Intergenerational Transmission of Maternal Health: Evidence from Cebu, the Philippines

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We examine intergenerational transmission of health from mothers to children, using a unique dataset that tracks a cohort of children born in the 1980s in the Philippines. We provide causal estimates of the impact of maternal health on child health, and examine the persistence of this relationship from birth through adolescence. Our results suggest that mother's health continues to impact child health throughout childhood; previous estimates gauging transmission at a single point in time therefore underestimate the full impact of maternal health on child health. While the effect of maternal health on weight-related outcomes is relatively constant, loosing significance as children age, the effect of maternal health on child height-for-age z-score increases over childhood until adolescence at which point it falls. This pattern does not appear to be explained by the persistent effect of birthweight, by socioeconomic mechanisms or by parental inputs. Maternal health stock may shape the childhood growth trajectory until the point of puberty.

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1 Introduction

Robust evidence worldwide demonstrates that the socioeconomic status of parents is a powerful determinant of that of their children (Björklund, Jäntti and Lindquist, 2009; Behrman et al., 2017; Mulder et al., 2009) and a growing literature explores how the intergenerational transmission of human capital may underpin that relationship (Björklund, Lindahl and Plug, 2006; Lefgren, Sims and Lindquist, 2012). The impact of parental health on child health is one such mechanism, but this relationship is intertwined with the intergenerational transmission of multiple other dimensions of human capital. For example, schooling is also correlated across generations in both rich and poor countries, reflecting both heritable and nurtured aspects of cognitive and noncognitive abilities, and endogeneity to other aspects of welfare (Akresh et al., 2017; Black, Devereux and Salvanes, 2005; Björklund, Lindahl and Plug, 2006; Grönqvist, Öckert and Vlachos, 2017; Agüero and Ramachandran, 2018; Hertz et al., 2007). Parent education also impacts child health (Chou et al., 2010; Currie and Moretti, 2003), lower-income children are generally less healthy (Case, Lubotsky and Paxson, 2002; Currie and Lin, 2007), and poor women have more variable cortisol levels, with implications for child cognition, health, and education (Aizer, Stround and Buka, 2015). Less healthy children, on average, obtain less schooling, earn lower wages, and own less assets as adults (Bharadwaj, Lundborg and Rooth, 2018; Victora et al., 2008; Walker et al., 2007).

While a large, cross-disciplinary literature examines the intergenerational transmission of health, most papers focus on correlations. For instance, Bhalotra and Rawlings (2011) show that maternal height and body size is associated with child birthweight, risk of mortality, and child height-for-age z-score across the developing world. Victora et al. (2008) document the same in an extensive review of nutrition studies in poor countries. The association between maternal health and child health is stronger for mothers in poor socioeconomic conditions (Bhalotra and Rawlings, 2011; Currie and Moretti, 2007). Yet these associations could reflect a variety of causal processes, and only a few notable papers explore their underlying mechanisms, all in rich countries. Currie and Moretti (2007) show that low birthweight mothers are 50% more likely to have low birthweight children. Royer (2009) use twin fixed effects to find a similar transmission of birthweight with stronger effects for smaller born mothers. Thompson (2014) finds that morbidity in adopted US children increases with the presence of morbidity in both biological and adoptive parents. Biological transmission accounts for only 20-30% of baseline transmission for non-adoptive kids.

Our paper contributes to this literature in three key ways. First, we provide a causal estimate of the relationship between mother and child health in Cebu, the Philippines by instrumenting mother's health with random weather variation around the time of her birth and early childhood. Second, we examine the persistence of this relationship from birth through adolescence, which to our knowledge has not been done before. Finally, we examine and compare the persistent effect of maternal health across multiple dimensions of child health. This comparison leads to an interesting result: while the effect of maternal health on weight-related outcomes is relatively constant, loosing significance as children age, the effect of maternal health on child height-for-age z-score increases over childhood until adolescence at which point it falls. This pattern does not appear to be explained by the persistent effect of birthweight, by socioeconomic

mechanisms, or by parental inputs. Rather, evidence suggests that maternal health stock may shape childhood growth trajectory until the point of puberty.

Previous papers attempt to estimate a causal transmission of human capital across generations using sibling fixed effects, twin fixed effects, or adoption studies.[1] All use data from rich countries. The necessary data for such identification strategies is scarce in developing countries; moreover, recent evidence shows that twin birth is systematically related to maternal health.[2] We know of only one other attempt to causally identify health transmission in a developing country: Venkataramani (2011) uses rainfall, grandparent socioeconomic status and regional fixed effects to isolate health-based variation in height in Vietnam. However, it is unclear whether these instruments meet the exclusion restriction necessary for causality.

We obtain exogenous variation in maternal health by employing instruments that capture wind, rainfall and temperature variation relevant to early life health and agricultural conditions in Cebu, the Philippines in the 1940s-1960s, when our cohort of mothers were born or very young. This means that we identify health transmission off women whose adult health is influenced by early life weather, and thus estimate a local average treatment effect (LATE) (Imbens and Angrist, 1994). However, the prevalence of non-compliers is likely low for two reasons. First, 40% of our sample mothers list agriculture as their parents' primary occupation. Second, agriculture was highly protected prior to trade liberalization in the 1990s, resulting in little to no food imports, despite the Filipino diet's high reliance on rice (see Appendix Figures B.1 and B.2). Consequently, growing season weather patterns likely impacted food prices, thus affecting the food security of both agricultural and non-agricultural households. Hence, our LATE is likely close to an average treatment effect. Moreover, unlike much of the literature recording the long-term health effects of weather shocks (Maccini and Yang, 2009; Caruso and Miller, 2015; Deuchert and Felfe, 2015; Fuller, 2014; Kim et al., 2014; Currie and Rossin-Slater, 2013), we identify off of fairly minor weather variation at the intensive margin, rather than a single, dramatic event such as a hurricane, earthquake, or drought. Thus, our instruments are less likely to have lasting, generational impacts on income, infrastructure, or schooling outcomes outside of the pathway between mother and child health. Nonetheless, such contamination is impossible to rule out, and we address this possibility in our analysis.

Because we view maternal health transmission across many ages throughout childhood, we are uniquely positioned to investigate the factors that drive this transmission. These mechanisms matter for policy. Maternal health transmission is commonly measured at birth, through the association between maternal birthweight or height and child birthweight or risk of mortality (Thomas, Strauss and Henriques, 1990; Emanuel et al., 1992; Conley and Bennett, 2000; Currie and Moretti, 2007; Royer, 2009). If the process through which maternal health shapes child health occurs primarily in utero, then subsequent correlations between maternal health and child nutritional status — as observed by a variety of authors such as Subramanian et al. (2009), Venkataramani

[1] See Bharadwaj, Lundborg and Rooth (2018); Grönqvist, Öckert and Vlachos (2017); Aizer, Stroud and Buka (2015); Thompson (2014); Royer (2009); Björklund, Lindahl and Plug (2006); Currie and Moretti (2007).

[2] Across 72 countries, Bhalotra and Clarke (2018) find that twin birth is systematically correlated with mother health, health-related behaviors, and the prenatal environment.

(2011), Bhalotra and Rawlings (2011), and ourselves — could merely result from the persistent impact of health at birth. Our results indicate that while birthweight explains much of the persistent effect of maternal health on child weight, it only explains approximately 15—30% of the persistent transmission to child height, post-infancy. This result holds when we use sequential g-estimation to estimate a controlled direct effect (Acharya, Blackwell and Sen, 2016). We might alternatively hypothesize that persistent transmission occurs because smaller, less healthy mothers end up in lower-income households, have a lower cognitive or physical capacity for childcare, or have access to inferior inputs to child health. In our data, however, socioeconomic status and parental investments in child health similarly only explain 15—30% of the persistent relationship between maternal and child height. Together, these two mechanisms appear to account for approximately half of the transmission of maternal health to child height-for-age z-scores.

This leads us to explore a third mechanism behind the persistent impact of maternal health on child health – maternal constraints to childhood *growth*.[3] The effect of maternal constraints on intrauterine growth, postnatal growth potential and growth trajectories during pre-pubescent childhood is well-documented in human and animal studies (Gluckman and Harding, 1997; Gluckman and Hanson, 2007). And while it is possible that maternal constraints might impede child growth potential without implications for other dimensions of child health, this is unlikely since these same factors are associated with long-term health outcomes including cardiovascular problems, metabolic disorders, hormonal imbalance, and organ dysfunction (Chiarelli et al., 1999; Wu, Imhoff-Kunsch and Girard, 2012; Lazar et al., 2003; Gicquel, 2008; Mullis and Tonella, 2008). If maternal constraints shape childhood growth trajectories, we should see a change in maternal health transmission to child growth around the time of puberty onset—when the child transitions to a new growth regime dictated by a different set of growth regulating hormones. This is precisely what we see for both boys and girls. These results support, though cannot confirm, a growth trajectory mechanism.

Finally, we also estimate all results under three different first stage specifications. Because we have too many potential instruments, we need to reduce first-stage dimensionality. To do so, we employ a different technique in each specification: Lasso (*least absolute shrinkage and selection operator*) following Belloni et al. (2012) and Belloni, Chernozhukov and Hansen (2014), a combination of principal component analysis and Lasso (Winkelried and Smith, 2011; Bai and Ng, 2010; Ng and Bai, 2009; Amemiya, 1966), and a novel method of our own based on singular value analysis. Our preferred method, singular value analysis, should sort the signal of the instrumental variables from their associated noise by defining a cut-off point that separates noise from signal in a rotated space of orthogonal vectors (Abu-Shikhah and Elkarmi, 2011; Zhang et al., 2016; Lawson and Hanson, 1974). While these three methods of dimensionality reduction differ slightly in terms of first stage parsimony and the instruments retained, second stage estimates are virtually identical across the three methods.

Section 2 provides an overview on three mechanisms that potentially drive mother-to-child health transmission, with an emphasis on the determinants of childhood

[3]Maternal constraint is a term used to describe the non-genetic and non-pathological pathways through which a mother constrains the growth of her fetus. These pathways include maternal size, age, and parity as well as her physiological status, placental function, maternal diet and maternal programming.

growth. Section 3 provides an overview of our survey and climate data. Section 4 lays out our strategy for identifying maternal-to-child health transmission, as well as for exploring the mechanisms behind that transmission. Appendix A gives a detailed explanation of singular value analysis, which underlies our preferred method for first stage dimensionality reduction. Section 5 reports our results, primarily in figure form. Appendix B provides all additional results and robustness checks. Appendix C and Appendix D report core results using alternative methods of reducing first stage dimensionality. Finally, Section 6 concludes.

2 Background on Maternal Health Transmission and Child Growth Trajectory

In addition to estimating the causal transmission of health from mother to child, we explore the potential channels through which this transmission may operate. We suggest that maternal-child health transmission may work through three mechanisms: (1) the effect of maternal health on socioeconomic status or capacity for child care, (2) the effect of maternal health on fetal development, manifest through an impact on child birth health only, and (3) the effect of maternal health on the growth trajectory of a child, manifest through a persistent impact on child growth velocity. The first represents a socioeconomic and/or behavioral mechanism, while the latter two represent biological mechanisms. While we cannot isolate the causal impact of these mechanisms, we conduct analysis that hints at their respective roles.

First, mother's health may transmit to her child's through its effect on her socioeconomic status or her parenting ability. A large literature documents that health, particularly early childhood health, improves a broad range of adult outcomes including numerous dimensions of socioeconomic status and ability. For example, evidence finds that early childhood health affects later cognitive outcomes, school attainment as well as adult earnings and consumption (Black, Devereux and Salvanes, 2007; Bütikofer, Løken and Salvanes, forthcoming; Bharadwaj, Lundborg and Rooth, 2018). Additionally, adverse shocks in utero can result in lower educational attainment, lower cognitive ability, and reduced earnings (Almond, Currie and Herrmann, 2012; Aizer, Stround and Buka, 2015; Black et al., forthcoming). It is therefore plausible that improvements in mothers' early life health improves her adult socioeconomic status and/or parenting ability. This, in turn, may improve child health status by relaxing resource constraints on child health investments or simply making mothers more adept at these investments. Indeed, parental socioeconomic status and schooling are associated with improved child health outcomes such as child morbidity, height, and mental health (Case, Lubotsky and Paxson, 2002; Currie and Moretti, 2003; Currie and Lin, 2007; Chou et al., 2010; Behrman et al., 2017; Luca and Bloom, 2018). It is not surprising, therefore, that mothers' early life health is associated with child health (Currie and Moretti, 2007; Conley and Bennett, 2000; Royer, 2009), and that this association is mitigated by improved parental socioeconomic status (Bhalotra and Rawlings, 2013; Currie and Moretti, 2003).

Second, any ongoing transmission of mother-to-child health may primarily be due to the effect of mother's health on child *in utero* and birth health. We know that maternal

health impacts child birthweight and mortality (Black et al., 2013; Luca and Bloom, 2018), and ample evidence demonstrates the importance of birth health to later health (Almond and Currie, 2011; Aizer, Stround and Buka, 2015; Bütikofer, Løken and Salvanes, forthcoming; Bharadwaj, Lundborg and Rooth, 2018). It is therefore possible that the causal effect of mother's health on child's health in later childhood and adolescence is due to a transmission at birth and the subsequent effects of birth health on health at later ages.

Finally, maternal health can affect childhood health by impacting the factors that regulate growth velocity. Greater growth velocity will cause an increasing height advantage as children age. Growth velocity is set by growth potential and duration, both of which are determined by the epiphyseal growth plates, the cartilage plates found at the end of each long bone where growth takes place. Therefore, the growth plates, formed *in utero*, are one of the primary determinates of growth velocity throughout childhood and adolescence (Boersma and Wit, 1997; Ballock and O'Keefe, 2003; Gat-Yablonski, Yackobovitch-Gavan and Phillip, 2011).

The events regulating linear growth take place in three distinct phases: uterine/infant growth[4], childhood growth, and pubertal growth (Karlberg, 1987; Hindmarsh et al., 2008). During each phase, growth plates are targeted by distinct sets of mechanisms that include complex systems of hormones, genes, and proteins. Uterine/infant growth is primarily determined by maternal physiology, placental function, and maternal and child nutrition, which interact with key fetal growth regulating hormones, proteins, and genes such as insulin, IGF-I, and IGF-II (insulin-like growth factors I and II) (Gluckman and Harding, 1997; Mullis and Tonella, 2008; Hindmarsh et al., 2008). Childhood growth is, instead, primarily dependent on growth hormone (GH) (Karlberg, 1987; Mullis and Tonella, 2008). GH affects growth by binding to receptors on the growth plates and by orchestrating the production of IGF-I (Gat-Yablonski, Yackobovitch-Gavan and Phillip, 2009; Mullis and Tonella, 2008). Pubertal growth is also GH-dependent; however, it is also heavily regulated by sex hormones such as estrogen and androgen (Karlberg, 1987; Hindmarsh et al., 2008; Gluckman and Harding, 1997). Linear growth is therefore most dependent on GH after infancy and before pubertal growth begins.

The timing of these three growth phases is fairly predictable, but varies slightly by child. Childhood growth, dictated primarily by GH, begins between ages 6 months to 1 year (Karlberg, 1987; Hindmarsh et al., 2008). There is greater variation in the age where a child transitions to pubertal growth. This age also depends on sex. Girls begin pubertal growth between 9.5 and 14.5—after the onset of puberty around 8, but before menarche (i.e., the first period). The transition begins later for boys, generally around 11, again after the onset of puberty. For both girls and boys, a delay in the onset of puberty means a delay in the transition from childhood growth to pubertal growth (Abassi, 1998; Stang and Story, 2005).

Maternal health, physiology, and size during pregnancy can impact childhood growth trajectory due to the influence of the uterine environment on growth regulating factors. Evidence from human and animal studies indicates that these characteristics affect

[4]Growth in infancy is thought to be largely a continuation of fetal growth.

uterine growth rates through multiple mechanisms including by influencing the presence of GH, insulin, IGF-1 and IGF-II (Gluckman and Hanson, 2007; Gluckman, Hanson and Beedle, 2007), which, in turn, are associated with childhood growth, at least in part through an effect on sensitivity to GH and IGF-I during childhood. Adverse fetal environments and/or restricted uterine growth is also associated with numerous later health problems, including metabolic disorders, dysfunction and abnormal development of organs, cardiovascular disorders, and numerous problems related to hormonal imbalance (Chiarelli et al., 1999; Lazar et al., 2003; Gicquel, 2008; Mullis and Tonella, 2008; Wu, Imhoff-Kunsch and Girard, 2012). In fact, the fetal environment can induce many of these problems even without affecting birth size (Gluckman, Hanson and Beedle, 2007; Lillycrop and Burdge, 2012). Additionally the growth plates themselves are formed in utero, and maternal characteristics and well-being can affect the height of the growth plate, which impacts growth potential (Gat-Yablonski, Yackobovitch-Gavan and Phillip, 2011). Therefore it is possible for a mother's health to affect postnatal growth trajectories, with implications for attained height as well as long-term health.

3 Data

3.1 Cebu Longitudinal Health and Nutrition Survey

To explore mother-to-child transmission of health we exploit unique data collected by the Cebu Longitudinal Health and Nutrition Survey (CLHNS). The CLHNS is a rich longitudinal dataset collected from the island of Cebu, the Philippines that follows 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 in northeast Cebu and includes several urban, mountainous, and coastal regions.[5] 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. Three follow up surveys were additionally conducted during childhood and adolescence, in 1991-1992, 1994-1996, and 1998-2000. During these surveys, index children were approximately 8, 11, and 15 years old.

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 household, and his/her mother's household (if it is not the same as the child's). Anthropometric measures were taken in each wave for both mother and child. These data are therefore uniquely suited to investigate the relationship between maternal and child health.

Height-for-age z-score (HAZ) is often considered the gold standard for measuring early life health stock. Deficits in child HAZ typically signify long-term, cumulative health and/or nutrition shortfalls resulting in a failure to reach growth potential (WHO, 1995). Low child HAZ is associated with increased morbidity and mortality and reduced long-term health, and negatively associated with a wide range of socioeconomic outcomes such as educational achievement, cognitive ability, and adult economic productivity (Strauss and Thomas, 2008; Victora et al., 2008; Vogl, 2014; WHO, 1995).

[5] A barangay is the smallest administrative unit in the Philippines.

Table 1 provides summary statistics on baseline characteristics and health outcomes for sample children across observed ages, including HAZ.[6] Sample children are short, with HAZ hovering around two standard deviations below what is considered healthy. Approximately 64% of the sample experienced stunting (i.e., HAZ $\leq$ -2) at some point in their first year of life. Stunting rates rise to 76% at age 2 and then reduce to around 50% and below at ages 8, 11, and 15.[7]

Child weight is more sensitive to current nutritional inputs and morbidity than is child height, and is therefore used to capture health flow rather than health stock. Approximately 12% of the sample was born at a low birthweight. At later ages, weight-for-height z-score (WHZ) and body mass index z-score (ZBMI) capture how thin a child is for his/her height. Average WHZ and ZBMI range between a half and one standard deviation below what is considered healthy during all the ages examined. Wasting (i.e., WHZ, ZBMI $\leq$ -2) is most prevalent when the sample children are young. Approximately a third of children experience wasting at some point during their first year of life.[8]

The CLHNS also contain information on the pubertal development of the sample children. Using this information we approximate the timing of the onset of pubertal growth for both girls and boys. We then parse our 1994 sample of girls (who are approximately 11) into those who likely began pubertal growth and those who likely did not. We parse our 1998 sample of boys (who are approximately 15) in the same way. We parse girls around age 11 because girls generally begin pubertal growth around 9.5, and so heterogeneity in growth regime exists around age 11, while little variation exists at ages 8 or 15. We define a girl as likely to have begun pubertal growth in 1994 if menarche occurred by the 1994 interview date or within 2 years after that interview date (Abassi, 1998; Stang and Story, 2005). Information on the precise timing of menarche is gathered in both the 1994 and 1998 surveys.[9]

Unfortunately, the 1994 surveys do not collect information on pubertal indicators for boys, and so we cannot parse boys by pubertal development at age 11 as we do with girls. However, we can parse boys at age 15 as the 1998 survey gathered a series of indicators regarding male pubertal development. From this information we generate four indicators for male pubertal development at 15: an indicator for visible underarm hair, whether the boy shaves, whether his voice has changed, or has high levels of pubic hair (level four or five on the pubic development drawings).[10] If any of these variables

[6] World Health Organization reference data was used to standardize all anthropometric z-scores. For ages 1 and 2 HAZ is calculated using recumbent length rather than height.

[7] To contextualize, 2015-2016 and 2016 Demographic and Health Survey (DHS) data from Malawi and Nepal show that 12.7 and 12.4 percent of children are stunted at age 1, respectively, 25.3 and 31.8 percent at age 2, and 33.9 and 37.6 percent at age 5. However, these statistics are not strictly comparable. For the CLHNS data on children 1 and 2 we list a child as experiencing stunting or wasting if he or she was stunted or wasted at any of multiple visits during the 1st and 2nd year of life. The DHS data view each child only once at a given age.

[8] For comparison, again, 7.7 and 14.6 percent of children are wasted at age 1, in Malawi and Nepal. Wasting rates continue to hover between 10 and 20% at ages 2-15.

[9] Ample evidence demonstrate the reliability of recall data for menarche, especially when the window of time between recall and menarche is short (Adair, 2001; Koo and Rohan, 1997). Indeed, the two surveys exhibit high concordance in their recalled date of menarche.

[10] Sample boys looked at five drawings of male genital areas, each indicating a stage of pubertal development, and chose the drawing that best matched the thickness and spread of their pubic hair. The high

are equal to one, the sample boy is more likely to have already begun pubertal growth by the age of 15. However, it is worth noting that none of these indicators are as accurate an indicator of pubertal growth as is the timing of menarche for girls.

3.2 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 early childhood. 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 (see Appendix Figure B.3). Therefore, to investigate the effect of climatic conditions on maternal health, we use three geospatial datasets that all stretch 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 heavily 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 3-hour estimates within day, and across the cells that overlay the island of Cebu. We use gridded windspeed data, rather than modeling windspeed based on historical cyclone trajectories as do Anttila-Hughes and Hsiang (2013) or Hsiang (2010), for two reasons. Historical cyclone trajectories are less accurate prior to the advent of satellites.[11] Also, we wish to identify on high windspeeds that occur outside the presence of cyclones.

Our temperature data, also from the 20th Century Re-Analysis Project, contains global surface temperature estimates at a 2 degree spatial resolution in 6 hour intervals (4 observations per day). We again create daily average temperature for the island of Cebu. We procure precipitation data from the Global Precipitation Climatology Centre, also at ESRL. This dataset provides monthly, average precipitation estimates at a 0.5 degree spatial resolution, which we extract for the island of Cebu.[12] Appendix Figures B.4-B.7 illustrate monthly averages for rainfall, temperature, and windspeed over Cebu.

Using this gridded climate data, we create three types of weather shock instruments. First, we create instruments that capture potential morbidity due to high windspeeds or typhoons around the time of birth. The Philippines is one of the most intensely typhoon-exposed countries in the world. Typhoons around the time of birth increases 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, 2013; DOH, 2017; Salas, 2015). High winds not qualifying

level of pubic development would coincide with a Sexual Maturity Rating (SMR) of 4 or 5. Pubertal growth in males generally begins somewhere in SMR 3 (Stang and Story, 2005).

[11] They are even less accurate prior to the 1940s, when the United States began flying reconnaissance to detect cyclones. Even after 1940, such missions were rare in the Western Pacific basin.

[12] We weight grid cells by area overlaying the island of Cebu. For robustness, we also conduct analysis using: (1) only grid cells directly overlying Cebu city, and (2) grid cells overlaying the island of Cebu, surrounding islands, and the island of Luzon (known as the "rice bowl" because it produces most of the country's rice). While these two extractions produced nearly identical results (available upon request) to those reported in the paper, accuracy is best when we extract at the level of Cebu island only.

as typhoons can do the same. We therefore create a monthly measure of windspeed intensity by squaring the maximum windspeed observed in each month, between 1927 and 1970. We 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.

We next construct instruments that impact rice production during our sample mothers' early childhood. The Filipino diet is highly dependent on rice.[13] Prior to the 1990s when agricultural trade was liberalized, the country's rice was largely self-supplied. Appendix Figure B.2 illustrates that Filipino rice consumption was almost entirely supplied by own-production. Therefore, short-term fluctuations in rice production have implications for local food supply, household food security, family income, and early life health.

Our second set of instruments are thus constructed to capture wind damage to rice crops during early childhood. High windspeeds cause significant damage to rice crops, particularly during the reproductive and ripening phase that occurs during the two months prior to harvest (Blanc and Strobl, 2016).[14] Accordingly, high wind speeds during this period had potentially severe consequences for food security and the health of small children or even as-yet-unborn children. Our second set of instruments therefore consists of maximum squared windspeed ($(m/s)^2$) observed during the two months proceeding rice harvest for the two years prior to our sample mothers' birth year, their birth year, and for the five years following their birth.[15]

Additionally, temperature and precipitation during the monsoon months impact rice and other crop production. Thus, we create year-specific measures of growing conditions that include average monsoon season temperature, average monsoon season rainfall, and interactions between the two. Appendix Figure B.4 shows that the rainiest months are March-December. Accordingly, we define monsoon months as May-December. Our year-specific rainfall and temperature variables are therefore given as average rainfall (mm/day) and average temperature (degrees Celcius) over the course of May-December in any given year. Our third set of instruments consists of these measures for the two years prior to, for the year of, and for five years after the births of mothers in our dataset.[16] To avoid capturing spurious correlations between longer-term time trends in temperature and precipitation (e.g., driven by el nino or climate change) and time trends in mother's health, we de-trend monsoon variables by year, allowing for a quadratic shape (Christian and Barrett, 2018).

Our fourth and last set of instruments consists of mother's birth month dummies. Appendix Figure B.7 plots maternal height as a function of birth months,

[13] Between the years 1961 and 2017, Filipinos consumed between 0.13 (286.60) and 0.21 (467.38) metric tons (lbs.) per capita per year. See appendix Figure B.1.

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

[15] The reproductive/ripening phase for rice crops in Cebu occurs over the months of August and September (season 1) and January and February (season 2). We use the squared maximum wind speed in Cebu across these four months for our windspeed instruments. We also conducted analysis using squared maximum windspeeds for each crop cycle separately, resulting in two measures per year instead of one. This gave almost identical results.

[16] We include pre-harvest and monsoon season conditions for two years prior to the mother's birth to allow potential measurement error in mothers' recalled year of birth.

demonstrating seasonality in maternal health stock. This last set of instruments thus captures the predictable seasonality of health, driven by typhoons, monsoon seasons, disease vectors or other factors. Our sample mothers' birth months are fairly evenly distributed (Appendix Figure B.8), and so mothers born in a particular month or season are not overweighted in later estimations.

All together our four sets of instruments consist of 69 instruments in total. We are therefore concerned that we may have a many, weak instrument problem which will yield biased estimates in the traditional 2SLS environment. We discuss and and address this issue in Section 4.1. We also address and provide a series of checks for violations of the the exclusion restriction in Section 5.1.

4 Estimation Strategy

4.1 Identifying Transmission in a Data Rich Environment

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_{jv}, and the point estimate $\widehat{\gamma}^a_1$ therefore captures the coefficient of intergenerational transmission of health to the child at age a. We use mother's height, measured in cm at baseline when the women were in their third trimester of pregnancy, as a proxy for mother's health stock. However, because we identify transmission from the component of mother's height that is driven by early life shocks to health, we interpret γ^a_1 as the effect of mother's multidimensional health, not mother's height. Specifically, γ^a_1 can be interpreted as the effect of a change in any of the dimensions of mother's adult health that is associated with a 1 cm change in her height as a result of early life health realizations.

$$C^a_{ijv} = \gamma^a_1 M_{jv} + \kappa^a_1 X^a_{ijv} + \lambda^a_v + \varepsilon^a_{ijv} \tag{1}$$

We estimate Equation 1 separately for child ages $a \in \{0, 1, 2, 8, 11, 15\}$. The matrix X^a_{ijb} includes mother's age, mother birth cohort dummies indicating whether she was under 20, 20-35 years old, or older than 35 at baseline, a dummy for child gender, child barangay of birth fixed effects, and child month of birth fixed effects. Note that because all sample children were born in one year, controlling for maternal age is equivalent to controlling for maternal birth year time trends. At birth only ($a = 0$), X^a_{ijb} includes an indicator for whether the child's gestational age is in question.[17] λ^a_v is a vector of baranagay of residence fixed effects.[18]

We consider two dimensions of child health: health stock and flow. We proxy health stock with HAZ at each age a (length-for-age for $a < 2$). We proxy health flow using three weight measures, as appropriate by age: weight at birth, weight-for-length z-scores (WHZ) for $a = 1, 2$, and body-mass-index-for-age z-scores (ZBMI) for $a = 8, 11, 15$.

[17] Approximately, 500 of our sample children were born with at least of the following: (1) low birthweight, (2) mother had diabetes during pregnancy. (3) Mother experienced bleeding during early pregnancy, or (4) mother does not remember her last regular menstrual period. If any one of these circumstances were true, then the child's gestational age at birth was uncertain.

[18] This is equivalent to barangay of birth for $a \leq 2$.

Estimating Equation 1 using OLS will return a biased estimate of γ_1^a, as mother's health is correlated with numerous, omitted dimensions of socioeconomic status that also affect child health. We therefore instrument for mother's health in Equation 1 using variation in weather from the time of her birth and early childhood (as described in Section 3.2): 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 effects, λ_{jb}. If we retained the entire set of exogenous weather instruments, the first stage equation would be specified as Equation 2, except that it would additionally include all Equation 1 covariates.

$$M_{jv} = \beta_1 WB_{jv} + \beta_2 MN_{jv} + \beta_3 HW_{jv} + \lambda_{jb} + u_{jv}, \tag{2}$$

However, this large set of 69 instruments is weak (Stock and Yogo, 2005; Belloni et al., 2012); tests of the joint first stage significance of these instruments return F-statistics that hover around one. Therefore, while our instruments likely meet the exclusion restriction (addressed in Section 5.1), they do not meet the relevance condition, and estimated two-stage least squares (2SLS) parameters may be biased towards OLS parameters. This well-known bias, which increases with the number of weak instruments, stems from the first stage over-fitting to random noise within the endogenous variable and the instruments. When the first stage model is over-fit, the predicted first stage outcome retains a degree of the endogenous variation that biases the 2SLS parameters towards OLS estimates (Winkelried and Smith, 2011).

Since our instruments are both many and weak, we reduce first-stage dimensionality using three separate machine learning methods, each of which chooses or creates one or more "optimal" first stage instruments. First, we estimate Equation 2 via Lasso which creates a subset of optimal first stage instruments (Belloni et al., 2012; Belloni, Chernozhukov and Hansen, 2014). Lasso is a shrinkage estimator designed to reduce model dimensionality by isolating key sources of 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. We choose Lasso's penalty function via cross-validation, as is most commonly done (Tibshirani, 1996).

Second, we decompose the entire set of instruments into 69 new, orthogonal vectors via principal component analysis (PCA). Kloek and Mennes (1960) first proposed principal components as potentially optimal linear combinations of instruments. Choosing principal components lowers dimensionality, retaining the bulk of a matrix's original variation, and increases prediction stability. Principal components also satisfy several desirable properties of a theoretically optimal linear combination of instruments (Amemiya, 1966). We choose the components to retain in the first stage via Lasso, following the boosting procedure employed by (Ng and Bai, 2009).[19] Again, we choose the Lasso penalty function via cross-validation.

Third, we propose a new but related method employing singular value analysis (SVA). SVA is often used in signal processing to de-noise data in a least squares environment

[19] While boosting is an algorithm rather than an estimator, the subset chosen by boosting is very similar to that chosen by Lasso (Ng and Bai, 2009).

(Abu-Shikhah and Elkarmi, 2011; Zhang et al., 2016), making it appropriate for the first stage of a 2SLS estimation. While PCA decomposes the instrument covariance matrix, SVA decomposes the original instrument matrix itself into eigenvectors and "singular values" (a direct transformation of eigenvalues). An optimal subset of these principal axes is then used to solve the original, first-stage minimization problem in the rotated space of orthogonal vectors (Lawson and Hanson, 1974). This "optimal" subset is determined by a cutpoint k that theoretically separates the eigenvectors representing signal from those providing noise.

This single separating point between noise and signal differentiates SVA from the first two methods. Lasso subsets instruments or principal components in no particular order – if the first and third principal component are chosen by Lasso, the second need not be. Under SVA all eigenvectors of index less than or equal to k are included in the solution, while those above k are discarded as noise. This ordering reflects a belief that the largest components of variation reflect signal, the smaller components reflect noise, and a cut-point k separates the two. This theory makes SVA particularly appropriate for noisy data such as the gridded climate data we are working with. PCA performs best when data have an underlying factor structure (Ng and Bai, 2009; Bai and Ng, 2010). Lasso performs best when the endogenous variable is directly driven by a small subset of the potential instruments, and the other instruments carry little information (Ng and Bai, 2009; Kapetanios and Marcellino, 2010*b*,*a*).

We employ SVA in our primary specification because the underlying assumptions make sense for our data. However, recognizing that the reality of our data structure may not fall cleanly into any of the three camps, we replicate all core results using Lasso (Appendix C) and PCA-Lasso (Appendix D). In all three cases, the optimal linear combination or subset of instruments is then used to estimate Equation 1 via 2SLS.

While Lasso chooses a subset of instruments (or principal components) for the first stage, SVA results in only one optimal linear combination of the original instruments. This instrument, $\widehat{M}_j^{k^*}$, is given by the linear combination of the original instruments defined in Equation 3. The coefficients in Equation 3 are defined critically by the chosen cut-point k that separates presumed signal (eigenvectors retained) from presumed noise (eigenvectors discarded) in the rotated space. It therefore determines the parsimony of the solution. The cutpoint k can vary from 1 to k_{max} and determines the total number of non-zero singular values (Lawson and Hanson, 1974). While k is often chosen via rules of thumb (Abu-Shikhah and Elkarmi, 2011; Van Der Veen, Deprettere and Swindlehurst, 1993; Zhang et al., 2016), we choose $k = k^*$, where k^* is chosen through group-wise cross validation.[20] More details regarding the SVA estimation of $\widehat{\beta}_1^{k^*}, \widehat{\beta}_2^{k^*} \widehat{\beta}_3^{k^*}$ and $\widehat{\lambda}^{k^*}$ can be found in Appendix A.

$$\widehat{M}_j^{k^*} = \widehat{\beta}_1^{k^*} WB_{jv} + \widehat{\beta}_2^{k^*} MN_{jv} + \widehat{\beta}_3^{k^*} HW_j + \widehat{\lambda}_{jv}^{k^*}. \tag{3}$$

[20] We choose the k that optimizes predictive power for out-of-sample mothers. Instrument variation that predicts mother's health in sample but does not contribute to forecast power, under $k > k^*$, is likely to be over-fitting noise in the data, picking up endogeneity that will bias second stage results.

4.2 Persistence and Mechanisms

The causal transmission of mother-to-child health identified by $\widehat{\gamma}_1^a$ might operate through a variety of mechanisms, and isolating mechanisms in non-experimental data is often possible only under stringent assumptions (Acharya, Blackwell and Sen, 2016; Imai et al., 2011). We therefore explore the three mechanisms outlined in Section 2 with some caution regarding the interpretation of our findings.

Mother's health may impact child health outside of any biological transmission, because healthier mothers enjoy higher income levels, greater educational attainment, or for other reasons invest differently in their child's health. Thus, socioeconomic status and/or parenting ability may serve as mediating factors between treatment (mother's health) and outcome (child's health). To explore this possibility, we estimate the mother-to-child health transmission at each age conditioning on a large set of socioeconomic variables and inputs to child health.

$$C_{ijv}^a = \gamma_2^a M_{jv} + \delta_2^a S_{ijv}^a + \kappa_2^a X_{ijv}^a + \lambda_{ijv}^a + \eta_{ijv}^a. \tag{4}$$

where S_{ijv}^a includes numerous measures of socioeconomic status and parental inputs including household income, mother's education, child vaccination status, child expenditures, child time allocation, and many others. We estimate Equation 4 first including all controls that capture socioeconomic status (listed in Column 1, Table 3), and then including those socioeconomic controls alongside controls capturing parental investment in children (listed in Column 2, Table 3).[21]

Ideally, if mother's health transmits to her child's health partly through improving her socioeconomic status/parenting ability, then γ_2^a estimates the "direct effect" of mother's health—the effect that operates through any other pathway. However, γ_2^a will not estimate this direct effect if unobserved, intermediate confounders exist, affected by the treatment (mother's health) and also correlated with both the outcome (child health) and the mediator (some aspect of socioeconomic status/ parenting ability) (Acharya, Blackwell and Sen, 2016).[22]

Any of the socioeconomic or input variables included in S_{ijv}^a might serve either as mediator or intermediate control. Therefore, as a robustness check, we estimate the average direct controlled effect (ACDE) using sequential g-estimation as proposed by Acharya, Blackwell and Sen (2016).[23] To do so we choose a single variable, mother's

[21] Note, the socioeconomic and parental input controls we employ differ by age, partly because survey questions differed slightly by round, and also because some inputs are important at one age but not another (e.g., arental help with homework may be an important input at 11, but not during infancy).

[22] However, if controls S_{ijv}^a include mediators and also all intermediate confounders, then γ_2^a estimates the residual effect of mother's health that operates through neither the mediators nor the intermediate confounders, which is again different from the direct effect (Acharya, Blackwell and Sen, 2016). If this residual effect is non-zero, we can infer that additional mechanisms exist. That is, if S_{ijv}^a include all mediators and cofounders, then Equation 4 examines whether alternative mechanisms exist, rather than providing support for any particular mechanism.

[23] Acharya, Blackwell and Sen (2016) propose sequential g-estimation of the ACDE as a way of "de-mediating" the dependent variable to address intermediate variable bias. Under certain assumptions, the ACDE avoids this bias by not conditioning the demediated regression on either the mediator or intermediate controls.

education, as a mediator.[24] We choose mother's education since it is highly correlated with both socioeconomic status and ability. We then treat all other socioeconomic and input controls listed in Table 3 as intermediate confounders. We treat all controls included in Equation 1 as pretreatment controls.

Next, we turn to potential biological channels for the transmission of health. If health transmission occurs at birth, and health at birth continues to impact child health over childhood, then birthweight, a widely used measure of health at birth, may serve as a mediator for health transmission at all ages. We therefore estimate Equation 5 for ages $a \in \{1, 2, 8, 11, 15\}$, continuing to control for socioeconomic status and parental inputs S^a_{ijv}, and now also holding constant birthweight, C^0_{ijv}.

$$C^a_{ijv} = \gamma^a_3 M_{jv} + \phi^a C^0_{ijv} + \delta^a_3 S^a_{ijv} + \kappa^a_3 X^a_{ijv} + \lambda^a_{ijv} + \omega^a_{ijv}. \tag{5}$$

The parameter γ^a_3 will only capture the direct effect of mother's health, net of socioeconomic status, parent inputs, and now birth health, if no intermediary confounders exist. Because such confounders may exist, we again estimate the ACDE using sequential g-estimation treating birthweight as the mediator. In this case, our situation more closely mirrors the appropriate context for estimating an ACDE: birthweight is our only mediator of interest. We include controls from Equation 1 as pre-treatment variables, and all controls in S^a_{ijv} as intermediary confounders. If both γ^a_3 and the ACDE are non-zero, we infer that alternative mechanisms likely exist, underpinning health transmission across ages. One such mechanism could be a persistent biological transmission through child growth trajectory/velocity.

We explore the possibility of a transmission of maternal health to child growth regulation and velocity in two ways. First, we exploit the differential timing of puberty onset and the subsequent transition to pubertal growth across sexes, by re-estimating Equation 5 for girls and boys separately. Girls begin pubertal growth earlier. Therefore, if transmission is working through factors that are specific to childhood growth, we should see a change in the pattern of estimated marginal effects of maternal health earlier for girls than for boys.

Second, we exploit information on the timing of the transition from childhood growth to pubertal growth, within sex. As explained in Section 3.1, we use the timing of menarche for 11 year old girls and visible signs of puberty for 15 year old boys to define a binary indicator for likely pubertal growth P^a_{ijv}. In Equation 6 we interact maternal height M_{jv} with P^a_{ijv}, for $a = 11$ for girls and $a = 15$ for boys, to investigate whether the impact of mother's health varies with probable growth regime.[25]

$$C^a_{ijv} = \gamma^a_4 M_{jv} + \alpha^a P^a_{ijv} + \psi^a (M_{jv} \times P^a_{ijv}) + \delta^a_4 S^a_{ijv} + \kappa^a_4 X^a_{ijv} + \lambda^a_v + \epsilon^a_{ijv} \tag{6}$$

[24] We also estimated the ACDE treating household per capita income as the socioeconomic mediator and included mother's education as an intermediate confounder. This produced nearly identical results, available upon request.

[25] This gives us an additional endogenous variable. We create an additional exogenous instrument by interacting the SVA-optimal instrument with the pubertal growth dummy.

5 Results

5.1 First-Stage Specification and Identification

The $\widehat{M}_j^{k^*}$ compiled by singular value analysis (our SVA-optimal instrument) is a linear combination of all 69 original instruments. However only 45 rotated, orthogonal vectors are chosen (i.e., $k^* = 45$). The bulk of the variation in the SVA-optimal instrument is provided by just over a dozen instruments, all capturing monsoon agricultural conditions. Appendix Figure B.9 illustrates the relative weight of these instruments, i.e., their average contribution to $\widehat{M}_j^{k^*}$.[26] Because these weights are clustered around zero with long tails in either direction, we only include in the figure instruments that fall below the 10th weight percentile (i.e., those with a highly negative effect on $\widehat{M}_j^{k^*}$) and instruments that fall above the 90th weight percentile (i.e., those with a highly positive effect on $\widehat{M}_j^{k^*}$). Remaining weights are available upon request. Figure B.9 illustrates that while monsoon temperature and simultaneously high monsoon temperatures and rainfall positively predict maternal health, high monsoon rainfall on it's own negatively predicts maternal health, perhaps indicating flooding or storms.

Identifying variation comes from an overlapping but distinct set of instruments when we use Lasso or PCA-Lasso to reduce first stage dimensionality. The 28 first stage instruments chosen by Lasso feature every category of instrument (Appendix Table B.1). However, weights representing the average contribution of each instrument to first stage prediction illustrate that 6 instruments, bolded in Table B.1, provide the bulk of the first stage variation.[27] As with the SVA-optimal instrument, monsoon temperature positively predicts maternal health, while high pre-harvest winds — likely capturing rice crop destruction — negatively predict maternal health. Last, Appendix Figures B.10 and B.11 illustrate the relative weights of the instruments that provide the bulk of the variation in the first two of the Lasso-chosen components, under PCA-Lasso. Again, we include only the instruments that fall below the 10th weight percentile or above the 90th weight percentile.[28] A similar graphic for every component is available upon request. Figures B.10 and B.11 suggest that PCA-Lasso instruments share identifying variation with both the SVA-optimal instrument and the Lasso-chosen instruments, which makes sense since this method includes both linear combination and subsetting.[29]

While these three methods of dimensionality reduction result in overlapping but distinct sets of instruments, estimating the first stage of Equation 1 is effective under all three methods and provides similar second-stage estimates. Appendix Table B.2 reports the first-stage estimates using our SVA-optimal instrument. Tables B.3 and B.4 reports the first-stage estimates using Lasso-chosen instruments and Lasso-chosen principal components.[30]

[26] Weights are given by the instrument's coefficient from Equation 3 (elements of $\widehat{\beta}_1^{k^*}$, $\widehat{\beta}_2^{k^*}$, $\widehat{\beta}_3^{k^*}$, or $\widehat{\lambda}_{jv}^{k^*}$) multiplied by the mean of the instrument itself.

[27] As with the SVA-optimal instrument, weights are given by the mean of each instrument multiplied by it's coefficient from a regression of mother's height on the Lasso-chosen instruments. The bolded instruments are those that fall below the 10th weight percentile or above the 90th weight percentile.

[28] In this case weights are given by the instrument-specific elements of the eigenvectors/components

[29] However, interpretation is most difficult with this method since the influence of any given instrument is spread across all Lasso-chosen components.

[30] Table B.2 reports first stage estimates for each child age, while Tables B.3 and B.4 so so only at birth. The first stage is identical at all ages, except that sample size and second stage covariates differ.

The causal effect of mother health on child health is only identified if the exclusion restriction for our instruments holds, for any first-stage specification. If early-life weather impacts the long-term socioeconomic status of mothers' families, this would violate the exclusion restriction, as child health might be effected by aspects of maternal welfare other than health. Because we identify on minor year-to-year weather variation rather than major weather shocks, such a violation seems unlikely. However, to further dispel this concern, we check for a relationship between grandparent socioeconomic status (grade attainment and occupation type) and the SVA-optimal instrument. While grandparent socioeconomic status is associated with mother's (endogenous) adult height, it is not associated with the SVA-optimal instrument. These checks can be found in Appendix Figures B.12 and B.13 and Tables B.6 and B.7.

If mother's birth month is endogenous to grandparent socioeconomic status, this would also violate the exclusion restriction. However, scarce contraceptive use makes it unlikely that birth month was endogenous to socioeconomic status. As a predominately Roman Catholic country modern contraceptive use is low in the Philippines, and was even lower prior to the 1970s when our mothers were born. For example, the 1968 National Demographic Survey found that only 15.5% of married women in the Philippines used any contraceptive practice and the majority of those that did (almost 70%) used traditional methods such as withdrawal or rhythm methods. Less than 4% of contraception users used modern contraception such as pills, IUD, sterilization, or condoms (Laing, 1984). Regardless, we check for correlation between mother's birth month and grandparent grade attainment in Appendix Table B.8. Mother's birth months do not individually or jointly predict grandparent school attainment.

5.2 Mother-to-Child Health Transmission

Figure 1 and Table 4 estimate Equation 1 via 2SLS, using the SVA-optimal instrument, to report the estimated causal effect of maternal health on child health. Panel A of Figure 1 and Table 4 report the transmission of maternal health to child HAZ from birth to approximately age 15. Panel B reports the corresponding transmission effects to child weight outcomes: birthweight, weight-for-length z-scores (WHZ) at ages 1 and 2, and BMI-for-age z-scores (ZBMI) at ages 8, 11, and 15. Birthweight is included in Panel B of Table 4 but not in Panel B of Figure 1 as its scale, grams, differs from that of the other weight outcomes.

Again, the coefficients reported in Figure 1 and Table 4 can be interpreted as the causal effect of a change in all aspects of mother's health associated with a 1 cm change in her adult height, as predicted by early life weather shocks. The effect on child HAZ persists and in fact increases through childhood until adolescence (Panel A of Figure 1). That is, transmission increases child HAZ by 0.09, 0.13, 0.13, 0.15, and 0.11 standard deviations at ages 1, 2, 8, 11, and 15, respectively. Birth length is not significantly affected by maternal health, but birthweight (the most widely used measure of birth health) is: a one unit increase in mother's health increases birthweight by 41.72 grams. The transmission of maternal health to child weight outcomes persists through childhood, with significant estimated effects on WHZ and ZBMI of 0.08, 0.10 and 0.08 at ages 1, 2, and 8, respectively. At ages 11 and 15, this effect decreases slightly to 0.06 but is not significant at standard levels (p-value=0.102, 0.120).

Given the well-documented association between maternal health and child birth health, we expected to find the strongest transmission in early life, which then diminishes with child age. Instead, we find a fairly stable (maybe slightly diminishing) maternal health transmission to child weight, and a transmission to child HAZ that increases through childhood before decreasing slightly in adolescence. To better understand this pattern of transmission across childhood stages, we examine the mechanisms through which it may operate in Section 5.3.

Before moving to mechanisms, however, it is worth noting that Appendix Figures C.1 and D.1 hold virtually identical health transmission estimates, produced by 2SLS estimations with instruments subset/combined by Lasso and PCA-Lasso, respectively. The similarity in second stage outcomes is notable given that first stage identifying variation differs slightly under each of the three dimensionality reduction methods.

Appendix Table B.5 reports OLS and traditional 2SLS estimates (using all instruments) of maternal health transmission to both child height and weight outcomes, at all ages. Comparing these estimates (Columns 1, 2, 4, and 6) to the estimates obtained with our SVA-optimal instrument (Columns 4 and 8) demonstrates that indeed, a weak first stage biases traditional 2SLS estimates towards OLS. Table B.5 illustrates that early life weather shocks to mother's health also directly predict child health, in a reduced form regression (Columns 3 and 7).

5.3 Socioeconomic and Parenting Ability Mechanisms

The intergenerational health transmission displayed in Figure 1 and Table 4 may operate through the ongoing effect of mother's health on her socioeconomic status and/or her parenting ability. If this socioeconomic/ability channel represents the primary mechanism through which maternal health transmission operates, we might expect the transmission to be strongest during early childhood. The first few years of life are known to be a sensitive period for child health (particularly for child height) when the effects of parental and environmental inputs are strongest (Villa, 2017; Grantham-McGregor et al., 2007).[31] However, instead of seeing the strongest transmission to child height occurring at ages 1 and 2, we see an increasing transmission coefficient as the child ages up until early adolescence. This hints that the socioeconomic channel may not the primary mechanism at work.

Figures 2 and 3 report Equation 4 estimates, exploring the possibility that maternal health transmission operates through socioeconomic or parenting ability. Each subplot provides point estimates of the effect of maternal health associated with a 1 cm height increase at a given child age, with 95% confidence intervals. Within each subplot, we first provide the baseline transmission coefficient from Table 4, then the coefficient estimated with socioeconomic controls included, and last the coefficient estimated with all socioeconomic and parental input controls. Estimating a non-zero transmission coefficient after including all controls suggests that socioeconomic status and parenting ability does not fully explain the transmission.[32]

[31] Sensitive periods refer to stages in childhood during which inputs (e.g., parental investment, home environment) exert a stronger impact on a child's development of a trait or skill than in other periods.

[32] Appendix Table B.9 reports the corresponding point estimates illustrated in Figures 2 and 3.

Adding this rich set of controls does little to mitigate the transmission to child height at any age (Figure 2). We do however, see slightly larger reductions in the magnitude of the transmission coefficients during sensitive periods for growth than in others (ages 1, 2, and 11).[33] Similarly, the transmission to child weight (Figure 3) appears more sensitive to external inputs during the sensitive periods of early childhood. At all other ages, including additional socioeconomic or input controls does almost nothing to move our point estimates for the effect of maternal height on child weight.

Overall, this analysis suggests that socioeconomic status and parental ability/inputs may explain part of maternal health transmission, perhaps most importantly during sensitive periods, but that other mechanisms are also at play. Our results are almost identical when using Lasso or PCA-Lasso instead of SVA (Appendix Figures C.2, C.3, D.2 and D.3). Estimates of the ACDE are nearly identical or actually higher than the conditional effect estimated by Equation 4 (Appendix Figures B.14 and B.15 and Table B.10), for all ages and health outcomes, likely because Equation 4 underestimates the direct effect by controlling for intermediate confounders directly. All results suggest that mother-to-child health transmission operates through additional mechanisms outside of the socioeconomic/ability channel.

5.4 Biological Mechanisms

We next turn to examining biological mechanisms. First we examine whether the persistent transmission through childhood and into adolescence is due primarily to a biological transmission of health at birth, and the subsequent importance of birth health to later health. We then examine the possibility that this persistence is due to a transmission to growth velocity, possibly due to a biological transmission to the factors regulating childhood growth.

To explore transmission to birth health as a potential channel, we estimate Equation 5, controlling for birthweight in addition to all our socioeconomic and input controls. Figure 4 reports the estimated transmission coefficients of maternal health to child height (Panel A) and weight outcomes (Panel B)—conditional on the health transmission at birth.[34] Again, a non-zero transmission coefficient suggests other mechanisms likely exist.

For child weight outcomes, the transmission of health at birth appears to explain much of the persistent effect of maternal health. At almost every age, the transmission coefficient reduces in both magnitude and significance.[35] Conversely, the transmission of health at birth does not appear to explain the persistent effect of maternal health on child height outcomes. That is, Panel A of Figures 4 and 1 are quite similar. The magnitude of the transmission at each age declines slightly, indicating that transmission at birth may explain part of the persistence. However, coefficients on maternal health remain statistically and clinically significant at each observed age except age 1. These results indicate that the transmission of maternal health to child height, specifically, operates through additional mechanisms beyond birth health, socioeconomic, and parenting ability channels. Results are similar when instruments are subset using Lasso

[33]Early adolescence is a second sensitive period for linear growth due to the onset of puberty.
[34]Appendix Table B.11 reports corresponding coefficients.
[35]The effect at age 2 is weakly significant.

and PCA-Lasso (Appendix Figures C.4 and D.4). And again, the ACDE of mother's health, excluding the pathway working through birthweight, is higher than the conditional effect estimated by Equation 5 at every age, for both child weight and height (Appendix Figures B.16 and B.17 and Table B.12).

Furthermore, Figure 4 again suggests an accumulating (rather than diminishing) transmission of mother health to child HAZ as children age. While the difference between transmission coefficients to child HAZ across ages are unlikely statistically significant, we estimate the same inverse-U shape when pooling our data across ages, and allowing for a quadratic shape in the effect of maternal health on child HAZ across ages (Appendix Table B.13, Figure B.18). One possible explanation for this pattern is that increased mother health transmits not only increased child height (in the form of a size advantage at birth) but also an increased childhood growth velocity. Higher growth velocity would translate into a marginal child height advantage that increases with age—such as we observe. In other words, if improved maternal health causes higher growth velocity, then the marginal impact of maternal health on child HAZ will increase as the child ages. It is worth noting at this point that if child height was measured in terms of height deficit, rather than height-for-age, as proposed by Leroy et al. (2015), then this height advantage would increase even more as children age.[36]

Next, we explore the possibility that maternal health transmits to child growth trajectories by exploiting sex-specific variation in pubertal development. For this reason we will only focus on child HAZ. Figure 5 reports the marginal effect of maternal health on child HAZ across ages, separated by sex. We continue to control for all pretreatment controls, all socioeconomic and parental input controls, and birthweight. Most of the estimates of the effect of maternal health on boy HAZ are statistically insignificant. However, this likely represents a lack of precision due to the large number of included controls and reduced sample size rather than a true zero. The effect on age 11 girl HAZ is significant at the 10% level.[37] If we exclude all but the pretreatment controls, then we see a similar pattern with similar point estimates that are all statistically significant.[38]

Figure 5 displays a pattern of transmission that matches up remarkably with sex-specific growth trajectories and pubertal development.[39] The transmission of maternal health to girl height increases until it peaks at age 8 (the average age of female puberty onset) and then declines. Similarly, the transmission coefficient to boy height increases until it peaks at age 11 (the average age of male puberty onset) and then declines. Again, the difference between coefficients are likely insignificant. Yet, the presence of this pattern is at least suggestive that the transmission to height may peak just before the transition to the pubertal growth regime.

The patterns of transmission illustrated in Figure 5 suggest that maternal health (as

[36]Leroy et al. (2015) propose measuring child height in terms of height deficits (HAD) rather than height-for-age (HAZ), where HAD is not normalized by age-sex-specific standard deviation (SD) as is HAZ. That is, HAD=HAZ*SD. Since SD grows as children age, this has the potential to hide growing deficits as measured in standard units (Leroy et al., 2015). Since the marginal effect of maternal health on child health happens through an effect on the numerator in HAZ, any impact on HAD will be even larger, and larger in a way that increases with SD as children age.

[37]The effects for age 1 and age 2 girl HAZ insignificant but are close to significant at accepted levels.

[38]Results available upon request.

[39]Coefficients corresponding to Figure 5 are reported in Appendix Tables B.14 and B.15

determined by her early life weather experience) may transmit to the regulating factors of childhood growth. We further explore this possibility by exploiting variation in indicators of pubertal development (Equation 6). Figure 6 plots the different transmission coefficients for girls who are more likely to be in childhood versus pubertal growth depending on menarche timing.[40]

If maternal health transmission to height operates through childhood growth regulating factors, we would expect health transmission to be greater in magnitude for girls likely still experiencing childhood growth. Indeed this magnitude is higher and statistically significant for girls likely in the childhood growth stage where as that for girls likely in pubertal growth is smaller and insignificant.[41] We must interpret these results with caution as effects for the two groups are not statistically different. However, this difference is statistically significant at the 1% level if we employ PCA-Lasso in our first stage (Appendix Figure D.6). These results are in line with a health transmission mechanism working through childhood growth regulation.

Figure 7 plots the different transmission coefficients for boys who are more likely to be in childhood versus pubertal growth.[42] Overall, it is hard to distinguish a discernible pattern for boys. This is likely because by the time of the 1998 (Age 15) survey, the vast majority of boys would have begun pubertal growth, making any differences hard to identify.

Overall, our results indicate a reduced effect of maternal health on child height after puberty onset. The results for girls suggest that the transmission may operate partly through the factors regulating pre-pubertal growth. The results for boys provide no evidence against this hypothesis. Yet, we must be cautious as we cannot directly test this interpretation. The evidence is merely in line with a biological transmission to growth trajectories; it does not clearly identify such an underlying mechanism.

6 Conclusion

We contribute to a small but growing literature on the intergenerational transmission of health, using unique data from the Philippines that track a cohort of mother-child pairs as the children age from birth to adolescence. This allow us to examine the causal impact of maternal health on child health at multiple ages throughout childhood. The CLHNS . Additionally, the unique climatology of the Philippines allows us to obtain exogenous variation in maternal health through early life weather variation, which impacted disease environment and food availability. Jointly, these data allows us to tackle a critical question — at which ages does maternal health most critically impact child health, and why?

Previous papers generally estimated maternal health transmission at a single age, often birth, and our results suggest that doing so dramatically underestimates magnitude and complexity of this transmission. Mother's health continues to impact child health

[40] Table B.16 reports corresponding estimated coefficients and marginal effects.

[41] Again, we define girls to be in childhood growth if their first period occurs 2 years or more after their 1994 interview date. These results are robust to different specifications of early versus late menarche.

[42] Corresponding coefficients and marginal effects are in Appendix Table B.17.

throughout childhood. The transmission of health at birth explains part of the ongoing relationship between mother and child health, and so does maternal socioeconomic status and ability. Jointly, these two mechanisms seem to account for approximately 30—50% of the transmission, depending on child age. Maternal ability and inputs to child health may be slightly more influential during "sensitive periods", when child health is more responsive to health environment.

After controlling for these two pathways, or "demediating" maternal health by removing their effect, we still observe a persistent, significant effect of maternal health on child HAZ, which rises with age, up until the age of puberty onset. The effect of maternal health on child weight outcomes does not display this pattern but is fairly constant over age, and non-zero but insignificant once birthweight and socioeconomic/ability controls are added.

We hypothesize that effect of maternal health on child HAZ rises with age due to an effect on childhood growth velocity. Human and animal studies find that maternal health, physiology and size during pregnancy may impact the factors regulating childhood growth such as GH, insulin, IGF-I and -II, and growth plates. These same factors are linked to later health in a number of ways, largely related to metabolism, hormone regulation, and organ development. Observational evidence on the subject is scarce, and we cannot confirm that the patterns we estimate do indeed stem from the influence of maternal health on childhood growth trajectory. However, if this mechanism is at play, we would expect to see the effect of maternal health on child HAZ increase over childhood until puberty onset, which is exactly what we observe.

More work is needed to explore the mechanisms behind intergenerational transmission of health, and how it plays into socioeconomic mobility as a whole. In particular, more studies are needed in poor countries, where health may play a especially strong role in determining human productivity and adult welfare.

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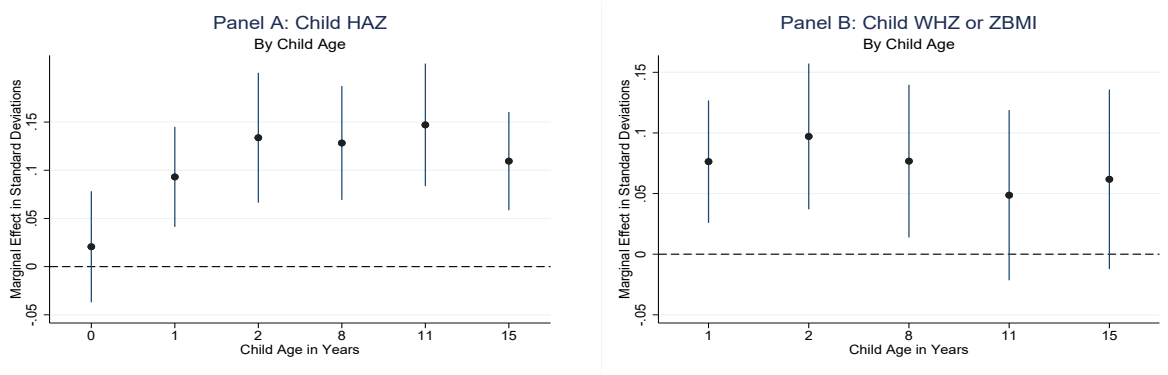
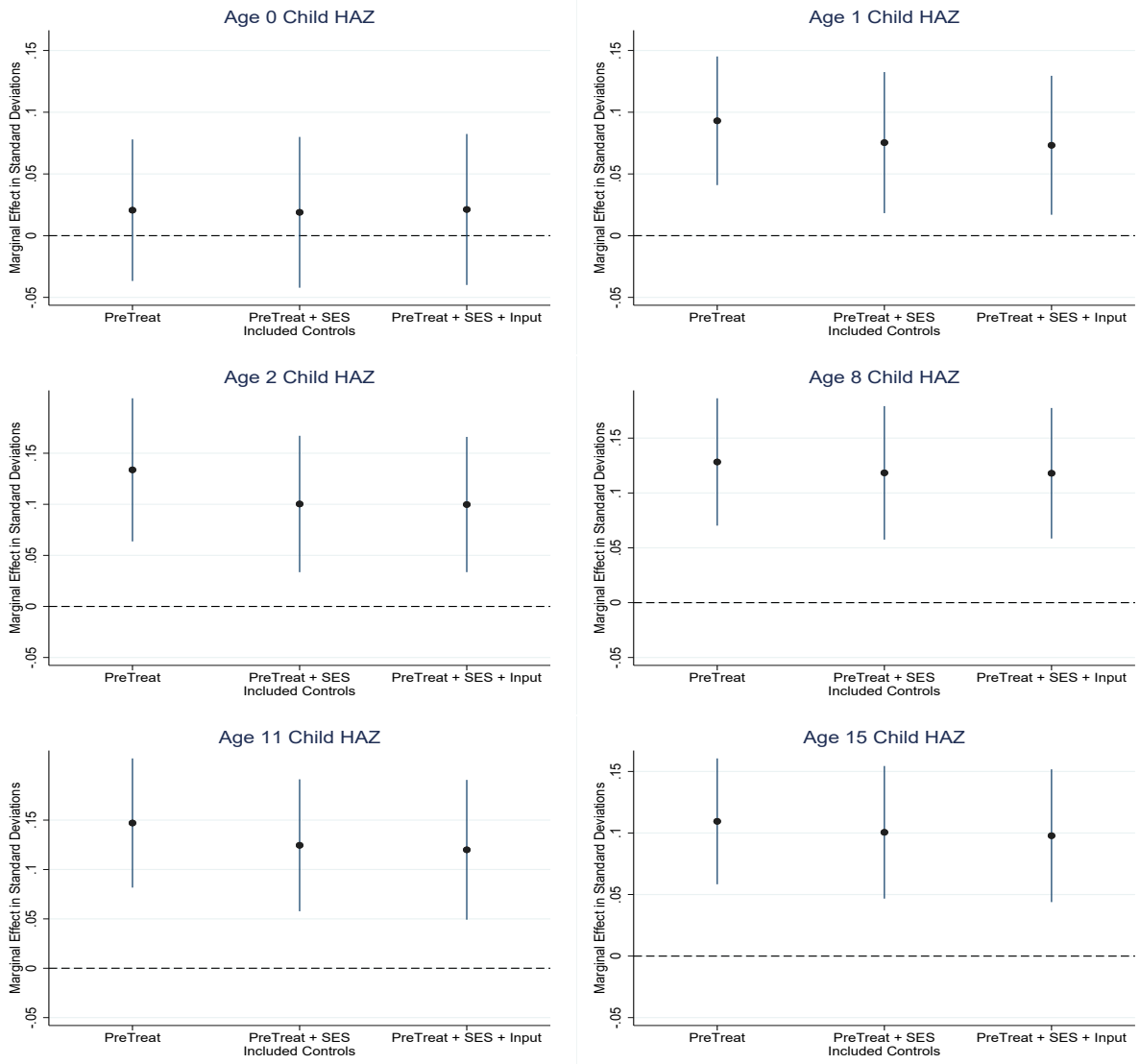
Figure 1: Marginal Effect of Maternal Health on Child Health across Ages



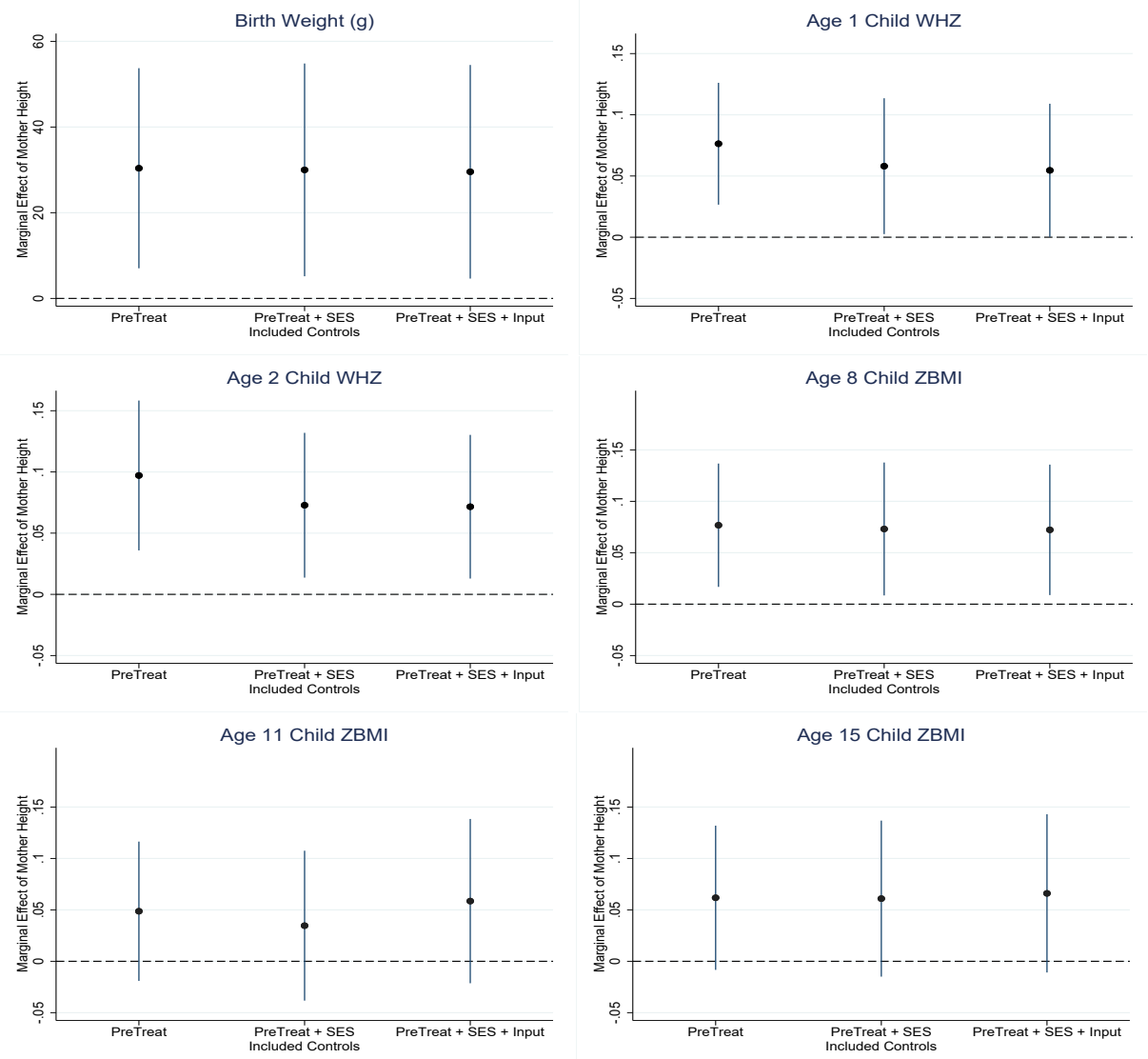
Bars represent 95% confidence intervals.

Figure 2: Marginal Effect of Maternal Health on Child Height Outcomes across Ages—Controlling for Different Categories of Socioeconomic Characteristics



Note: *PreTreat* includes pretreatment controls, X^a_{ijb} , *SES* includes socioeconomic controls (Column 1 of Table 3), and *Input* includes parental input variables (Column 2 of Table 3).

Figure 3: Marginal Effect of Maternal Health on Child Weight Outcomes across Ages—Controlling for Socioeconomic Characteristics



Note: *PreTreat* includes pretreatment controls, X^a_{ijb} , *SES* includes socioeconomic controls (Column 1 of Table 3), and *Input* includes parental input variables (Column 2 of Table 3).

Figure 4: Marginal Effect of Maternal Health on Child Health Controlling for Socioeconomic Characteristics and Birthweight

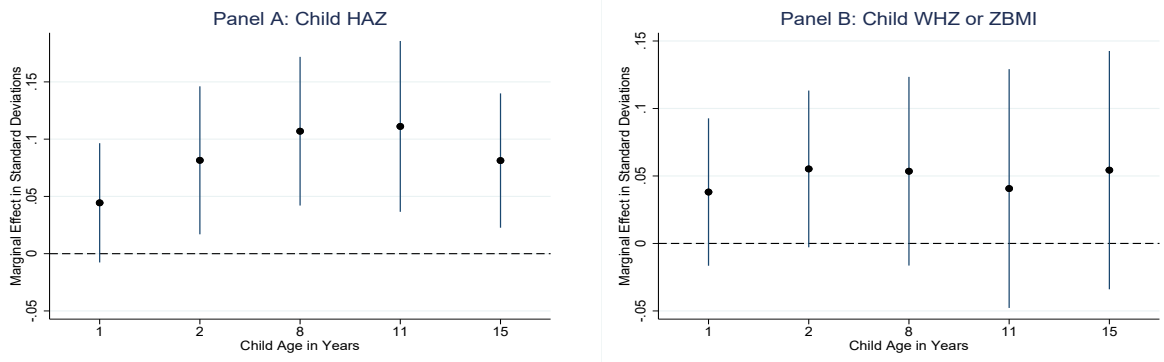


Figure 5: Marginal Effect of Maternal Health on Child Height-for-Age By Sex Controlling for Socioeconomic Characteristics and Birthweight

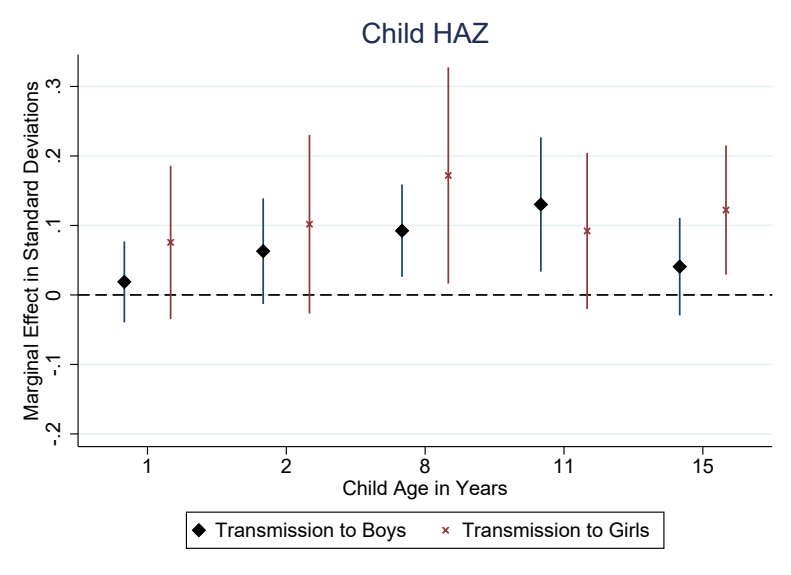


Figure 6: Marginal Effect of Maternal Health on Age 11 Girl HAZ Depending Probable Growth Stage Controlling for Socioeconomic Characteristics and Birthweight

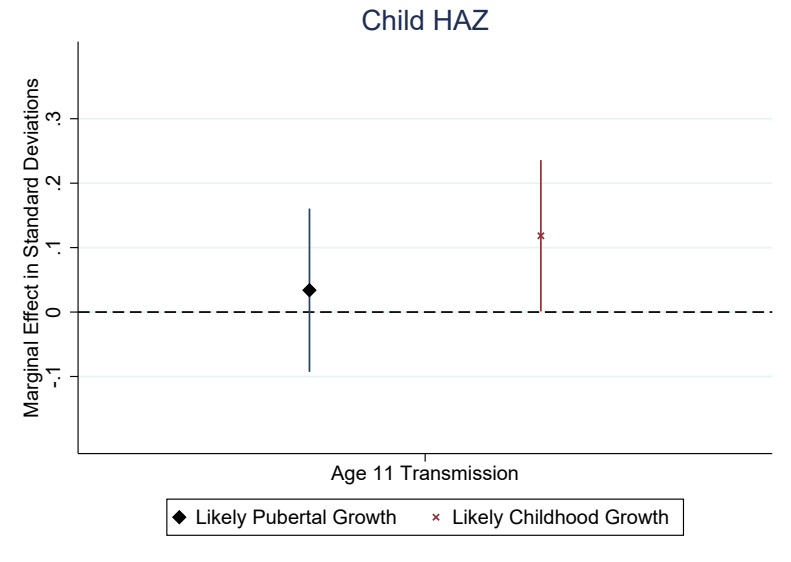


Figure 7: Marginal Effect of Maternal Health on Age 15 Boy HAZ at Age 15 Depending Probable Growth Stage Controlling for Socioeconomic Characteristics and Birthweight

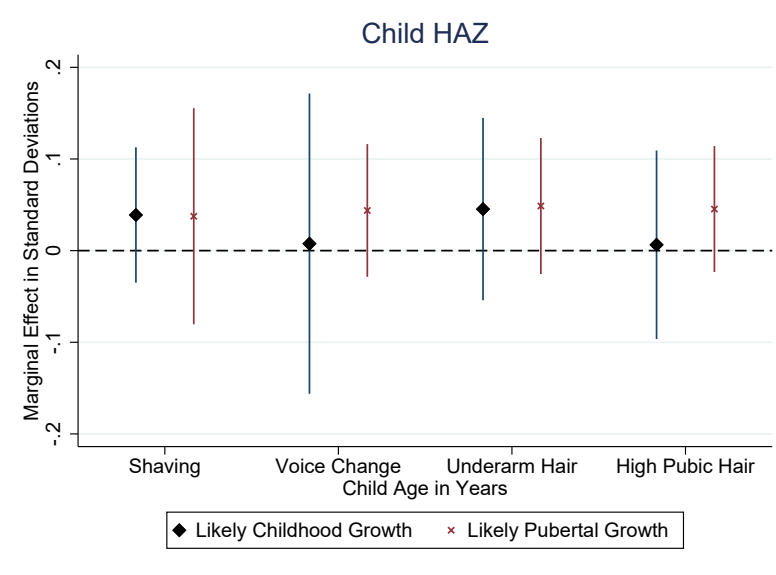


Table 1: Summary Statistics I (Birth – Age 2)

	Females	Males	Total
Birth			
Height-for-age Z-score	-0.47	-0.64	-0.56
	(1.08)	(1.08)	(1.08)
Birth Weight	2,964.96	3,025.90	2,997.31
	(433.56)	(455.76)	(446.44)
Low birth Weight	0.13	0.11	0.12
	(0.33)	(0.31)	(0.32)
Per Capital Income	2,750.23	2,796.54	2,774.81
	(4,085.68)	(3,977.72)	(4,028.13)
Household Size	5.71	5.64	5.67
	(2.88)	(2.75)	(2.81)
Age 1			
Height-for-age Z-score	-1.21	-1.41	-1.32
	(0.98)	(1.02)	(1.00)
Weight-for-age Z-score	-0.57	-0.58	-0.58
	(0.79)	(0.89)	(0.85)
Experienced Stunting in First Year	0.62	0.66	0.64
	(0.49)	(0.47)	(0.48)
Experienced Wasting in First Year	0.29	0.34	0.32
	(0.46)	(0.48)	(0.47)
Per Capital Income	2,704.47	2,693.98	2,698.90
	(4,755.89)	(3,649.03)	(4,203.78)
Household Size	6.69	6.92	6.81
	(2.81)	(2.82)	(2.82)
Age 2			
Height-for-age Z-score	-2.23	-2.38	-2.31
	(1.08)	(1.14)	(1.11)
Weight-for-age Z-score	-0.71	-0.73	-0.72
	(0.84)	(0.92)	(0.88)
Experienced Stunting in Second Year	0.73	0.78	0.76
	(0.44)	(0.41)	(0.43)
Experienced Wasting in Second Year	0.21	0.22	0.21
	(0.40)	(0.41)	(0.41)
Per Capital Income	3,272.52	3,362.11	3,319.70
	(7,993.72)	(5,267.41)	(6,696.52)
Household Size	6.73	6.88	6.81
	(2.74)	(2.77)	(2.76)

Summary Statistics II (Ages 8 – 11)

	Females	Males	Total
	Age 8		
Child age in 1991	8.51	8.51	8.51
	(0.04)	(0.05)	(0.05)
Height-for-age Z-score	-1.99	-2.08	-2.04
	(0.95)	(0.94)	(0.95)
BMI-for-age Z-score	-0.81	-0.81	-0.81
	(0.84)	(0.94)	(0.89)
Stunted	0.52	0.54	0.53
	(0.50)	(0.50)	(0.50)
Wasted	0.08	0.09	0.09
	(0.27)	(0.29)	(0.28)
Per Capital Income	10,462.59	10,583.86	10,526.52
	(10,330.76)	(12,291.09)	(11,403.84)
Household Size	6.86	6.91	6.88
	(2.24)	(2.28)	(2.26)
	Age 11		
Child age in 1994	11.55	11.53	11.54
	(0.40)	(0.40)	(0.40)
Height-for-age Z-score	-1.93	-2.00	-1.96
	(1.10)	(0.97)	(1.03)
BMI-for-age Z-score	-1.02	-1.14	-1.09
	(1.05)	(1.13)	(1.09)
Stunted	0.48	0.52	0.50
	(0.50)	(0.50)	(0.50)
Wasted	0.17	0.20	0.18
	(0.38)	(0.40)	(0.39)
Per Capital Income	14,479.75	15,178.44	14,845.31
	(15,480.39)	(19,616.55)	(17,764.33)
Household Size	7.06	7.11	7.09
	(2.40)	(2.46)	(2.43)
	Age 15		
Child age in 1998	15.09	16.12	15.63
	(0.35)	(0.33)	(0.62)
Height-for-age Z-score	-1.83	-1.87	-1.85
	(0.80)	(0.86)	(0.84)
BMI-for-age Z-score	-0.67	-1.01	-0.85
	(0.99)	(1.07)	(1.05)
Stunted	0.42	0.42	0.42
	(0.49)	(0.49)	(0.49)
Wasted	0.09	0.16	0.13
	(0.29)	(0.37)	(0.34)
Per Capital Income	23,493.57	27,125.65	25,389.81
	(21,960.64)	(26,766.31)	(24,648.01)
Household Size	6.95	6.75	6.84
	(2.41)	(2.44)	(2.43)

Table 2: Pubertal Timing Indicators

	Mean	Standard Dev	Min	Max
Mother's Age	26.80	5.87	17.00	47.00
Male	0.53	0.50	0.00	1.00
Girls				
Age at menarche	13.05	0.98	10.25	15.33
Menarch four years or more after 1991 interview date	0.72	0.45	0.00	1.00
Menache 2+ yrs post 1994 survey	0.35	0.48	0.00	1.00
Menarche 1+ yrs before 1998 survey	0.84	0.37	0.00	1.00
Boys				
Begun shaving	0.21	0.40	0.00	1.00
Voice has changed in recent years	0.95	0.22	0.00	1.00
Has visible underarm hair	0.61	0.49	0.00	1.00
Has high level of pubic hair	0.42	0.49	0.00	1.00

Table 3: Socioeconomic and Parental Input Controls at each Age

	Socio-Economic Controls 1	Parental Input Controls 2
Birth	Per capita household income, Household size, Mother's education, Baseline asset value; Access to piped water, Flushable toilet, Electricity; Garbage taken away after disposal; Uses clean cooking fuel, Food area kept clean; Excreta visible around HH	Took prenatal vitamins; Received prenatal care; Baby delivered by doctor; Baby delivered in hospital
Age 1	Per capita household income, Household size, Mother's education, Year 1 asset value; Access to piped water, Flushable toilet, Electricity; Garbage taken away after disposal; Household uses clean cooking fuel, Food area kept clean; Excreta visible around HH; Animals kept inside HH	In last year child given vitamins or minerals, vaccinations, or non-treated water; Child breastfed for full first year
Age 2	Per capita household income, Household size, Mother's education, Baseline asset value; Access to piped water, Flushable toilet, Electricity; Garbage is taken away after disposal; Uses clean cooking fuel, Food area kept clean; Excreta visible around HH; Animals kept inside HH	In last year child given vitamins or minerals, vaccinations, or non-treated water
Age 8	Per capita household income, Household size, Mother's education; Access to piped water, Flushable toilet, Electricity; Garbage taken away after disposal; Uses clean cooking fuel, Food area kept clean; Excreta visible around HH	Since last survey child given vitamins or minerals or vaccinations; Child given worm medication; Child's food consumption score for average week; Extended family in HH
Age 11	Per capita household income, Household size, Mother's education; Access to piped water, Flushable toilet, Electricity; Garbage taken away after disposal; Uses clean cooking fuel, Food area kept clean; Excreta visible around HH	Parent usually helps child with homework; Hours spend on chores in avg week; Child's food consumption score for average week; Extended family in HH; Per capita expenditure on food, child allowances, and school fees
Age 15	Per capita household income, Household size, Mother's education; Access to piped water, Flushable toilet, Electricity; Garbage taken away after disposal; Uses clean cooking fuel, Food area kept clean; Excreta visible around HH	Child's food consumption score for average week; Extended family in HH; Per capita expenditure on food, child allowances, and school fees

Table 4: Transmission of Maternal Health to Child Health

	Panel A: Child Height Outcomes					
	(1) Birth HAZ	(2) Age 1 HAZ	(3) Age 2 HAZ	(4) Age 8 HAZ	(5) Age 11 HAZ	(6) Age 15 HAZ
Mother's height	0.0206	0.0931***	0.134***	0.128***	0.147***	0.109***
	(0.0294)	(0.0265)	(0.0344)	(0.0301)	(0.0325)	(0.0260)
Observations	2988	2996	2604	2210	2133	2045
	Panel B: Child Weight Outcomes					
	Birth Weight	Age 1 WHZ	Age 2 WHZ	Age 8 ZBMI	Age 11 ZBMI	Age 15 ZBMI
Mother's height	30.38**	0.0764***	0.0971***	0.0768**	0.0487	0.0618
	(12.23)	(0.0258)	(0.0307)	(0.0321)	(0.0358)	(0.0378)
Observations	2990	2980	2606	2210	2133	2045

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1
Controls include gender, mother's age, mother age cohorts, 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.

Appendix A 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 $\widehat{\gamma}$ to minimize $||\mathbf{S}\gamma - \mathbf{g}||^2$. The original parameter vector $\widehat{\beta}$ is calculated as $\mathbf{V}\widehat{\gamma}$.

Predicted outcome $\widehat{\mathbf{y}}$ may be equivalently calculated as either $\mathbf{X}\widehat{\beta}$ or $\mathbf{US}\widehat{\gamma}$, since $\mathbf{X}\beta \simeq \mathbf{y}$ and $\mathbf{S}\widehat{\gamma} \simeq \mathbf{g} = \mathbf{U}^T\mathbf{y}$. The residual $\widehat{\mathbf{r}}$ may be equivalently calculated as either $\mathbf{y} - \mathbf{X}\widehat{\beta}$ or $\mathbf{U}(\mathbf{g} - \mathbf{S}\widehat{\gamma})$ 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 $\widehat{\gamma}$ become increasingly insignificant to $\widehat{\beta} = \mathbf{V}\widehat{\gamma}$. The candidate solution $\gamma^{(k)}$ may therefore be considered, where each element of $\gamma^{(k)}$ is identical to that of the full solution $\widehat{\gamma}$ up until the k'th element, and all subsequent elements are zero, as below.

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

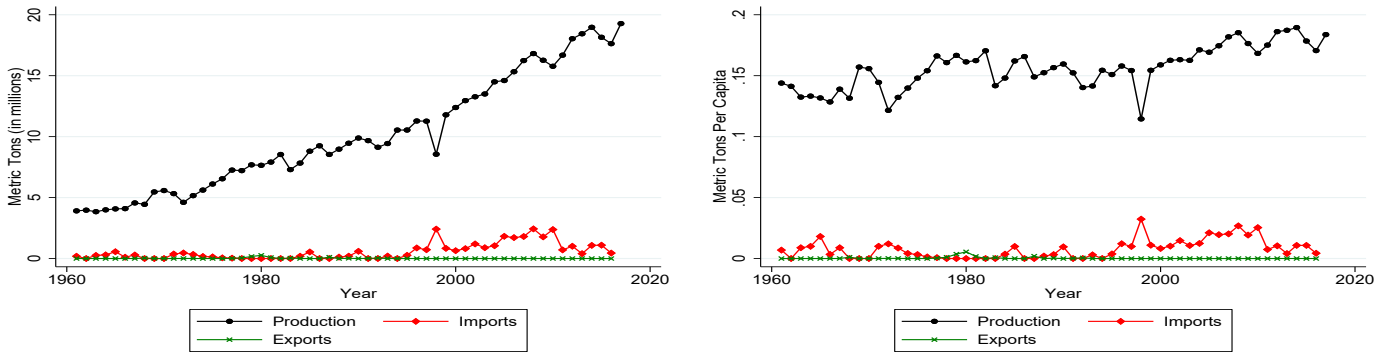
In many cases the candidate solution $\gamma^{(k)}$, for some particular $k < n$, minimizes mean squared forecasting error (MSFE) better than the full solution γ. This is because the rows of matrix $\mathbf{V}$ hold "averages" for the columns of matrix $\mathbf{X}$, but with each row explaining less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

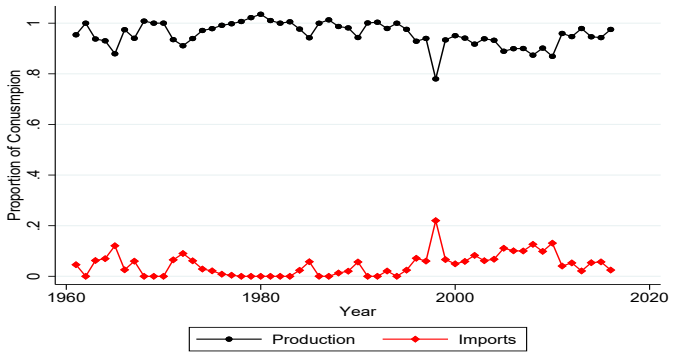
Appendix B Extra Results and Robustness Checks

Figure B.1: Rice Production and Trade, the Philippines 1961—2017



Source: Food and Agricultural Organization (FAO): http://www.fao.org/faostat/en/#data

Figure B.2: Rice Production and Trade as Proportion of Consumption, the Philippines 1961—2017



Source: Food and Agricultural Organization (FAO): http://www.fao.org/faostat/en/#data

Figure B.3: Distribution of Maternal Year of Birth

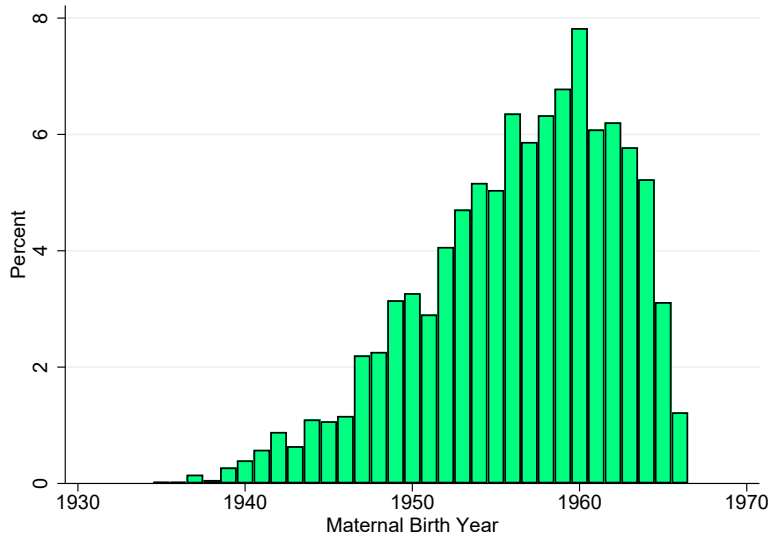


Figure B.4: Monthly Average Rainfall

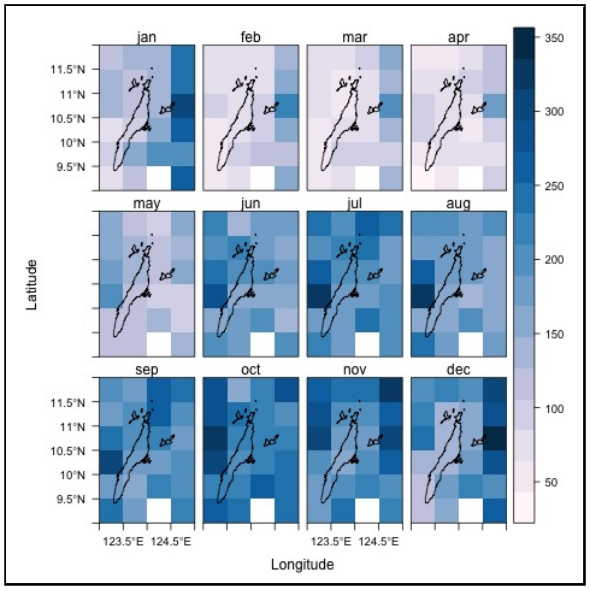


Figure B.5: Monthly Average Temperature

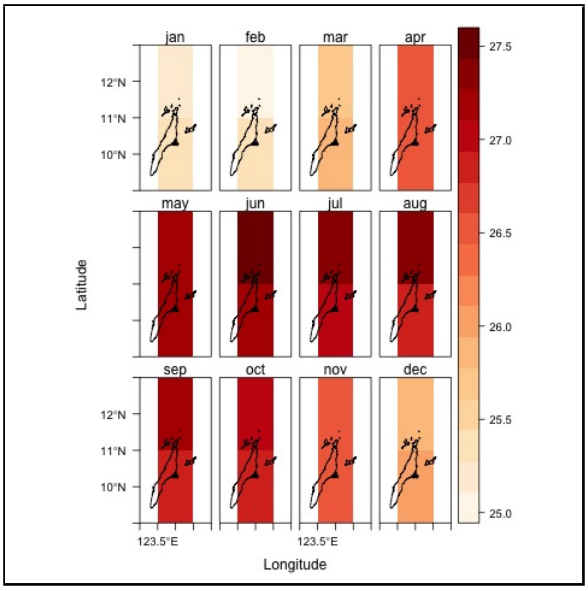


Figure B.6: Monthly Average Windspeed

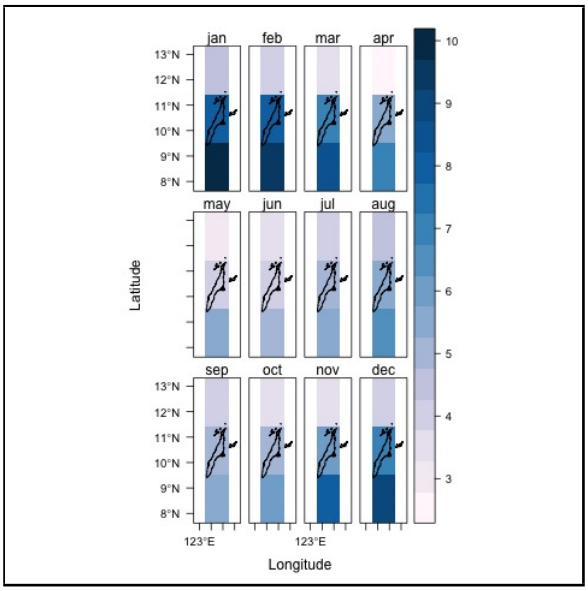


Figure B.7: Mother's Height by Month of Birth

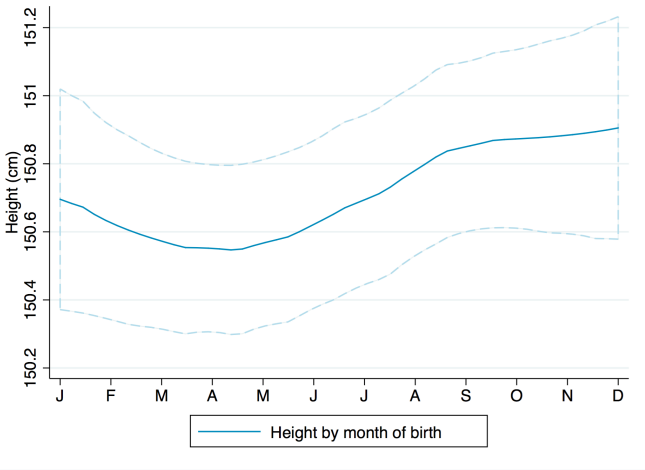


Figure B.8: Maternal Month of Birth

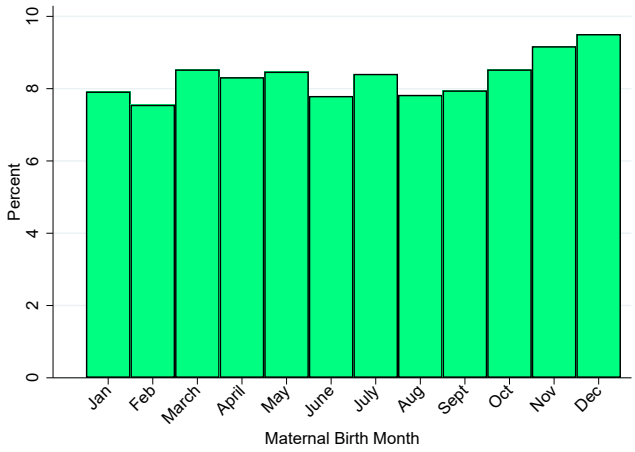


Figure B.9: Variables Providing Main Identifying Variation for SVA instrument

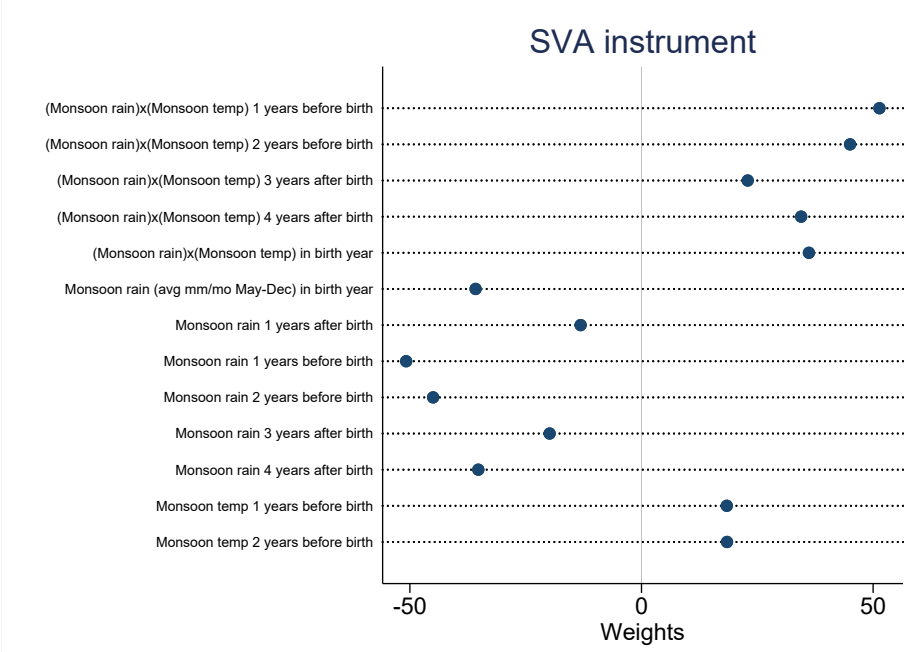


Table B.1: Instruments Chosen by Lasso

Variables Chosen	Weights
Max windspeed 12 mo prior to birth	0.30
Max windspeed 11 mo prior to birth	0.27
Max windspeed 10 mo prior to birth	-0.49
Max windspeed 9 mo prior to birth	-0.38
Max windspeed 8 mo prior to birth	-0.25
Max windspeed 5 mo prior to birth	-0.13
Max windspeed 4 mo prior to birth	-0.37
Max windspeed 3 mo prior to birth	0.19
Max windspeed 1 mo prior to birth	-0.47
Max windspeed 2 mo after birth	-0.29
Max windspeed 3 mo after birth	0.20
Max windspeed 6 mo after birth	0.40
Max windspeed 8 mo after birth	-0.23
Max windspeed 10 mo after birth	0.30
Monsoon rain 2 years before birth	0.12
Monsoon rain 3 years after birth	0.93
Monsoon rain 5 years after birth	0.60
Monsoon temp 1 years before birth	**36.99**
Monsoon temp 3 years after birth	**49.64**
Monsoon temp 4 years after birth	**36.44**
(Monsoon rain)x(Monsoon temp) 2 years after birth	0.46
Harvest high winds 2 years before birth	**-1.38**
Harvest high winds the year of birth	-0.57
Harvest high winds 2 years after birth	**-0.83**
Harvest high winds 3 years after birth	**-0.65**
Born in month 3	-0.05
Born in month 5	-0.02
Born in month 11	0.07

Instruments providing main identifying variation are bolded

Figure B.10: Variables Providing Main Identifying Variation for 1st PCA-Lasso Instrument

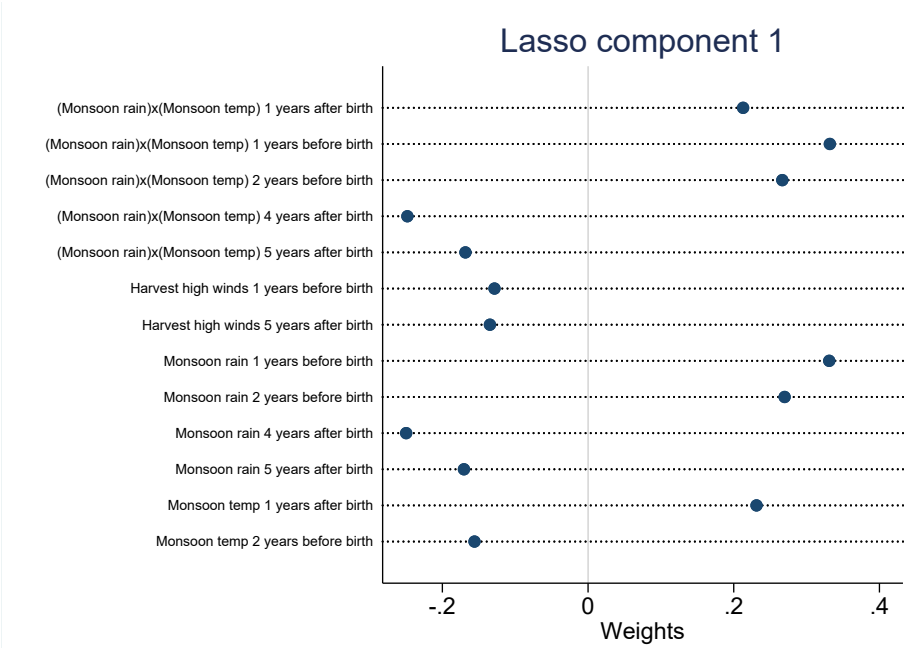


Figure B.11: Variables Providing Main Identifying Variation for 2nd PCA-Lasso Instrument

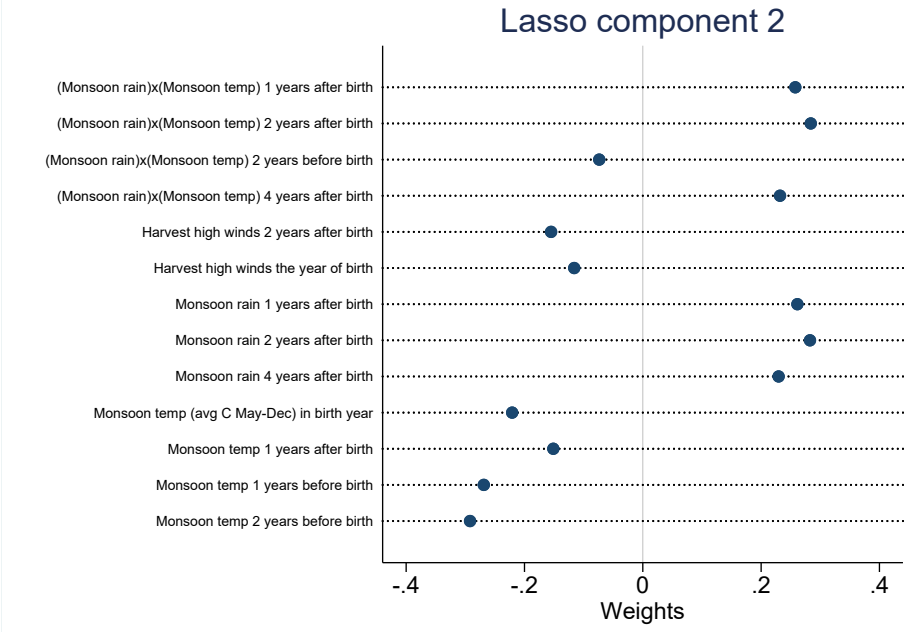


Table B.2: First Stage Results: SVA-Optimal Instrument

	(1) Birth	(2) Age 1	(3) Age 2
Optimal SVA for Mother's Height (k=45)	0.872***	0.910***	0.821***
	(0.129)	(0.129)	(0.139)
Observations	2988	2996	2604
R^2	0.0529	0.0443	0.0481
	Age 8	Age 11	Age 15
Optimal SVA for Mother's Height (k=45)	0.930***	0.957***	0.922***
	(0.157)	(0.158)	(0.164)
Observations	2210	2133	2045
R^2	0.114	0.125	0.135

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1
Controls include gender, mother's age, mother age cohorts, 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.

Table B.3: First Stage Results at Birth: Lasso-Chosen Instruments

	(1) Mother's height
Max windspeed 12 mo prior to birth	0.00177 (0.00200)
Max windspeed 11 mo prior to birth	0.00103 (0.00192)
Max windspeed 10 mo prior to birth	-0.00312* (0.00178)
Max windspeed 9 mo prior to birth	-0.00406** (0.00195)
Max windspeed 8 mo prior to birth	-0.00212 (0.00198)
Max windspeed 5 mo prior to birth	0.00000677 (0.00181)
Max windspeed 4 mo prior to birth	-0.00211 (0.00192)
Max windspeed 3 mo prior to birth	0.00117 (0.00192)
Max windspeed 1 mo prior to birth	-0.00390* (0.00202)
Max windspeed 2 mo after birth	-0.00187 (0.00190)
Max windspeed 3 mo after birth	0.00358* (0.00190)
Max windspeed 6 mo after birth	0.00380** (0.00188)
Max windspeed 8 mo after birth	-0.00115 (0.00195)
Max windspeed 10 mo after birth	0.00354* (0.00190)
Monsoon rain 2 years before birth	-0.00382 (0.00419)
Monsoon rain 3 years after birth	0.00710* (0.00374)
Monsoon rain 5 years after birth	-0.000937 (0.00475)
Monsoon temp 1 years before birth	1.260 (0.803)
Monsoon temp 3 years after birth	1.179 (0.842)
Monsoon temp 4 years after birth	1.921** (0.865)
(Monsoon rain)x(Monsoon temp) 2 years after birth	0.0000951 (0.000166)
Harvest Max Winds 2 Years before Birth	-0.00939*** (0.00359)
Harvest Max Winds Year of Birth	-0.00609 (0.00483)
Harvest Max Winds 2 Years after Birth	-0.00441 (0.00432)
Harvest Max Winds 3 Years after Birth	-0.00872 (0.00617)
Born in month 3	-0.523 (0.356)
Born in month 5	-0.525 (0.367)
Born in month 11	0.704* (0.368)
Observations	2988
R^2	0.0554

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1
Controls include gender, mother's age, mother age cohorts, birth month fixed effects, baseline/current barangay fixed effects, and an indicator for whether gestational age is in question
Child outcome is length-for-age z-scores.

Table B.4: First Stage Results at Birth: Lasso-Chosen Principal Components

	(1) Mother's height
Scores for component 2	0.0286 (0.0495)
Scores for component 3	0.0792 (0.0763)
Scores for component 4	0.0474 (0.0510)
Scores for component 5	0.0511 (0.0545)
Scores for component 8	-0.0434 (0.0575)
Scores for component 9	0.109* (0.0629)
Scores for component 11	0.118 (0.0876)
Scores for component 14	-0.203** (0.0872)
Scores for component 19	0.0126 (0.0854)
Scores for component 20	0.0944 (0.0863)
Scores for component 21	0.169* (0.0873)
Scores for component 24	0.182** (0.0874)
Scores for component 25	-0.136 (0.0886)
Scores for component 27	0.0738 (0.0930)
Scores for component 29	0.212** (0.0985)
Scores for component 32	0.224** (0.103)
Scores for component 34	-0.150 (0.139)
Scores for component 35	-0.248** (0.117)
Scores for component 38	-0.121 (0.122)
Scores for component 42	0.250* (0.138)
Scores for component 47	-0.458*** (0.147)
Scores for component 49	-0.345** (0.169)
Scores for component 50	0.353* (0.185)
Scores for component 51	0.367** (0.178)
Scores for component 59	1.187 (0.758)
Observations	2988
R^2	0.0578

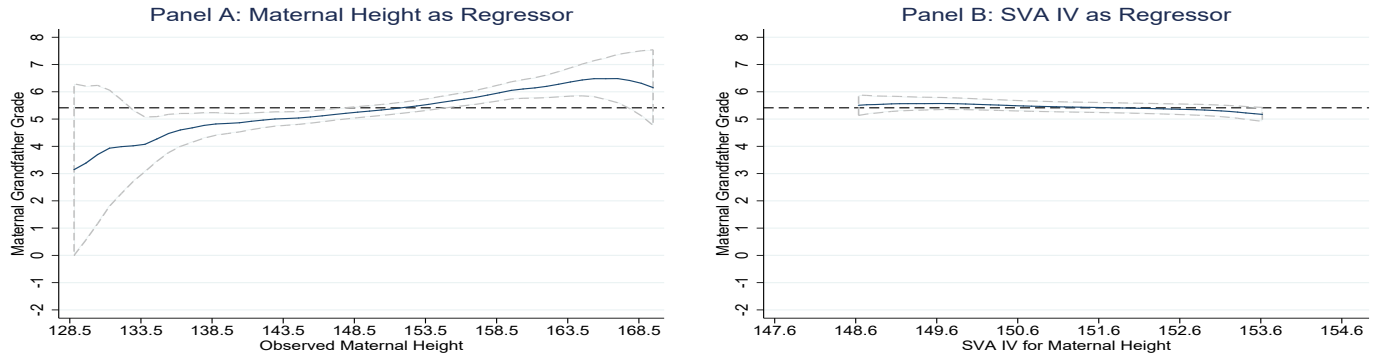
Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1
Controls include gender, mother's age, mother age cohorts, birth month fixed effects, baseline/current barangay fixed effects, and an indicator for whether gestational age is in question
Child outcome is length-for-age z-scores.

Table B.5: Effects of Maternal Height on Child Height and Weight Outcomes using OLS, 2SLS, Reduced Form and SVA

	Birth Height Zscore				Birth Weight			
	(1) OLS	(2) 2SLS (All IVs)	(3) Reduced Form	(4) 2SLS (SVA IV)	(5) OLS	(6) 2SLS (All IVs)	(7) Reduced Form	(8) 2SLS (SVA IV)
Mother's height	0.0336***	-0.00222		0.0206	17.16***	24.05***		30.38**
	(9.33)	(-0.09)		(0.70)	(10.48)	(2.59)		(2.48)
Optimal SVA for Mother's Height (k=45)			0.0180				62.35***	
			(0.69)				(6.19)	
Observations	2988	2988	2988	2988	2990	2990	2990	2990
IV F-stat		1.199		45.87		1.204		46.39
	Age 1 Height Zscore				**Age 1 Weight Zscore**			
Mother's height	0.0582***	0.0684***		0.0931***	0.0129***	0.0610***		0.0764***
	(16.87)	(3.24)		(3.52)	(4.14)	(3.10)		(2.96)
Optimal SVA for Mother's Height (k=45)			0.0847***				0.0690***	
			(3.39)				(3.07)	
Observations	2996	2996	2996	2996	2980	2980	2980	2980
IV F-stat		1.240		49.93		1.224		48.95
	Age 2 Height Zscore				**Age 2 Weight Zscore**			
Mother's height	0.0702***	0.0758***		0.134***	0.0244***	0.0495**		0.0971***
	(17.19)	(3.29)		(3.89)	(7.15)	(2.55)		(3.17)
Optimal SVA for Mother's Height (k=45)			0.110***				0.0801***	
			(3.77)				(3.37)	
Observations	2604	2604	2604	2604	2606	2606	2606	2606
IV F-stat		1.476		34.97		1.526		35.34
	Age 8 Height Zscore				**Age 8 Weight Zscore**			
Mother's height	0.0680***	0.0737***		0.128***	0.00636	0.0394**		0.0768**
	(16.94)	(3.61)		(4.26)	(1.53)	(1.97)		(2.39)
Optimal SVA for Mother's Height (k=45)			0.119***				0.0714**	
			(4.03)				(2.44)	
Observations	2210	2210	2210	2210	2210	2210	2210	2210
IV F-stat		1.499		35.12		1.499		35.12
	Age 11 Height Zscore				**Age 11 Weight Zscore**			
Mother's height	0.0667***	0.0718***		0.147***	0.0117**	0.0224		0.0487
	(14.82)	(3.57)		(4.53)	(2.35)	(0.93)		(1.36)
Optimal SVA for Mother's Height (k=45)			0.141***				0.0466	
			(4.43)				(1.31)	
Observations	2133	2133	2133	2133	2133	2133	2133	2133
IV F-stat		1.564		36.72		1.564		36.72
	Age 15 Height Zscore				**Age 15 Weight Zscore**			
Mother's height	0.0713***	0.0675***		0.109***	0.00679	0.0121		0.0618
	(19.75)	(4.06)		(4.21)	(1.34)	(0.51)		(1.64)
Optimal SVA for Mother's Height (k=45)			0.101***				0.0569	
			(3.67)				(1.61)	
Observations	2045	2045	2045	2045	2045	2045	2045	2045
IV F-stat		1.458		31.76		1.458		31.76

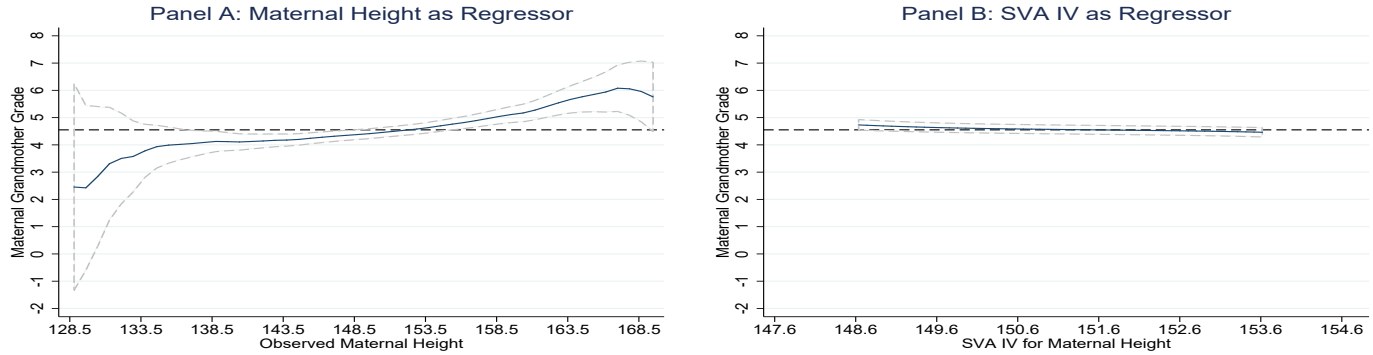
Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1
Regression includes fixed effects for mother's month of birth and for baseline barangay

Figure B.12: Association between Grandfather Grade Attainment and Maternal Height and SVA IV



Note: Reference line on the sample mean of maternal grandfather grade attainment

Figure B.13: Association between Grandmother Grade Attainment and Maternal Height and SVA IV



Note: Reference line on the sample mean of maternal grandmother grade attainment

Table B.6: Effect of Maternal Maternal Height and SVA IV on Grandparent Grade Attainment

	(1) Maternal Height	(2) SVA IV
Maternal grandmother education	0.129***	-0.00454
	(0.0440)	(0.00627)
Maternal grandfather education	0.110***	0.000124
	(0.0402)	(0.00573)
Observations	1751	1751
Fstat p-value	7.42e-09	0.685

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table B.7: Effect of Grandparent Occupation on Maternal Height and SVA IV

	Maternal Grandfather		Maternal Grandmother	
	(1) Maternal Height	(2) SVA IV	(3) Maternal Height	(4) SVA IV
Administrative	0.329	-0.0200	-2.838	-0.00158
	(1.095)	(0.156)	(1.469)	(0.209)
Clerical	0.608	-0.0263	-2.160	-0.120
	(1.122)	(0.160)	(1.773)	(0.252)
Retail/Vendor	-0.370	-0.143	-1.878*	0.0632
	(0.936)	(0.133)	(0.747)	(0.106)
Agriculture	-0.968	-0.0569	-2.227**	0.127
	(0.851)	(0.121)	(0.763)	(0.108)
Miner	0.529	0.0125		
	(1.148)	(0.163)		
Transport	0.00440	-0.0832	-0.864	-0.322
	(0.893)	(0.127)	(2.218)	(0.315)
Craftsmen	-0.863	-0.0678	-2.009*	0.110
	(0.869)	(0.124)	(0.810)	(0.115)
Laborer	-1.895*	-0.175	-2.446*	0.00265
	(0.926)	(0.132)	(0.969)	(0.138)
Service	-1.074	-0.109	-2.419**	0.0381
	(0.923)	(0.131)	(0.867)	(0.123)
Non-Participant	-0.741	-0.215	-2.111**	0.0877
	(1.567)	(0.223)	(0.740)	(0.105)
Constant	150.2***	149.8***	151.4***	149.6***
	(1.228)	(0.175)	(1.122)	(0.160)
Observations	2017	2017	2047	2047
Fstat p-value	0.0130	0.723	0.277	0.685

Robust standard errors in parentheses
Controls include mother's age, mother age, and baseline barangay fixed effects.
*** p<0.01, ** p<0.05, * p<0.1

Table B.8: Association between Maternal Birth Month and Grandparent Grade Attainment

	(1) Grandmother	(2) Grandfather
February	0.146	0.0336
	(0.422)	(0.471)
March	-0.137	0.489
	(0.405)	(0.450)
April	0.109	0.164
	(0.419)	(0.464)
May	-0.0294	0.182
	(0.414)	(0.462)
June	0.302	0.889*
	(0.428)	(0.472)
July	-0.0902	-0.194
	(0.420)	(0.466)
August	0.0116	0.0569
	(0.442)	(0.491)
September	0.332	0.720
	(0.425)	(0.475)
October	-0.520	0.0535
	(0.414)	(0.462)
November	-0.141	-0.130
	(0.414)	(0.461)
December	-0.135	0.406
	(0.401)	(0.450)
Constant	4.574***	5.187***
	(0.301)	(0.337)
Observations	1912	1823
Fstat p-value	0.821	0.389

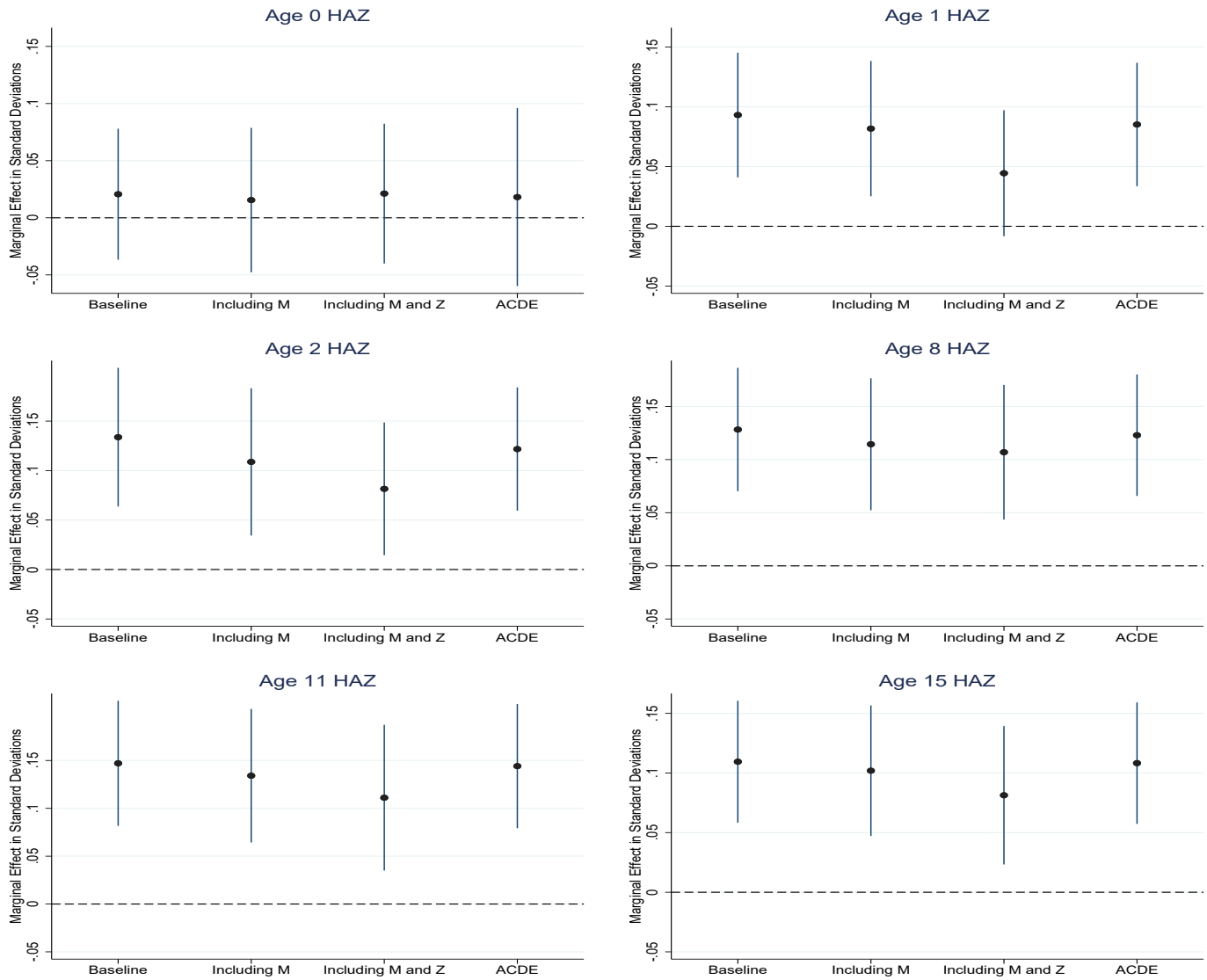
Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table B.9: Maternal Transmission to Child Health Controlling for Socioeconomic Characteristics—Coefficients Corresponding to Figures 2 and 3

	Birth HAZ			Birthweight		
	(1)	(2)	(3)	(4)	(5)	(6)
Mother's height	0.0206	0.0189	0.0212	30.38**	30.00**	29.56**
	(0.0293)	(0.0312)	(0.0312)	(11.91)	(12.67)	(12.72)
Observations	2988	2988	2988	2990	2990	2989
R^2	0.166	0.169	0.172	0.177	0.184	0.189
	Age 1 HAZ			Age 1 WHZ		
	(1)	(2)	(3)	(4)	(5)	(6)
Mother's height	0.0931***	0.0754***	0.0732**	0.0764***	0.0580**	0.0546**
	(0.0266)	(0.0292)	(0.0287)	(0.0254)	(0.0283)	(0.0278)
Observations	2996	2599	2599	2980	2595	2595
R^2	0.121	0.189	0.208	.	.	0.00925
	Age 2 HAZ			Age 2 WHZ		
	(1)	(2)	(3)	(4)	(5)	(6)
Mother's height	0.134***	0.100***	0.0998***	0.0971***	0.0728**	0.0716**
	(0.0357)	(0.0341)	(0.0338)	(0.0312)	(0.0302)	(0.0299)
Observations	2604	2461	2461	2606	2462	2462
R^2	0.0867	0.221	0.231	.	0.0271	0.0374
	Age 8 HAZ			Age 8 ZBMI		
	(1)	(2)	(3)	(4)	(5)	(6)
Mother's height	0.128***	0.118***	0.118***	0.0768**	0.0731**	0.0723**
	(0.0296)	(0.0310)	(0.0304)	(0.0306)	(0.0330)	(0.0324)
Observations	2210	2209	2205	2210	2209	2205
R^2	0.134	0.206	0.222	.	.	0.00366
	Age 11 HAZ			Age 11 ZBMI		
	(1)	(2)	(3)	(4)	(5)	(6)
Mother's height	0.147***	0.124***	0.120***	0.0487	0.0346	0.0585
	(0.0334)	(0.0341)	(0.0361)	(0.0346)	(0.0372)	(0.0408)
Observations	2133	2133	2016	2133	2133	2016
R^2	0.0536	0.193	0.212	0.0928	0.141	0.118
	Age 15 HAZ			Age 15 ZBMI		
	(1)	(2)	(3)	(4)	(5)	(6)
Mother's height	0.109***	0.101***	0.0978***	0.0618*	0.0609	0.0660*
	(0.0261)	(0.0275)	(0.0275)	(0.0358)	(0.0387)	(0.0393)
Observations	2045	2038	2038	2045	2038	2038
R^2	0.231	0.275	0.290	0.0800	0.0888	0.0847
Baseline Controls	Yes	Yes	Yes	Yes	Yes	Yes
SES Controls	No	Yes	Yes	No	Yes	Yes
Parent Input Controls	No	No	Yes	No	No	Yes

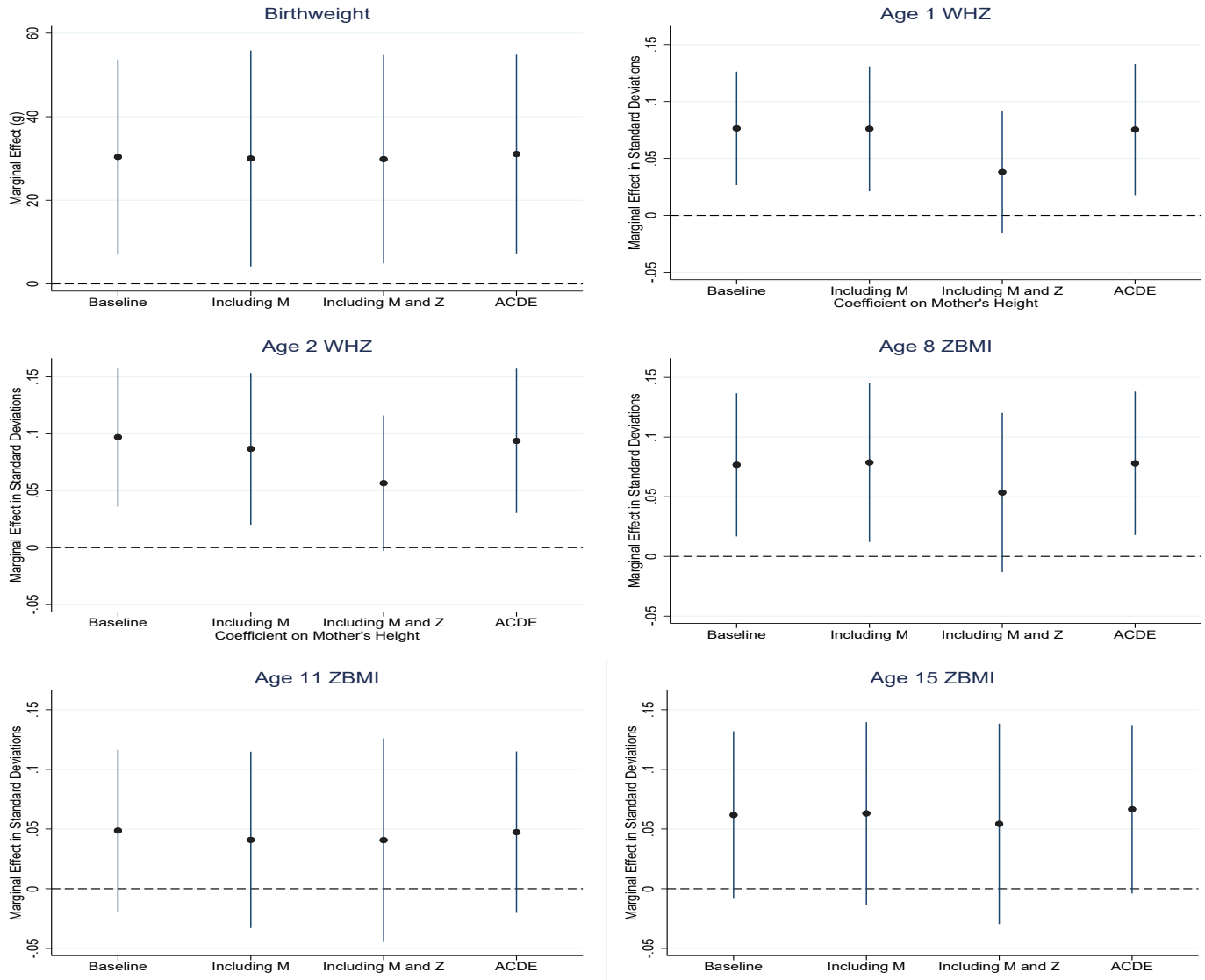
Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1
All controls from Table 4 are included.
Additionally, controls include all socioeconomic characteristics and parental inputs from Table 3.

Figure B.14: Comparison of Conditional Marginal Effect with ACDE of Maternal Height on Child Height Outcomes across Ages—Using Maternal Education as Mediator



Baseline refers to a regression with only pretreatment controls included. "'Including M" includes pretreatment controls and the mediator. "Including M and Z" pretreatment controls, mediator, and intermediating confounders.

Figure B.15: Comparison of Conditional Marginal Effect with ACDE of Maternal Height on Child Weight Outcomes across Ages—Using Maternal Education as Mediator



Baseline refers to a regression with only pretreatment controls included. "'Including M" includes pretreatment controls and the mediator. "Including M and Z" pretreatment controls, mediator, and intermediating confounders.

Table B.10: Comparison of Conditional Marginal Effect with ACDE of Mother Height on Child Height and Weight Outcomes Using Maternal Education as Mediator

	Age 1 HAZ				Age 1 WHZ			
	Baseline	Including M	Including M and Z	ACDE	Baseline	Including M	Including M and Z	ACDE
Mother's Height	0.0931***	0.0817***	0.0444*	0.0851***	0.0764***	0.0760***	0.0381	0.0753***
	(0.0266)	(0.0289)	(0.0269)	(0.0270)	(0.0254)	(0.0280)	(0.0275)	(0.0216)
Observations	2996	2996	2593	2996	2980	2980	2589	2980
R^2	0.121	0.151	0.385	0.115	.	.	0.139	.
	Age 2 HAZ				Age 2 WHZ			
	Baseline	Including M	Including M and Z	ACDE	Baseline	Including M	Including M and Z	ACDE
Mother's Height	0.134***	0.109***	0.0815**	0.122***	0.0971***	0.0868**	0.0567*	0.0938***
	(0.0357)	(0.0380)	(0.0342)	(0.0322)	(0.0312)	(0.0340)	(0.0303)	(0.0326)
Observations	2604	2604	2457	2604	2606	2602	2455	2602
R^2	0.0867	0.177	0.303	0.0919	.	.	0.134	.
	Age 8 HAZ				Age 8 ZBMI			
Mother's Height	0.128***	0.114***	0.107***	0.123***	0.0768**	0.0787**	0.0535	0.0780**
	(0.0296)	(0.0317)	(0.0323)	(0.0292)	(0.0306)	(0.0340)	(0.0340)	(0.0307)
Observations	2210	2210	2202	2210	2210	2210	2202	2210
R^2	0.134	0.190	0.266	0.133	.	.	0.0870	.
	Age 11 HAZ				Age 11 ZBMI			
Mother's Height	0.147***	0.134***	0.111***	0.144***	0.0487	0.0409	0.0407	0.0474
	(0.0334)	(0.0356)	(0.0388)	(0.0331)	(0.0346)	(0.0377)	(0.0435)	(0.0345)
Observations	2133	2133	2013	2133	2133	2133	2013	2133
R^2	0.0536	0.109	0.243	0.0526	0.0928	0.107	0.166	0.0929
	Age 15 HAZ				Age 15 ZBMI			
Mother's Height	0.109***	0.102***	0.0813***	0.108***	0.0618*	0.0631	0.0543	0.0666*
	(0.0261)	(0.0279)	(0.0296)	(0.0260)	(0.0358)	(0.0390)	(0.0428)	(0.0360)
Observations	2045	2045	2035	2045	2045	2045	2035	2045
R^2	0.231	0.256	0.338	0.230	0.0800	0.0771	0.121	0.0742

*** p<0.01, ** p<0.05, * p<0.1

Robust standard errors in parentheses

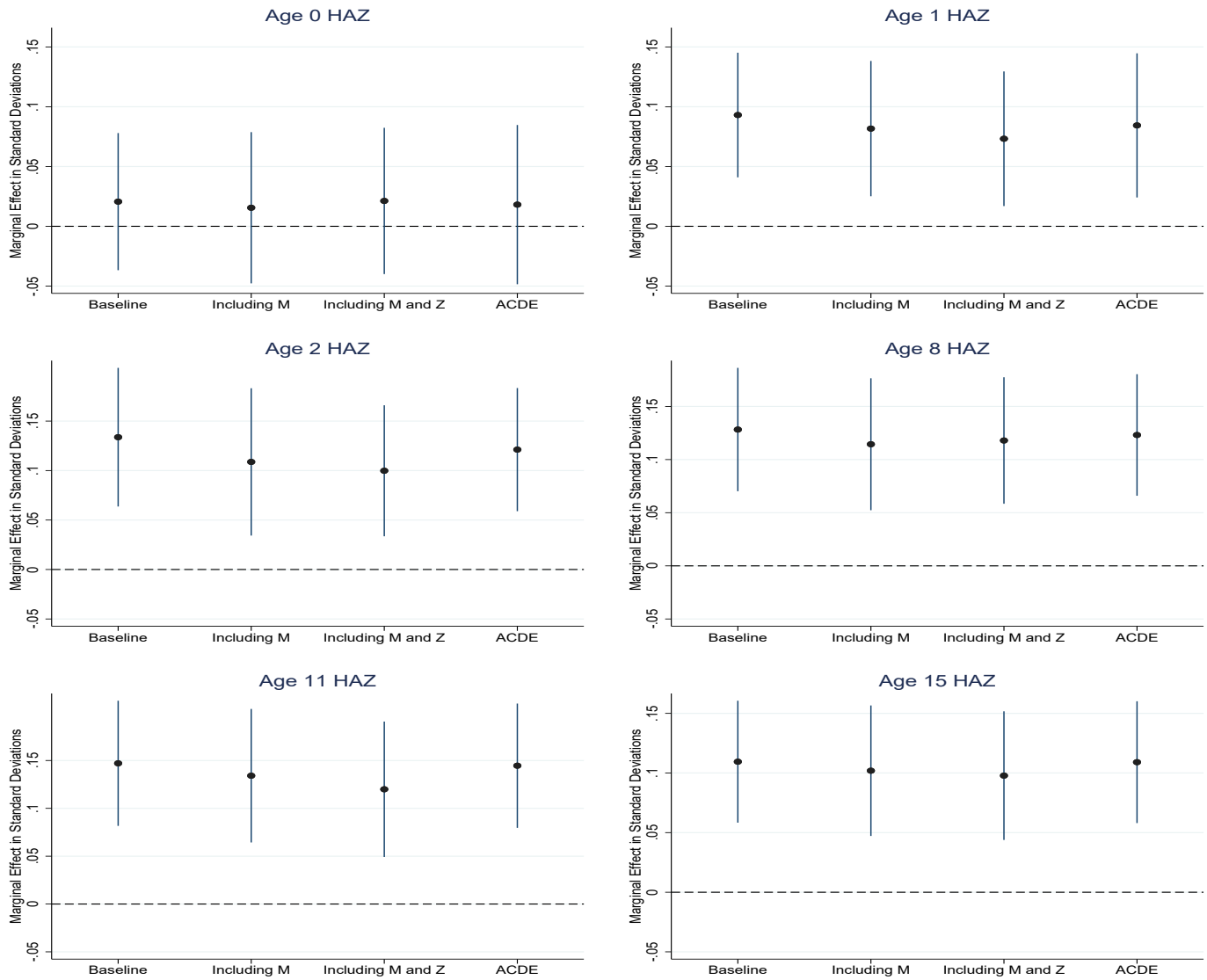
Baseline refers to a regression with only pretreatment controls included. "'Including M" includes pretreatment controls and the mediator. "Including M and Z" pretreatment controls, mediator, and intermediating confounders.

Table B.11: Transmission of Maternal Height to Child Health, Controlling for Socioeconomic Characteristics and Birthweight—Coefficients Corresponding to Figure 4

	Panel A: Child Height Outcomes					
	(1) Birth HAZ	(2) Age 1 HAZ	(3) Age 2 HAZ	(4) Age 8 HAZ	(5) Age 11 HAZ	(6) Age 15 HAZ
Mother's height	-0.0256	0.0444*	0.0815**	0.107***	0.111***	0.0813***
	(0.0276)	(0.0266)	(0.0330)	(0.0331)	(0.0381)	(0.0299)
Birth Weight (g)	0.00157***	0.000929***	0.000591***	0.000282***	0.000241***	0.000318***
	(0.0000749)	(0.0000623)	(0.0000766)	(0.0000776)	(0.0000917)	(0.0000695)
Observations	2988	2593	2457	2202	2013	2035
IV F-stat						
	Panel B: Child Weight Outcomes					
	Birth Weight	Age 1 WHZ	Age 2 WHZ	Age 8 ZBMI	Age 11 ZBMI	Age 15 ZBMI
Mother's height	29.56**	0.0381	0.0552*	0.0535	0.0407	0.0543
	(13.02)	(0.0279)	(0.0296)	(0.0357)	(0.0451)	(0.0450)
Birth Weight (g)		0.000573***	0.000494***	0.000344***	0.000401***	0.000267***
		(0.0000628)	(0.0000669)	(0.0000824)	(0.000107)	(0.0000997)
Observations	2989	2589	2458	2202	2013	2035

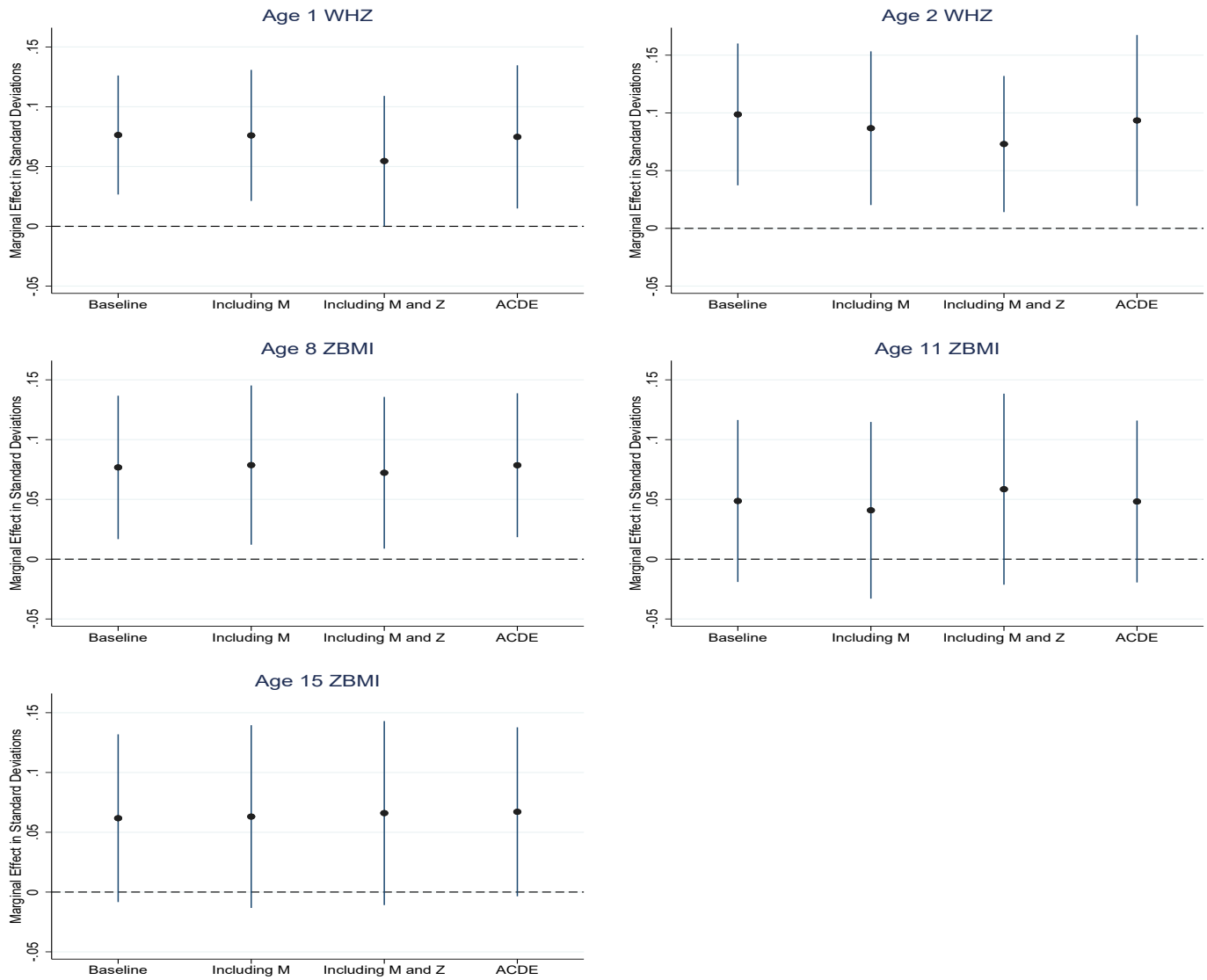
Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1
All controls from Table 4 are included.
Additionally, controls include all socioeconomic characteristics and parental inputs from Table 3.

Figure B.16: Comparison of Conditional Marginal Effect with ACDE of Maternal Height on Child Height Outcomes across Ages Using Birthweight as Mediator



Baseline refers to a regression with only pretreatment controls included. "'Including M" includes pretreatment controls and the mediator. "Including M and Z" pretreatment controls, mediator, and intermediating confounders.

Figure B.17: Comparison of Conditional Marginal Effect with ACDE of Maternal Height on Child Weight Outcomes across Ages Using Birthweight as Mediator



Baseline refers to a regression with only pretreatment controls included. "'Including M" includes pretreatment controls and the mediator. "Including M and Z" pretreatment controls, mediator, and intermediating confounders.

Table B.12: Comparison of Conditional Marginal Effect with ACDE of Mother Height on Child Height and Weight Outcomes Using Birthweight as a Mediator

	Age 1 HAZ				Age 1 WHZ			
	Baseline	Including M	Including M and Z	ACDE	Baseline	Including M	Including M and Z	ACDE
Mother's Height	0.0931***	0.0817***	0.0732**	0.0844***	0.0764***	0.0760***	0.0546**	0.0748**
	(0.0266)	(0.0289)	(0.0287)	(0.0261)	(0.0254)	(0.0280)	(0.0278)	(0.0321)
Observations	2996	2996	2599	2996	2980	2980	2595	2980
R^2	0.121	0.151	0.208	0.115	.	.	0.00925	.
	Age 2 HAZ				Age 2 WHZ			
Mother's Height	0.134***	0.109***	0.0998***	0.121***	0.0971***	0.0868**	0.0730**	0.0935***
	(0.0357)	(0.0380)	(0.0338)	(0.0346)	(0.0312)	(0.0340)	(0.0301)	(0.0340)
Observations	2604	2604	2461	2604	2606	2602	2459	2602
R^2	0.0867	0.177	0.231	0.0922	.	.	0.0314	.
	Age 8 HAZ				Age 8 ZBMI			
Mother's Height	0.128***	0.114***	0.118***	0.123***	0.0768**	0.0787**	0.0723**	0.0786**
	(0.0296)	(0.0317)	(0.0304)	(0.0292)	(0.0306)	(0.0340)	(0.0324)	(0.0307)
Observations	2210	2210	2205	2210	2210	2210	2205	2210
R^2	0.134	0.190	0.222	0.133	.	.	0.00366	.
	Age 11 HAZ				Age 11 ZBMI			
Mother's Height	0.147***	0.134***	0.120***	0.145***	0.0487	0.0409	0.0585	0.0482
	(0.0334)	(0.0356)	(0.0361)	(0.0331)	(0.0346)	(0.0377)	(0.0408)	(0.0345)
Observations	2133	2133	2016	2133	2133	2133	2016	2133
R^2	0.0536	0.109	0.212	0.0527	0.0928	0.107	0.118	0.0928
	Age 15 HAZ				Age 15 ZBMI			
Mother's Height	0.109***	0.102***	0.0978***	0.109***	0.0618*	0.0631	0.0660*	0.0671*
	(0.0261)	(0.0279)	(0.0275)	(0.0261)	(0.0358)	(0.0390)	(0.0393)	(0.0360)
Observations	2045	2045	2038	2045	2045	2045	2038	2045
R^2	0.231	0.256	0.290	0.231	0.0800	0.0771	0.0847	0.0736

*** p<0.01, ** p<0.05, * p<0.1

Robust standard errors in parentheses

Baseline refers to a regression with only pretreatment controls included. "'Including M" includes pretreatment controls and the mediator. "Including M and Z" pretreatment controls, mediator, and intermediating confounders.

Table B.13: Nonlineariy in Marginal Effect on Child Height with Pooled Data

	Maternal Grandfather
	(1) Maternal Height
Mother's Height	0.0853***
	(0.0237)
Mother's Height × Child Age	0.00912
	(0.00692)
Mother's Height × Child Age × Child Age	-0.000516
	(0.000367)
Child Age	-2.450*
	(1.061)
Child Age × Child Age	0.115*
	(0.0557)
Constant	-13.19***
	(3.588)
Observations	11342

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Figure B.18: Nonlineariy in Marginal Effect on Child Height with Pooled Data

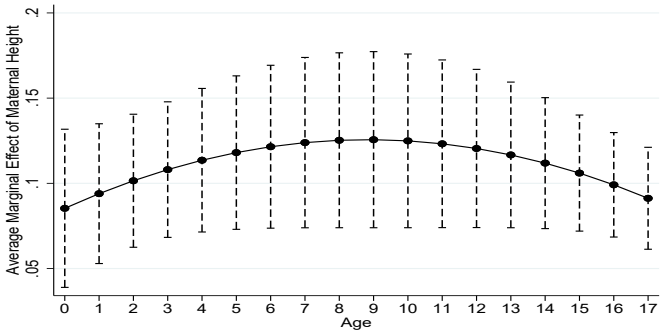


Table B.14: Maternal Transmission to Girl Height—Coefficients Corresponding in Figure 5

	Panel A: Child Height Outcomes				
	(1) Age 1 HAZ	(2) Age 2 HAZ	(3) Age 8 HAZ	(4) Age 11 HAZ	(5) Age 15 HAZ
Mother's height	0.0757	0.102	0.172**	0.0921	0.122**
	(0.0562)	(0.0656)	(0.0794)	(0.0573)	(0.0474)
Observations	1216	1164	1041	975	972
	Panel A: Child Weight Outcomes				
	(1) Age 1 WHZ	(2) Age 2 WHZ	(3) Age 8 ZBMI	(4) Age 11 ZBMI	(5) Age 15 ZBMI
Mother's height	0.0191	0.0125	0.0561	-0.0102	-0.00536
	(0.0520)	(0.0537)	(0.0670)	(0.0575)	(0.0607)
Observations	1214	1164	1041	975	972

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1
All controls from Table 4 are included.
Additionally, controls include all socioeconomic characteristics and parental inputs from Table 3.

Table B.15: Maternal Transmission to Boy Height—Coefficients Corresponding in Figure 5

	Panel A: Child Height Outcomes				
	(1) Age 1 HAZ	(2) Age 2 HAZ	(3) Age 8 HAZ	(4) Age 11 HAZ	(5) Age 15 HAZ
Mother's height	0.0188	0.0630	0.0924***	0.130***	0.0407
	(0.0297)	(0.0388)	(0.0339)	(0.0493)	(0.0358)
Observations	1377	1293	1161	1038	1063
	Panel A: Child Weight Outcomes				
	(1) Age 1 WHZ	(2) Age 2 WHZ	(3) Age 8 ZBMI	(4) Age 11 ZBMI	(5) Age 15 ZBMI
Mother's height	0.0479	0.0730**	0.0621	0.0539	0.103*
	(0.0314)	(0.0357)	(0.0395)	(0.0591)	(0.0556)
Observations	1375	1294	1161	1038	1063

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1
All controls from Table 4 are included.
Additionally, controls include all socioeconomic characteristics and parental inputs from Table 3.

Table B.16: Maternal Transmission to Girl Height by Early/Late Menarche—Coefficients Corresponding to Figure 6

	Estimated Average Marginal Effects
Average Marginal Effect of Mother's Height	
Menache 2+ yrs post 1994 survey=0	0.0339
	(0.0646)
Menache 2+ yrs post 1994 survey=1	0.118**
	(0.0599)
Observations	892
	Estimated Coefficients
Mother's height	0.0339
	(0.0646)
Age at menarche	-0.611***
	(0.0509)
Menache 2+ yrs post 1994 survey=1 × Mother's height	0.0845
	(0.0709)
Menache 2+ yrs post 1994 survey=1	-12.72
	(10.66)
Birth Weight (g)	0.000371***
	(0.000127)
Observations	892

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1
All controls from Table 4 are included.
Additionally, controls include all socioeconomic characteristics and parental inputs from Table 3.

Table B.17: Maternal Transmission to Boy Height by Early/Late Menarche—Coefficients Corresponding to Figure 6

	Calculated Average Marginal Effects			
	Shaving	Voice Change	Underarm Hair	High Pubic
Mother's Height				
Begun shaving=0	0.0389			
	(0.0377)			
Begun shaving=1	0.0375			
	(0.0602)			
Voice changed=0		0.00759		
		(0.0836)		
Voice changed=1		0.0438		
		(0.0369)		
Visible underarm hair=0			0.0453	
			(0.0508)	
Visible underarm hair=1			0.0487	
			(0.0379)	
Has high level of pubic hair=0				0.00636
				(0.0525)
Has high level of pubic hair=1				0.0453
				(0.0351)
Observations	1056	1059	1056	1041
	Estimated Coefficients			
	Shaving	Voice Change	Underarm Hair	High Pubic
Mother's Height	0.0389	0.00759	0.0453	0.00636
	(0.0377)	(0.0836)	(0.0508)	(0.0525)
Begun shaving=1 × Mother's Height	-0.00139			
	(0.0593)			
Begun shaving=1	0.331			
	(8.932)			
Voice changed=1 × Mother's Height		0.0362		
		(0.0868)		
Voice changed=1		-5.386		
		(13.07)		
Visible underarm hair=1 × Mother's Height			0.00343	
			(0.0528)	
Visible underarm hair=1			-0.179	
			(7.959)	
Has high level of pubic hair=1 × Mother's Height				0.0390
				(0.0512)
Has high level of pubic hair=1				-5.579
				(7.730)
Observations	1056	1059	1056	1041

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1
All controls from Table 4 are included.
Additionally, controls include all socioeconomic characteristics and parental inputs from Table 3.

Appendix C Results with Lasso Selection of IVs

Figure C.1: Marginal Effect of Maternal Health on Child Health across Ages

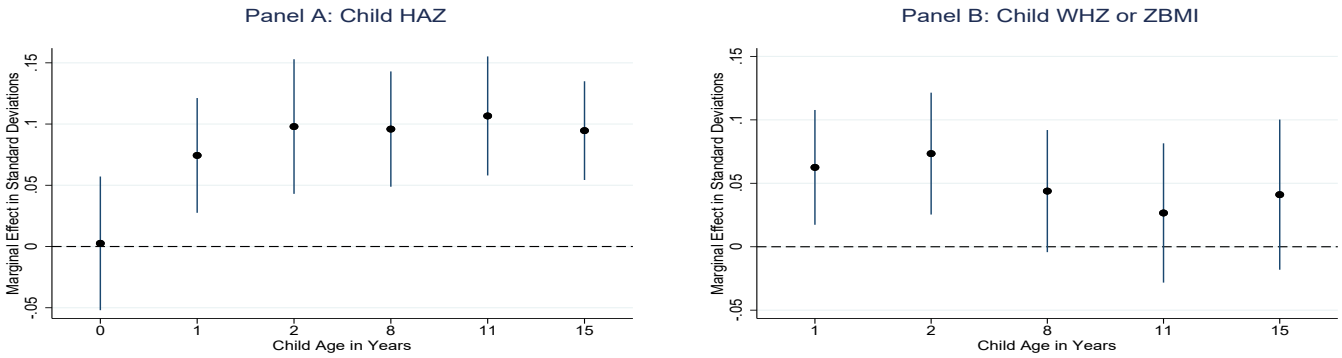
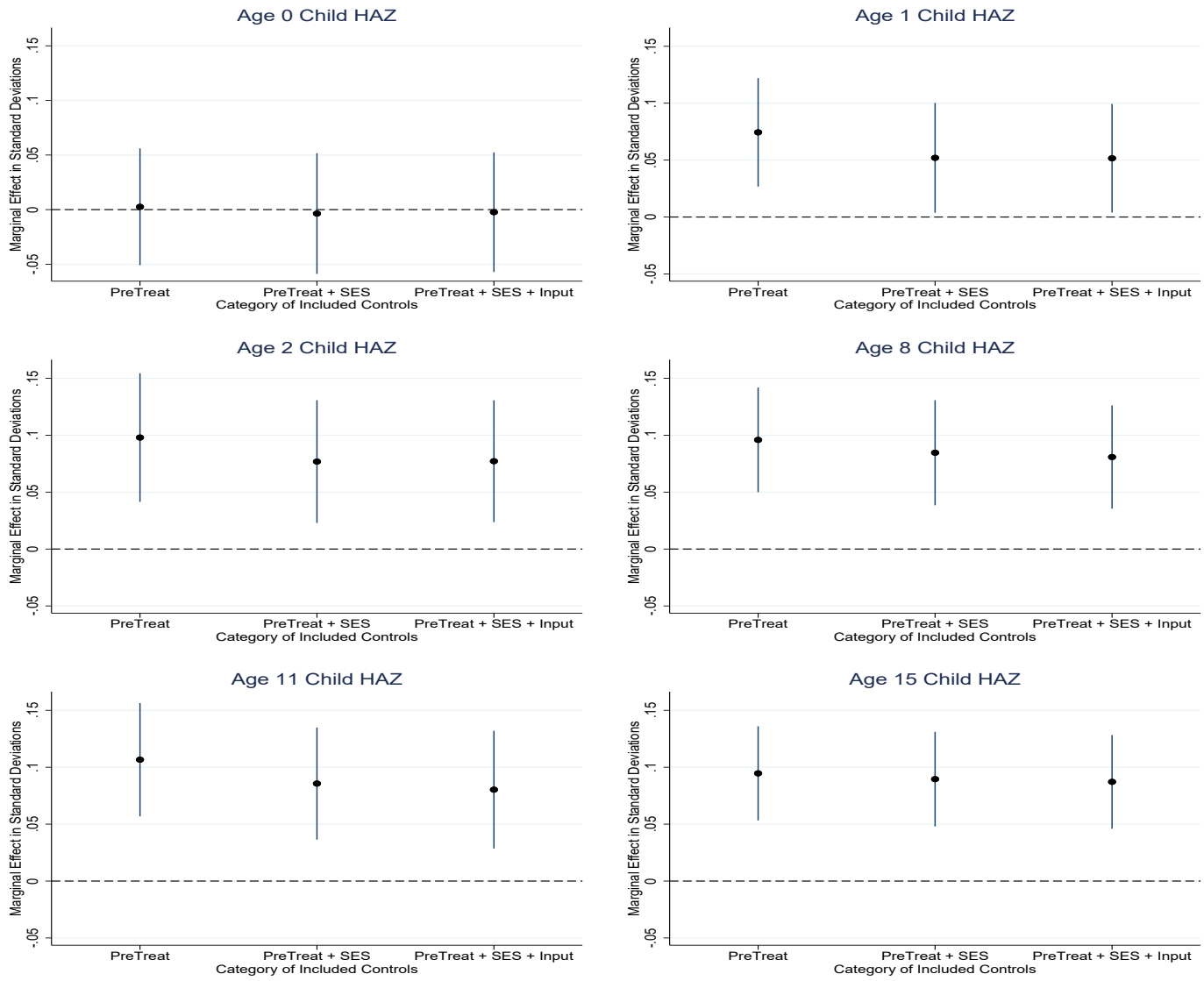
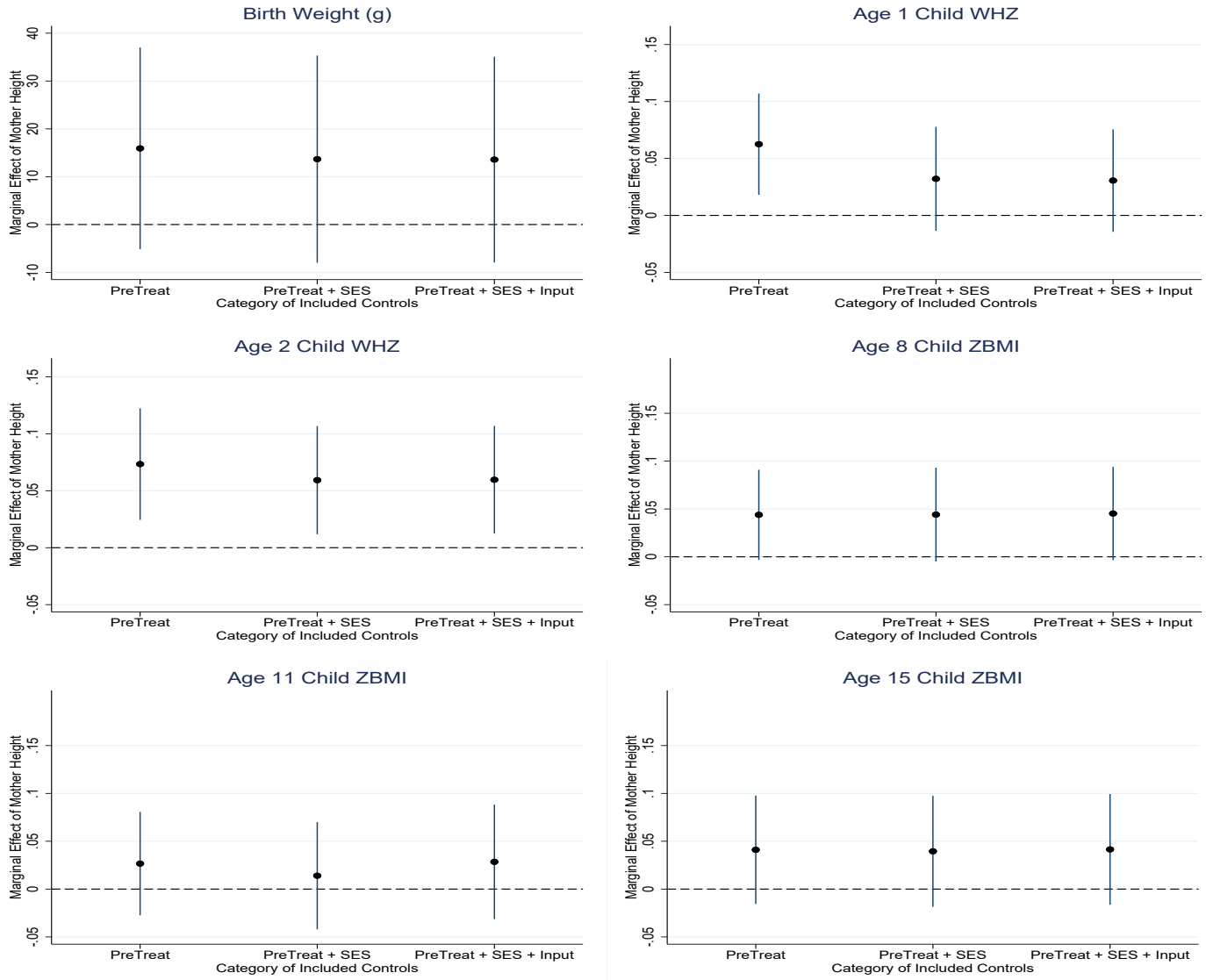


Figure C.2: Marginal Effect of Maternal Health on Child Height Outcomes across Ages—Controlling for Different Levels of Socioeconomic Characteristics



Note: *PreTreat* includes pretreatment controls, X^a_{ijb} , *SES* includes socioeconomic controls (Column 1 of Table 3), and *Input* includes parental input variables (Column 2 of Table 3).

Figure C.3: Marginal Effect of Maternal Health on Child Weight Outcomes across Ages—Controlling for Different Levels of Socioeconomic Characteristics



Note: The vertical axis unit at birth is grams while that at other ages is standard deviations

Note: *PreTreat* includes pretreatment controls, X^a_{ijb} , *SES* includes socioeconomic controls (Column 1 of Table 3), and *Input* includes parental input variables (Column 2 of Table 3).

Figure C.4: Marginal Effect of Maternal Health on Child Health Controlling for Socioeconomic Characteristics and Birthweight

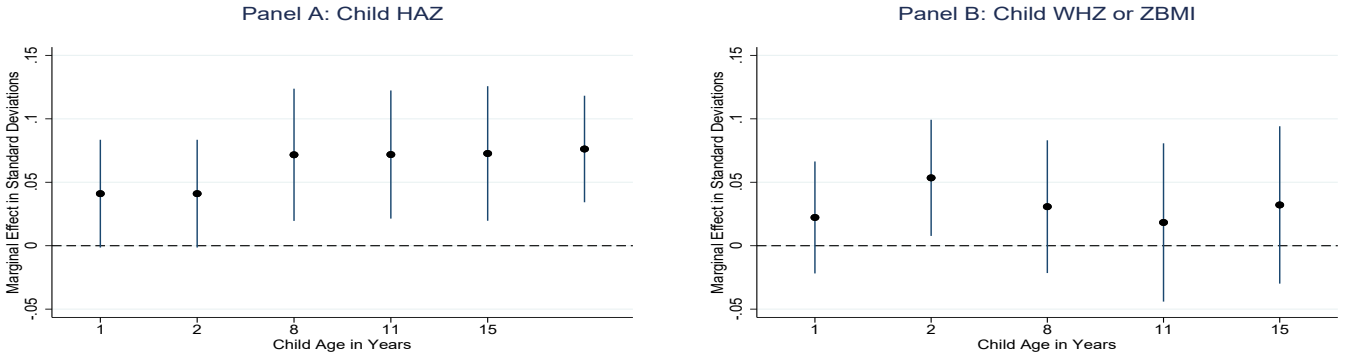


Figure C.5: Marginal Effect of Maternal Health on Child Health for Boys and Girls Separately Controlling for Socioeconomic Characteristics and Birthweight

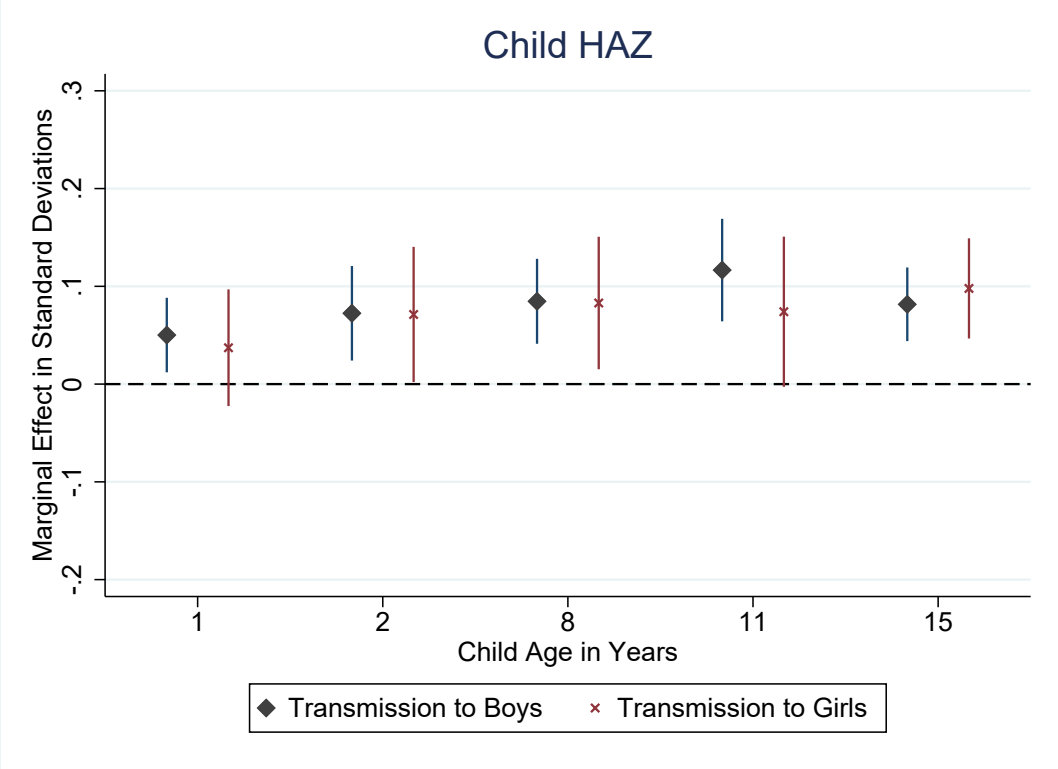


Figure C.6: Marginal Effect of Maternal Health on Age 11 Girl HAZ Depending on Probable Growth Stage Controlling for Socioeconomic Characteristics and Birthweight

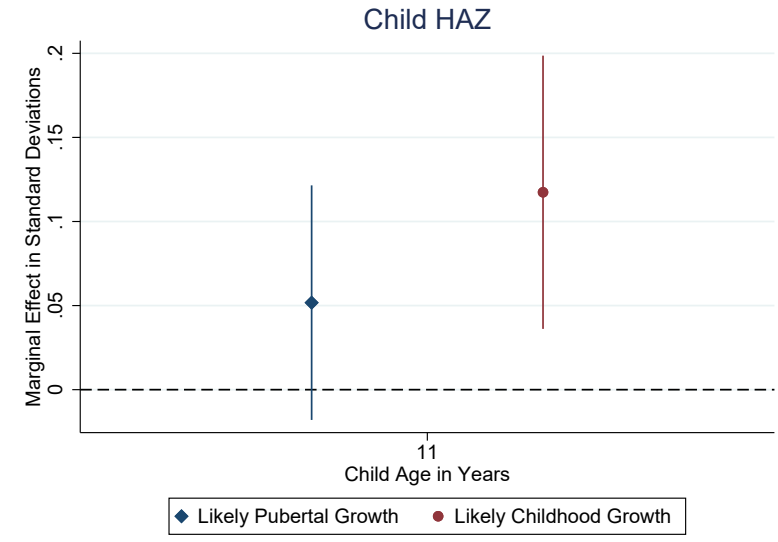
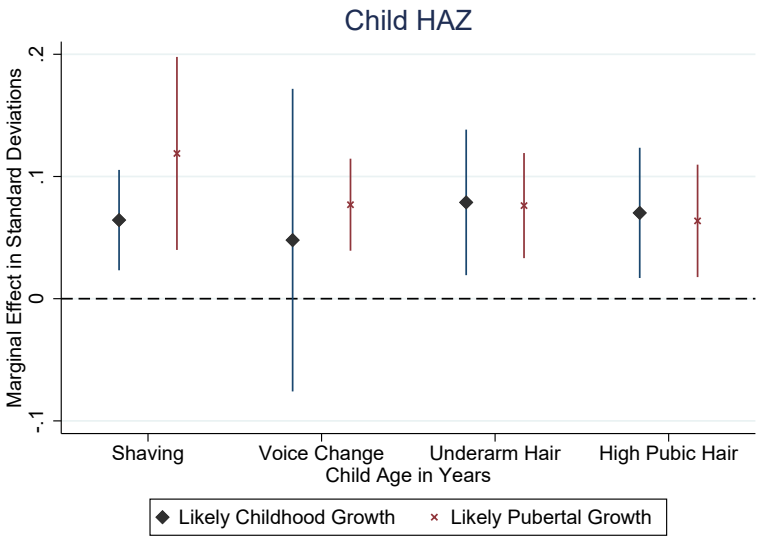


Figure C.7: Marginal Effect of Maternal Health on Age 15 Boy HAZ Depending on Probable Growth Stage Controlling for Socioeconomic Characteristics and Birthweight



Appendix D Results with Lasso Selection of PCA-IVs

Figure D.1: Marginal Effect of Maternal Health on Child Health across Ages

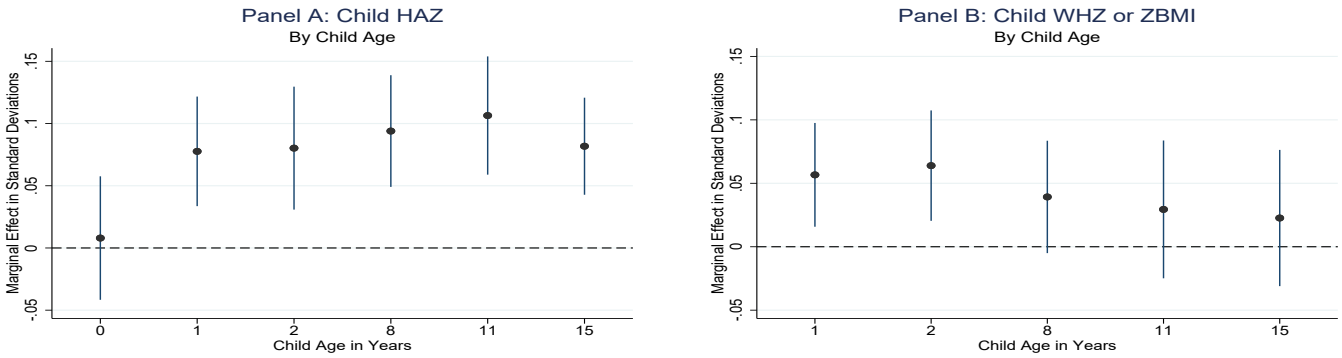
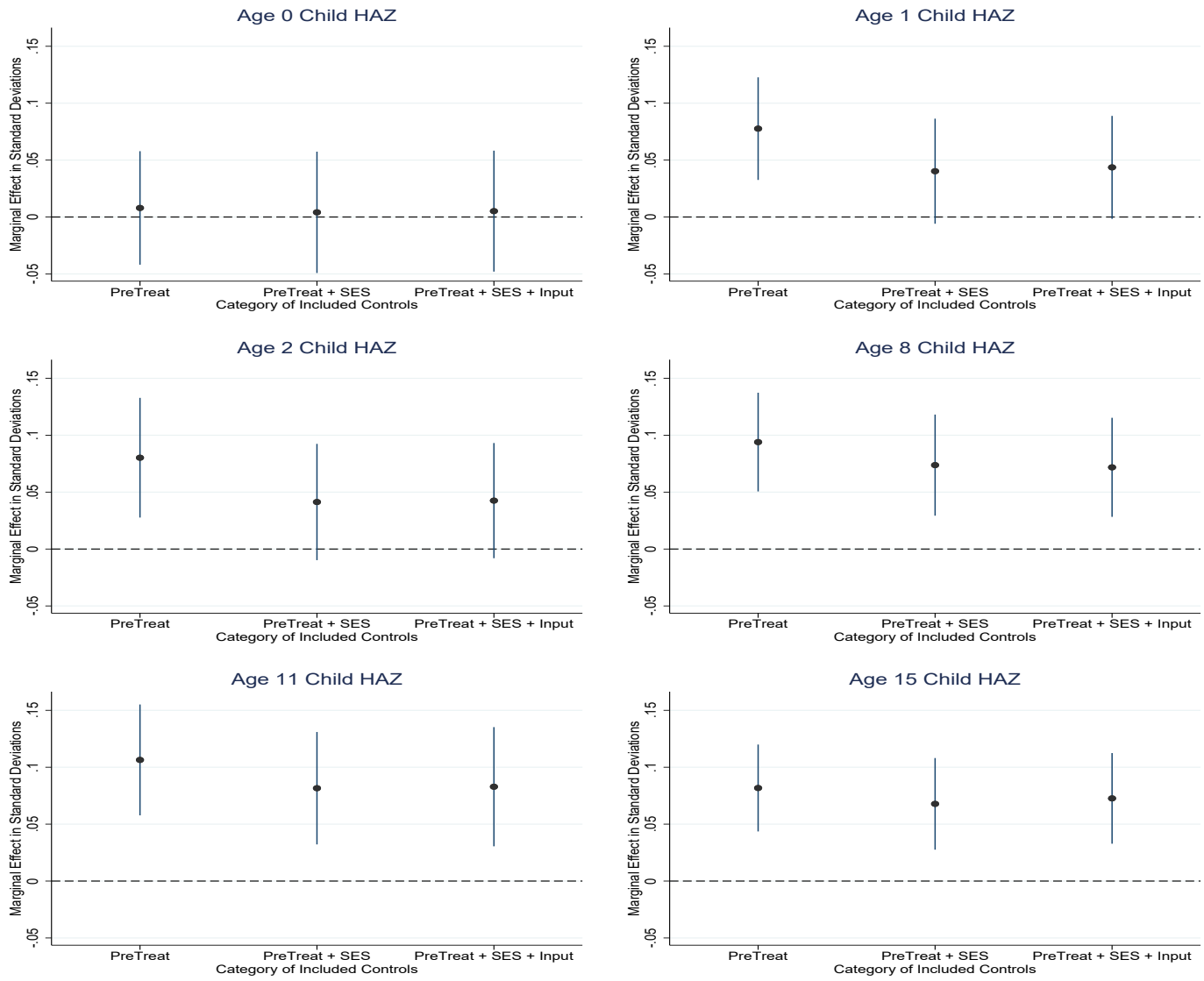
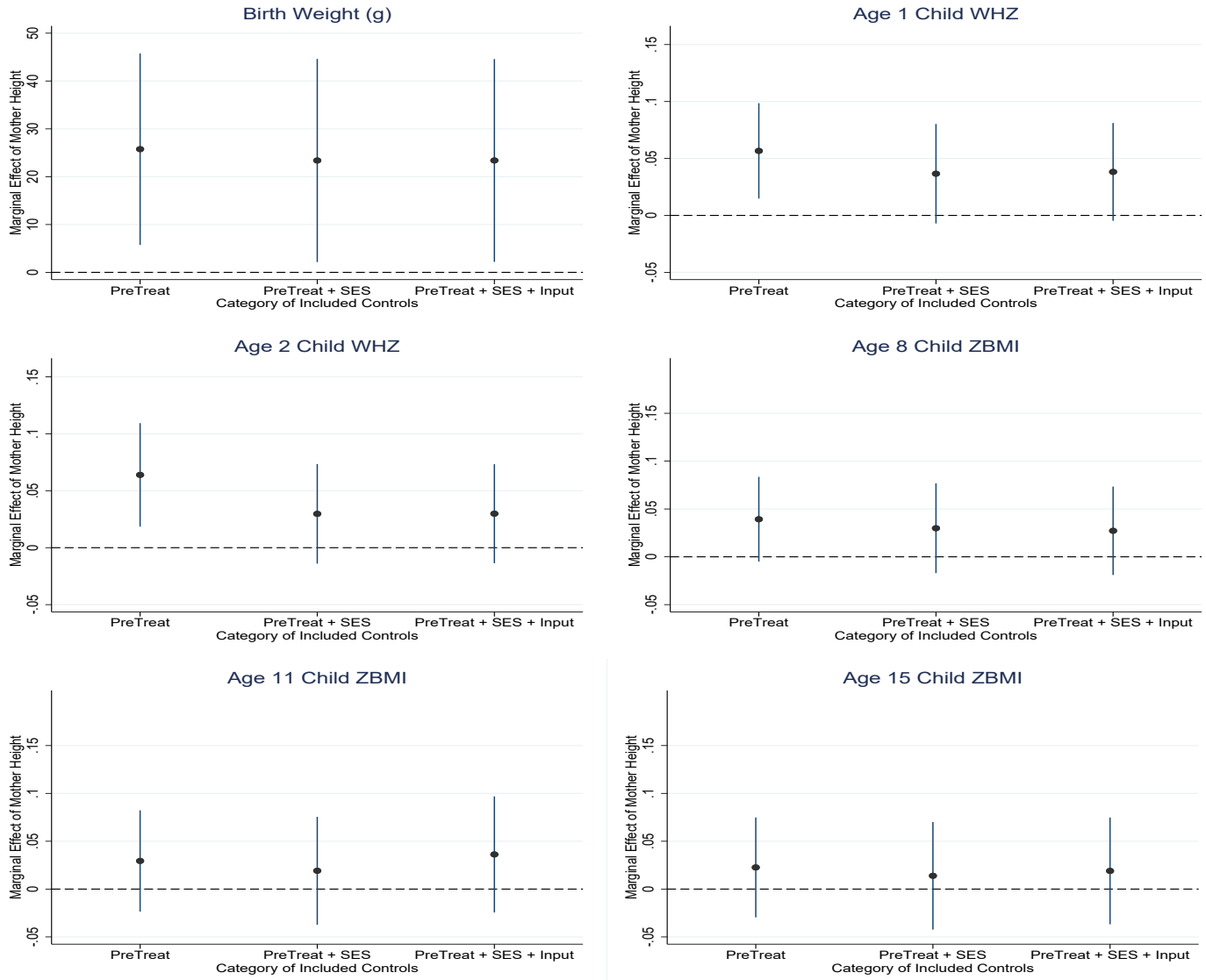


Figure D.2: Marginal Effect of Maternal Health on Child Height Outcomes across Ages—Controlling for Different Levels of Socioeconomic Characteristics



Note: *PreTreat* includes pretreatment controls, X^a_{ijb} , *SES* includes socioeconomic controls (Column 1 of Table 3), and *Input* includes parental input variables (Column 2 of Table 3).

Figure D.3: Marginal Effect of Maternal Health on Child Weight Outcomes across Ages—Controlling for Different Levels of Socioeconomic Characteristics



Note: The vertical axis unit at birth is grams while that at other ages is standard deviations

Note: *PreTreat* includes pretreatment controls, X^a_{ijb} , *SES* includes socioeconomic controls (Column 1 of Table 3), and *Input* includes parental input variables (Column 2 of Table 3).

Figure D.4: Marginal Effect of Maternal Health on Child Health Controlling for Socioeconomic Characteristics and Birthweight

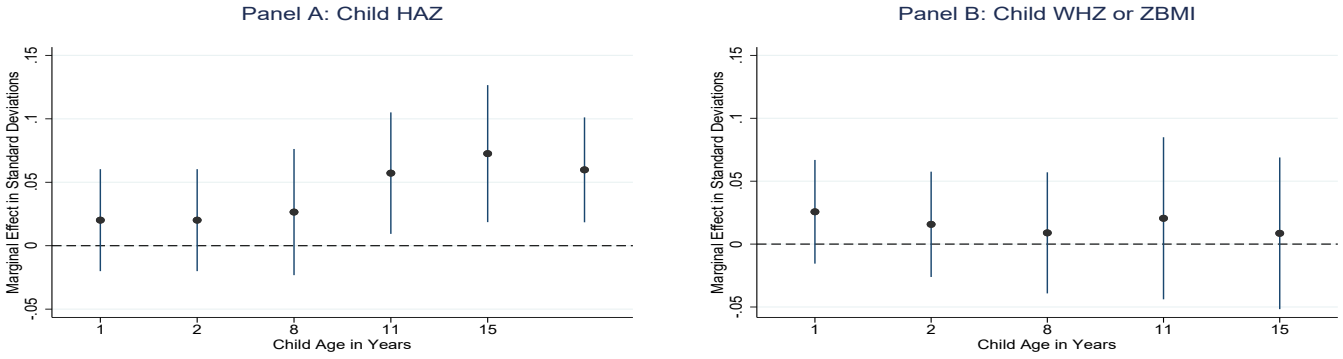


Figure D.5: Marginal Effect of Maternal Health on Child Health for Boys and Girls Separately Controlling for Socioeconomic Characteristics and Birthweight

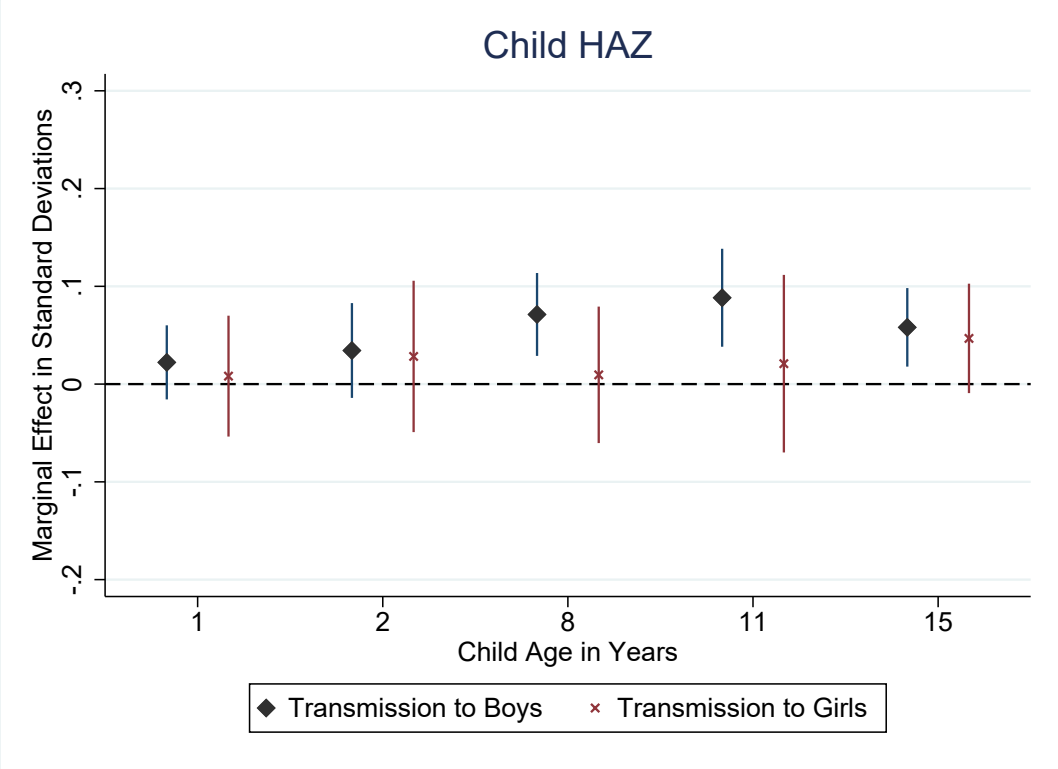


Figure D.6: Marginal Effect of Maternal Health on Age 11 Girl HAZ Depending on Probable Growth Stage Controlling for Socioeconomic Characteristics and Birthweight

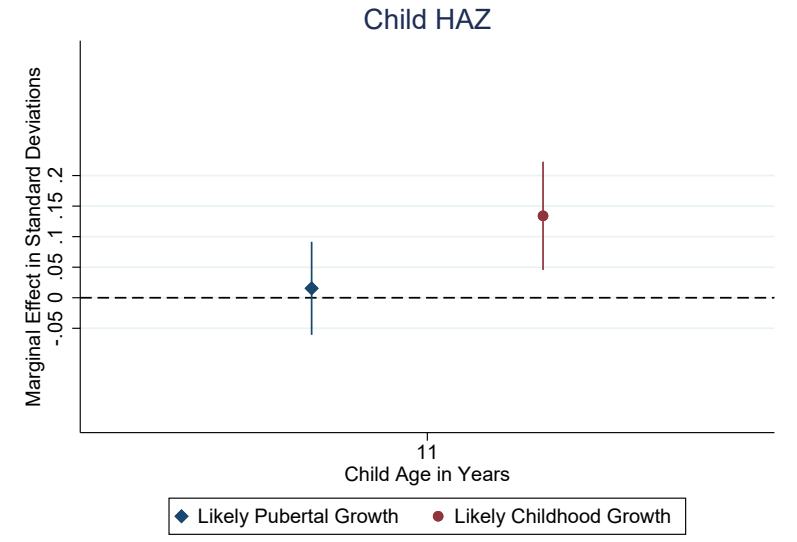


Figure D.7: Marginal Effect of Maternal Health on Agw 15 Boy HAZ Depending on Probable Growth Stage Controlling for Socioeconomic Characteristics and Birthweight