. Typically both methods deliver similar results.

We display the scree plots for the first set of variables in panel (a),(c) and (e) of Figure 1 and for the second set of discrete outcomes in panel (b),(d) and (f) . The Kaiser criterion favors retaining three components from the first set of proxies but only two from the second set of proxies although the third component in Figure 1 is close to unity in all three panels. Since only the first two principal components have eigenvalues significantly above one, we focus primarily on these principal components to represent the key dimensions of latent health. However, because it appears as if the first three components of health might contain some meaningful information, we also employ the sum of the first two and the first three of these components as alternative measures of latent health. This contrasts with [Andersen \(2021\)](#) and [Chang et al. \(2024\)](#) who only identified *one* principal component based on the variables in their analyses.

After identifying the principal components that account for the bulk of the variance in health outcomes, we also examined the factor loads of each variable within each component. These factor loadings are shown in Appendix Tables [A1](#) and [A2](#) for the first set of primarily continuous health outcomes and for the second set of exclusively discrete outcomes. We find that the factor loadings differ somewhat between the groups but that some patterns are evident. For example, in Table [A1](#), QALY tends to show up strongest in the third component, whereas systolic blood pressure tends to show up in the first component. In contrast, hemoglobin

⁸The common distribution in which we rank health is a gender/cohort.

and BMI are most prevalent in the second component.

4 Results

We now present our findings on intergenerational health persistence for each of the individual health components including QALYs, biomarkers, and anthropometric measures in Section 4.1. This section highlights how persistence varies across different dimensions of health and helps identify which components drive overall transmission. We then turn to the results from our PCA analysis in Section 4.2, where we examine persistence in the latent health index and assess how well this composite measure captures intergenerational patterns relative to the individual components.

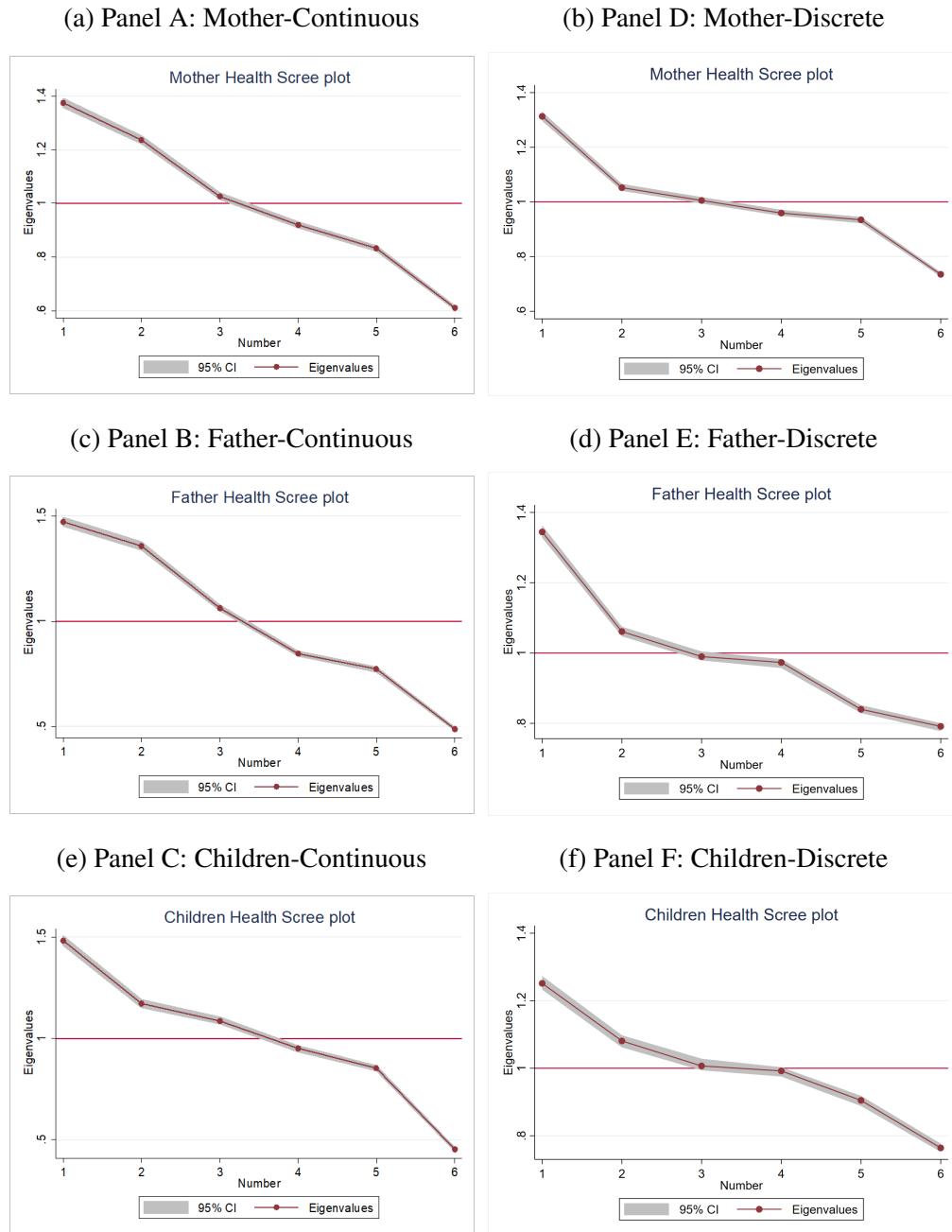
4.1 QALY, Biomarkers and Anthropomorphic measures

We begin our analysis by estimating intergenerational persistence in the QALY which was previously described in Section 2.1. These estimates can easily be compared to existing estimates from the US, the UK, and Australia that use similar QALY-based health measures (Halliday et al., 2021; Bencsik et al., 2023; Vera-Toscano and Brown, 2022). In Figure 2, we display binscatter plots along with regression coefficients relating the child QALY to the parent QALY using both levels in Panel A and ranks in Panel B. In both figures, we pool all children and both parents. For the duration of this paper, we will refer to the case in which we pool all children and both parents as the *baseline sample*.

Our estimate of the IHA is 0.18 in Panel A and our analogous estimate for the rank-rank slope is 0.12 in Panel B. These estimates are generally lower than comparable estimates from the US, the UK, and Australia. For example, in the US, Halliday et al. (2021) estimate an IHA of 0.23 and a rank-rank slope of 0.26. In the UK, Bencsik et al. (2023) estimate an IHA of 0.19 and a rank-rank slope of 0.17. In Australia, Vera-Toscano and Brown (2022) estimate an IHA of 0.20. At first glance, this might suggest that Indonesia has greater health mobility than these other richer anglophone countries. However, we will show that we obtain *greater* estimates once we take a broader approach to measuring latent health in Indonesia by incorporating both biomarkers and anthropometric measures. One interpretation of this is that the SRHS measures upon which the QALY is built do not do an adequate job capturing the multifaceted nature of latent health in the Indonesian context.⁹

⁹We do emphasize that both Halliday et al. (2021) and Bencsik et al. (2023) employ a variety of more objective health measures in alternative health indices and they obtain IHA estimates that are no larger than the QALY-based estimates in the US and UK, respectively.

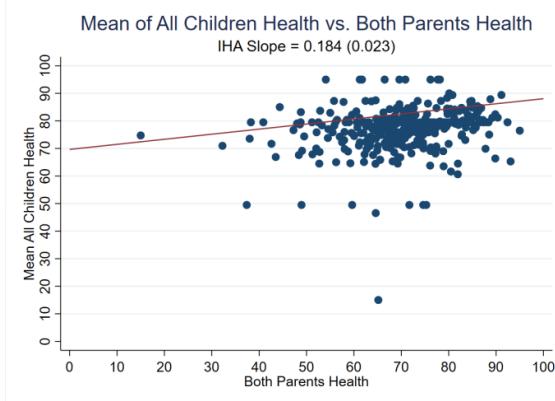
Figure 1: PCA: Continuous Outcomes vs. Discrete Outcomes



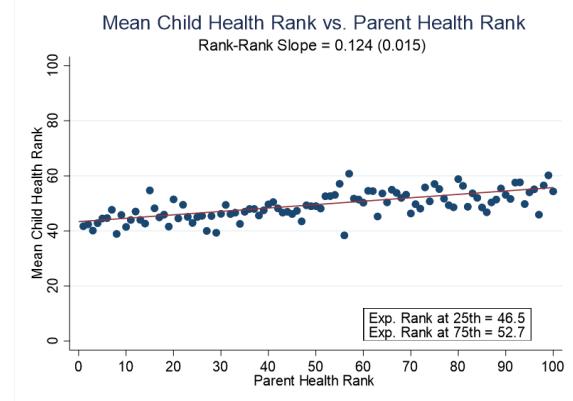
Notes: Panel (a), (c) and (e) display scree plots for fathers, mothers and children using continuous health measures. And Panel (b), (d) and (f) display scree plots for fathers, mothers and children using discrete health measures.

Figure 2: Intergenerational Transmission of Quality Adjusted Life Years (QALY)

(a) Panel A: Intergenerational Health Association



(b) Panel B: Rank-rank slope



Notes: We converted SRHS to the QALY value ranges for each health status category are as follows: 1 very healthy is [90,100]; 2 somewhat healthy is [70,90); 3 somewhat unhealthy [30,70); 4 unhealthy [1,30); We assign the midpoint of the interval for each reported health category in each year and then average these values over all available years for each individual. Panel A shows the shows the regression of the child QALY on parent QALY in levels and Panel B shows the regression in ranks. Sons and daughters are pooled and parent health is the average of both parents.

We also report estimates of health persistence in the QALY across each parent–child gender pair (e.g. mother-son, father-daughter). Panel A of Table 3 shows this for the IHA while analogous estimates of the rank–rank slopes are reported in Panel A of Table 4 and displayed visually in Figure A1. A notable finding is that the IHA in the QALY is strongest from mother to daughter at 0.17, compared with 0.11 from mother to son. Transmission from fathers is somewhat weaker, with estimates of 0.14 for father–son pairs and 0.13 for father–daughter pairs. Similarly, in Table 4, the rank–rank slopes reinforce the pattern of stronger transmission within same-gender pairs: 0.10 from mother to daughter and 0.11 from father to son, compared with 0.09 from mother to son and 0.07 from father to daughter. Finally, we find that the IHA for the QALY is highest when combining both parents. This is a common finding in the literature (Bencsik et al., 2023; Halliday et al., 2021; Chang et al., 2024) and is largely due to averaging health across parents which helps mitigate attenuation bias arising from measurement errors.

Table 3: Intergenerational health associations by parent-child samples

	All	Sons	Daughters	All	Sons	Daughters	All	Sons	Daughters
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Panel A: QALY									
Mother's Health Only	0.135 (0.020)	0.109 (0.027)	0.166 (0.029)	0.163 (0.018)	0.101 (0.020)	0.222 (0.028)	0.230 (0.024)	0.200 (0.028)	0.261 (0.026)
Father's Health Only	0.132 (0.023)	0.136 (0.031)	0.132 (0.030)	0.056 (0.020)	0.034 (0.023)	0.080 (0.032)	0.076 (0.029)	0.096 (0.030)	0.065 (0.026)
Both Parents' Health	0.184 (0.023)	0.175 (0.032)	0.199 (0.029)	0.185 (0.020)	0.122 (0.023)	0.243 (0.031)	0.227 (0.031)	0.218 (0.033)	0.236 (0.031)
Y-Mean	77.14	77.86	76.42	0.23	0.13	0.34	13.58	14.74	12.51
Observations	21,390	10,495	10,895	17,137	8,233	8,914	17,137	8,233	8,914
Panel D: Hypertension									
Mother's Health Only	0.185 (0.018)	0.172 (0.027)	0.199 (0.025)	0.216 (0.020)	0.195 (0.028)	0.238 (0.023)	0.344 (0.018)	0.294 (0.023)	0.381 (0.024)
Father's Health Only	0.144 (0.019)	0.117 (0.026)	0.173 (0.027)	0.203 (0.021)	0.223 (0.028)	0.189 (0.028)	0.336 (0.028)	0.310 (0.032)	0.354 (0.039)
Both Parents' Health	0.267 (0.022)	0.242 (0.031)	0.291 (0.029)	0.321 (0.022)	0.325 (0.033)	0.321 (0.027)	0.488 (0.022)	0.441 (0.030)	0.518 (0.029)
Y-Mean	0.22	0.21	0.22	76.94	74.63	79.13	23.55	22.50	24.55
Observations	16,574	7,999	8,575	14,242	6,816	7,426	18,119	8,693	9,426
Panel G: Overweight									
Mother's Health Only	0.272 (0.021)	0.241 (0.028)	0.293 (0.028)	0.133 (0.017)	0.150 (0.027)	0.122 (0.019)			
Father's Health Only	0.197 (0.029)	0.209 (0.039)	0.182 (0.039)	0.109 (0.018)	0.146 (0.030)	0.070 (0.018)			
Both Parents' Health	0.384 (0.026)	0.360 (0.037)	0.393 (0.035)	0.173 (0.019)	0.226 (0.031)	0.132 (0.020)			
Y-Mean	0.33	0.23	0.42	0.09	0.11	0.07			
Observations	18,119	8,693	9,426	18,119	8,693	9,426			
Panel H: Underweight									

Notes: Each cell shows the estimates and s.e. on the parent health measure from a separate regression. All the regressions are weighted using the sample weights for the children. The dependent variable of all the regressions is the child's time-averaged health measure.

Table 4: Health rank mobility by parent-child samples

	RR Slope	Obs.	RR Slope	Obs.	RR Slope	Obs.	RR Slope	Obs.
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Panel A: QALY		Panel B: Anemia		Panel C: Hemoglobin		Panel D: Hypertension	
Mother-Son	0.091 (0.021)	9,970	0.104 (0.021)	7,739	0.184 (0.022)	7,739	0.095 (0.024)	7,523
Mother-Daughter	0.100 (0.020)	10,171	0.16 (0.021)	8,221	0.22 (0.023)	8,221	0.132 (0.023)	7,902
Father-Son	0.114 (0.024)	8,105	0.028 (0.022)	6,198	0.131 (0.025)	6,198	0.058 (0.025)	5,966
Father-Daughter	0.073 (0.023)	8,095	0.052 (0.023)	6,415	0.083 (0.026)	6,415	0.111 (0.025)	6,106
Both Parents-All Children	0.124 (0.015)	20,405	0.131 (0.015)	16,804	0.203 (0.016)	16,804	0.154 (0.016)	16,234
Panel E: Pulse		Panel F: BMI		Panel G: Overweight		Panel H: Underweight		
Mother-Son	0.175 (0.024)	6,402	0.308 (0.024)	8,275	0.233 (0.032)	8,275	0.004 (0.021)	8,275
Mother-Daughter	0.221 (0.022)	6,855	0.321 (0.021)	8,821	0.263 (0.028)	8,821	0.100 (0.019)	8,821
Father-Son	0.195 (0.025)	5,118	0.284 (0.025)	6,760	0.347 (0.035)	6,760	0.039 (0.023)	6,760
Father-Daughter	0.199 (0.026)	5,373	0.239 (0.025)	6,960	0.097 (0.034)	6,960	0.087 (0.019)	6,960
Both Parents-All Children	0.245 (0.016)	13,928	0.343 (0.015)	18,119	0.240 (0.019)	18,119	0.079 (0.012)	18,119

Notes: Each row of Table 3 reports the rank-rank slope and number of observations for each parent-child sample for all the 9 health outcome variables. All the regressions are weighted using the child's individual sample weights. Standard errors are clustered by family.

Using the rank-rank framework, we can also calculate estimates of absolute mobility based on conditional expected ranks, such as p_{25} and p_{75} which deliver the expected health ranks of children whose parents are at either the 25th or 75th percentile. These estimates are shown in Appendix Figure A1. We see that a child whose parent is at the 25th percentile can expect to rise to the 46.5th percentile whereas a child whose parent is at the 75th percentile can expect to fall to the 52.7th percentile. This indicates a large degree of health mobility when the QALY is the health measurement. However, as we show below, health appears substantially more persistent in Indonesia once we consider additional health measures, particularly biomarkers and anthropometric indicators, suggesting that these dimensions may better capture underlying latent health in the Indonesian context.

We now turn to specific biomarkers including: anemia, hemoglobin, hypertension, and the pulse rate. The estimates of the IHA are shown in panels B through E of Table 3. For anemia, we estimate an IHA from both parents to all children of 0.18. However, as is well established in the medical literature, anemia is more prevalent among women. Consistent with this, we find that the association between mothers and daughters is

0.22, more than twice the corresponding estimate of 0.10 for mothers and sons. Next, looking at our baseline sample for the remaining biomarkers, we find that the IHA is 0.24 for hemoglobin, 0.27 for hypertension and 0.32 for the pulse rate. These coefficients are substantially higher than what we obtained by simply looking at the QALY. This highlights the importance of biomarker data in the Indonesian context. Finally, in columns B through E of Table 4 we show the rank-rank associations for the biomarkers. These tend to be a bit smaller than the analogous IHA estimates.

We now turn to the anthropometric outcomes in panels F through H. We estimate that the IHA for our baseline sample is 0.49 for BMI, 0.38 for overweight, and 0.17 for underweight. The large estimate for the BMI is very consistent with prior work in the US (Classen, 2010; Classen and Thompson, 2016). As with the biomarkers, we tend to estimate higher intergenerational persistence in adiposity-based outcomes.

Across all outcomes in Table 3, we continue to observe a notable gender difference. The IHA between parents and daughters exceeds the corresponding estimates for parents and sons in six of the eight outcomes, with pulse rate and underweight status as the exceptions. This is also depicted graphically in Panel A of Figure A5. Overall, daughters' health appears more closely linked to their parents' health than sons' health.

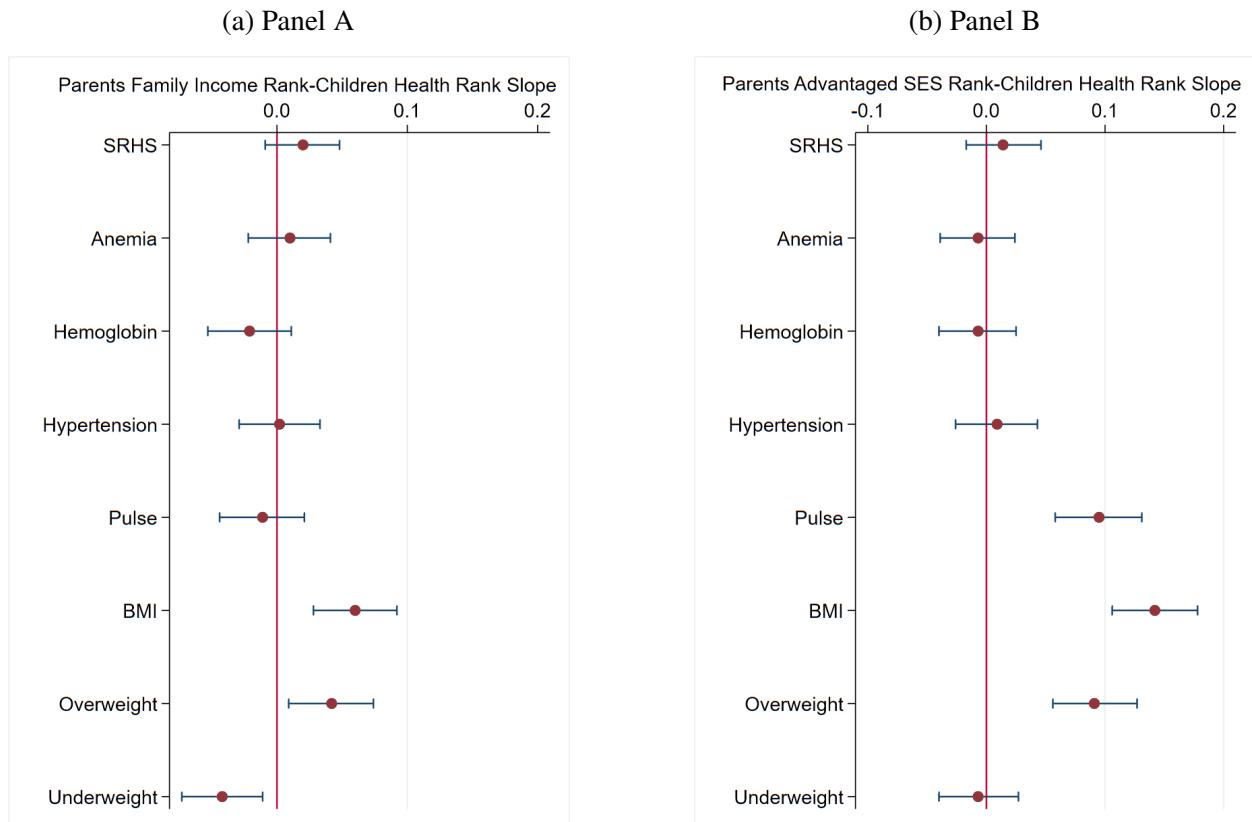
Similarly, a child's health appears to be more strongly influenced by the mother than by the father. This pattern is evident in Table 3; for all eight outcomes, estimated health transmission from mothers to children exceeds that from fathers to children. This is also seen graphically in Panel B of Figure A5. Much of this difference is driven by substantially stronger transmission from mothers to daughters than from mothers to sons, especially for anemia and hemoglobin. Table 4 reinforces this pattern, as the rank-rank slopes for mother-daughter pairs exceed those for mother-son pairs.

Finally, in Figure 3, we report the rank-rank slopes from regressions of child health onto either parent income rank in Panel A or an index of parent SES advantage in Panel B. The index of parent SES advantage is constructed from the first principle component of several SES variables including: whether the household has access to clean water, clean fuel, electricity, a toilet, a TV as well an indicator of whether or not they are low-educated. Higher values of the parent SES index indicate more advantaged households. For half of the outcomes including QALYs, anemia, hemoglobin, and hypertension, there is no statistically significant association between parental income or SES and child health. This is an interesting result in light of the seminal work by Case et al. (2002) that documented very strong income/health gradients in the United States. On the other hand, for BMI and overweight, we observe a *reverse* gradient in the sense that higher SES or income is significantly associated with children with worse adiposity outcomes. We also see a reverse gradient for pulse when using our SES advantage index but not when using income. This suggests that, as Indonesia develops economically, the prevalence of obesity-related diseases may rise, at least in the short-term.

4.2 Latent Health

In the previous subsection, we examined intergenerational health persistence across three domains: a QALY derived from self-reported health status, biomarkers, and anthropometric measures. In this subsection, we combine information across these disparate outcomes to construct comprehensive measures of latent health using principal components analysis (PCA), an approach previously used by Andersen (2021) and Chang

Figure 3: Parents Income/Advantaged SES Rank vs Children's health Rank



Notes: This set of figures estimates the rank-rank slopes of parents' family income or an index of parent SES advantage on their children's health. The index of parent SES advantage is constructed from the first principle component of several SES variables, including whether the household has access to clean water, clean fuel, electricity, a toilet, a TV as well an indicator of whether or not they are low-educated. Higher values of the parent SES index indicate more advantaged households.

et al. (2024). As discussed in Section 2.1, the Kaiser criterion supported retaining up to three components, though the eigenvalue for the third component was close to one, making its inclusion somewhat marginal. Moreover, Figure 1 provided stronger support for retaining the third component for the continuous health outcomes than for the discrete indicator outcomes. Accordingly, we use four primary dependent variables: the first, second, and third components, as well as the sum of all three. For the binary indicator outcomes (e.g., anemia, obesity), we also consider the sum of the first two components, since the corresponding scree plots displayed a clear “elbow” at the second component (again, see Figure 1).

We report estimates of the IHA using these latent health measures in Tables 5 and 6, which correspond to the continuous and discrete outcomes, respectively. We report binscatters for these estimations in Figures A2 and A3. Table 5 includes four panels—one for each of the four dependent variables—while Table 6 contains five panels to accommodate the additional measure based on the sum of the first two components. Each panel in both tables reports nine sets of estimates for all parent–child gender pairings, exactly as in Table 3. As before, we refer to the sample that pairs both parents with all children as the baseline sample.

The principal component–based measures of latent health reveal even stronger intergenerational persistence than the individual health outcomes. In Table 5, using the first principal component, we estimate an IHA of 0.266 in the baseline sample. The corresponding estimates for the second and third components are 0.270 and –0.085, respectively. To construct the most comprehensive measure of latent health, we sum the three components, yielding an estimated IHA of 0.303 which is larger than any of the estimates based on specific health measures in the baseline sample.¹⁰ As in Table 3, transmission from mothers to children exceeds transmission from fathers for the first and second components (Panels A and B) as well as for the sum of components (Panel D). Once again, this pattern suggests that mothers may play a larger role than fathers in transmitting health to their children.

Next, in Table 6, we present IHA estimates based on PCA applied to the discrete health indicators. Although we prefer the PCA constructed from the continuous variables, we found that indicator variables that reflect whether specific biomarkers or anthropometric measures cross established diagnostic thresholds, also provide some valuable information for interpreting PCA components, as we discuss below.

The results in Table 6, which use the discrete proxies, are broadly in line with those in Table 5 which employs the continuous proxies. For example, when using only the first component, we estimate that the IHA is 0.287 in Table 6 in the baseline sample, whereas the corresponding estimate in Table 5 was 0.266. The IHA estimates in this table using the second and third components are small.

A useful benefit of the indicator based PCA is that it potentially provides an interpretation of what each principal component represents. This can be seen in Table A2 which shows the factor loading for each component (variables have had their signs converted so that higher values correspond to worse health). If we look, for example, at the PC1 for associations between mothers and all children (column 1), it is dominated by being overweight (factor loading of 0.65), being underweight (0.63), and having anemia (0.39). In contrast, PC2 appears to reflect cardiovascular conditions: abnormal pulse (0.67), hypertension (0.55). PC3 is dominated by reporting being in poor health (0.87). An important caveat is that the interpretation of

¹⁰Table A1 shows the factor loadings for each variable for each component.

these components can differ a bit depending on the parent or child gender combination. For example, PC 3 for fathers is dominated by abnormal pulse (0.93). This may not be especially surprising given that the scree plot in Figure 1 supports retaining at most two components for the discrete measures, indicating that the case for including a third component is quite weak in this setting. Overall, Tables 5 and 6 indicate that the IHA in latent health lies in the range of roughly 0.28 to 0.30.

Table 5: Intergenerational Health Associations of PC1–PC3 and Sum (Continuous Health Measures)

	All Children (1)	Sons (2)	Daughters (3)	All Children (4)	Sons (5)	Daughters (6)
Panel A: PC1				Panel B: PC2		
Mother's Health Only	0.265 (0.017)	0.234 (0.023)	0.290 (0.023)	0.201 (0.020)	0.221 (0.028)	0.192 (0.022)
Father's Health Only	0.083 (0.016)	0.093 (0.019)	0.079 (0.019)	0.167 (0.027)	0.208 (0.032)	0.114 (0.031)
Both Parents' Health	0.266 (0.020)	0.269 (0.024)	0.266 (0.024)	0.270 (0.023)	0.317 (0.031)	0.233 (0.027)
Y-Mean	0.592	0.682	0.505	0.120	-0.345	0.568
Observations	11,373	5,478	5,895	11,373	5,478	5,895
	(7)	(8)	(9)	(10)	(11)	(12)
Panel C: PC3				Panel D: Sum		
Mother's Health Only	-0.079 (0.021)	-0.076 (0.029)	-0.096 (0.026)	0.228 (0.020)	0.221 (0.029)	0.234 (0.027)
Father's Health Only	-0.041 (0.026)	-0.034 (0.032)	-0.069 (0.034)	0.161 (0.027)	0.203 (0.033)	0.119 (0.032)
Both Parents' Health	-0.085 (0.026)	-0.075 (0.035)	-0.121 (0.031)	0.303 (0.025)	0.339 (0.034)	0.271 (0.031)
Y-Mean	0.098	0.449	-0.240	0.810	0.786	0.834
Observations	11,373	5,478	5,895	11,373	5,478	5,895

Notes: Each row reports the rank-rank slope and number of observations for each parent-child sample for PC1 (first principal component), PC2 (second principal component), PC3(third principal component) and the sum of all the above three components. All the regressions are weighted using the child's individual sample weights. Standard errors are clustered by family.

In Tables 7 and 8, we conduct similar exercises to those in Tables 5 and 6 except that now we estimate rank-rank coefficients rather than the IHA. As before, in Table 7, we apply the PCA to the continuous proxies and in Table 8, we apply the PCA to the discrete proxies. Once again, we report binscatters for these estimations in Figures A2 and A3 in the right panel. Before we discuss the results, one technical point is that, when summing the components, we first summed the raw components and then we ranked them to ensure the variables had uniform distributions.

Rank–rank correlations in latent health are somewhat smaller than the corresponding IHA estimates. In Table 7, the rank–rank slope in the baseline sample is 0.25 when using the first component and 0.228 when

Table 6: Intergenerational Health Associations for PC1–PC3 and Sum (Discrete Health Measures)

	All Children (1)	Sons (2)	Daughters (3)	All Children (4)	Sons (5)	Daughters (6)	All Children (7)	Sons (8)	Daughters (9)
	PC1			PC2			PC3		
Mother's Health Only	0.219 (0.017)	0.212 (0.024)	0.232 (0.022)	-0.078 (0.021)	-0.091 (0.031)	-0.064 (0.028)	0.080 (0.023)	0.073 (0.036)	0.078 (0.028)
Father's Health Only	0.173 (0.021)	0.213 (0.026)	0.128 (0.028)	0.021 (0.025)	0.028 (0.029)	0.020 (0.035)	0.009 (0.022)	0.027 (0.034)	-0.002 (0.028)
Both Parents' Health	0.287 (0.020)	0.319 (0.028)	0.265 (0.025)	-0.054 (0.027)	-0.071 (0.038)	-0.034 (0.035)	0.077 (0.024)	0.091 (0.038)	0.061 (0.030)
Y-Mean Observations	-0.449 11,373	-0.246 5,478	-0.645 5,895	0.192 11,373	0.032 5,478	0.347 5,895	0.070 11,373	0.210 5,478	-0.065 5,895
	(10)	(11)	(12)	(13)	(14)	(15)			
	Sum of PC1+PC2			Sum of PC1+PC2+PC3					
Mother's Health Only	0.151 (0.019)	0.128 (0.027)	0.173 (0.026)	0.151 (0.019)	0.141 (0.029)	0.163 (0.024)			
Father's Health Only	0.137 (0.021)	0.155 (0.026)	0.118 (0.030)	0.095 (0.023)	0.118 (0.031)	0.071 (0.031)			
Both Parents' Health	0.213 (0.022)	0.213 (0.030)	0.216 (0.029)	0.190 (0.022)	0.203 (0.034)	0.183 (0.028)			
Y-Mean Observations	-0.257 11,373	-0.213 5,478	-0.298 5,895	-0.187 11,373	-0.003 5,478	-0.363 5,895			

Notes: Each row reports the rank–rank slope and number of observations for each parent–child sample for PC1 (first principal component), PC2 (second principal component), PC3 (third principal component) and the sum of all the above three components. All the regressions are weighted using the child's individual sample weights. Standard errors are clustered by family.

using the sum of the first three components. In Table 8, relying on the discrete proxies, the rank–rank estimate is 0.236 in the baseline sample when using only the first component. We do not observe large rank–rank estimates for the second or third components, which is consistent with the scree plot indicating that these components contribute relatively little information when based on the discrete measures.

5 Heterogeneity analysis

In this section, we explore heterogeneity in intergenerational health persistence along several key socioeconomic and demographic dimensions. Understanding whether the strength of the parent–child correlation varies systematically across groups is critical because it sheds light on the mechanisms that generate health inequality and persistence. Consequently, differences by income, education, ethnicity, and geographic context may reveal the role of resources and other structural conditions in shaping health mobility.

In Figure 4, we present the rank–rank relationship between parent and child PC1 separately by four quantiles of parental socioeconomic status. Panel A stratifies families by the distribution of parent income while Panel B stratifies by using the socioeconomic advantage index, which was constructed using PCA, as an alternative proxy for SES. Across both measures, the estimated slopes are remarkably similar. This suggests that intergenerational health persistence operates with roughly the same strength across the socioeconomic

Table 7: Health rank mobility of PC1–PC3 and Sum (Continuous Health Measures)

	RR Slope (1)	Exp.25th (2)	Exp.75th (3)	Obs. (4)	RR Slope (5)	Exp.25th (6)	Exp.75th (7)	Obs. (8)
	PC1				PC2			
Mother-Son	0.209 (0.026)	45.529 (0.952)	55.956 (0.988)	5,169	0.198 (0.026)	45.194 (1.030)	55.102 (0.959)	5,169
Mother-Daughter	0.233 (0.023)	45.563 (0.878)	57.210 (0.896)	5,477	0.196 (0.023)	45.388 (0.921)	55.202 (0.854)	5,477
Father-Son	0.142 (0.029)	48.803 (1.057)	55.917 (1.091)	4,145	0.205 (0.027)	44.411 (1.114)	54.643 (1.018)	4,145
Father-Daughter	0.105 (0.026)	50.434 (0.978)	55.664 (1.019)	4,282	0.112 (0.028)	47.846 (1.132)	53.455 (0.998)	4,282
Both Parents-All Children	0.25 (0.017)	45.954 (0.583)	58.466 (0.732)	11,373	0.225 (0.017)	45.517 (0.653)	56.751 (0.669)	11,373
	PC3				Sum			
Mother-Son	-0.066 (0.025)	52.138 (0.960)	48.842 (0.928)	5,169	0.187 (0.026)	45.607 (0.979)	54.968 (0.981)	5,169
Mother-Daughter	-0.08 (0.023)	52.724 (0.930)	48.721 (0.896)	5,477	0.194 (0.024)	45.981 (0.924)	55.657 (0.895)	5,477
Father-Son	0.003 (0.029)	50.307 (1.088)	50.457 (1.046)	4,145	0.218 (0.028)	45.29 (1.071)	56.173 (1.038)	4,145
Father-Daughter	-0.037 (0.029)	51.289 (1.039)	49.44 (1.113)	4,282	0.116 (0.028)	48.667 (1.063)	54.475 (1.054)	4,282
Both Parents-All Children	-0.073 (0.018)	52.458 (0.677)	48.824 (0.656)	11,373	0.228 (0.017)	45.733 (0.609)	57.114 (0.712)	11,373

Notes: Each row reports the rank-rank slope and number of observations for each parent-child sample for PC1 (first principal component), PC2 (second principal component), PC3 (third principal component) and the sum of all the above three components. All the regressions are weighted using the child's individual sample weights. Standard errors are clustered by family.

distribution. Although families at the bottom and top of the SES distribution differ in many observable ways, these differences do not appear to translate into sizable variation in the degree of health mobility in our data.

Next, in Tables 9 through 11, we provide rank-based mobility estimates across a range of health outcomes stratified by ethnicity, region, and education. These estimates are plotted in Figure A4. Specifically, we rank each variable in the *full* population. Subsequently, we estimate rank-rank regressions in subpopulations that are stratified by either ethnicity, region, or education. Doing this, allows us to see if certain subgroups exhibit more or less upward or downward mobility than other subgroups. One critical point that we emphasize is that when we compute either p_{25} (for upward mobility) or p_{75} (for downward mobility) in this manner, we are holding the parent fixed at the *same* percentile for the full population across *all* subgroups. This allows us to make a statement about whether or not a given subgroup is more or less mobile in absolute terms than another (Chetty et al., 2014; Halliday et al., 2021).

Tables 9 through 11 are organized as follows. Each table contains eight panels, one for each health outcome. Within each panel, we report the rank-rank slope as well as p_{25} and p_{75} for subsamples defined by region (rural and urban), ethnicity (Javanese, Sundanese, and other), and education (unschooled, less than

Table 8: Health rank mobility of PC1–PC3 and Sum (Discrete Health Measures)

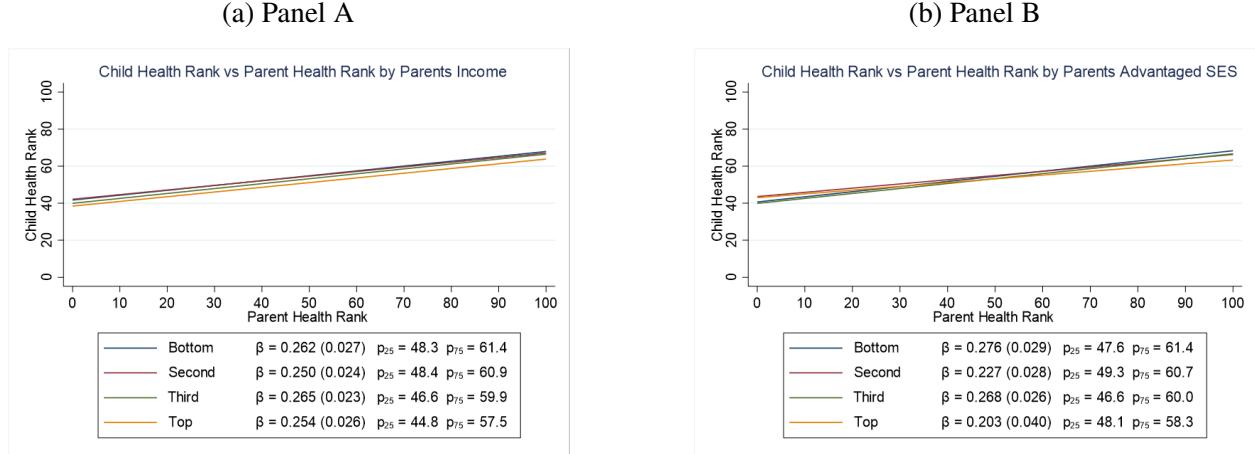
	RR Slope	Exp.25th	Exp.75th	RR Slope	Exp.25th	Exp.75th	RR Slope	Exp.25th	Exp.75th	Obs. (10)
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	
	PC1			PC2			PC3			
Mother-Son	0.195 (0.024)	45.020 (0.971)	54.749 (0.928)	-0.057 (0.026)	51.534 (0.942)	48.668 (0.961)	0.071 (0.024)	48.077 (0.960)	51.623 (0.887)	5,169
Mother-Daughter	0.213 (0.023)	44.452 (0.884)	55.111 (0.908)	-0.059 (0.024)	51.480 (0.916)	48.546 (0.892)	0.082 (0.023)	48.119 (0.888)	52.232 (0.864)	5,477
Father-Son	0.163 (0.027)	45.881 (1.062)	54.055 (1.047)	0.018 (0.026)	49.847 (1.089)	50.737 (0.955)	0.002 (0.027)	50.290 (1.021)	50.371 (1.032)	4,145
Father-Daughter	0.105 (0.027)	46.827 (1.027)	52.054 (1.076)	0.009 (0.027)	50.916 (0.994)	51.365 (1.049)	-0.008 (0.026)	50.572 (1.077)	50.193 (0.924)	4,282
Both Parents-All Children	0.236 (0.016)	42.745 (0.714)	54.535 (0.571)	-0.032 (0.017)	51.123 (0.659)	49.528 (0.645)	0.054 (0.016)	49.004 (0.619)	51.727 (0.630)	11,373
	PC1+PC2			PC1+PC2+PC3						
	(11)	(12)	(13)	(14)	(15)	(16)				
Mother-Son	0.114 (0.024)	46.802 (0.929)	52.479 (0.948)	0.118 (0.023)	46.856 (0.884)	52.739 (0.934)				
Mother-Daughter	0.161 (0.023)	45.609 (0.905)	53.654 (0.866)	0.132 (0.022)	46.507 (0.886)	53.103 (0.845)				
Father-Son	0.126 (0.026)	46.702 (1.003)	52.988 (1.041)	0.069 (0.026)	48.272 (1.026)	51.746 (0.979)				
Father-Daughter	0.117 (0.027)	47.198 (1.001)	53.046 (1.050)	0.072 (0.026)	48.324 (0.994)	51.918 (1.033)				
Both Parents-All Children	0.168 (0.016)	44.974 (0.677)	53.366 (0.590)	0.137 (0.016)	46.223 (0.641)	53.093 (0.584)				

Notes: Each row reports the rank–rank slope and number of observations for each parent-child sample for PC1 (first principal component), PC2 (second principal component), PC3 (third principal component) and the sum of all the above three components. All the regressions are weighted using the child’s individual sample weights. Standard errors are clustered by family.

junior high school, junior high school, and high school or college). For each grouping, we formally test whether the mobility measures differ across categories and report the associated p-values.

We begin with Table 9 where we investigate heterogeneity in three outcomes: QALYs, hemoglobin, and anemia. We see that the rank–rank slopes are essentially identical across subgroups defined by region, ethnicity, and education. However, these similarities in the slopes mask important heterogeneity in both upward and downward mobility, particularly, in hemoglobin and anemia. For example, in column (7) of Table 9, we see that urban dwellers whose parents were in the 75th percentile for hemoglobin can expect to be at the 57.14 percentile of the overall hemoglobin distribution. In contrast, the corresponding estimate for rural dwellers is 55.04. This difference is statistically significant at the 5% level and suggests that urban dwellers experience less downward mobility in this domain. Next, we see that the Javanese experience less downward mobility than the Sundanese with p_{75} equal to 56.65 for the former and p_{75} equal to 52.31 for the latter. Once again, these differences are significant at the 5% level. Consistent with this pattern, the Javanese experience more downward mobility in anemia, with $p_{75} = 50.89$, compared with $p_{75} = 55.43$ for the Sundanese. Likewise, we observe greater upward mobility in hemoglobin among the Javanese which is reflected in $p_{25} = 47.03$ versus $p_{25} = 42.38$ for the Sundanese. The Javanese also experience less upward mobility in anemia with $p_{25} = 45.56$ compared with $p_{25} = 48.44$ for the Sundanese. These differences are

Figure 4: Intergenerational Persistence of PC1 by Parental Family Income and Parental Advantaged SES



Notes: In Panel A, we divide parents' family income into 4 quantiles and the each line in Panel A represents the rank-rank slopes for each of the four groups. In Panel B, we first construct the first principal component (PC1) of parents' advantaged social-economic status, including whether have access to clean water, clean fuel, electricity, toilet, TV, poor and lower educated. We further divide the PC1 of parents advantaged social-economic status into 4 quantiles. The lines in Panel B represents the rank-rank slopes for each of the four groups.

statistically significant at the 1% and 5% levels, respectively.

Next, we consider mobility in hypertension and the pulse rate by region, ethnicity, and education. As with QALYs, hemoglobin, and anemia, we do not see any statistically significant differences in the rank slopes. However, we do see that there is more downward mobility in hypertension among the Javanese ($p_{75} = 50.83$) than the Sundanese ($p_{75} = 53.66$). In addition, we also see statistically significant differences in both upwards and downwards mobility in pulse rate across rural and urban dwellers. Specifically, we see more upward mobility among urban dwellers than rural dwellers with p_{25} equal to 46.81 for the former versus 42.59 for the latter. We also see *less* downward mobility in pulse among urban dwellers than rural dwellers. The estimate of p_{75} is 59.00 for the urban sub-sample and is 54.10 for the rural subsample.

In Table 11, we consider heterogeneity in intergenerational mobility in our adiposity outcomes: BMI, overweight, and underweight. As in Tables 9 and 10, we do not see any meaningful differences in the rank slopes, but we do see some interesting differences in upward and downward mobility. First, looking across ethnicity, we see that there is more upward mobility in overweight among the Sundanese than the Javanese. We estimate that p_{25} is 48.97 for the Sundanese but only 44.09 for the Javanese. This suggests that Sundanese children are more likely to be overweight than Javanese children born to parents at the 25th percentile of the Indonesian distribution. Next, we also see statistically significant differences across rural and urban dwellers for upwards mobility in underweight with urban dweller experiencing more mobility than their rural counterparts. We estimate that p_{25} is 46.33 for rural dwellers and 48.38 for urban dwellers.

Finally, we also see important heterogeneity by education in this table. First, we see an interesting gradient in p_{25} by education in overweight. Specifically, we see that upward mobility declines with educational

attainment. For example, we estimate that p_{25} is 45.87 for the unschooled, 43.99 for those with less than junior high school, 42.15 for those with junior high school, and 39.42 for those with high school or college. These patterns suggest that overweight is becoming a serious concern across all education groups, including those with relatively low levels of schooling. We see an analogous pattern in p_{75} for underweight with one interesting difference. For overweight, we estimate that p_{75} is 53.48 for the unschooled, 50.75 for those with less than junior high school, and 51.58 for those with junior high school. This indicates that the unschooled have substantially more downward mobility in underweight than people with at least some schooling through junior high school. Again, this might indicate that malnourishment is becoming less of a problem among those with the least amount of education. However, p_{75} is the *highest* among the most educated children; we estimate that p_{75} is 56.56. Thus, among the well-educated, parents in the top quartile of the underweight distribution tend to have children who also remain toward the upper end of this distribution. Given the relatively advantaged status of these households, this pattern is likely less about resource constraints and more about other factors.

6 Conclusion

An emerging literature across a range of mostly high income countries has highlighted the degree of intergenerational persistence in broad-based measures of latent health. These studies either use survey based measures of self-reported health (Halliday et al., 2021; Bencsik et al., 2023; Vera-Toscano and Brown, 2022) or administrative health records (Andersen, 2021; Chang et al., 2024). We provide a novel contribution to this literature by using biomarkers (e.g. pulse, blood pressure) and by studying a middle income country, Indonesia. The use of biomarkers appears to be quantitatively important. Using only self-reported health, our estimates of the intergenerational health association are 0.18 and broadly in line with estimates from many other countries. However, when we include biomarkers and some other health measures, our estimates rise by two-thirds to 0.3. This is the largest estimate of persistence in a broad-based measure of health that we are aware of, and suggest Indonesia has perhaps higher intergenerational persistence and lower intergenerational health mobility than other higher income countries.

Another contribution is that we find a second quantitatively important principal component of health that is intergenerationally transmitted, suggesting that multiple dimensions of health matter. We further show that transmission appears to be stronger from mothers and to daughters. On the other hand, we find very little heterogeneity in intergenerational persistence though there are some differences in absolute health mobility by urbanicity and ethnicity. We also highlight some potential health issues as developing countries transition to middle income status. We find a notable gradient in the pulse rate and obesity in Indonesia by measures of socioeconomic status.

While it is important for future research to further document patterns in intergenerational health persistence across a wider range of countries, it would also be useful for future studies to use high quality research designs to better understand the degree to which this relationship is causal and what factors drive the persistence. This may provide greater information for policy makers to try to prevent the perpetuation of poor

Table 9: Health rank mobility by ethnicity, region and education

	Panel A: QALY			Panel B: Hemoglobin			Panel C: Anemia					
	RR Slope (1)	Exp.25th (2)	Exp.75th (3)	Obs. (4)	RR Slope (5)	Exp.25th (6)	Exp.75th (7)	Obs. (8)	RR Slope (9)	Exp.25th (10)	Exp.75th (11)	Obs. (12)
Region												
Rural	0.125 (0.021)	46.479 (0.796)	52.709 (0.802)	9,545 (0.022)	0.215 (0.022)	46.01 (0.787)	56.777 (0.868)	7,919 (0.021)	0.144 (0.021)	45.489 (0.917)	52.714 (0.727)	7,919 (0.727)
Urban	0.123 (0.017)	46.469 (0.696)	52.612 (0.649)	10,860 (0.020)	0.188 (0.768)	45.675 (0.736)	55.095 (0.736)	8,884 (0.019)	0.112 (0.019)	46.311 (0.749)	51.934 (0.733)	8,884 (0.733)
Test of Equality P-value	0.947	0.992			0.332	0.746	0.12		0.244	0.473	0.426	
Ethnicity												
Java	0.139 (0.023)	47.21 (0.978)	54.149 (0.771)	8,563 (0.026)	0.182 (0.966)	47.034 (0.952)	56.138 (0.952)	6,788 (0.026)	0.086 (0.026)	46.577 (0.993)	50.889 (0.904)	6,788 (0.904)
Sunda	0.094 (0.046)	47.275 (1.581)	51.988 (1.840)	2,121 (0.048)	0.208 (1.719)	42.377 (1.785)	52.79 (1.785)	1,607 (0.043)	0.140 (0.043)	48.439 (1.745)	55.434 (1.554)	1,607 (1.554)
Others	0.1 (0.019)	45.289 (0.756)	50.266 (0.817)	9,721 (0.020)	0.223 (0.786)	45.762 (0.795)	56.934 (0.795)	8,409 (0.019)	0.174 (0.019)	44.241 (0.820)	52.942 (0.717)	8,409 (0.717)
Test of Equality P-value	0.395	0.227			0.003	0.448	0.061	0.104	0.022	0.041	0.028	
Education												
Unschooled	0.1 (0.035)	47.796 (1.475)	52.793 (1.363)	3,172 (0.037)	0.18 (1.367)	45.47 (1.458)	54.464 (1.458)	2,605 (0.036)	0.146 (0.036)	44.855 (1.681)	52.179 (1.297)	2,605 (1.297)
Less than Junior HS	0.129 (0.022)	45.713 (0.850)	52.143 (0.820)	10,216 (0.024)	0.201 (0.856)	46.141 (0.906)	56.211 (0.906)	8,382 (0.023)	0.111 (0.023)	46.569 (0.898)	52.116 (0.805)	8,382 (0.805)
Junior High School	0.13 (0.042)	46.861 (1.550)	53.383 (1.539)	2,394 (0.052)	0.222 (1.917)	45.224 (1.789)	56.318 (1.789)	1,994 (0.047)	0.164 (0.047)	45.939 (1.739)	54.122 (1.631)	1,994 (1.631)
High School or College	0.155 (0.044)	46.382 (1.653)	54.124 (1.587)	2,297 (0.052)	0.201 (2.030)	44.541 (1.880)	54.58 (1.880)	1,811 (0.054)	0.092 (2.003)	47.301 (2.003)	51.879 (1.994)	1,811 (1.994)
Test of Equality P-value	0.803	0.654	0.689		0.924	0.876	0.678		0.614	0.773	0.721	

Notes: Each row reports the rank-rank slope for each subgroup and number of observations for each parent-child sample for SRH, Hemoglobin and Anemia. All the regressions are weighted using the child's individual sample weights. Standard errors are clustered by family.

Table 10: Health rank mobility by ethnicity, region and education

	RR Slope (1)	Exp.25th (2)	Exp.75th (3)	Obs. (4)	RR Slope (5)	Exp.25th (6)	Exp.75th (7)	Obs. (8)
	Panel D: Hypertension				Panel E: Pulse			
Region								
Rural	0.144 (0.021)	45.586 (0.730)	52.804 (0.854)	7,563	0.23 (0.022)	42.59 (0.794)	54.104 (0.873)	6,573
Urban	0.167 (0.021)	45.246 (0.828)	53.601 (0.748)	8,671	0.244 (0.021)	46.813 (0.868)	59.009 (0.712)	7,355
Test of Equality P-value	0.417	0.746	0.461		0.64	0.000	0.000	
Ethnicity								
Java	0.118 (0.026)	44.953 (0.889)	50.834 (1.003)	6,516	0.242 (0.026)	44.581 (1.026)	56.669 (0.938)	5,146
Sunda	0.173 (0.045)	44.991 (1.474)	53.657 (1.908)	1,553	0.307 (0.053)	42.149 (1.937)	57.485 (2.067)	1,220
Others	0.179 (0.021)	46.423 (0.841)	55.397 (0.753)	8,165	0.231 (0.020)	44.375 (0.816)	55.913 (0.760)	7,562
Test of Equality P-value	0.166	0.437	0.001		0.4	0.521	0.683	
Education								
Unschooled	0.187 (0.041)	46.43 (1.406)	55.775 (1.765)	2,593	0.258 (0.040)	43.945 (1.593)	56.824 (1.494)	2,237
Less than Junior HS	0.149 (0.022)	44.817 (0.781)	52.269 (0.881)	8,015	0.248 (0.023)	43.974 (0.849)	56.35 (0.885)	6,925
Junior HS	0.125 (0.045)	47.843 (1.811)	54.086 (1.653)	1,900	0.339 (0.055)	41.983 (2.193)	58.932 (1.770)	1,596
HS degree or College	0.221 (0.057)	41.896 (1.869)	52.941 (2.037)	1,725	0.256 (0.045)	46.109 (1.843)	58.909 (1.713)	1,460
Test of Equality P-value	0.489	0.098	0.319		0.491	0.541	0.402	

Notes: Each row reports the rank-rank slope for each subgroup and number of observations for each parent-child sample for hypertension and pulse. All the regressions are weighted using the child's individual sample weights. Standard errors are clustered by family.

health across generations.

Table 11: Health rank mobility by ethnicity, region and education

	RR Slope (1)	Exp.25th (2)	Exp.75th (3)	Obs. (4)	RR Slope (5)	Exp.25th (6)	Exp.75th (7)	Obs. (8)	RR Slope (9)	Exp.25th (10)	Exp.75th (11)	Obs. (12)
	Panel F: BMI				Panel G: Overweight				Panel H: Underweight			
Region												
Rural	0.354 (0.022)	41.961 (0.684)	59.66 (0.930)	8,456	0.22 (0.029)	44.253 (0.711)	55.247 (1.196)	8,681	0.091 (0.016)	46.325 (0.723)	50.867 (0.763)	8,681
Urban	0.335 (0.021)	41.92 (0.952)	58.678 (0.732)	9,226	0.242 (0.025)	45.528 (0.935)	57.626 (0.855)	9,437	0.071 (0.016)	48.384 (0.565)	51.94 (0.867)	9,437
Test of Equality P-value	0.517	0.972	0.386		0.543	0.261	0.088		0.366	0.019	0.332	
Ethnicity												
Java	0.342 (0.025)	42.176 (0.951)	59.278 (0.936)	7,114	0.24 (0.032)	44.093 (0.958)	56.078 (1.183)	7,257	0.078 (0.020)	47.657 (0.729)	51.581 (1.019)	7,257
Sunda	0.339 (0.047)	43.093 (1.954)	60.057 (1.657)	1,659	0.195 (0.051)	48.97 (1.835)	58.703 (1.924)	1,685	0.119 (0.036)	45.96 (1.383)	51.915 (1.861)	1,685
Others	0.342 (0.020)	41.373 (0.741)	58.459 (0.842)	8,910	0.251 (0.025)	44.004 (0.748)	56.552 (1.026)	9,177	0.068 (0.016)	47.436 (0.688)	50.849 (0.769)	9,177
Test of Equality P-value	0.999	0.626	0.635		0.613	0.036	0.501		0.418	0.549	0.778	
Education												
Unschooled	0.297 (0.039)	41.698 (1.314)	56.532 (1.625)	2,719	0.19 (0.047)	45.867 (1.307)	55.376 (1.880)	2,829	0.081 (0.024)	49.451 (1.180)	53.481 (1.177)	2,829
Less than Junior HS	0.374 (0.022)	41.743 (0.793)	60.45 (0.911)	8,793	0.258 (0.030)	43.994 (0.813)	56.897 (1.197)	8,927	0.075 (0.018)	47.012 (0.695)	50.752 (0.913)	8,927
Junior High School	0.337 (0.045)	42.676 (2.117)	59.526 (1.395)	2,176	0.286 (0.051)	42.152 (2.162)	56.445 (1.597)	2,205	0.091 (0.039)	47.029 (1.409)	51.579 (1.969)	2,205
High school or college	0.398 (0.052)	37.405 (2.529)	57.295 (1.596)	1,928	0.310 (0.058)	39.424 (2.411)	54.93 (1.933)	1,947	0.177 (0.046)	47.723 (1.501)	56.56 (2.387)	1,947
Test of Equality P-value	0.285	0.384	0.106		0.361	0.09	0.803		0.223	0.342	0.068	

Notes: Each row reports the rank-rank slope for each subgroup and number of observations for each parent-child sample for BMI, overweight and underweight. All the regressions are weighted using the child's individual sample weights. Standard errors are clustered by family.

References

Akbulut-Yuksel, M. and A. D. Kugler (2016). Intergenerational persistence of health: do immigrants get healthier as they remain in the us for more generations? *Economics & Human Biology* 23, 136–148.

Andersen, C. (2021). Intergenerational health mobility: Evidence from danish registers. *Health Economics* 30(12), 3186–3202.

Bencsik, P., T. J. Halliday, and B. Mazumder (2023). The intergenerational transmission of mental and physical health in the united kingdom. *Journal of Health Economics* 92, 102805.

Blanchflower, D. G. and A. Bryson (2022). Taking the pulse of nations: A biometric measure of well-being. *Economics & Human Biology* 46, 101141.

Bütikofer, A., R. Ginja, K. Karbownik, and F. Landaud (2024). (breaking) intergenerational transmission of mental health. *Journal of Human Resources* 59(S), S108–S151.

Case, A., D. Lubotsky, and C. Paxson (2002). Economic status and health in childhood: The origins of the gradient. *American Economic Review* 92(5), 1308–1334.

Chang, H., T. J. Halliday, M.-J. Lin, and B. Mazumder (2023). Estimating intergenerational health transmission in taiwan with administrative health records. Technical report, IZA Discussion Papers.

Chang, H., T. J. Halliday, M.-J. Lin, and B. Mazumder (2024). Estimating intergenerational health transmission in taiwan with administrative health records. *Journal of Public Economics* 238, 105194.

Chetty, R., N. Hendren, P. Kline, and E. Saez (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.

Classen, T. J. (2010). Measures of the intergenerational transmission of body mass index between mothers and their children in the united states, 1981–2004. *Economics & Human Biology* 8(1), 30–43.

Classen, T. J. and O. Thompson (2016). Genes and the intergenerational transmission of bmi and obesity. *Economics & Human Biology* 23, 121–133.

Corak, M. (2013). Income inequality, equality of opportunity, and intergenerational mobility. *Journal of Economic Perspectives* 27(3), 79–102.

Deutscher, N. and B. Mazumder (2023). Measuring intergenerational income mobility: A synthesis of approaches. *Journal of Economic Literature* 61(3), 988–1036.

Durlauf, S. N., A. Kourtellos, and C. M. Tan (2022). The great gatsby curve. *Annual Review of Economics* 14, 571–605.

Fletcher, J. and K. M. Jajtner (2021). Intergenerational health mobility: Magnitudes and importance of schools and place. *Health economics* 30(7), 1648–1667.

Graeber, D. (2023). Intergenerational health mobility in germany.

Halliday, T. (2023). Intergenerational health mobility. In *Handbook of labor, human resources and population economics*, pp. 1–22. Springer.

Halliday, T., B. Mazumder, and A. Wong (2021). Intergenerational mobility in self-reported health status in the us. *Journal of Public Economics* 193, 104307.

Halliday, T. J. and B. Mazumder (2017). An analysis of sibling correlations in health using latent variable models. *Health economics* 26(12), e108–e125.

Halliday, T. J., B. Mazumder, and A. Wong (2020). The intergenerational transmission of health in the united states: A latent variables analysis. *Health economics* 29(3), 367–381.

Johnson, R. C. and R. F. Schoeni (2011). The influence of early-life events on human capital, health status, and labor market outcomes over the life course. *The BE journal of economic analysis & policy* 11(3).

Jones, C. I. and P. J. Klenow (2016, September). Beyond gdp? welfare across countries and time. *American Economic Review* 106(9), 2426–57.

Kumar, S., T. Halliday, and B. Mazumder (2025). Cycles of malnutrition: Intergenerational health transmission in india.

Kumar, S. and B. Nahlen (2023). Intergenerational persistence of health: Evidence from india. *Economics Letters* 224, 111023.

Mazumder, B. (2005). Fortunate sons: New estimates of intergenerational mobility in the united states using social security earnings data. *Review of Economics and Statistics* 87(2), 235–255.

Mazumder, B. (2024). What do we know about the intergenerational transmission of health. In J. E. Elina Kilpi-Jakonen, Jo Blanden and L. Macmillan (Eds.), *Research Handbook on Intergenerational Inequality*, Elgar Handbooks on Inequality, pp. 150–163. Cheltenham, UK: Edward Elgar Publishing Limited.

Omran, A. R. (1971). *The Milbank Memorial Fund Quarterly* 49(4), 509–538.

Rachmi, C., M. Li, and L. A. Baur (2017). Overweight and obesity in indonesia: prevalence and risk factors—a literature review. *Public health* 147, 20–29.

Sen, A. (2002). Health: perception versus observation. *British Medical Journal* 324(7342), 860–1.

Song, L. (2022). Examining the relationship between intergenerational upward mobility and inequality: Evidence from panel data. *Social Indicators Research* 163(1), 1–27.

Subramanian SV, Subramanyam MA, S. S. K. I. (2009). Are self-reports of health and morbidities in developing countries misleading? evidence from india. *Soc Sci Med.* 68(2), 260–5.

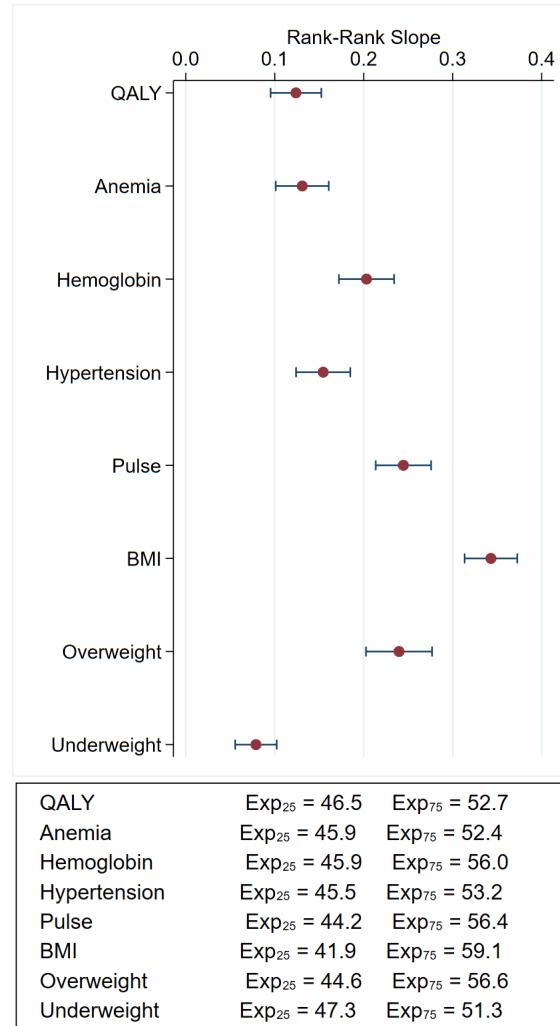
Sungkar, A., S. Bardosono, R. Irwinda, N. Manikam, R. Sekartini, B. Medise, S. Nasar, S. Helmyati, A. Ariani, J. Nurihsan, et al. (2022). A life course approach to the prevention of iron deficiency anemia in indonesia. *nutrients* 2022, 14, 277.

Thomas, D., E. Frankenberg, and J. P. Smith (2001). Lost but not forgotten: Attrition and follow-up in the indonesia family life survey. *Journal of Human resources*, 556–592.

Vera-Toscano, E. and H. Brown (2022). The intergenerational transmission of mental and physical health in australia: Evidence using data from the household income and labor dynamics of australia survey. *Frontiers in Public Health* 9, 763589.

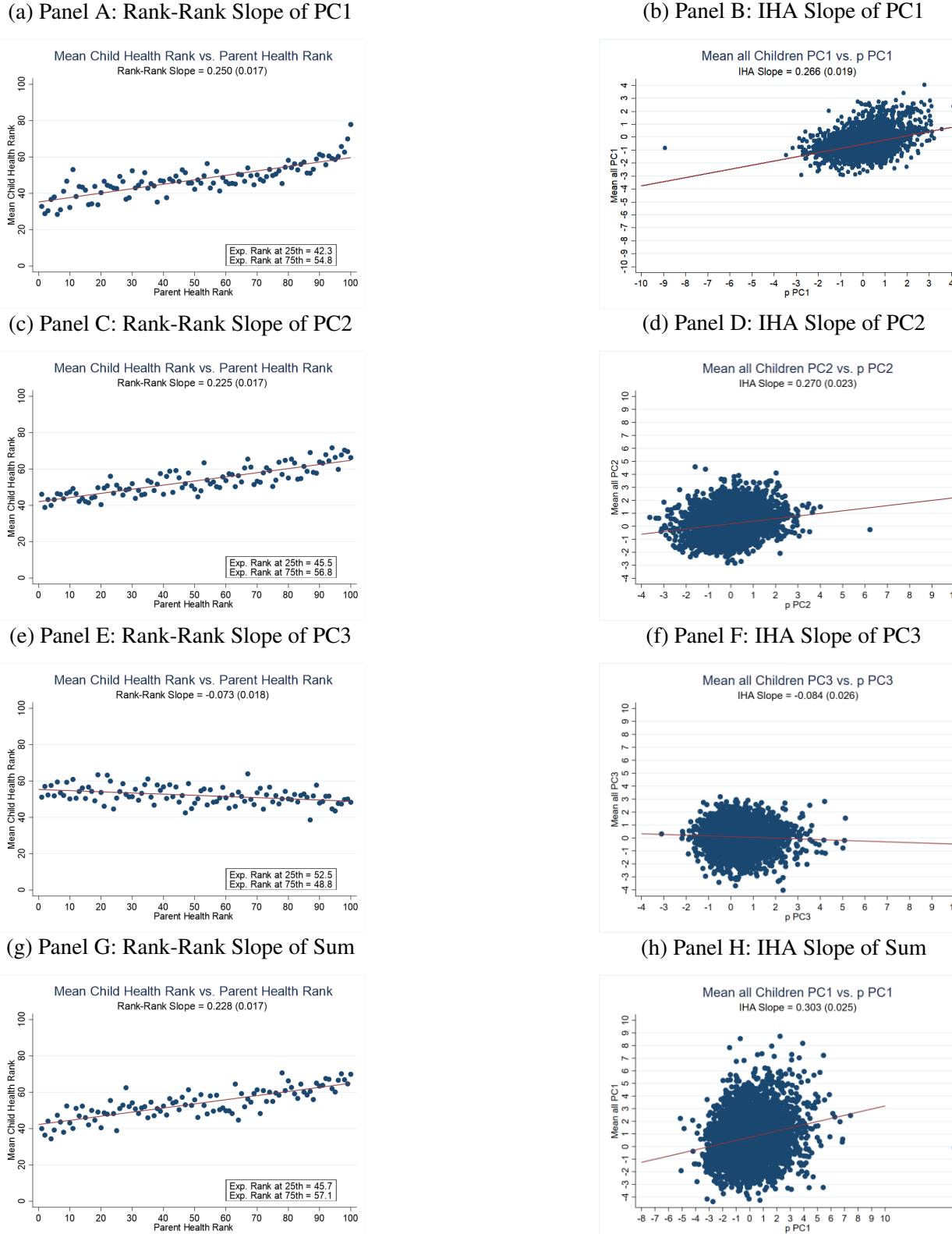
Zhang, J. (2021). A survey on income inequality in china. *Journal of Economic Literature* 59(4), 1191–1239.

Figure A1: Intergenerational Rank-Rank Slopes in Health Measures



Notes: This figure shows the rank-rank slopes for each of the health outcome variables and corresponds to the results in Table 4. The whiskers represent 95 percent confidence intervals of the estimates. The table shows the expected ranks of children whose parents were at the 25th or 75th percentiles.

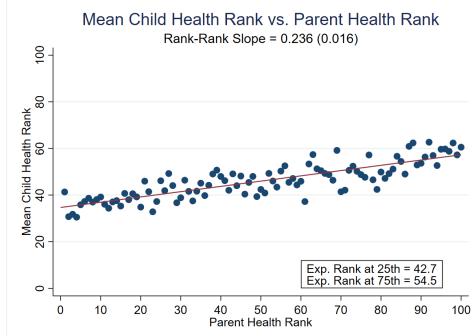
Figure A2: Rank-Rank and IHA slopes of PC1-PC3 and Sum (Continuous Health Measures)



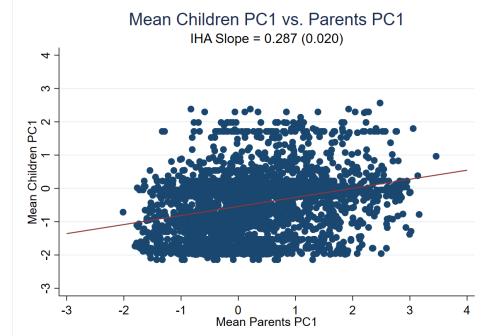
Notes: The figures show intergenerational relationships in the first three principal components of the health measures and correspond to results in Tables 5 and Table 7.

Figure A3: Rank-Rank and IHA slopes of PC1-PC3 and Sum (Discrete Health Measures)

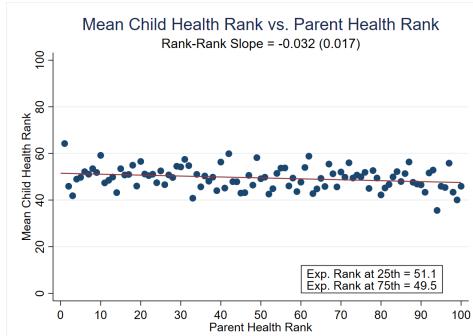
(a) Panel A: Rank-Rank Slope of PC1



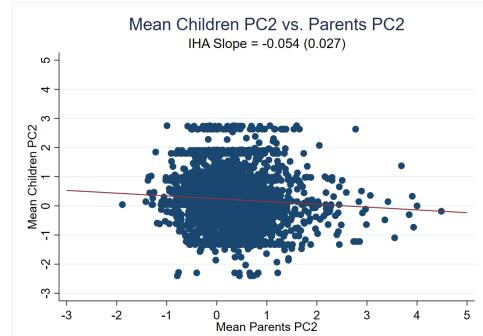
(b) Panel B: IHA Slope of PC1



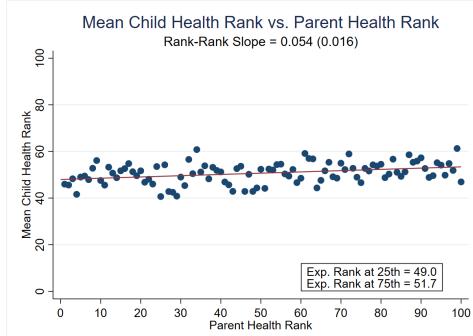
(c) Panel C: Rank-Rank Slope of PC2



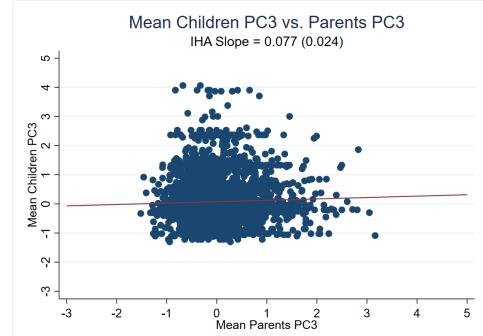
(d) Panel D: IHA Slope of PC2



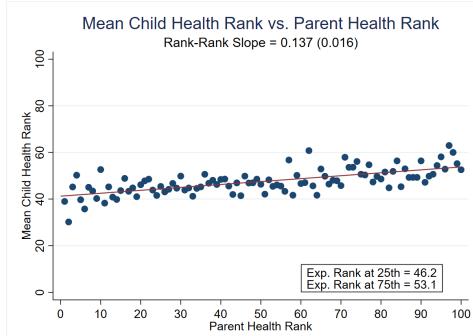
(e) Panel E: Rank-Rank Slope of PC3



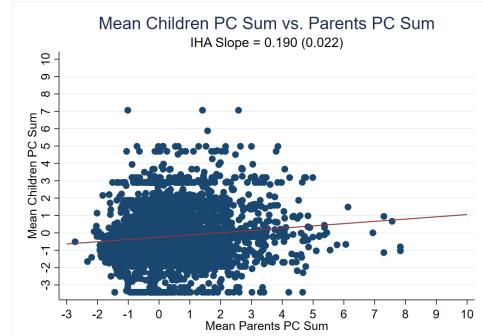
(f) Panel F: IHA Slope of PC3



(g) Panel G: Rank-Rank Slope of Sum

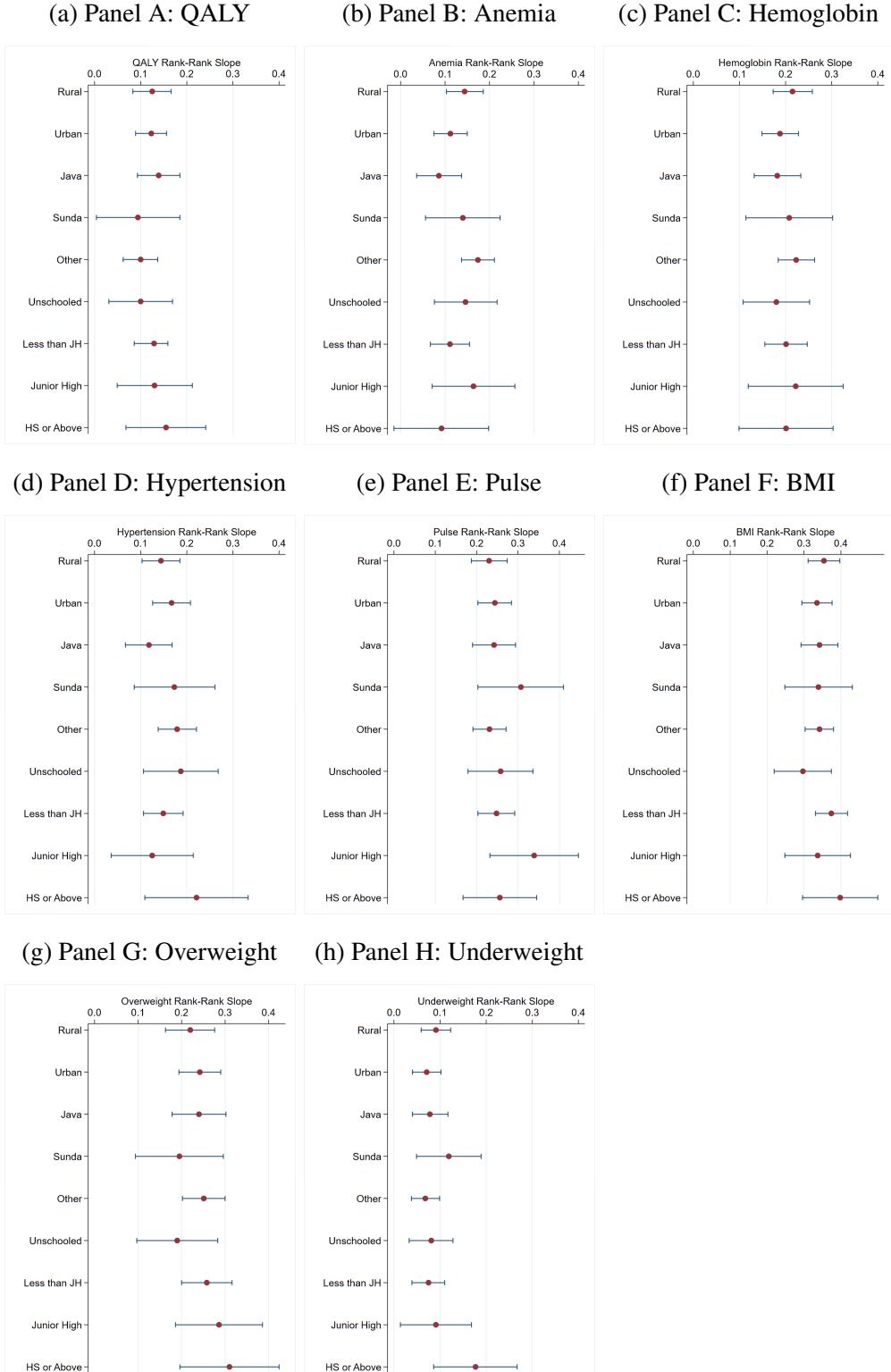


(h) Panel H: IHA Slope of Sum



Notes: The figures show intergenerational relationships in the first three principal components of the health measures and correspond to results in Tables 6 and Table 8

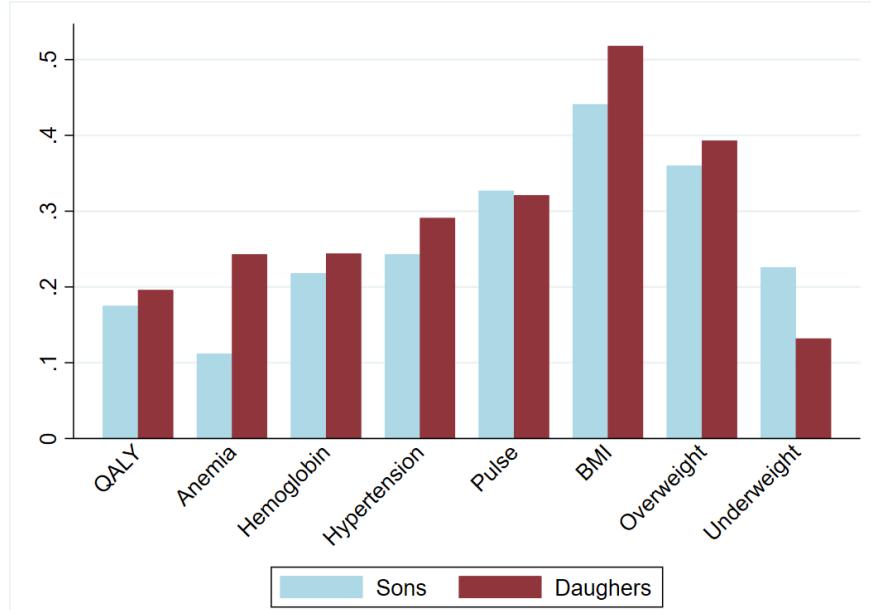
Figure A4: Heterogeneity in Rank-Rank Slopes



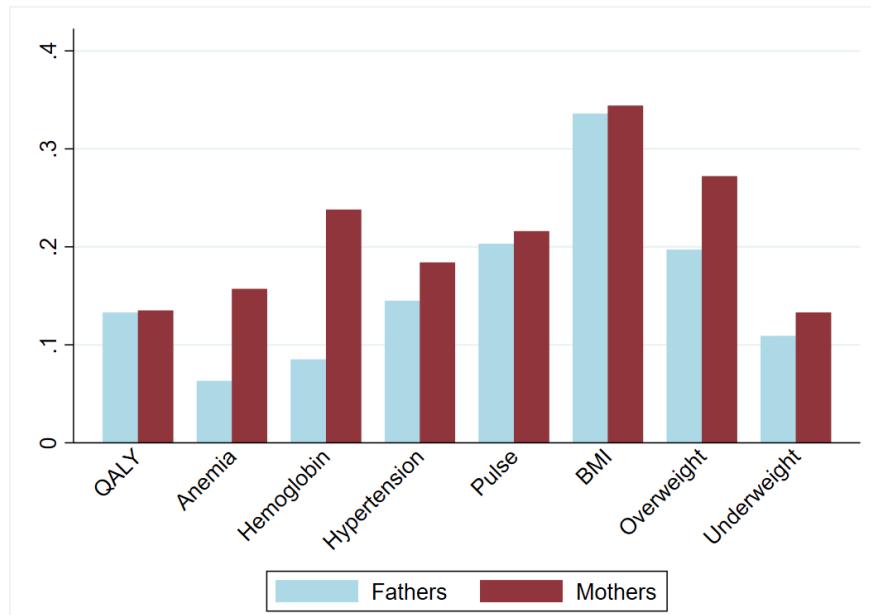
Notes: Each figure in the panels presents the rank-rank slopes when combining both parents health and when pooling sons and daughters. Results are by urban vs. rural, ethnicity (Javanese, Sundanese or other ethnicity), and education levels and correspond to estimates in Tables 9, Tables 10 and Tables 11. The whiskers represent 95 percent confidence intervals.

Figure A5: Intergenerational Health Association by Gender of Parents and Children

(a) Panel A: Intergenerational Health Associations, Both Parents on Sons vs Daughters

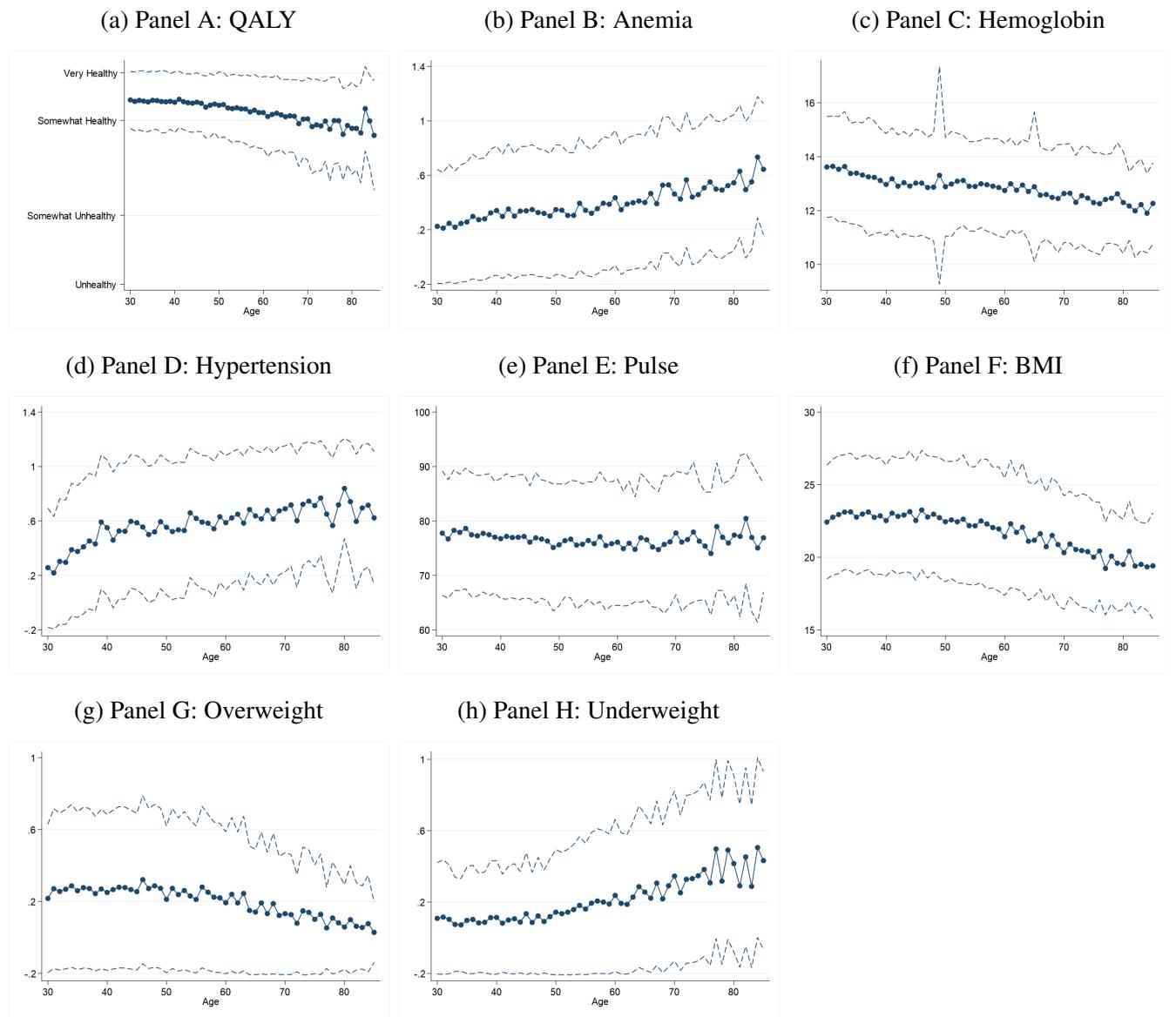


(b) Panel B: Intergenerational Health Associations, Mothers vs Fathers on Pooled Sons and Daughters



Notes: The figure in Panel A shows the intergenerational health associations of both parents health on sons and daughters, respectively. Panel B presents the estimates of the intergenerational health associations of mothers and fathers on pooled sons and daughters.

Figure A6: Health Measures Over the Life cycle



Notes: These figures plot each health outcomes over life cycle from 30 to 85 years old.

Table A1: Factor loading for Mothers, Fathers and Children

	Comp1 (1)	Comp2 (2)	Comp3 (3)	Comp4 (4)	Comp5 (5)	Comp6 (6)
Panel A: Mothers						
QALY	0.29	0.16	0.66	0.63	0.9	-0.20
BMI	0.39	-0.50	-0.19	-0.05	0.66	-0.34
Diastolic	-0.49	-0.48	0.24	0.23	0.30	0.57
Systolic	0.71	0.02	0.03	-0.14	-0.05	0.69
Hemoglobin	-0.15	0.53	0.34	-0.48	0.59	0.07
Pulse	-0.04	0.45	-0.59	0.54	0.34	0.20
Panel B: Fathers						
QALY	0.02	0.29	0.73	0.60	-0.05	-0.13
BMI	0.52	-0.37	0.01	0.13	0.66	-0.38
Diastolic	-0.35	-0.61	0.28	0.11	0.25	0.59
Systolic	0.63	0.35	0.11	-0.17	0.13	0.66
Hemoglobin	-0.38	0.37	0.33	-0.56	0.54	-0.13
Pulse	-0.28	0.39	-0.52	0.52	0.45	0.19
Panel C: Children						
QALY	0.13	0.11	0.53	0.83	-0.02	-0.04
BMI	0.36	0.55	-0.31	0.06	0.56	-0.38
Diastolic	-0.55	0.31	-0.39	0.32	0.17	0.56
Systolic	0.72	0.07	-0.09	-0.04	-0.13	0.67
Hemoglobin	-0.16	0.33	0.68	-0.43	0.38	0.27
Pulse	0.06	-0.69	-0.03	0.12	0.70	0.14
Panel D: Sons						
QALY	0.07	-0.15	0.90	0.38	0.10	-0.02
BMI	0.38	0.50	-0.04	0.01	0.69	-0.36
Diastolic	-0.53	0.53	0.07	0.12	0.23	0.61
Systolic	0.70	-0.06	0.02	-0.18	0.01	0.69
Hemoglobin	0.27	0.39	-0.20	0.72	-0.47	-0.04
Pulse	0.06	0.54	0.37	-0.54	-0.49	-0.16
Panel E: Daughters						
QALY	0.20	-0.20	0.57	0.77	0.11	-0.05
BMI	0.49	0.46	-0.04	0.06	-0.55	-0.49
Diastolic	-0.46	0.55	0.12	0.26	-0.38	0.51
Systolic	0.71	0.07	0.04	-0.16	0.07	0.68
Hemoglobin	0.07	0.53	-0.42	0.34	0.65	-0.09
Pulse	-0.06	0.41	0.69	-0.45	0.34	-0.17

Notes: The magnitudes of the factor loading are derived from the scoring coefficients of the principal components for mothers, fathers and children respectively.

Table A2: Factor loading for Mothers, Fathers and Children (convert all the variables to negative health conditions)

	Comp1 (1)	Comp2 (2)	Comp3 (3)	Comp4 (4)	Comp5 (5)	Comp6 (6)
Panel A: Mothers						
Poor Health	0.06	0.38	0.87	0.26	-0.05	-0.19
Anemia	0.39	-0.24	0.11	0.14	0.87	0.08
Hypertension	-0.12	0.55	-0.44	0.68	0.16	-0.06
Abnormal pulse	0.04	0.67	-0.09	-0.67	0.29	-0.05
Underweight	0.63	0.20	-0.02	0.06	-0.30	0.68
Overweight	-0.65	0.03	0.20	0.00	0.21	0.70
Panel B: Fathers						
Poor Health	0.27	0.39	-0.13	0.82	0.13	-0.26
Anemia	0.48	0.41	0.11	-0.18	-0.73	0.14
Hypertension	-0.12	0.75	0.23	-0.39	0.44	-0.14
Abnormal pulse	0.18	-0.21	0.93	0.20	0.11	0.10
Underweight	0.58	-0.04	-0.23	-0.09	0.47	0.62
Overweight	-0.55	0.27	0.04	0.31	-0.15	0.71
Panel C: Children						
Poor Health	-0.06	0.60	0.52	0.20	0.56	-0.08
Anemia	0.12	0.36	-0.36	0.79	-0.32	-0.02
Hypertension	-0.07	-0.63	-0.09	0.50	0.58	0.00
Abnormal pulse	0.17	-0.32	0.77	0.28	-0.45	0.02
Underweight	0.69	0.05	-0.03	-0.05	0.17	0.70
Overweight	-0.69	0.04	0.06	0.08	-0.11	0.71
Panel D: Sons						
Poor Health	0.08	-0.59	0.46	0.45	0.48	-0.01
Anemia	0.32	0.35	0.55	0.42	-0.53	-0.12
Hypertension	-0.18	0.71	-0.01	0.32	0.61	0.00
Abnormal pulse	0.10	-0.13	-0.68	0.69	-0.18	0.04
Underweight	0.66	0.09	-0.04	-0.12	0.16	0.72
Overweight	-0.64	-0.03	0.19	0.16	-0.24	0.68
Panel E: Daughters						
Poor Health	-0.09	-0.38	0.77	-0.24	0.43	-0.10
Anemia	0.23	-0.36	0.13	0.89	0.02	0.09
Hypertension	0.02	0.70	0.06	0.25	0.66	0.05
Abnormal pulse	0.19	0.48	0.61	0.07	-0.59	-0.04
Underweight	0.67	-0.06	0.01	-0.27	0.09	0.69
Overweight	-0.68	0.03	0.12	0.10	-0.11	0.71

Notes: The magnitudes of the factor loading are derived from the scoring coefficients of the principal components for mothers, fathers and children respectively.