Intergenerational Transmission of Mother-to-Child Health: Evidence from Cebu, the Philippines

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Using rich longitudinal data from the Philippines, we causally estimate the transmission of maternal health to child health, from birth through preadolescence. To obtain exogenous variation in maternal health we exploit an array of data on climate (temperature, precipitation and cyclone) shocks surrounding the time of the mother's birth and during her early childhood. This large set of instruments is weak, with first stage F-statistics hovering around one. We contribute a new machine learning method of choosing/forming optimal instruments in a setting of many valid but weak instrumental variables, a method based on singular value analysis (SVA). This method out-performs other options, and results in first stage F-statistics hovering around 50. Using this optimal instrument, we show that mother's health causally transmits to child health from birth through childhood and into adolescences. Some of this transmission is due to the effect of mother's health on child health at birth and then the effect of birth health on later health. Yet, the transmission of maternal height to child height persists into adolescence beyond its effect on health at birth and in early life. Moreover, the transmission of height increases rather than diminishes as the child ages. This may be, in part, due to a transmission of growth velocity that causes the health advantage of having a taller mother to widen with age.

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1 Introduction

Intergenerational correlations in socio-economic status characterize an important dimension of inequality and poverty in a society. They provide evidence on the extent to which children's future economic well-being is tied to the socio-economic status of their parents: is there truly social and economic mobility in a society or are children born to poor families destined to be poor adults? The transmission of health from one generation to the next may be an important pathway underlying these correlations. Less healthy children, on average, obtain less schooling, earn lower wages, and own less assets as adults.[1] However, obtaining causal estimates of the transmission of health is difficult, particularly since causality can run in both directions. The transmission of poor health, for example, likely causes a transmission of poverty and, simultaneously, the transmission of poverty likely causes a transmission of poor health. Either or both directions of causality are plausible in that health is both genetically and environmentally determined.

A large body of literature demonstrates an association between poor maternal health and poor child health outcomes.For example, see Bhalotra and Rawlings (2011), Currie and Moretti (2007), Eriksson, Bratsberg and Raaum (2005), Victora et al. (2008), and Yashin and Iachine (1997). However, evidence regarding the mother-to-child health transmission is plagued by omitted variable bias that obscures the causal relationship between mother and child health. Causal estimates of the transmission of maternal health to child human capital are sparse. While nutritionists and economists have in a few cases traced the children of women who participated in supplementation trials, new immunization regimes or other randomized, health-related trials, such research measures the intergenerational impact of a particular trial, only.[2] It cannot provide evidence regarding the more general, intergenerational transmission of health from mothers who fall within the normally-observed distribution of health. The best estimates using observational data tend to come from within-mother or within-siblings estimates of maternal-child health transmission.[3] Understanding the extent that a child's human capital is determined by that of his or her mother enables policymakers to better work towards breaking the transmission of poor human capital outcomes across generations, particularly in the context of child poverty. Moreover, improved understanding of the extent that health is causally transmitted from one generation to the next tells us the degree to which the consequences of a health shock or the benefits of a health intervention will cross into the next generation.

Using rich longitudinal data from Cebu, the Philippines, we estimate the causal transmission of health from mother to child. Specifically we estimate the effect of mother's health stock (proxied by height) and health flow (proxied by skinfold thickness) on her child's health stock and health flow. We do so across multiple childhood stages from birth through early childhood and adolescence. To obtain exogenous variation in maternal health, we capitalize on random climate variation in Cebu around the time of the mother's birth and in her early childhood. Specifically, we instrument mothers baseline health using information on typhoon exposure (proxied by windspeed) from the year prior to her birth, her birth year, and the year following her

[1]For example, see Victora et al. (2008) and Walker et al. (2007).
[2]See Black et al. (2013) for a good summary of this evidence.
[3]For example, see Currie and Moretti (2007).

birth. We also use harvest season typhoon exposure as well as monsoon season precipitation and temperature covering the period from the two years preceding through the five years following her birth.

The far-reaching consequences of early life health shocks to adult human capital and productivity are well-documented.[4] Further, the association between maternal health and child outcomes (especially child health) is similarly well-documented.[5] However, very little evidence traces the impact of early childhood health shocks across multiple generations. In this paper, we add to the literature on the long-term consequences of early life health by demonstrating that the impact of early life climate variations extends not only to our sample mothers' adult health but is evident in the health of their children through adolescence.

We also add nuance to previous literature on the intergenerational transmission of health by considering multiple dimensions of both mother's and child's health as well as by examining multiple windows of transmission over many stages of childhood. This allows us to pinpoint where health transmission is most crucial, and to consider the conditional persistence of transmission. Is transmission of mother-to-child health strongest at birth and diminishes as the child ages and is exposed to a widening array of health inputs? Are observations of mother-to child health transmission in later childhood due to a primary transmission occurring at birth, which in turn, affects later health?

We further provide an important contribution to the limited but growing literature on the health effects of climate shocks. Much of the previous work in this body of literature examines the short- or long-term impact of a climate shock such as a hurricane or earthquake.[6] However, our instruments are not necessarily climate shocks but rather measure climate variation on the intensive margin. Our analysis therefore establishes that even simple year-to-year variation in climate can have far-reaching consequences that can span generations.

Finally, we add to the literature on dealing with many, but weak, instruments. The climate variables we employ for our instruments occur during the period from the 1930s to the 1960s—two to four decades prior to the birth of our sample children. We also de-trend them by time and time squared. It is, therefore, unlikely that our instruments affect child health outside of their effect on maternal health. Thus, our climate variables likely satisfy the exclusion restriction required for an instrument to be valid.[7] However, our instruments are both many and weak with first-stage F-Statistics hovering around one. We are therefore unlikely to satisfy the relevance condition and may suffer from two-stage-least-squares (2SLS) bias. To address this problem, we propose a new method of forming an optimal instrument from many weak instruments using a machine learning method based on singular value analysis (SVA). This method far out-performs other methods and results in first-stage F-Statistics ranging from approximately 35 to 75.

[4]For example, see Alderman, Hoddinott and Kinsey (2006), Almond and Currie (2011), Barker (1998), Maluccio et al. (2009), Strauss and Thomas (1998), and Victora et al. (2008).

[5]See footnote 1

[6]For example, see Caruso and Miller (2015), Currie and Rossin-SLater (2013), Deuchert and Felfe (2015), and Fuller (2014).

[7]We provide further evidence below to support this claim.

Our results indicate that maternal health exhibits a persistent effect on child health years after birth. We find that mother's health flow measured in her third trimester of pregnancy significantly predicts her child's health from birth through childhood. However, this transmission seems to be primarily due to the mother's health at pregnancy transmitting to her child's health at birth and then the birth health's effect on subsequent realizations of child health. Conversely, the transmission of maternal health stock to child health stock (proxied by height-for-age z-scores) persists throughout childhood and into adolescence even after controlling for health at birth. Not only does the transmission persist, but the magnitude of the transmission increases as the child ages. One possible explanation for this finding is that increased mother's height not only translates into increased height in her child but also into an increased growth velocity. This results in a widening height advantage as the child gets older. Indeed, the estimated transmission of maternal health stock to child height only reduces to near zero once we control for all previously accumulated height.

2 Climate, Early Life Health, and the Intergenerational Transmission of Health

A growing literature documents intergenerational correlations of poverty and income in both rich and poor countries (Asadullah, 2012; Behrman, Gaviria and Székely, 2001; Behrman et al., 2017; Bevis and Barrett, 2015; Black and Devereux, 2011; Harper, Marcus and Moore, 2003; Lefgren, Sims and Lindquist, 2012; Takahashi, 2013). Due to the documented effect of early life health on a wide range of adult economic, health, demographic, and behavioral outcomes (Alderman, Hoddinott and Kinsey, 2006; Almond and Currie, 2011; Maluccio et al., 2009; Victora et al., 2008), the intergenerational transmission of health is one potential channel through which the transmission of poverty operates. Poor maternal health is associated with numerous adverse child health outcomes such as low birth weight (Currie and Moretti, 2007; Victora et al., 2008), infant mortality (Bhalotra and Rawlings, 2011), shorter lifespan (Yashin and Iachine, 1997), and increased morbidity (Eriksson, Bratsberg and Raaum, 2005).

The majority of evidence concerns the association between maternal health and birth weight, or some other indicator of infant health at birth. For instance, using five cohort studies from Brazil, Guatemala, India, the Philippines, and South Africa, Victora et al. (2008) find that maternal undernutrition — using a range of indicators — is associated with low birthweight. They cannot parse, however, whether this correlation reflects a biological mechanism or a transmission of socioeconomic status. Bhalotra and Rawlings (2011) examine the relationship between mother's height and infant survival using a large dataset of 2.24 million children born to 600,000 mothers spanning 38 developing countries. They find that, within mothers, improvements in socioeconomic status and health environment mitigate the transmission of health between mother and child. Yet they are only able to examine this transmission along the dimension of infant survival.

Using data from California, Currie and Moretti (2007) show that a child is almost 50 percent more likely to be low birthweight if his or her mother was low birthweight. This is true even after controlling for the birthweight of mothers' siblings and for mothers'

socioeconomic status, suggesting that this transmission reflects a biological mechanism. However, they also find that the likelihood that a woman transmits her low birthweight to her child increases at higher levels of poverty. This suggests that the biological transmission of low birthweight can be mitigated by improved economic circumstances.

The evidence on the relationship between mother and child health suggests that policies aimed improving maternal health outcomes will hold important benefits for child well-being. Programs targeting early childhood health hold promise as effective instruments for impacting women's health in adulthood. A large body of compelling evidence demonstrates the importance of early childhood health to a wide range of adult human abilities and characteristics, including adult health. During the early years of childhood, individuals experience rapid growth and brain development. By the age of two a child typically reaches approximately half of her adult height and her brain will be approximately 90% of its adult size (Michaelsen et al., 2008). Consequently, both positive and negative health shocks during this period can have far-reaching consequences for an individual's long-term health status. The fetal programming literature documents a robust relationship between *in utero* or infant health and adult health. Early life health outcomes such as birth weight, fetal and maternal nutrition, and growth in the first two years of life are all predictive of later adult health (Almond and Currie, 2011; Victora et al., 2008). Further, due to the multidimensional nature of human capital formation, adverse health conditions in early life can impair learning and socio-emotional development. A robust literature documents the long-term effects of early childhood malnutrition and health shocks on school attainment, impaired cognitive development, decreased future productivity and earnings, and participation in crime and other risky behaviors (Alderman, Hoddinott and Kinsey, 2006; Victora et al., 2008; Maluccio et al., 2009).

Recently, a related and growing literature specifically examines the effects of climate shocks and natural disasters on child health and long-term human capital formation. Weather events can impact individual health through multiple channels including their effect on physical infrastructure, food security, rural incomes, disease environment, and maternal stress. Weather events such as hurricanes, floods, frost, and rainfall and temperature shocks are associated with reduced child height and weight (Pörtner, 2010; Skoufias and Vinha, 2012; Tiwari, Jacoby and Skoufias, 2017), increased birth complications (Currie and Rossin-SLater, 2013; Simeonova, 2009), and poorer performance on cognitive achievement tests (Fuller, 2014). Demonstrating the longer-term effects of weather events, Maccini and Yang (2009) find that early life rainfall affects the adult health, schooling, and socio-economic status of women in Indonesia.

If early life health holds such profound influence on adult well-being, and the well-being of parents, particularly mothers, is an important determinant of child health; then it stands to reason that the effects of early childhood health shocks will not only impact the adult health of individuals exposed to them but will reach into the next generation affecting their children's health as well. One of the only studies we are aware of that traces the effects of a natural disaster to the next generation is Caruso and Miller (2015), who examine the long-term effects of the 1970 earthquake in Ancash, Peru. They find that individuals exposed to the earthquake completed less schooling by adulthood and exposed women had a higher probability of being divorced or single

parents. They further find that children whose mother was exposed to the earthquake had lower school attainment, on average. They found no schooling effects of having a father exposed to the earthquake.

3 Many but Weak Instruments

In our analysis we exploint information on climate during the time of our smaple mothers' birth and in their early childhood to obtain exogenous variation in their health. However, our climate instruments are both many and weak. A growing literature deals with the asymptotic properties of various estimators in settings with many weak instrumental variables (IVs). It is well known that parameters estimated via two stage least squares are biased towards the parameters that would be estimated via OLS when instruments are weak. Additionally, instrumental variable estimators making use of many, weak instruments can have poor asymptotic properties — when the concentration parameter is small relative to sample size, neither two stage least squares nor limited information maximum likelihood estimators are consistent, and the usual standard errors are too small (Stock and Yogo, 2005; Staiger and Stock, 1997; Chao and Swanson, 2005; Hansen, Hausman and Newey, 2008).

A related literature attempts to choose or formulate a small number of optimal IVs in settings with many weak IVs, thereby avoiding (rather than correcting) the asymptotic problems associate with many weak IVs. Belloni et al. (2012) propose using the *least absolute shrinkage and selection operator* (Lasso) or the related Post-Lasso estimator in such a setting. Originally proposed by Tibshirani (1996), Lasso is a shrinkage estimator, designed to reduce model dimensionality by isolating key sources of model variation. Lasso estimates coefficients by minimizing the sum of squares subject to a penalty on large coefficients, forcing the majority of coefficients to zero and thereby leaving only the strongest predictive covariates in the model.[8] Belloni et al. (2012) propose and demonstrate the use of Lasso in the first stage of a two stage least squares model, as a method of subsetting the strongest IVs while estimating the first stage equation.

The number of IVs chosen by Lasso depends on the specification of the penalty function, but generally first stage Lasso results in a very small subset of optimal IVs. For instance, Belloni et al. (2012) and Belloni, Chernozhukov and Hansen (2014) consider the effects of federal appellate court decisions regarding eminent domain on four types of housing prices, instrumenting for court decisions with judge characteristics. From 138, 143, 147, and 138 potential, price type specific instruments, Lasso chooses only 1, 2, 2, and 4 optimal instruments. Mueller-Smith (2014) similarly applies Lasso and Post-Lasso to estimate the effects of incarceration on subsequent behavior and labor market activity, instrumenting for court decisions with interactions between judge characteristics and defendant characteristics. From likely hundreds of potential instruments, Lasso subsets 5 for use in Post-Lasso estimation. Mueller-Smith (2014) points out that that not only does Lasso estimation reduce dimensionality, but examining the selected covariates lead to insight about the data generating process.

[8]Because Lasso results in downwardly biased coefficients even for the "chosen" covariates, Post-Lasso simply re-estimates the least squares problem again with only the smaller subset of covariates, using ordinary least squares.

Other machine learning procedures can be used in the first stage with similar effect. Okui (2011) selects IVs in the first stage with a shrinkage procedure that uses a ridge penalty. This ridge penalty shrinks coefficients towards zero, but doesn't set them to zero as Lasso does. Ng and Bai (2009) select IVs via boosting, a model selection procedure that produces solutions similar to those produced by Lasso.

Rather than choosing an optimal *subset* of potential IVs, one might instead chose optimal linear combinations of all potential IVs. Kloek and Mennes (1960) first proposed principal components as just such a set of linear combinations. Using principal components in the first stage lowers dimentionality while retaining the bulk of original variation. Additionally, the fact that each principal component is orthogonal to the next increases prediction stability. Amemiya (1966) later supported the proposition by showing that principal components satisfy several desirable properties when it comes to choosing linear combinations of IVs. The use of principal components in the first stage was subsequently embraced in the macroeconomics literature, where observations were scarce and the accompanying sparsity was particularly useful (Klein, 1969; Mitchell, 1971; Giles and Morgan, 1977).

More recently, Ng and Bai (2009), Kapetanios and Marcellino (2010*a*), and Kapetanios and Marcellino (2010*b*) evaluate the performance of principal component IVs under various data generating structures. In a setting where the endogenous variable is truly driven by a small subset of many potential IVs, subsetting the original IVs results in better estimation outcomes than subsetting the principal component IVs (Ng and Bai, 2009). This is because the primary principal components will capture variation from both the "true" IVs and the less relevant IVs.

Conversely, if the endogenous variable is driven by a small number of unobservable, exogenous common factors, all correlated with the entire set of original IVs, principal component IVs will perform well, as they proxy for the true common factors (Ng and Bai, 2009; Bai and Ng, 2010). A factor structure is not necessary for principal components IVs to perform well, however. If the original IVs are themselves imperfect proxies for the true data generating process (i.e., only correlated with the true drivers of the endogenous variable), then a subset of the principal component IVs will still be consistent, and will perform better than a subset of the original IVs (Ng and Bai, 2009; Kapetanios and Marcellino, 2010*b*),

Most authors subset potential IVs (whether principal component IVs or otherwise) based on explanatory power. Lasso and other shrinkage estimators choose "optimal" IVs by minimizing mean squared error, or maximizing explanatory power, but with an added penalty for high first stage coefficients. Ng and Bai (2009) choose optimal IVs via boosting as well as two types of "ranking." Boosting maximizes explanatory power like Lasso, though it works by iteratively selecting predictors that minimize the squares of the current residuals. The first ranking procedure simply keeps only IVs with t-values over a certain power, thus subsetting on first stage relevance rather than explanatory power. The second ranking procedure combines both relevance and explanatory power.

Winkelried and Smith (2011) subset principal components based on the magnitude of associated eigenvalues, rather than explanatory power, and evaluate two thresholds for

eigenvalue magnitude.[9] This is similar, though not equivalent, to subsetting on first stage relevance — principal components with greater eigenvalues capture more variability in the original IV matrix, and thus are likely but not guaranteed to be better predictors of the endogenous variable.

We propose a new method of formulating optimal IVs in settings with many weak IVs, using singular value decomposition rather than principal component analysis (PCA) to form optimal linear combinations of the original IVs. Singular value decomposition decomposes the matrix itself (rather than the covariate matrix as PCA does) into the eigenvectors, or principal axes, and also into "singular values," which are a direct transformation of eigenvalues. Singular value *analysis*(SVA) solves a minimization problem equivalent to the original ordinary least squares minimization problem, but in a space defined by the new axes, or eigenvectors, obtained by singular value decomposition. The parameter estimated by this minimization can be transformed into the parameter that solves the equivalent ordinary least squares minimization problem. We then apply machine learning procedures to the SVA procedure in order to form one "optimal" IV from our full set of instruments.

4 Data

4.1 Cebu Longitudinal Health and Nutrition Survey

To explore the transmission of mother-to-child human capital we exploit unique data collected by the Cebu Longitudinal Health and Nutrition Survey (CLHNS). The CLHNS is a rich longitudinal data set collected from the metropolitan area of Cebu, the Philippines. Originally designed to study infant feeding patterns and diets, the CLHNS is part of an ongoing study of a cohort of Filipino women who gave birth during the one year period between May 1, 1983 and April 30, 1984. The study area encompasses 17 urban and 16 rural randomly selected barangays[10] in metropolitan Cebu and includes several urban, mountainous, and coastal regions.

Within these barangays, all pregnant women due to give birth during the designated time frame were canvassed to participate in the study. Women were surveyed in their third trimester, at birth, and then every 2 months for the first 24 months of their child's life. Additional rounds were later added and the mothers, index children, and other caregivers were subsequently surveyed throughout childhood, adolescence and into early adulthood. In order to study the transmission of maternal health to child human capital we exploit information collected at birth and the first two years of the index children's lives. We also use information from when the index children were 8, 11, and 15 years old.[11]

The CLHNS provide broad and detailed information on numerous dimensions of human capital. In each round, the surveys collected extensive information on the child, the mother, the child's primary caregiver if that is not his/her mother, the child's

[9] Doran and Schmidt (2006) do the same when using principal components to solve a GMM estimation problem, not in an instrumental variable context.

[10] A barangay is the smallest administrative unit in the Philippines

[11] The first three survey waves conducted after the age two survey were conducted at ages 8, 11, and 15.

household, and his/her mother's household (if if is not the same as the child's). Anthropometric measures were taken in each wave on both mother and child. Therefore these data are uniquely suited towards investigating the relationship between maternal and child health.

Table 1 provides summary statistics on some baseline characteristics and child health outcomes for the sample children through age 11. Height-for-age is often considered the gold standard for measuring early life health and health stocks. The children in this sample are relatively short with height-for-age z-scores (HAZ) hovering around two standard deviations below median height for what is considered healthy for their age.[12] Approximately 60% of the sample is stunted[13] at some point during the first 6 months of life. Stunting rates spike at approximately 75% at age 2 and then reduce to around 50% at ages 8 and 11.

Measures related to weight are more sensitive to current nutritional inputs and morbidity and are thus in part able to capture aspects of current health flows rather than stocks. Just over 10% of the sample was born at a low birth weight.[14] Weight-for-height (WHZ) and body mass index (BMI) z-scores capture how thin a child is for his/her height and hover around one standard deviation below what is considered healthy during all the ages examined. Wasting rates are highest when the sample children are young. Approximately 30% of the sample children are wasted at age 1. Wasting rates decrease to around 20%at age 2 and 10% at age 8 but increase again to almost 20% at age 11.

4.2 Climate Data and Instruments

In order to causally identify the intergenerational transmission of human capital in this setting we exploit climate information on windspeed, temperature and rainfall around the time of the mother's birth and during the early years of her childhood. Figure 1 plots the distribution of maternal birth year. Mothers in our sample were all born between the years 1936 and 1966, with most born in the 1950s and early 1960s, approximately two to four decades before the birth of our sample children.

To investigate the effect of climatic conditions on maternal health, we use three geospatial datasets, all stretching back to the 1930s. The majority of geospatial re-analysis data begins in 1979, with the advent of satellites. Re-analysis data beginning prior to this relies on global records of pressure levels.

We obtain data on windspeed from the 20th Century Re-Analysis project, run by the Earth Science Research Laboratory (ESRL) at the National Oceanic and Atmospheric Administration. It contains global 10-meter windspeed estimates at a 2 degree spatial resolution in 3 hour intervals (8 observations per day). We average these estimates within day, and across the cells that overlay the island of Cebu.

Our temperature data also come from the 20th Century Re-Analysis project, and contains global surface temperature estimates at a 2 degree spatial resolution in 6 hour

[12]World Health Organization reference data was used to standardize all anthropometric z-scores.
[13]A child is considered stunted if his/her height-for-age z-Score is below -2.
[14]A birth weight is considered low if it is below 2500 grams.

intervals (4 observations per day). We procure precipitation data from the Global Precipitation Climatology Centre, also generated by ESRL. This dataset provides monthly, average precipitation estimates at a 0.5 degree spatial resolution.

4.3 Typhoons in the Philippines

The Philippines is one of the most intensely exposed countries in the world to typhoons (also known as tropical cyclones or hurricanes). Typhoons are characterized by heavy rainfall and intense, damaging winds. Anttila-Hughes and Hsiang (2014) demonstrate the heavy economic and health cost of typhoons in the Philippines. They find that typhoons increase infant mortality and reduce household income a year after they strike pointing to significant economic losses due to unearned income long after exposure. They further find that households often cope with typhoon shocks by reducing expenditure on human capital inputs such as medicine, education, and nutritious foods. Salas (2015) further finds that typhoon exposure increases pregnancies post-typhoon event with the fertility effect concentrated in agricultural areas where child labor is more prevalent.

Typhoons around the time of an infant's birth increases his/her risk of morbidity and mortality by destroying household assets and spreading waterborne disease through contaminated flood waters due to ocean surges (Anttila-Hughes and Hsiang, 2014; DOH, 2017). We therefore create a monthly measure of typhoon likelihood/windspeed intensity by squaring the maximum windspeed observed in each month, between 1927 and 1970. We then use as our first set of instruments these measures of monthly maximum wind speeds for the month of our sample mothers' births, each of the 12 months preceding their births, and each of the 12 months following their births. This allows us to control for windspeed intensity in the months directly before and after the births of mothers in our dataset.

Typhoons can also cause significant damage to rice crops, the primary staple in Philippines. Rice crops are most susceptible to environmental damage during the reproductive and ripening phase, which occurs during the two months prior to harvest (Blanc and Strobl, 2016).[15] The country underwent massive trade liberalization in agriculture during during the 1990s. Prior to this, however, rice was largely self-supplied. Accordingly, typhoons during the ripening period can have severe consequences for food security and the health of small children or even as-yet-unborn children. Our second set of instruments therefore captures potential typhoon-driven rice destruction for the two years prior to our sample mothers' birth year, the year of their birth, and the five years following their birth. This potential destruction is captured by squaring the period-maximum windspeed observed for the two months preceding rice harvest, for two harvest periods per year.

Similarly, growing conditions (temperature and precipitation) during the monsoon months of each year impact rice production, driving local food supply and family income in the short run and therefore impacting early life health. We create a year-specific measure of growing conditions by controlling for average monsoon season

[15] Strong typhoon winds can damage rice crops through lodging, striping and injuring plant organs. Rice plants can also suffer from water stress due to enforced transpiration (Blanc and Strobl, 2016).

temperature, average monsoon season rainfall, and interactions between temperature and rainfall. We use as our third set of instruments these measures for the two years prior to, year of, and five years after the births of mothers in our dataset. To avoid capturing any spurious correlations between time trends in monsoon season temperature and precipitation (e.g., driven by el nino or by climate change) and time trends in mother's health, we de-trend monsoon variables by year, allowing for a quadratic shape.

Figure 2 plots maternal height as a funtion of birth months and clearly demonstrates a distinct seasonality in maternal health stock. Therefore, to capture the predictable seasonality of health (related to the typhoon and monsoon seasons as well as disease vectors and perhaps other factors) we use a vector of maternal month of birth fixed effects as our fourth set of instruments. As seen in Figure 3, our sample mothers' birth months appear to be fairly evenly distributed meaning that mother's born in a particular month or season are not overweighted in our later estimations. Appendix 1 holds greater detail on gridded wind, temperature and precipitation data, as well as on the variables created with these climate data.

4.4 Maternal Health and Wind, Rain, and Temperature

While we do not use the above raw climate variables directly as instruments for maternal health (we instead use an optimal linear combination of them described below), Tables B1—B3 in Appendix 2 demonstrate the persistent effect these variables exert on the adult height and skinfold thickness of our sample women. Tales B1, B2, and B3 report the estimated marginal effects of windspeed in the months preceding and following birth, windspeed during the rice harvest seasons early in life, and average precipitation and temperature in the monsoon seasons in early life, respectively. We do not estimate the effects of each of these variable sets together due to the large number of climate variables. While not reported, each specification additionally controls for maternal month of birth and baseline barangay fixed effects. In Table B1 we see that higher wind speeds around the time of conception and birth appear to result in women with shorter stature and lower skinfold thickness. Increased wind speed in the sixth and tenth month of life appears to improve maternal height. This result is somewhat puzzling and may simply reflect a spurious correlation.

Wind speed in the months surrounding birth appears to have a weaker effect on maternal adult health than that during early life harvest seasons and monsoon season conditions. This likely highlights the importance of the weather's effect on food security and household income in affecting early life, and subsequently adult, health outcomes. Generally, we find that increased wind speed during the rice ripening and harvest seasons reduces maternal adult health (Table B2) reflecting the destructive effects of typhoon winds on rice crops. Conversely, increased rain and temperature during the early life monsoon seasons improves growing conditions and thus improves the adult health of our sample mothers (Table B3).

5 Estimation Strategy

We wish to estimate the transmission of maternal health to child health. Accordingly, Equation 1 models the health of child i at age a born to mother j living in barangay v,

C^a_{ijv}. The health of mother j is captured by M^l_{jv}, and the point estimate $\hat{\gamma}^a_1$ therefore captures the intergenerational transmission of health at child age a. The superscript $l \in \{stock, flow\}$ defines mother's health as a stock (height) or a flow (skinfold thickness).

$$C^a_{ijv} = \gamma^a_1 M^l_{jv} + \gamma^a_2 X^a_{ijv} + \lambda^a_v + \varepsilon^a_{ijv} \tag{1}$$

We estimate Equation 1 separately for child ages $a \in \{0, 1, 2, 8, 11, 15\}$. The matrix X^a_{ijb} holds individual- and household-level controls and includes household per capita income, household size, mother's age, and mother birth cohort dummies that indicate whether the child's mother is younger than 20 years, between 20 and 35 years old, or older than 35. X^a_{ijv} also includes a vector of barangay of birth fixed effects as well as child month of birth fixed effects to control for seasonality in health based on month of birth. λ^a_v is a vector of baranagay of residence fixed effects (which is equivalent to barangay of birth during the first two years of life).

We consider two dimensions of child health: health stock and health flow. We proxy child health stock by height-for-age z-scores (HAZ) at each age we examine. For ages two and younger HAZ is calculated using recumbent length rather than height. We proxy child health flow using weight measures. Specifically, these measures include birth weight[16], weight-for-height z-scores (WHZ) for ages 1 and 2, and body-mass-index-for-age z-scores (BMIZ) for ages 8, 11, and 15. All z-scores are calculated using World Health Organization (WHO) reference data. We similarly consider a measure of maternal health stock, M^{stock}_{jv}, and maternal health flow, M^{flow}_{jv}. Our measure of maternal health stock is her baseline height measured in centimeters (cm). Our measure of her health flow is her baseline skinfold thickness, also measured in cm. Both measures are taken at baseline when the women were in their third trimester of pregnancy.[17]

Estimating 1 using OLS will return a biased estimate of $\hat{\gamma}^a_1$ as mother's health is correlated with numerous, omitted dimensions of socio- economic status that also affect child health. To obtain a causal estimate of γ^a_1, we instrument mother's health using our climate information from around the time of her birth and in her early childhood. To do so, we would typically instrument for mother's health using our entire set of exogenous climate variables: windspeed in the months surrounding birth (WB_{jv}), monsoon conditions (temperature and rainfall and their interaction) for the years prior to and after birth (MN_{jv}), windspeed during harvest months for the years prior to and after birth (HW_{jv}), and maternal month of birth fixed effect λ^l_{jb}. The first stage equation of this two stage least squares estimation would be specified as follows:

$$M^l_{jv} = \beta^l_1 WB_{jv} + \beta^l_2 MN_{jv} + \beta^l_3 HW_{jv} + \lambda^l_{jv} + \beta^l_4 D_{jv} + u^l_{jv}, \tag{2}$$

[16]Birth outcomes were measured in the first survey round following the child's birth. Therefore, measurements were not necessarily taken on the day of birth but most were taken withing a few days of birth. However, a handful were taken over a month past their day of birth.

[17]Results do not substantially differ if we use mother's middle upper arm circumference as her health flow rather than skinfold thickness. Ideally, we would have liked to have used BMI as our measure of maternal health flow. However, any measure including maternal weight was not an option as our sample mother's were all pregnant at baseline and their weight thus included the sample child's weight, by construction.

where D_{jv} includes all the individual- and household-level covariates included in Equation (1).

However, this large set of 69 instruments seems to be weak (Stock and Yogo, 2005; Belloni et al., 2012). Indeed, tests on the joint significance of our instruments in (2) return F-statistics that hover around one. Therefore while our instruments likely meet the exclusion restriction required for an valid instrument, they do not appear to meet the necessary relevance condition. Therefore rather than estimating (2) with the full set of instruments, we propose a new new method that employs singular value analysis (SVA) and machine learning to choose the optimal linear combination of climate variables to use as our single instrument.

That is, rather than estimating Equation (2) using ordinary least squares (OLS), which maximizes explanatory power at the cost of predictive power, we estimate Equation (2) via singular value analysis (SVA). Singular value analysis minimizes mean squared forecast error (MSFE) by first decomposing the matrix of exogenous weather variables $\{WB_{jv};MN_{jv};HW_{jv}\}$ into a series of orthogonal eigenvectors and matching singular values (related to eigenvalues), and then solving a new minimization problem, equivalent to the original OLS minimization problem, in this rotated space. Candidate solutions to the SVA/OLS minimization are chosen by allowing the number of singular values and matching eigenvectors, k, to vary from 1 to k_{max}, where k_{max} is the total number of non-zero singular values (Lawson and Hanson, 1974). More information about SVA can be found in Appendix 3.

We denote Equation (2) parameters estimated via this SVA process as $\hat{\beta}_1^{l,k}$, $\hat{\beta}_2^{l,k}$, $\hat{\beta}_3^{l,k}$, and $\hat{\lambda}_{jv}^{l,k}$. A low k indicates a more parsimonious solution, modeling M_{jv}^l with less dimensions of variance in the original climate shock matrix. Parameter estimates with a higher k will provide a better fit to the original data, but they will also result in less stable predictions of $\hat{M}_{jv}^l$, and after a point will have lower forecast power as they will begin to overfit to random noise in our instruments and maternal health measures. Therefore, the choice of k plays a crucial role in shaping our instrumental variable.

We choose an optimal k^* by choosing the k that minimizes MSFE via group-wise cross validation. We could choose k in a different manner. For instance, we might choose k according to the associated singular values, choosing a cut-off for inclusion similar to the threshold for eigenvalue magnitude considered by Winkelried and Smith (2011). Theoretically, we could also choose k to maximize explanatory power, rather than forecast power — but this would simply lead us to choose $k = k_{max}$, including all eigenvalues with non-zero singular values in the rotated minimization problem, and would result in $\hat{\beta}_1^k$, $\hat{\beta}_2^k$, $\hat{\beta}_3^k$ estimates identical to those estimated under OLS. We might even choose k according to some measure of eigenvector relevance, in the rotated minimization problem.

However, we choose k to minimize MSFE because our goal is to differentiate signal, or causal associations between climate shocks and mother's health, from noise, or spurious/accidental associations between climate shocks and mother's health. Spurious correlations will increase explanatory power, but only meaningful signals will increase predictive power. Because eigenvectors capture primary directions/axes of the climate data, in order of decreasing importance, we expect that after some number k^*, all

remaining eigenvectors are associated with noise rather than signal, and therefore no longer increase predictive power.[18]

Having chosen the MSFE-minimizing k, we then predict our optimal instrumental variable as follows:

$$\hat{M}_j^{l,k^*} = \hat{\beta}_1^{l,k^*} WB_{jv} + \hat{\beta}_2^{l,k^*} MN_{jv} + \hat{\beta}_3^{k^*} HW_j + \hat{\lambda}_{jv}^{l,k^*}.$$

This gives us one optimal instrumental variable, $\hat{M}_j^{l,k^*}$, for each maternal health dimension to obtain the causal estimate of the transmission coefficient, $\hat{\gamma}_1^a$, in Equation (1). This is quite different from the principal component IV approach, where a subset of principal components (that subset being chosen according to some threshold for eigenvalues or relevance) are all included in the first stage of a two stage least squares estimation. Instead, we use a matrix decomposition approach to choose the optimal linear combination of instrumental variables. We do not use rotated vectors as instruments themselves.

5.1 Persistence and Mechanisms

After obtaining causal estimates of of the mother-to-child health transmission at each observed age, we additionally examine the persistence of maternal-child health transmission by estimating the ongoing, causal impact of baseline maternal health on child health at later ages, holding earlier child health constant. Ample evidence demonstrates that early health is an important determinant of later health. Therefore if we find in (1) that maternal height significantly impacts child height at birth and at, say, age 11, we do not know if the latter effect is due to the direct transmission of maternal health at age 11, an indirect transmission operating through the effect on birth health, which in turn affects age 11 health, or some combination of the two.

Equation (3) estimates the transmission of mother-to-child health for ages $a \in \{1, 2, 8, 11, 15\}$ in the same manner as Equation (1) except that we also control for lagged child health outcomes.

$$C_{ijv}^a = \delta_1^a M_{jv}^k + \delta_2^a C_{ijv}^{a-1} + \delta_3^a X_{ijv}^a + \lambda_{ijv}^a + \eta_{ijv}^a. \tag{3}$$

We estimate (3) increasing the lagged time period for included child health. For example if we estimate (3) for child HAZ at age $a = 8$, we first include child birth length as our lagged child health outcome. We then estimate (3) again including age 1 child HAZ and then age 2 child HAZ as our lagged child health outcome. If, for example, $\hat{\delta}_1^a = 0$ after controlling for birth health, then any transmission found at that age in Equation (1) is due to the effect of mother's health on birth health. If, on the other hand, $\hat{\delta}_1^a$ significantly differs from zero in Equation (3), we know that maternal health continues to causally transmit to child health at age a beyond its indirect effect through transmissions that occurred at the age of the lagged health outcome and before.

[18] Note that if eigenvectors representing signal and eigenvectors representing noise were interspersed, this ordered approach to choosing all eigenvectors of index $\leq k$ would not work. This approach assumes an approximately smooth transition between "signal eigenvectors" and "noise eigenvectors."

Equation (3) allows us to pinpoint the windows of mother-to-child health transmission. This, in turn, can elucidate some of the mechanisms underlying this transmission. For example, mother's health may transmit to child health through a biological mechanism. If this is the case we would expect the coefficient of transmission either to be most prevalent at birth (suggesting a biological transmission that occurs *in utero*) or to be relatively constant through all stages (suggesting some sort inherited aspect of health and growth). There could also be a genetic component to the transmission of health. For example, height has a strong genetic component. However, genetic variation in height does not typically emerge until the second growth spurt during adolescence. Early growth is primarily determined by nutrition and health status.[19] Thus if this is the case then we would expect the mother-to-child health transmission to be strongest during adolescence.

Finally, mother's health may also transmit to child health through parenting ability or socio-economic status. We identify the mother-to-child transmission of health off of variation in the mother's early life health. As noted previously, early childhood health affects a broad range of adult outcomes including economic, demographic, health, and human capital outcomes. Therefore, early life health shocks due to climate variability may result in reduced parenting ability or socio-economic status for these women. While not biological, this still represents a causal transmission of maternal health to child health. Only, it is operating through economic mechanisms. If this is the primary mechanism, then we would expect to find the maternal health transmission to be strongest for child health flow outcomes, which are more responsive to contemporaneous inputs and during sensitive periods[20] for the production of health stock (i.e., during early childhood for the production of height).

6 Results

Tables 2—6 report the estimated causal effect of maternal height and skinfold thickness on child height and weight outcomes from birth through age fifteen. Tables B5 and B6 in Appendix 4 compare the transmission coefficients of Equation (1) as estimated by OLS, 2SLS using all 69 instruments in the first stage, LIML using all 69 instruments, PCA using PCA components chosen by a Scree test, and 2SLS using the SVA generated instrument in the first stage. What is most notable about the results reported here is that the SVA procedure for boosting the power of our instruments in the first stage was highly successful. Tests on the significance of the SVA generated instruments returned F-statistics ranging from 45.41 to 72.65 in the first stage predicting mother's height and 35.06 to 66.65 in the first stage predicting mother's skinfold thickness.[21] Conversely, those same F-statistics using the full set of instruments in the first stage range from 1.129 to to 1.202 and 0.991 to 1.298, respectively. Therefore SVA appears to hold much promise as a way of addressing situations in which instruments are many but weak.

[19] In environments where there are no adverse influences on growth, genetic differences results in a worldwide height variability of about one cm in five-year-old children (cite).

[20] A senstive period for the development of a particular trait or characteristic such as health is a stage of development during which the production of that trait is most sensitive to inputs.

[21] First-stage F-statistics vary because controls and sample size slightly differs at each childhood stage. Sample size differs due to attrition and interruption.

6.1 Transmission of Mother-to-Child Health

Table 2 reports the estimated transmission coefficients of mother's health stock (proxied her baseline height in cm) and health flow (proxied by her baseline skinfold thickness in cm) to child health stock (proxied by height measures). Height-for-age z-scores proxy for child health stock at each age examined.[22] With regard to the child's height, the transmission of mother-to-child health does not appear to be statistically or qualitatively significant at birth. However, the transmission of mother's health stock is significant at every other observed age. Increasing mother's height by 1 cm increases her child's HAZ by 0.06, 0.08, 0.10, 0.11, and 0.08 standard deviations at ages 1, 2, 8, 11, and 15, respectively.

The transmission of mother's health flow is significant in early childhood but ceases to have a statistically significant effect after age eight. Increasing mother's baseline (i.e., during pregnancy) skinfold thickness by 1 cm increases her child's HAZ by 0.08 standard deviations at ages 1 and 2 and by 0.11 standard deviations at age 8. The effect of mother's skinfold on child height at ages 11 and 15 is not statistically significant.

Table 3 reports the estimated transmission coefficients of mother's health stock and health flow to child health flow (proxied by child weight measures). Child health flow at birth is proxied by birth weight in grams. Weight-for-height z-scores (WHZ) proxy child health flow at ages 1 and 2. Finally, BMI-for-age z-scores (BMIZ) proxy child health flow at ages 8, 11, and 15. Unlike with birth length, both mother's health stock and health flow are significant determinants of child birth weight. Increasing maternal height and skinfold thickness by one cm increases birth weight by 26.45 and 30.04 grams, respectively. The transmission of maternal health appears to persist through early childhood. Increasing maternal height by 1 cm increases child WHZ by 0.04 and 0.05 standard deviations at ages 1 and 2, respectively. The corresponding effects of increasing skinfold thickness by 1 cm are approximately 0.06 standard deviations at each age. Increasing maternal height additionally increases child BMIZ at age 8 by 0.06 standard deviations. Maternal skinfold thickness does not exhibit a statistically significant effect on child weight at ages 8, 11, and 15.

Figure 4 graphically depicts the results reported in Tables 2 (Panel A of Figure 4) and 3 (Panel B of Figure 4). Birth weight is excluded from Panel B of Figure 4 as its scale, grams, differs from that of each of the other child health outcomes, which are measured in z-score standard deviations. There are a few things worth noting in these results. First, mother's health flow is a much less precise predictor of child health than her health stock. This is intuitive in that health flow, by definition, captures the mother's contemporaneous health state and is thus a less robust measure of her long-term health status. Given that our measure of health flow is skinfold thickness measured when our sample mothers were in their third trimester, it is a direct input into *in utero* health. Therefore, we would expect to see, as we do, that mother's third trimester health flow impacts health at birth and possibly even early childhood health. What it more surprising is that the transmission of mother's health flow at pregnancy continues into adolescence, particularly with regards to child height.

Second, although the transmission of maternal health to child weight is less precisely

[22]Recumbent length-for-age is used rather than height-for-age at birth, age one, and age two.

estimated as the child gets older, the transmission point estimate remains relatively stable at around 0.05 standard deviations across each childhood stage we observe. A measure of a child's health flow, such as weight, is sensitive to a wide range of biological and environmental inputs including nutrient intake, activity, and disease. The range of inputs an individual is exposed to increases as that individual ages. We would therefore expect this measure to be noisier at older ages. Consequently, it is unclear whether the lack of significance of the effect of maternal health on child weight at older ages is due to a zero-effect or due to lack of precision. Indeed the confidence bands around the effect (Panel B in Figure 4) increase substantially in later childhood and adolescence.

Finally, given that an association between maternal health and child health at birth is widely documented, we expected to find a transmission effect that is strongest at birth and in early childhood which subsequently diminishes as the child ages. However, instead we find a mother-to-child health transmission that persists into adolescence. We find a stable estimated transmission effect to child weight across ages (Panel B of Figure 4). Moreover, surprisingly, we find that the transmission effect to child height increases through childhood before decreasing slightly in adolescence (Panel A of Figure 4). In order to better understand the pattern of health transmission across childhood stages, we must think through some of the mechanisms through which it may operate.

While our estimated effects of maternal health on child health are causal, there are nonetheless a number of potential mechanisms underlying them. As noted, mother's health may transmit to child health directly through a biological mechanism or indirectly economic mechanisms operating through parenting ability or socio-economic status. Examining the persistence of the of the mother-to-child transmission can help to elucidate some of the mechanisms that may be at work.

6.2 Persistence of Mother-to-Child Health Transmission across Childhood Stages

In Figure 4 we find that mother's health transmits to her child not only only at birth but continues to affect child health through childhood and into adolescence. In this section we examine the persistence of the mother-to-child health transmission by attempting to pinpoint stages of transmission. For example, the effect of maternal health on child health at older ages may be due to a health transmission at birth. Then estimated effects at later ages may be due to the birth transmission and birth health's subsequent effect on later health. Certainly, ample evidence demonstrates that early health is an important determinant of later health. To explore this explanation, we first estimate Equation (3), controlling for birth weight—a widely used proxy for birth health.

Table 4 reports the estimated transmission coefficients to child health flow (proxied by weight) net of the health transmission at birth and Table 5 reports the corresponding effects on child health stock (proxied by HAZ). These results are also graphically depicted in Figure 5. In Table 4 we see that after controlling for birth weight, the transmission coefficients on both mother's height and skinfold thickness decrease in both significance and magnitude. Only the effect of mother's height on child WHZ at age two remains weakly significant after controlling for birth health. The second year of life is the period during which a child begins to transition to the family diet. Therefore,

the effect of mother's health stock on child weight at this age may reflect parenting ability or socio-economic contraints on her ability to provide a nutrious diet during this sensitive period. While we do not explicitly test for this explanation, it remains plausible.[23] The lack of significance of the effect of mother's health on child health flow at all other ages indicates that the maternal health transmission to this dimension of child health is largely due to the transmission to child's health flow at birth.

In Table 5 and Panel A of Figure 5 we see that after controlling for birth weight the effect of mother's health flow at pregnancy ceases to have a statistically significant effect on child height at any age after birth. Thus the primary mechanism through which mother's health flow during the late stages of pregnancy affects later childhood health appears to be through a transmission to health at birth.

Interestingly, the transmission of mother health stock to child health stock net of the birth effects (depicted in Table 2 and Panel A of Figure 5) look similar to that reported in Table 2 and Figure 4. The magnitude of the transmission at each age declines slightly indicating that the transmission at birth at least partly explains the later transmission of health stock. However, at each observed age after birth, the transmission coefficients on health stock remain statistically and clinically significant. Moreover, as previously, rather than observing an transmission that is strongest at birth that gradually diminishes as the child ages, the transmission effect increases in magnitude throughout childhood and then reduces slightly in adolescence.

To attempt to pinpoint the mechanism underlying this pattern we re-estimate Equation (3) controlling iteratively for each available child health lag. For example, to estimate the transmission of health stock at age 8, we first estimate (3) controlling for birth length. We then perform the same estimation controlling for age 1 HAZ rather than birth length. We then perform the same estimation controlling for age 2 HAZ rather than age 1 HAZ. Assuming that the realization of child HAZ at each age is a sufficient statistic for all realizations of HAZ that preceded it, this allows us to pinpoint the stage during which maternal height ceases to have a direct effect on child height. These results are reported in Table 6.

In Table 6 we see that the maternal height effects on age 1 and age 8 HAZ remain significant even after controlling any of the preceding child HAZ realizations. At age 1, the effect maternal health stock on child HAZ declines only modestly—from 0.0649 to 0.0586—once we control for birth length (the most recent child health lag). The first year of life is widely documented to be a sensitive period for linear growth. Thus the persistence of the health stock transmission at this age may be due to both a direct biological transmission as well as an indirect transmission through socio-economic mechanisms. The transmission effect on age 8 HAZ declines as more recent lags are included from a point estimate of 0.0999 with no lags included to 0.0444 with age 2 HAZ (the most recent child health lag available) included as a control. The sensitive period for linear growth is thought span from *in utero* to approximately 3 to 5 years of age. It is possible that the continued significant transmission we observe after controlling for age 2 HAZ may be due to growth during the latter end of this sensitive period. However, this is still a bit of a puzzle as maternal height does not statistically

[23]We will more explicitly test for this explanation in forthcoming analysis.

significantly affect age 2 HAZ when controlling for age 1 HAZ.

At every other observed age, we see that the magnitude of the health stock transmission declines as we control for more recent health lags. However, it does not decline to zero unless the most recent lag is controlled for. Figure 6 graphically depicts this pattern. Panels A and B correspond to Figures 4 and 5 which include no lag and a birth length lag, respectively. Panel C of Figure 6 controls for birth length at age 1, and age 1 HAZ for all other ages. Panel D controls for birth length at age 1, age 1 HAZ at age 2, and age 2 HAZ for all other ages. Panel E controls for birth length at age 1, age 1 HAZ at age 2, age 2 HAZ at age 8, and age 8 HAZ for all other ages. Finally, Panel F controls for birth length at age 1, age 1 HAZ at age 2, age 2 HAZ at age 8, age 8 HAZ at age 11, and age 11 HAZ at age 15.

In Figure 6 we see that as except for age 1 and age 8, the same general pattern exists as that depicted in Panel A of Figure 4 of marginal effects across ages but with declining magnitude as more recent lags are included. Once the most recent lag is included, the marginal effect of maternal height on child height at that age is no longer statistically different from zero. In Panel F of Figure 6, the most recent available lag is included as a predictor for HAZ at each age. At this point maternal height only has a statistically significant effect on child HAZ at age 1 and age 8.

Our findings of increasing marginal effects of maternal height on child height across ages suggest an accumulating (rather than diminishing) transmission of mother-to-child health stock. One possible explanation for the phenomenon is that increased mother height does not only translate into increased child height (both as genetic height and health stock) but also translates into increased growth velocity. This translates into a marginal child height advantage that increases with age. When we control for the most recent lagged child health, we are netting out the accumulated height advantage from previous periods, leaving only the effect on growth from the last period.

This finding is similar to the idea of dynamic complementarity discussed in Cunha and Heckman (2006). Dynamic complementarity describes the feature of human development where capabilities or traits produced in one stage of childhood raise the productivity of inputs into their production at later stages of childhood. For example, a child with higher initial cognitive ability is likely a more able learner and thus can produce more additional cognitive ability with the same inputs as a child with lower initial cognitive ability. Similarly, a healthier child's body may be better able to translate health inputs into increased additional health on the margin. Consequently, inputs into human development at different stages of childhood are complementary with earlier inputs making later inputs more productive on the intensive margin. If the transmission of maternal health stock can be thought of as an input into child health and continues to transmit throughout childhood, then we would expect to see accumulating levels of transmission as we observe in this sample.

7 Conclusion

In the paper, we bring together two important strands of literature. A large body of demonstrates the an association between mother and child health. Another substantial

body of literate documents the long-arm of early childhood in that early life health shocks affect a wide range of adult outcomes including adult health. However, very few studies exmaine the effects of an early life health shock on the next generation. In this paper we bring these two strands of literature together by tracing the impact of climate variation in the first few years of our sample women's lives to their adult health to the health of their children through adolescence. In doing so, we document the causal transmission of mother-to-child health across multiple stages of childhood into adolescence. We do so for measures of health stock and health flow in both our sample mothers and children. To obtain causality we exploit the unique climate conditions of the Philippines and use climate information from the time of our sample mothers' birth and in their early childhood.

We find that mother's health flow transmits to child health and the effects of this transmission can be felt into adolsecence, particularly with regards to child height. However, the mechanism of this transmission largely operates through the effect of mother's health flow on a child's health at birth and then the effect of birth health on later health. More surprisingly, we also find that mother's health stock transmits to child health stock through childhood and adolescence and this is net of its effect on birth weight. This transmission seems to be primarily due to a transmission of growth velocity, which leads to a mother-to-child health stock transmission that increases as the child ages rather than diminishes.

Finally, while our instruments satisfy the necessary exclusion restriction for a valid instrument. The are both many and weak. Therefore 2SLS estimates using our full set of instruments may be biased towards OLS estimates. Therefore, we propose a new method for addressing the problem of many but weak instruments using singular value analysis and machine learning methods. This method for boosting instrument power appears to hold much promise in that it increases first-stage F-statistics from around 1 to between 35 and 75.

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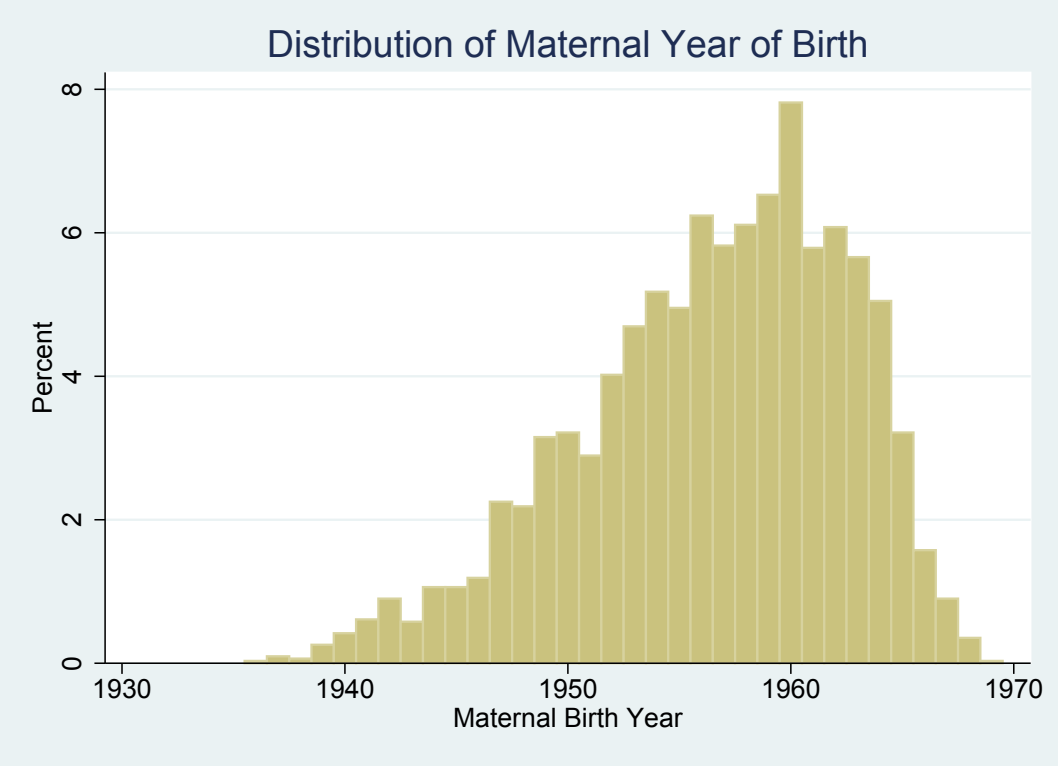
Figure 1: Maternal Year of Birth Distribution



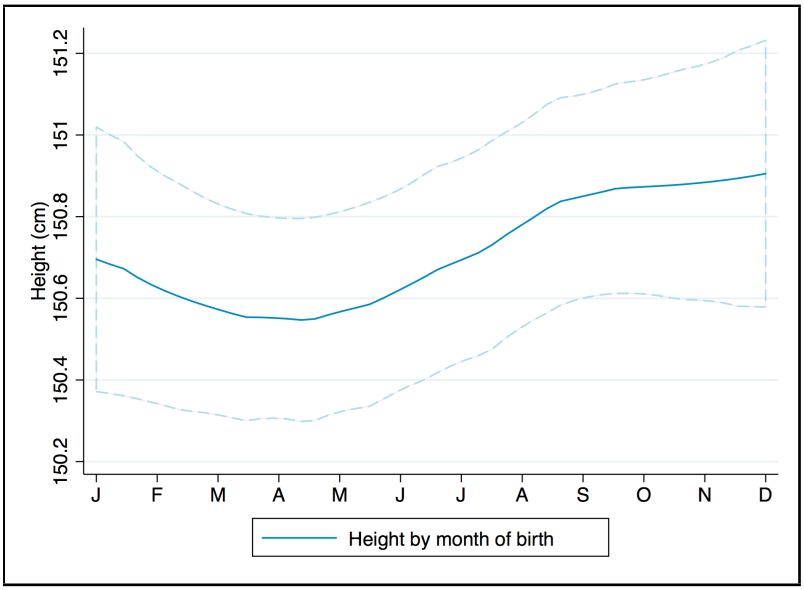


Figure 2: Mother's Height by Month of Birth

Figure 3: Maternal Month of Birth Distribution

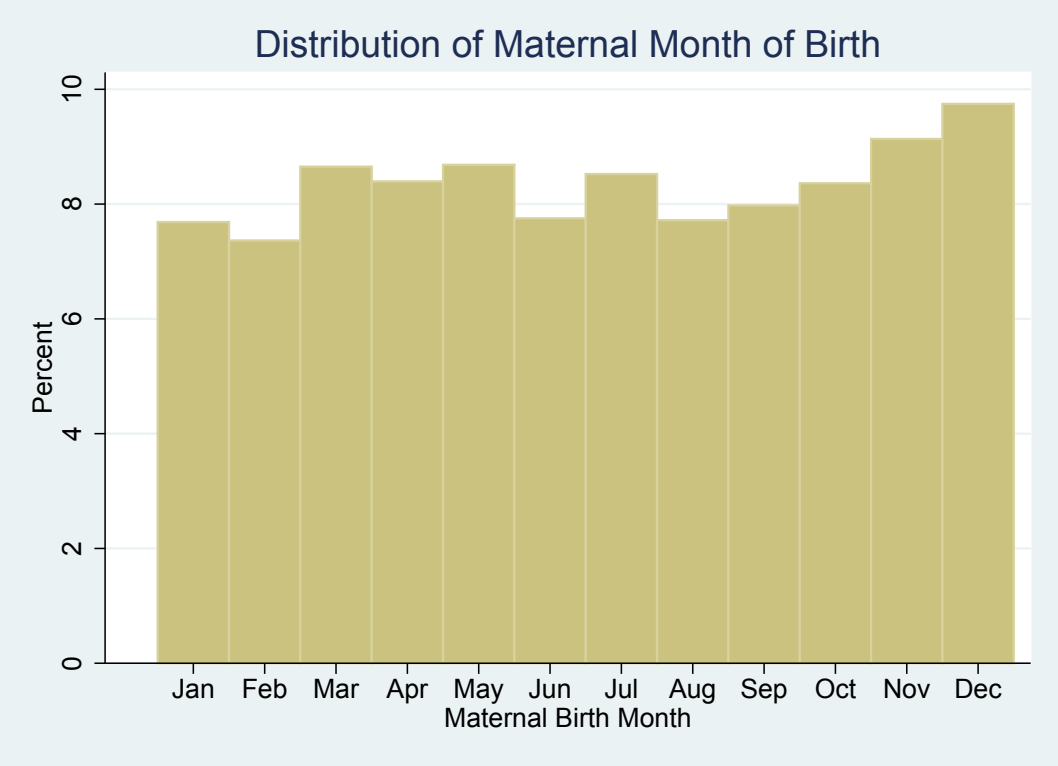


Figure 4: Marginal Effect of Maternal Health on Child Health across Ages

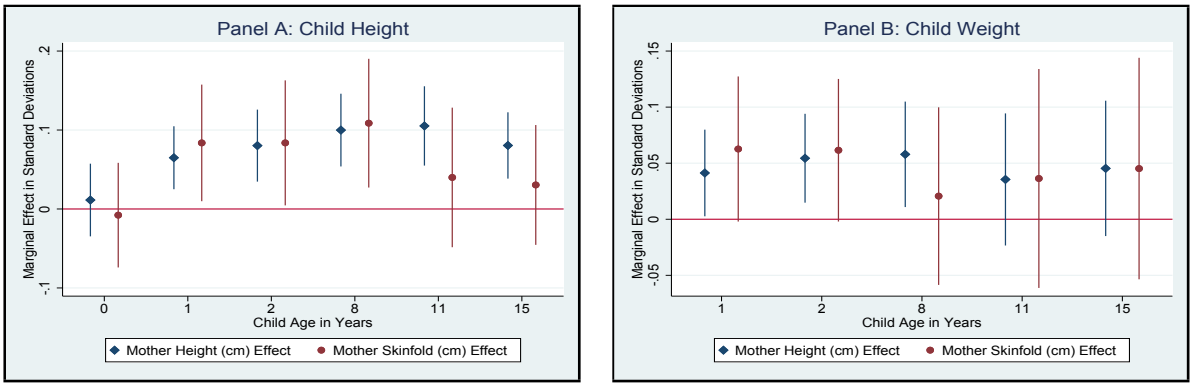


Figure 5: Marginal Effect of Maternal Health on Child Health Controlling for Birth Weight

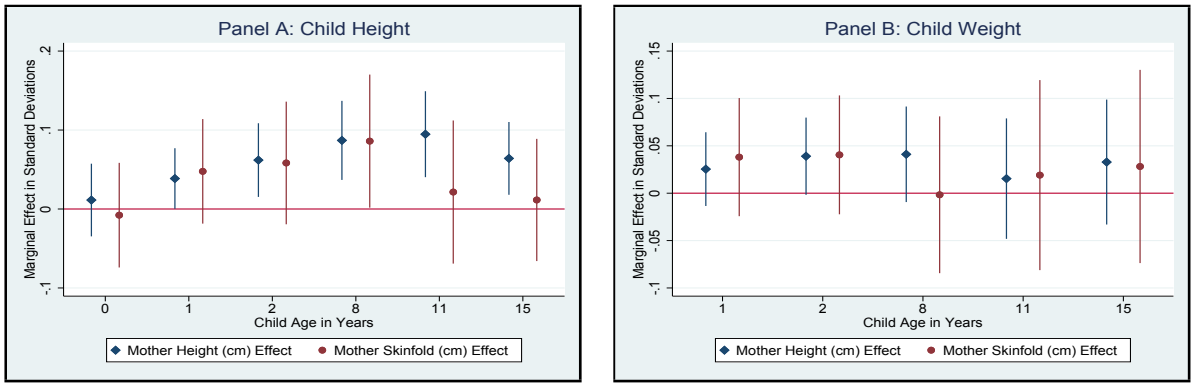


Figure 6: Marginal Effect of Maternal Health on Child Height—Controlling for Lagged Child Health Iteratively

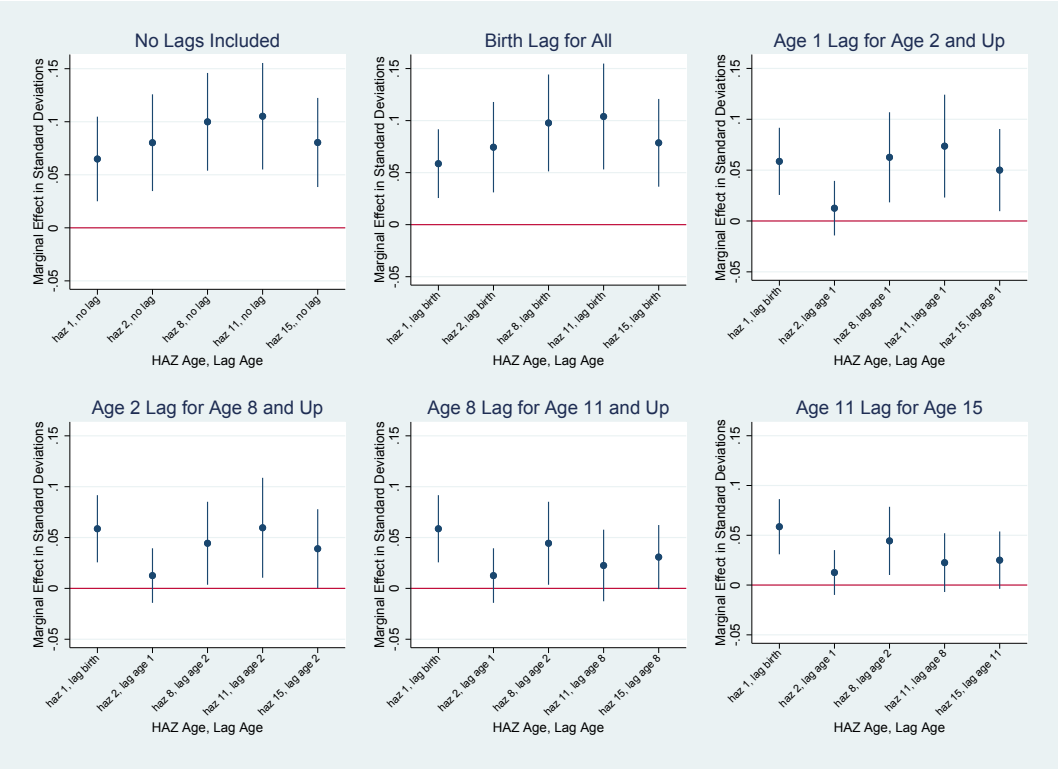


Table 1: Descritive Statistics

	Females	**Males**	**All**
Male	0.00	1.00	0.53
	(0.00)	(0.00)	(0.50)
Baseline Household Size	5.70	5.64	5.67
	(2.87)	(2.77)	(2.82)
Baseline Per Capita Household Income (Philippine Pesos)	2780.98	2822.49	2803.02
	(4263.69)	(4048)	(4149.94)
Health at Birth			
Height-for-Age Zscore	-0.47	-0.64	-0.56
	(1.08)	(1.08)	(1.08)
Birth Weight	2.97	3.03	3.00
	(0.43)	(0.46)	(0.45)
Weight-for-Height Zscore	-0.83	-0.87	-0.85
	(1.11)	(1.14)	(1.13)
Low Birth Weight	0.12	0.10	0.11
	(0.33)	(0.31)	(0.32)
Stunted in First 6 Months	0.59	0.63	0.61
	(0.49)	(0.48)	(0.49)
Health in First Year			
Height-for-Age Zscore	-1.21	-1.41	-1.32
	(0.98)	(1.02)	(1.00)
Weight-for-Height Zscore	-0.57	-0.58	-0.58
	(0.79)	(0.89)	(0.85)
Stunting Incidence in First Year	0.62	0.66	0.64
	(0.49)	(0.47)	(0.48)
Wasting Incidence in First Year	0.29	0.34	0.32
	(0.46)	(0.48)	(0.47)
Health in Second Year			
Height-for-Age Zscore	-2.23	-2.38	-2.31
	(1.08)	(1.14)	(1.11)
Weight-for-Height Zscore	-0.72	-0.73	-0.72
	(0.85)	(0.92)	(0.89)
Stunting Incidence in Second Year	0.73	0.78	0.76
	(0.44)	(0.41)	(0.43)
Wasting Incidence in Second Year	0.21	0.22	0.21
	(0.40)	(0.41)	(0.41)
Health in Year Eight			
Height-for-Age Zscore	-1.99	-2.08	-2.04
	(0.95)	(0.94)	(0.95)
Body Mass Index Zscore	-0.81	-0.81	-0.81
	(0.84)	(0.94)	(0.89)
Skinfold Measurement (cm)	7.27	6.34	6.78
	(1.95)	(1.99)	(2.02)
Subscapular Measurement (cm)	6.08	5.30	5.67
	(1.77)	(1.71)	(1.78)
Stunted at Age Eight	0.52	0.54	0.53
	(0.50)	(0.50)	(0.50)
Wasted at Age Eight	0.08	0.09	0.09
	(0.27)	(0.29)	(0.28)
Health in Year Eleven			
Height-for-Age Zscore	-1.93	-2.00	-1.96
	(1.10)	(0.97)	(1.03)
Body Mass Index Zscore	-1.02	-1.14	-1.09
	(1.05)	(1.13)	(1.09)
Skinfold Measurement (cm)	10.34	8.58	9.42
	(3.54)	(3.51)	(3.63)
Subscapular Measurement (cm)	8.34	6.74	7.50
	(3.42)	(3.04)	(3.33)
Stunted at Age Eleven	0.48	0.52	0.50
	(0.50)	(0.50)	(0.50)
Wasted at Age Eleven	0.17	0.20	0.18
	(0.38)	(0.40)	(0.39)

Table 2: Transmission of Maternal Health to Child Height—SVA Instrument

	Birth		Age 1		Age 2	
	(1)	(2)	(3)	(4)	(5)	(6)
Mother's height	0.0113		0.0649***		0.0802***	
	(0.0235)		(0.0203)		(0.0233)	
Mother's skinfold		-0.00779		0.0837**		0.0837**
		(0.0338)		(0.0377)		(0.0404)
IV F-stat	71.12	63.62	65.44	41.49	64.68	45.32
	Age 8		Age 11		Age 15	
Mother's height	0.0999***		0.105***		0.0804***	
	(0.0235)		(0.0256)		(0.0214)	
Mother's skinfold		0.109***		0.0399		0.0305
		(0.0416)		(0.0451)		(0.0387)
IV F-stat	50.87	33.87	46.95	33.56	43.47	31.35

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1 $^{+}$ p<0.15
Controls include current per capital income and household size, gender, mother's age, mother age cohorts, and birth month fixed effects, and baseline and current barangay fixed effects (which are equivalent at birth year).
For birth outcomes only an indicator for whether gestational age is in question is included.
Child outcomes are length-for-age z-scores from birth through age 2, and height-for-age z-scores for ages 8-15.

Table 3: Transmission of Maternal Health to Child Weight—SVA Instrument

	Birth		Age 1		Age 2	
	(1)	(2)	(3)	(4)	(5)	(6)
Mother's height	26.45***		0.0413**		0.0544***	
	(9.422)		(0.0197)		(0.0202)	
Mother's skinfold		30.04**		0.0627*		0.0616*
		(13.98)		(0.0330)		(0.0324)
IV F-stat	71.51	63.78	65.02	41.51	65.30	45.55
	Age 8		Age 11		Age 15	
Mother's height	0.0579**		0.0355		0.0454^{+}	
	(0.0240)		(0.0300)		(0.0308)	
Mother's skinfold		0.0206		0.0364		0.0452
		(0.0404)		(0.0498)		(0.0504)
Observations	2206	2206	2129	2129	2034	2034

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1 $^{+}$ p<0.15
Controls include current per capital income and household size, gender, mother's age, mother age cohorts, and birth month fixed effects, and baseline and current barangay fixed effects (which are equivalent at birth year).
For birth outcomes only an indicator for whether gestational age is in question is included.
Child outcomes are length-for-age z-scores from birth through age 2, and height-for-age z-scores for ages 8-15.

Table 4: Transmission of Maternal Health to Child Height Controlling for Birth Weight

	Birth		Age 1		Age 2	
	(1)	(2)	(3)	(4)	(5)	(6)
Mother's height	0.0113		0.0386**		0.0619***	
	(0.0235)		(0.0196)		(0.0238)	
Mother's skinfold		-0.00779		0.0477		0.0583^{+}
		(0.0338)		(0.0338)		(0.0396)
Mother's MUAC						
Birthweight(g)			0.000982***	0.00100***	0.000675***	0.000729***
			(0.0000573)	(0.0000574)	(0.0000693)	(0.0000686)
IV F-stat	71.12	63.62	61.33	39.67	60.89	43.56
	Age 8		Age 11		Age 15	
Mother's height	0.0869***		0.0947***		0.0641***	
	(0.0255)		(0.0278)		(0.0235)	
Mother's skinfold		0.0860**		0.0215		0.0114
		(0.0430)		(0.0462)		(0.0395)
Mother's MUAC						
Birthweight(g)	0.000331***	0.000397***	0.000280***	0.000451***	0.000362***	0.000477***
	(0.0000693)	(0.0000726)	(0.0000761)	(0.0000746)	(0.0000634)	(0.0000638)
IV F-stat	44.94	31.27	41.37	32.39	37.71	29.85

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1 $^{+}$ p<0.15
Controls include current per capital income and household size, gender, mother's age, mother cohorts, and birth month fixed effects, and baseline and current barangay fixed effects (which are equivalent at birth year).
For birth outcomes only an indicator for whether gestational age is in question is also included.
Child outcomes are birth length, height-for-age z-scores at ages 1-2, and BMI z-scores for ages 8-15.

Table 5: Transmission of Maternal Health to Child Weight Controlling for Birth Weight

	Birth		Age 1		Age 2	
	(1)	(2)	(3)	(4)	(5)	(6)
Mother's height	26.45***		0.0254		0.0390*	
	(9.422)		(0.0199)		(0.0208)	
Mother's skinfold		30.04**		0.0381		0.0405
		(13.98)		(0.0318)		(0.0320)
BirthWeight(g)			0.000619***	0.000624***	0.000552***	0.000580***
			(0.0000556)	(0.0000534)	(0.0000595)	(0.0000566)
IV F-stat	71.51	63.78	60.98	39.89	61.40	43.60
	Age 8		Age 11		Age 15	
Mother's height	0.0411^{+}		0.0154		0.0329	
	(0.0257)		(0.0324)		(0.0336)	
Mother's skinfold		-0.00165		0.0191		0.0282
		(0.0422)		(0.0512)		(0.0520)
BirthWeight(g)	0.000370***	0.000454***	0.000472***	0.000481***	0.000320***	0.000351***
	(0.0000705)	(0.0000682)	(0.0000898)	(0.0000833)	(0.0000871)	(0.0000837)
IV F-stat	44.94	31.27	41.37	32.39	37.71	29.85

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1 $^{+}$ p<0.15
Controls include current per capital income and household size, gender, mother's age, mother cohorts, and birth month fixed effects, and baseline and current barangay fixed effects (which are equivalent at birth year).
For birth outcomes only an indicator for whether gestational age is in question is also included.
Child outcomes are birth weight, weight-for-height z-scores at ages 1-2, and BMI z-scores for ages 8-15.

Table 6: Transmission of Maternal Health to Child Height Controlling for Lagged Height

	Birth	Age 1		Age 2			Age 8			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Mother's height	0.0113	0.0649***	0.0386**	0.0802***	0.0619***	0.0126	0.0999***	0.0869***	0.0626***	0.0444**
	(0.0235)	(0.0203)	(0.0196)	(0.0233)	(0.0238)	(0.0137)	(0.0235)	(0.0255)	(0.0226)	(0.0208)
BirthWeight(g)			0.000982***		0.000675***			0.000331***		
			(0.0000573)		(0.0000693)			(0.0000693)		
HAZ at Year 1						1.008***			0.523***	
						(0.0281)			(0.0436)	
HAZ at Year 2										0.543***
										(0.0365)
IV F-stat	71.12	65.44	61.33	64.68	60.89	57.30	50.87	44.94	43.97	42.97

	Age 11					Age 15				
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
Mother's height	0.105***	0.0947***	0.0736***	0.0596**	0.0225	0.0454	0.0641***	0.0500**	0.0390**	0.0308*	0.0250
	(0.0256)	(0.0278)	(0.0258)	(0.0251)	(0.0179)	(0.0308)	(0.0235)	(0.0206)	(0.0198)	(0.0161)	(0.0175)
BirthWeight(g)		0.000280***					0.000362***				
		(0.0000761)					(0.0000634)				
HAZ at Year 1			0.492***					0.468***			
			(0.0496)					(0.0413)			
HAZ at Year 2				0.516***					0.466***		
				(0.0441)					(0.0356)		
HAZ at Age 8					0.891***					0.667***	
					(0.0455)					(0.0385)	
HAZ at Age 11											0.597***
											(0.0315)
IV F-stat	46.95	41.37	41.83	39.34	38.38	43.47	37.71	38.74	37.52	38.88	34.77

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1
Controls include current per capital income and household size, gender, mother's age, mother cohorts, and birth month fixed effects, and baseline and current barangay fixed effects (which are equivalent at birth year).
For birth outcomes only an indicator for whether gestational age is in question is also included.
Child outcomes are length-for-age z-scores from birth through age 2, and height-for-age z-scores for ages 8, 11, and 15.

Appendix 1 Climate Instruments

Figures A1-A3 illustrate monthly averages for rainfall, temperature, and windspeed, respectively, around Cebu, Philippines. Figure A1 clearly shows that the rainiest months are March-December, or perhaps March-January. In Cebu, rice is grown in two cropping seasons — from various sources and some data exploration, it seems that planting begins around May, with the first harvest in late August or September, and that the second rice crop is planted shortly thereafter, with second harvest in late January or February.

Accordingly, we define monsoon months as May-December. This is the period when rice is growing, and rice yields most easily impacted by rainfall and temperature shocks. Our year-specific rainfall and temperature shock variables are therefore given as average rainfall (mm/day) and average temperature (degrees Celcius) over the course of May-December in any given year.

Figure A1: Monthly Average Rainfall

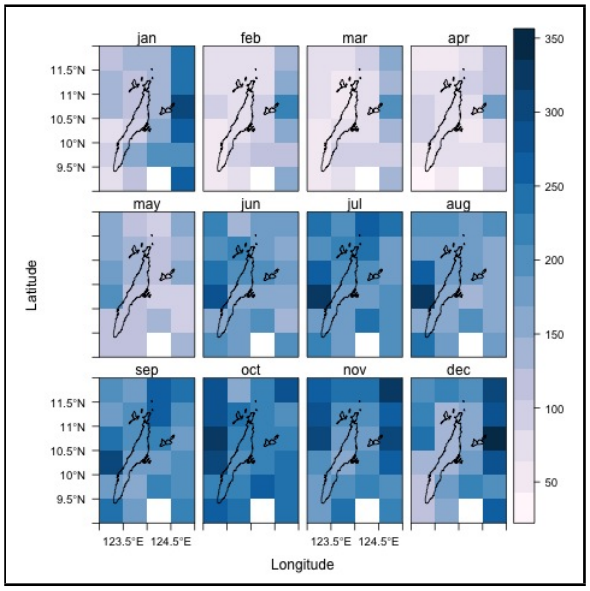


Figure A2: Monthly Average Temperature

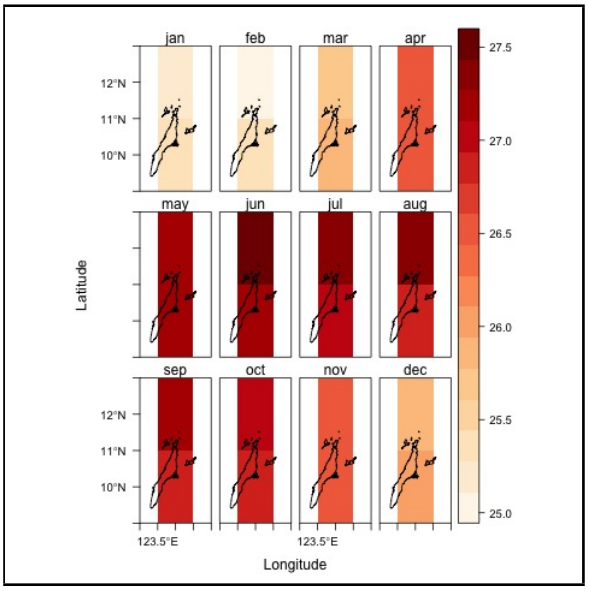
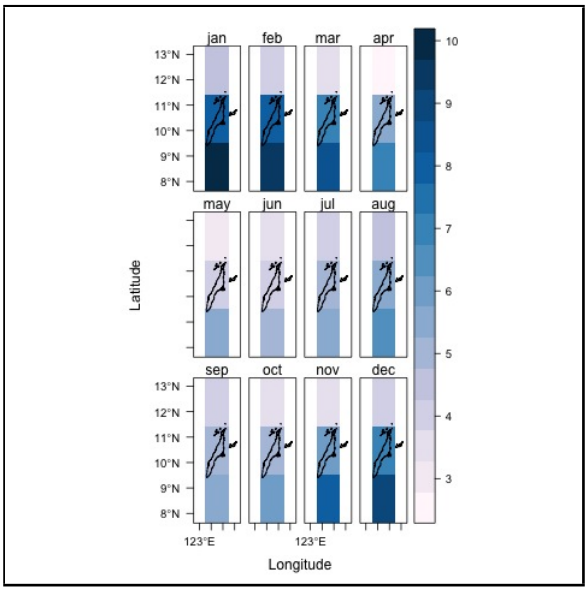


Figure A3: Monthly Average Windspeed



It is important to note that typically, climate shock variables are formulated as deviations from a region specific or grid specific mean. This is because most household datasets are spread over a wide geographic area, with varying levels of mean rainfall or temperature. Those varying climate averages may be correlated with other regional factors (e.g., high rainfall areas may be wealthier, or warmers areas more dependent on agriculture). If so, regressing a human outcome (e.g., educational achievement) on realized climate outcomes (e.g., yearly rainfall) may result in a spurious association (e.g., educational achievement may be higher in the region of a country with higher rainfall, simply because people are wealthier in that area). Grid-specific or region-specific deviations from long-run climate means are orthogonal to the long-run means themselves, and thus uncorrelated with other spatially-varying societal characteristics.

Because all women in our dataset are from Cebu, and because we do not know the specific area of Cebu in which they were born, our estimations do not suffer from this problem. That is, we create Cebu-specific rainfall and temperature realizations as area-weighted means of all grid cells that overlap Cebu. We cannot, therefore, estimate a spurious correlation between climate realizations and human outcomes due to spatially varying, correlated societal characteristics, because women in the north, south, and center of Cebu all receive the same, Cebu-average rainfall or temperature realizations. Otherwise stated, creating deviations from a long-run mean would simply entail subtracting the same mean from all realizations, since long-run means do not vary across mothers in our dataset.

However, we face a different form of spurious correlation. While women in our dataset were all born in close proximity to one another, they were born over a long period of years between the 1930s and the 1970s. We therefore de-trend rainfall and temperature realizations within the monsoon season by regressing these realizations on year and year squared, and subtracting non-linear monsoon trends (due perhaps to el niño or la niña) from the year-specific realizations.

Daily windspeed data is used to create two forms of variables. We proxy typhoon incidence in the 12 months before and after birth via month-specific maximum windspeeds. We also proxy for high winds or typhoons during the rice "heading" period, or directly before rice harvest, via maximum windspeeds in January and Febuary (the first harvest of the year) and maximum windspeeds in August and September (the second harvest in the year). Notably, maximum windspeeds in non-harvest months (November and December, or even December and January, rather than January and February) do not provide the same power in the first stage.

Appendix 2 Relationship between Climate Instruments and Maternal Health

Table B1: Effect of Windspeed in Months around Birth on Maternal Height and Skinfold Thickness

	(1) Mother's Height (cm)	(2) Mother's Skinfold (cm)
Max windspeed 12 mo prior to birth	0.00272 (1.28)	0.00104 (0.78)
Max windspeed 11 mo prior to birth	0.00124 (0.62)	-0.0000651 (-0.04)
Max windspeed 10 mo prior to birth	-0.00503*** (-2.59)	-0.00311** (-2.01)
Max windspeed 9 mo prior to birth	-0.00537*** (-2.58)	-0.00191 (-1.23)
Max windspeed 8 mo prior to birth	-0.00245 (-1.18)	-0.000222 (-0.14)
Max windspeed 7 mo prior to birth	0.00144 (0.71)	0.00148 (0.99)
Max windspeed 6 mo prior to birth	0.000393 (0.20)	0.000185 (0.12)
Max windspeed 5 mo prior to birth	-0.00000920 (-0.00)	-0.00109 (-0.70)
Max windspeed 4 mo prior to birth	-0.00257 (-1.26)	-0.00142 (-1.03)
Max windspeed 3 mo prior to birth	0.00240 (1.16)	0.00310* (1.80)
Max windspeed 2 mo prior to birth	0.00190 (0.87)	-0.000860 (-0.58)
Max windspeed 1 mo prior to birth	-0.00362* (-1.65)	-0.00102 (-0.63)
Max windspeed $(m/s)^2$ birth month	-0.0000593 (-0.03)	0.00262* (1.87)
Max windspeed 1 mo after birth	-0.000121 (-0.06)	-0.00215+ (-1.64)
Max windspeed 2 mo after birth	-0.00420** (-2.18)	0.00145 (0.89)
Max windspeed 3 mo after birth	0.00269 (1.33)	-0.00113 (-0.73)
Max windspeed 4 mo after birth	-0.000000335 (-0.00)	0.000226 (0.14)
Max windspeed 5 mo after birth	-0.000227 (-0.11)	-0.000711 (-0.49)
Max windspeed 6 mo after birth	0.00419** (2.14)	0.000660 (0.48)
Max windspeed 7 mo after birth	-0.000741 (-0.38)	-0.00000237 (-0.00)
Max windspeed 8 mo after birth	-0.00126 (-0.60)	0.000192 (0.13)
Max windspeed 9 mo after birth	0.00139 (0.75)	0.000420 (0.28)
Max windspeed 10 mo after birth	0.00461** (2.33)	0.00170 (0.97)
Max windspeed 11 mo after birth	0.000643 (0.31)	0.00126 (0.79)
Max windspeed 12 mo after birth	-0.000590 (-0.25)	0.000897 (0.55)
Constant	151.3*** (122.70)	12.09*** (13.51)
Observations	2991	2991
R^2	0.0316	0.0691

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1
Additional controls include current fixed effects for mother's month of birth and baseline barangay

Table B2: Effect of Early Life Harvest Season Wind Speed on Maternal Height and Skinfold Thickness

	(1) Mother's Height (cm)	(2) Mother's Skinfold (cm)
Monsoon high winds 2 years before birth	-0.0118***	0.000847
	(-3.98)	(0.39)
Monsoon high winds 1 years before birth	-0.00238	-0.00323
	(-0.82)	(-1.41)
(max) wndestr	-0.00482	-0.00639***
	(-1.24)	(-2.85)
Monsoon high winds 1 years after birth	-0.00496*	0.000556
	(-1.74)	(0.19)
Monsoon high winds 2 years after birth	-0.00494+	-0.00221
	(-1.49)	(-1.03)
Monsoon high winds 3 years after birth	-0.00991***	-0.00589**
	(-2.78)	(-2.45)
Monsoon high winds 4 years after birth	-0.00148	-0.00239
	(-0.46)	(-0.90)
Monsoon high winds 5 years after birth	0.00256	-0.00549**
	(0.57)	(-2.24)
Constant	156.9***	16.15***
	(92.36)	(11.74)
Observations	2991	2991
R^2	0.0248	0.0662

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1
Additional controls include current fixed effects for mother's month of birth and baseline barangay

Table B3: Effect of Early Life Monsoon Precipitation and Temperature on Maternal Height and Skinfold Thickness

	(1) Mother's Height (cm)	(2) Mother's Skinfold (cm)
Monsoon rain 2 years before birth	0.000439	-0.00126
	(0.13)	(-0.49)
Monsoon rain 1 years before birth	-0.00397	-0.000833
	(-1.13)	(-0.33)
Monsoon rain (avg mm/mo May-Dec) in birth year	0.00467	0.00399^{+}
	(1.34)	(1.49)
Monsoon rain 1 years after birth	-0.00313	0.000522
	(-0.73)	(0.17)
Monsoon rain 2 years after birth	0.00846**	0.00331
	(2.22)	(1.14)
Monsoon rain 3 years after birth	0.0105***	0.00260
	(3.10)	(1.05)
Monsoon rain 4 years after birth	-0.00305	-0.00325
	(-0.95)	(-1.36)
Monsoon rain 5 years after birth	0.00603*	0.00589**
	(1.80)	(2.32)
Monsoon temp 2 years before birth	0.824	-0.303
	(0.99)	(-0.45)
Monsoon temp 1 years before birth	2.067**	0.645
	(2.53)	(1.03)
Monsoon temp (avg C May-Dec) in birth year	1.188	0.996
	(1.25)	(1.38)
Monsoon temp 1 years after birth	-0.452	-0.433
	(-0.56)	(-0.71)
Monsoon temp 2 years after birth	1.333^{+}	1.113*
	(1.59)	(1.78)
Monsoon temp 3 years after birth	2.316***	-0.0463
	(2.66)	(-0.08)
Monsoon temp 4 years after birth	1.760**	0.212
	(2.14)	(0.36)
Monsoon temp 5 years after birth	-0.111	0.759
	(-0.13)	(1.22)
Constant	-91.03	-68.26
	(-1.04)	(-1.15)
Observations	2991	2991
R^2	0.0296	0.0693

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1
Additional controls include current fixed effects for mother's month of birth and baseline barangay

Table B4: First Stage Results

	Birth		Age 1		Age 2	
	(1)	(2)	(3)	(4)	(5)	(6)
Optimal SVA for Mother's Height	0.952***		0.975***		0.999***	
	(0.113)		(0.121)		(0.124)	
Optimal SVA for Mother's Skinfold		0.989***		0.874***		0.932***
		(0.124)		(0.136)		(0.138)
Observations	2981	2981	2592	2592	2455	2455
R^2	0.0673	0.102	0.0648	0.0954	0.0637	0.0955
	Age 8		Age 11		Age 15	
	(1)	(2)	(3)	(4)	(5)	(6)
Optimal SVA for Mother's Height	0.977***		0.939***		0.943***	
	(0.137)		(0.137)		(0.143)	
Optimal SVA for Mother's Skinfold		0.862***		0.864***		0.864***
		(0.148)		(0.149)		(0.154)
Observations	2206	2206	2129	2129	2034	2034
R^2	0.129	0.195	0.138	0.210	0.150	0.216

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1
Controls include current per capital income and household size, gender, mother's age, mother cohorts,
and birth month fixed effects, and baseline and current barangay fixed effects (which are equivalent at birth year).
For birth outcomes only an indicator for whether gestational age is in question is also included.
Child outcomes are length-for-age z-scores from birth through age 2, and height-for-age z-scores for ages 8, 11, and 15.

Appendix 3 Choosing an Optimal Instrument via Singular Value Analysis

The ordinary least squares (OSL) estimator minimizes $||\mathbf{X}\beta - \mathbf{y}||^2$, where $\mathbf{X}$ is an $m \times n$ matrix, β is an $m \times 1$ matrix, and $\mathbf{y}$ is an $m \times 1$ matrix. Yet singular value analysis (SVA) solves the same problem, by minimization an equivalent distance problem in a rotated space.

First, using singular value decomposition (SVD), we may decompose $\mathbf{X}$ as follows

$$\mathbf{X} = \mathbf{USV^T}$$

where $\mathbf{U}$ is an orthogonal $m \times n$ matrix, $\mathbf{S}$ is a diagonal $n \times n$ matrix with successive, positive and non-decreasing entries, and $\mathbf{V^T}$ is an orthogonal $n \times n$ matrix.

Because $\mathbf{U}$ is orthogonal, $\mathbf{U}^T$ is also orthogonal, and both are therefore distance preserving under multiplication. Thus,

$$\begin{aligned} ||\mathbf{X}\beta - \mathbf{y}||^2 &= ||\mathbf{U}^T(\mathbf{X}\beta - \mathbf{y})||^2 \\ &= ||\mathbf{U}^T(\mathbf{USV^T}\beta - \mathbf{y})||^2 \\ &= ||\mathbf{SV^T}\beta - \mathbf{U}^T\mathbf{y})||^2 \\ &= ||\mathbf{S}\gamma - \mathbf{g}||^2 \end{aligned}$$

where the third line follows from the fact that $\mathbf{U}$ is an orthogonal matrix, and we define $\gamma = \mathbf{V^T}\beta$ and $\mathbf{g} = \mathbf{U}^T\mathbf{y}$, both $n \times 1$ matrices.

To minimize $||\mathbf{X}\beta - \mathbf{y}||^2$, we can therefore choose a $\hat{\gamma}$ to minimize $||\mathbf{S}\gamma - \mathbf{g}||^2$. The original parameter vector $\hat{\beta}$ is calculated as $\mathbf{V}\hat{\gamma}$.

Predicted outcome $\hat{\mathbf{y}}$ may be equivalently calculated as either $\mathbf{X}\hat{\beta}$ or $\mathbf{US}\hat{\gamma}$, since $\mathbf{X}\beta \simeq \mathbf{y}$ and $\mathbf{S}\hat{\gamma} \simeq \mathbf{g} = \mathbf{U}^T\mathbf{y}$. The residual $\hat{\mathbf{r}}$ may be equivalently calculated as either $\mathbf{y} - \mathbf{X}\hat{\beta}$ or $\mathbf{U}(\mathbf{g} - \mathbf{S}\hat{\gamma})$ since $\mathbf{y} - \mathbf{X}\beta = \mathbf{Ug} - \mathbf{USV^T}\beta = \mathbf{U}(\mathbf{g} - \mathbf{S}\gamma)$.

However, because $\mathbf{S}$ holds successively non-increasing diagonal values, the elements of $\hat{\gamma}$ become increasingly insignificant to $\hat{\beta} = \mathbf{V}\hat{\gamma}$. The candidate solution $\gamma^{(k)}$ may therefore be considered, where each element of $\gamma^{(k)}$ is identical to that of the full solution $\hat{\gamma}$ up until the k'th element, and all subsequent elements are zero, as below.

$$\gamma^{(k)} = \begin{bmatrix} \hat{\gamma}_1 \\ \vdots \\ \hat{\gamma}_k \\ 0 \\ \vdots \\ 0 \end{bmatrix}$$

In many cases the candidate solution $\gamma^{(k)}$, for some particular $k < n$, minimizes mean squared forecasting error (MSFE) better than the full solution γ. This is because the rows of matrix $\mathbf{V}$ hold "averages" for the columns of matrix $\mathbf{X}$, but with each row explaining less and less of the variation in $\mathbf{X}$. One might imagine that, past some particular column k, the rows of matrix $\mathbf{V}$ hold only

sample-specific variation in $\mathbf{X}$, rather than variation that can be predicted out of sample. In other words, a solution vector $\hat{\beta}$ that captures such variation is over-fitting the model, leading to an increase in R^2 within sample, but also an increase in MSFE out of sample. A solution vector $\hat{\beta}$ that captures only the variation within the first k vectors of $\mathbf{V}$ will better minimize MSFE.

Often, k is chosen based on the condition number of the implied system, i.e., the instability of the solution. We instead use group-wise cross-validation to choose k, dividing the sample into 100 test/training groups, and measuring MSFE for each k in each of those 100 trials. We then choose the k that minimizes forecast error best, on average. It is worth nothing that this k is larger and results in far better predicts and F-statistics in the first stage, than a k based on solution instability.

Appendix 4 Comparing Transmission Estimates across Estimation Methods

Table B5: Regression of Child Height Measures on Mother's Health across Estimators

	Height Outcomes at Birth									
	(1) OLS	(2) OLS	(3) 2SLS	(4) 2SLS	(5) LIML	(6) LIML	(7) PCA	(8) PCA	(9) SVA	(10) SVA
Mother's height	0.0333***		0.000732		-0.0936*		-0.303		0.0113	
	(9.22)		(0.03)		(-1.82)		(-0.76)		(0.48)	
Mother's skinfold		0.0217***		-0.0469		-0.143***		-0.134		-0.00779
		(4.59)		(-1.54)		(-2.71)		(-1.32)		(-0.23)
Observations	2981	2981	2981	2981	2981	2981	2981	2981	2981	2981
R^2	0.171	0.153	0.148	0.0998	.	.	.	.	0.161	0.143
IV F-stat			1.157	1.297	1.157	1.297	0.567	1.414	71.12	63.62
	Height Outcomes at Age 1									
Mother's height	0.0583***		0.0586***		0.0516		-0.372		0.0649***	
	(16.46)		(2.94)		(0.58)		(-1.04)		(3.19)	
Mother's skinfold		0.0400***		0.0388		-2.620		0.116		0.0837**
		(8.24)		(1.27)		(-0.22)		(0.93)		(2.22)
Observations	2592	2592	2592	2592	2592	2592	2592	2592	2592	2592
R^2	0.169	0.0996	0.169	0.0996	0.168	.	.	0.0168	0.168	0.0721
IV F-stat			1.150	0.988	1.150	0.988	0.479	1.326	65.44	41.49
	Height Outcomes at Age 2									
Mother's height	0.0696***		0.0577**		-0.0950		-0.436		0.0802***	
	(16.81)		(2.55)		(-0.88)		(-1.30)		(3.45)	
Mother's skinfold		0.0524***		0.0478		-0.0217		0.165		0.0837**
		(9.16)		(1.48)		(-0.16)		(1.15)		(2.07)
Observations	2455	2455	2455	2455	2455	2455	2455	2455	2455	2455
R^2	0.188	0.118	0.185	0.117	.	0.0574	.	.	0.186	0.107
IV F-stat			1.156	1.160	1.156	1.160	0.582	1.738	64.68	45.32
	Height Outcomes at Age 8									
Mother's height	0.0647***		0.0716***		0.205*		-0.0475		0.0999***	
	(16.43)		(3.83)		(1.91)		(-0.23)		(4.25)	
Mother's skinfold		0.0503***		0.0878***		0.318***		0.117		0.109***
		(8.40)		(3.01)		(2.86)		(1.63)		(2.61)
Observations	2206	2206	2206	2206	2206	2206	2206	2206	2206	2206
R^2	0.270	0.195	0.269	0.177	.	.	.	0.138	0.238	0.151
IV F-stat			1.135	1.046	1.135	1.046	0.710	1.564	50.87	33.87
	Height Outcomes at Age 11									
Mother's height	0.0623***		0.0663***		-0.0130***		0.183		0.105***	
	(14.40)		(3.30)		(-9.09)		(0.67)		(4.11)	
Mother's skinfold		0.0555***		0.0702**		1.236		0.168*		0.0399
		(8.33)		(2.25)		(1.03)		(1.66)		(0.89)
Observations	2129	2129	2129	2129	2129	2129	2129	2129	2129	2129
R^2	0.258	0.206	0.257	0.204	0.136	.	.	0.0733	0.218	0.204
IV F-stat			1.202	1.083	1.202	1.083	0.795	1.289	46.95	33.56
	Height Outcomes at Age 15									
Mother's height	0.0685***		0.0609***		0.0330		-0.0848		0.0804***	
	(19.35)		(3.76)		(0.93)		(-0.86)		(3.76)	
Mother's skinfold		0.0341***		0.0508*		0.132*		0.122		0.0305
		(6.47)		(1.88)		(1.89)		(1.58)		(0.79)
Observations	2034	2034	2034	2034	2034	2034	2034	2034	2034	2034
R^2	0.305	0.170	0.303	0.166	0.264	0.0208	.	0.0485	0.300	0.170
IV F-stat			1.146	1.029	1.146	1.029	0.857	1.219	43.47	31.35

Controls include current per capital income and household size, gender, mother's age, mother cohorts, and birth month fixed effects, and baseline and current barangay fixed effects (which are equivalent at birth year).
For birth outcomes only an indicator for whether gestational age is in question is also included.
Child outcomes are length-for-age z-scores from birth through age 2, and height-for-age z-scores for ages 8, 11, and 15.
Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table B6: Regression of Child Weight Measures on Mother's Health across Estimators

	Weight Outcomes at Birth									
	(1) OLS	(2) OLS	(3) 2SLS	(4) 2SLS	(5) LIML	(6) LIML	(7) PCA	(8) PCA	(9) SVA	(10) SVA
Mother's height	12.61***		23.97***		93.99***		21.13		26.45***	
	(8.28)		(2.65)		(2.79)		(0.65)		(2.81)	
Mother's skinfold		14.13***		18.07		51.99		-30.33		30.04**
		(6.93)		(1.54)		(1.37)		(-0.98)		(2.15)
Observations	2983	2983	2983	2983	2983	2983	2983	2983	2983	2983
R^2	0.218	0.212	0.202	0.211	.	0.117	0.209	0.0807	0.194	0.195
IV F-stat			1.160	1.299	1.160	1.299	0.579	1.437	71.51	63.78
	Weight Outcomes at Age 1									
Mother's height	0.01000***		0.0321*		0.137**		-0.482		0.0413**	
	(3.17)		(1.72)		(2.53)		(-0.98)		(2.09)	
Mother's skinfold		0.0265***		0.0372		0.405		-0.205		0.0627*
		(5.79)		(1.38)		(1.34)		(-0.87)		(1.90)
Observations	2588	2588	2588	2588	2588	2588	2588	2588	2588	2588
R^2	0.0517	0.0616	0.0340	0.0594	.	.	.	.	0.0162	0.0364
IV F-stat			1.144	0.991	1.144	0.991	0.463	1.344	65.02	41.51
	Weight Outcomes at Age 2									
Mother's height	0.0239***		0.0405**		0.0852**		-0.134		0.0544***	
	(6.91)		(2.11)		(2.20)		(-1.21)		(2.69)	
Mother's skinfold		0.0348***		0.0227		-0.0247		0.0103		0.0616*
		(6.91)		(0.86)		(-0.41)		(0.16)		(1.90)
Observations	2456	2456	2456	2456	2456	2456	2456	2456	2456	2456
R^2	0.0799	0.0821	0.0710	0.0796	.	0.0212	.	0.0717	0.0496	0.0698
IV F-stat			1.170	1.162	1.170	1.162	0.607	1.754	65.30	45.55
	Weight Outcomes at Age 8									
Mother's height	0.00537		0.0405**		0.0944***		-0.00645***		0.0579**	
	(1.29)		(2.04)		(2.75)		(-5.83)		(2.41)	
Mother's skinfold		0.0350***		0.0171		-0.0411		-0.117		0.0206
		(5.89)		(0.60)		(-0.68)		(-1.14)		(0.51)
Observations	2206	2206	2206	2206	2206	2206	2206	2206	2206	2206
R^2	0.113	0.129	0.0768	0.125	.	0.0456	0.109	.	0.0324	0.126
IV F-stat			1.135	1.046	1.135	1.046	0.710	1.564	50.87	33.87
	Weight Outcomes at Age 11									
Mother's height	0.00864*		0.0194		0.0528		-0.00725***		0.0355	
	(1.77)		(0.85)		(1.14)		(-4.83)		(1.18)	
Mother's skinfold		0.0585***		0.0642*		0.0926		-0.170		0.0364
		(7.68)		(1.91)		(1.14)		(-0.76)		(0.73)
Observations	2129	2129	2129	2129	2129	2129	2129	2129	2129	2129
R^2	0.147	0.178	0.145	0.177	0.109	0.167	0.142	.	0.133	0.173
IV F-stat			1.202	1.083	1.202	1.083	0.795	1.289	46.95	33.56
	Weight Outcomes at Age 15									
Mother's height	0.00559		0.0143		0.0884		-0.365		0.0454	
	(1.11)		(0.64)		(1.20)		(-1.00)		(1.47)	
Mother's skinfold		0.0371***		0.0268		-0.367		0.198		0.0452
		(5.10)		(0.79)		(-1.03)		(0.83)		(0.90)
Observations	2034	2034	2034	2034	2034	2034	2034	2034	2034	2034
R^2	0.148	0.161	0.146	0.160	0.00631	.	.	.	0.115	0.160
IV F-stat			1.146	1.029	1.146	1.029	0.857	1.219	43.47	31.35

Controls include current per capital income and household size, gender, mother's age, mother cohorts, and birth month fixed effects, and baseline and current barangay fixed effects (which are equivalent at birth year).
For birth outcomes only an indicator for whether gestational age is in question is also included.
Child outcomes are length-for-age z-scores from birth through age 2, and height-for-age z-scores for ages 8, 11, and 15.
Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1