Grandmothers and Grandsons: Multigenerational effects of drought exposure in Peru

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Abstract

This paper examines the multigenerational effects of maternal grandmothers' exposure to drought during pregnancy, using a unique cohort study of Peruvian children and their families. I find that drought exposure has a persistent negative impact on the health stock of their daughter and grandchildren. Grandchildren have a lower height-for-age, first becoming apparent in early childhood and persisting through adolescence, with the height gap widening as they enter puberty. Additionally, grandchildren display lower early-life weight-for-age, however this effect diminishes as children age. These effects are strongest for grandsons, and are isolated to grandmothers living in rural areas during exposure, with exposure during early pregnancy having the largest impact. The first generation are also affected, with mothers being shorter in stature in adulthood. Mediation analysis indicates that effects are transmitted across generations through a biological channel rather than an economic channel, with mothers' long-term physical health acting as the primary mediator.

JEL codes: I10, J13, O15, Q54
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1 Introduction

There is a well established literature addressing the effect of prenatal shocks on life-long health and human capital outcomes (See Almond & Currie, 2011; Almond et al., 2018; Currie & Vogl, 2013, for reviews of the literature). It builds on the "foetal origins" hypothesis (Barker, 1990), which posits that the intrauterine environment is critical for long-term development, with shocks and investments experienced during this period having effects which persist long after birth, through "programming" the expression of parts of the genome crucial for healthy growth and cognitive function (Petronis, 2010). Furthermore, there is a growing cross-discipline literature that posits that effects are not limited to the generation exposed to these insults, but can echo down to subsequent generations (Aiken & Ozanne, 2014; Doyle & Jernström, 2023; Drake & Liu, 2010; East & Page, 2020).

Although there is an established body of evidence derived from lab-based animal studies that this developmental programming can affect subsequent non-exposed generations (Aiken & Ozanne, 2014), as well as ample evidence of strong correlations in health, educational attainment, and socio-economic outcomes across generations (Almond et al., 2012; Behrman & Rosenzweig, 2002; Bevis & Barrett, 2015; Bhalotra & Rawlings, 2013; Currie & Moretti, 2003, 2007; Emanuel et al., 1992), causal evidence of multigenerational effects within humans is very limited. Although this is in-part due to practical data limitations (Almond et al., 2018), it is also due to the difficulty of disentangling a causal effect from other confounding factors across generations (East & Page, 2020). Furthermore it can be difficult to identify the mechanisms underpinning the transmission of these effects across generations, given many commonly experienced shocks are correlated with background characteristics.

Understanding the potential for multigenerational effects has important policy implications. If a negative effect of a shock experienced by one generation has a lasting impact on the next generation, even in the absence of further shocks, it is likely that policy-makers do not fully account for these consequences for later generations, underestimating the true cost of shocks, as well as the cost-benefit ratio of any subsequent policy interventions aimed at mitigation (Doyle & Jernström, 2023). It is therefore of benefit to quantify the presence and magnitude of cross-generational effects, as well as identify the likely underlying channels of transmission, to inform future mitigation strategies.

Using novel data from the Peruvian sample of the Young lives study including the birth location and date of the mother of respondents, I match external climate data to identify their prenatal exposure to drought. I find that the exposure of a gestating grandmother to a drought shock has a negative impact on the long-term health of her daughter, who is 0.75cm shorter on average in adulthood than non-exposed individuals. This effect is also transmitted to her grandchild, who is also less healthy. Considering the dynamic effects, exposure is associated with grandchildren having a lower height-for-age z-score

(HAZ), an effect that is persistent from early childhood into late adolescence (between -0.076 and -0.173 S.D. across ages 5-15). While the grandchildren of exposed grandmothers also display a lower weight-for-age z-score (WAZ) in early childhood (-0.179 S.D. and -0.109 S.D. at age 1 and 5, respectively), this impact diminishes by age 8. Effects are also realised at the extensive margin, with shock exposure being associated with a higher incidence of stunting in mid-childhood and adolescence, and a higher probability of being classed as underweight in early childhood. Given weight is more sensitive to current health inputs, while height better captures the long-term health stock of an individual, this indicates that prenatal drought exposure can have a lasting multigenerational impact beyond the initial health endowment. This suggests the effects on subsequent generations may not be easily addressed by post-exposure investments, highlighting the importance of mitigating the initial shock exposure. In contrast to health outcomes, I find little evidence of a multigenerational effect of drought exposure on cognitive ability or educational attainment.

This chapter contributes to the nascent literature documenting causal multigenerational effects in three key ways. First, it exploits exogenous spatial and temporal variation in exposure to drought conditions experienced by the grandmother (zero generation) while pregnant, to establish the multigenerational effect of prenatal drought exposure on the health and cognitive outcomes of i) their offspring, who was exposed in-utero (first generation) and ii) of their grand-offspring (second generation).[1] Second, this study examines the dynamics of how these multigenerational effects manifest in the second generation from birth into adolescence, as well as exploring the potential for heterogeneity in the transmission of effects. Third, through formal mediation analysis, I provide evidence that effects are predominantly transmitted through a biological pathway, impacting the long-term health of the exposed first generation, however results may suggest that either a direct effect (via an effect on the second generation germ-line) or the role of other unobserved environmental/economic pathways cannot be fully discounted.

These multigenerational effects on health outcomes are driven by the impact of exposure of grandmothers living in rural areas, with a large and significant negative effect on the HAZ of their grandchild, first appearing at age 5 and remaining into late adolescence, compared to a null and insignificant effect for grandmothers living in urban areas at time of exposure. Early impacts on WAZ are also isolated to the offspring of rural grandmothers. This suggests that the direct effect of exposure is larger in rural areas, where a higher proportion of households would be reliant on local food sources and agriculture-related

[1]This follows commonly used notation from the epidemiology literature, set out by Skinner (2008), which describes a gestating female (F0 or zero generation) being exposed to an environmental insult, resulting in the embryo (F1 or first generation) and potentially the germ-line/reproductive cells (F2 or second generation) being exposed in-utero. Following Skinner (2008) and Drake and Liu (2010), I refer to effects on the F1 and F2 generation as "multigenerational", rather than "transgenerational", which is reserved for the impacts on the F3 generation (i.e. the great-grandchild of the F0 generation) and beyond.

income, likely directly impacting resources available for the first generation in-utero or immediately after birth. Additionally, I assess if the effects of exposure during the grandmothers' pregnancy differ when shock exposure is disaggregated by trimester. Results indicate that both the effects on grandchildren HAZ and early years WAZ are strongest for exposure to a shock during the first trimester, consistent with existing evidence that exposure earlier in the pregnancy has the largest impact on second generation outcomes (Khan, 2021; Stein & Lumey, 2000).

Considering heterogeneity amongst the second generation, I look at potential sex-specific differences in the transmission of effects between boys and girls, finding that the second generation effects are primarily exhibited in grandsons of exposed grandmothers, compared with small, insignificant effects for granddaughters. This is consistent with previous second generation findings where effects on HAZ and WAZ are isolated to the grandsons of shock-exposed maternal grandmothers (Fung & Ha, 2010). Additionally, using self-reported data on indicators of puberty, I assess how shock exposure interacts with entering in to pubertal growth in adolescent years. Results suggest that the gap in height-for-age between the grandchildren of unexposed and exposed grandmothers widens once in the pubertal stage of growth, with a large negative effect on HAZ estimated for those reporting signs of puberty at age 12, compared with a smaller insignificant impact for those not reporting signs of pubertal growth.

Finally, I conduct formal mediation analysis, estimating the average controlled direct effect (Acharya et al., 2016; Joffe & Greene, 2009; VanderWeele, 2009) of shock exposure, net of the effect of the shock operating indirectly through some mediator. Results suggest that measures of the household environment experienced by the second generation seem to account for very little of the effect of shock exposure, while the long-term health of the first generation accounts for the whole of the baseline effect for outcomes at almost all ages, supporting a biological transmission of health across generations as the primary mechanism.

The rest of this study is as follows: Section 2 provides a summary of the evidence for multigenerational effects of prenatal shocks, as well examining the likely biological and environmental mechanisms which account for these effects. Section 3 summarises the data sources, defining the key variables and providing sample descriptive statistics, with the empirical strategy described in Section 4. Results, including additional analyses of heterogeneous effects, sensitivity, and robustness checks, are provided in Section 5. Finally, results from the mediation analysis are presented in Section 6, and Section 7 concludes.

2 Background

2.1 Literature Review

An extensive literature of "first generation" studies links prenatal and early life shocks with later life outcomes within a single generation (Almond & Currie, 2011; Almond et al., 2018; Currie & Vogl, 2013). Given evidence from animal studies that the impacts of early life shocks can echo across generations (Aiken & Ozanne, 2014), there is a clear incentive to assess the potential for this phenomenon in human studies, leading to the emergence of a "second generation" literature. Recent reviews of this burgeoning literature are provided by East and Page (2020) and Doyle and Jernström (2023) for the impacts on health and education/labour market outcomes respectively, therefore I will summarise only those most relevant to this analysis.

While a large body of cross-disciplinary evidence correlates maternal birth-weight and disease exposure with offspring birth-weight and educational outcomes (Almond et al., 2012; Bhalotra & Rawlings, 2013; Bhalotra & Rawlings, 2011; Currie & Moretti, 2007; Drake & Walker, 2004; Emanuel et al., 1992), these likely reflect a wide range of causal mechanisms, and cannot separate environmental or epigenetic effects from cross-child variation in growth due to inherited genetic endowments. Additionally, some studies exploit twins or adoptees to control for genetic inheritance and isolate the impact of early life shocks (Royer, 2009; Thompson, 2014), however it is likely that these studies may have limited external validity, and are difficult to conduct outside of high-income, data-rich contexts.

Alternatively, several works attempt to identify a causal relationship between conditions experienced by the first generation in-utero (that is, during the grandmother's pregnancy) and their children's outcomes.[2] Early contributions exploit differences between cohorts exposed to famine and starvation prenatally and surrounding cohorts who were not exposed. Lumey (1992) studies the inter-generational effects of the Dutch Hunger Winter using hospital records, finding the children of mothers exposed to war-induced famine in-utero in the first and second trimester were more likely to be low birth-weight (LBW), with exposed mothers also more likely to be low birth-weight. However, a subsequent study using the same data-set found no significant relationship between maternal birth-weight and offspring birth-weight after adjusting for confounders, compared with a positive relationship for non-exposed mothers (Stein & Lumey, 2000). Painter et al. (2008) also study the Dutch hunger winter, finding women exposed in-utero become mothers at a younger age, give birth to more offspring, and have more twins than those not exposed. In contrast, male reproductivity was unaffected.

[2] A broader literature studies the causal relationship between parent and child income and educational attainment (See Black & Devereux, 2011), however these studies focus only on the effect of shocks or investments experienced post-birth, in early childhood.

Similarly, a number of studies assess the impact of in-utero exposure to the 1959-1961 Great Chinese Famine. Almond et al. (2007), while mainly focused on the cohort exposed to famine, find that women exposed to the famine in early years and whose parents subsequently migrated to Hong Kong in 1962 had a higher ratio of female to male births than unexposed native-born mothers. Distinguishing exposure for mothers and fathers, Fung and Ha (2010) find that the children of mothers exposed in-utero have lower weight- and height-for-age (HAZ and WAZ), with no significant effect of fathers' in-utero exposure. Kim et al. (2014) also find a gendered effect of exposure, with the children of mothers exposed in-utero 5-7 percentage points less likely to enter middle school. These results may indicate that maternal shock exposure is more important than that of the father, however, given the extent of the famine, maternal and paternal exposure is highly correlated, making it difficult to disentangle effects.[3] Fung and Ha (2010) also find sex-specific differences for second generation outcomes, with the effect on HAZ and WAZ of mother's in-utero exposure limited to boys, compared with a null effect for girls. A major limitation with the studies above are that famines are extreme events.[4] The estimated effects are therefore likely to suffer from selection bias, often with only survivors of extreme malnutrition, starvation, or sickness observed (Royer & Witman, 2014). Additionally, the estimates obtained by the above studies of the Dutch winter famine may be confounded by other effects of the Second World War and its aftermath.

More relevant for this study is a strand of literature exploring the impact of short-run unexpected deviations in climate conditions, including drought, experienced by the grandmother while pregnant or in the early years of the parents' life. In an early contribution, Venkataramani (2011) assesses the inter-generational transmission of health in Vietnam, as measured by height, including using an instrumental variable approach, using early life rainfall, grandparent socioeconomic status and regional fixed effects to capture non-genetic components of parent height variation. They find a strong relationship between maternal height variation and child height, while the association with paternal height is near zero under the instrumental variables approach.[5]

Using the India Household Development Survey, Khan (2021) assesses the impact of a rainfall deficit during the grandmother's pregnancy on her grandchildren's health and cognitive outcomes. They find that the grandchildren of exposed grandmothers have a lower HAZ amongst a pooled sample aged 0-5. Similarly, Hyland and Russ (2019) match DHS data in 19 Sub-Saharan countries to historical temperature and precipitation time series, finding that the children of mothers exposed in-utero to extreme drought are more

[3]Indeed, considering intensity of exposure for both parents, Li and An (2015) find negative effects of more intensive exposure to the famine on childrens' HAZ, regardless of which parent is exposed.

[4]The Great Chinese Famine for example, is estimated to have caused between 16.5-30 million deaths (Li & Yang, 2005).

[5]However, this study suffers from a weak multiple instruments problem, while it is also unclear if several instruments satisfy the exclusion restrictions required for causal interpretation.

likely to be born with low birth weight. A shortcoming of all previously mentioned studies is that they only give a snapshot of the effects, either using at-birth outcomes or by pooling respondents across a wide range of ages. This likely hides the potential for transmitted effects to alter as offspring age, either due to biological growth faltering or catch-up, or perhaps due to subsequent investments during their lifetime.

To the best of my knowledge, only two studies provide age-disaggregated results. Tafere (2017) uses the 1983-1985 Ethiopian famine as a natural experiment in a subsample of households located in famine-affected clusters of the Young Lives study. using panel data methods, they find that mothers exposed to the famine either in-utero or within the first three years of life are shorter on average and complete less schooling. Their children are also more likely to be likely to be shorter (5% less than average), with the intensity of exposure also being important. Additionally, pooled OLS results using a triple interaction between a famine-cohort dummy, number of months of early life exposure, and the survey round for each observation, show a U-shaped relationship between famine exposure duration and height as children age, although only statistically significant at ages 1 and 12.

Most closely related to this analysis, Bevis and Villa (2022) use an instrumental variable approach to estimate the potential transmission of health between mothers and children on Cebu island in the Philippines. They instrument variation in health with an array of early-life weather conditions, using a novel dimensionality reduction technique to derive a single value instrument for early-life weather variation. They find an early-life weather-induced 1cm increase in mother's adult height is associated with a persistent effect on their child's health stock (the long-term cumulative health of an individual), measured by HAZ across childhood from age 1 until age 15. They also find an effect on birthweight and early age WAZ, their measure of health flow (which remains more sensitive to current inputs, such as maternal health), however this effect diminishes and disappears by age 8.

This study expands the evidence base for the multigenerational impacts of early life shock exposure in three key ways. First, I provide evidence of the multigenerational effects of shocks experienced specifically in the place of birth of the mother. A key limitation of the previous studies is that their identification strategy relies on defining shock exposure using the place of residence of the household during interview, often assuming zero migration between the zero and second generation, or restricting analysis to never-migrating households.[6] In the context of Peru, where significant rural-urban migration has occurred in the later half of the 20th century, this would likely introduce

[6]For example, both Tafere (2017) and Khan (2021) assume no migration in Ethiopia and India, respectively. This is unlikely to hold, particularly in India, where a large proportion of women migrate for marriage (Rosenzweig & Stark, 1989). Hyland and Russ (2019) restrict their analysis to only never-migrated households, which represents less than half of their full pooled sample. Alternatively, Bevis and Villa (2022) do not have information on where mothers are born within Cebu island, therefore weather shocks are defined for the entire study area, limiting spatial variation in shock exposure.

significant measurement error.[7] Combined with data on the month and year of birth of the mother, I am able to accurately identify shock exposure of mothers in-utero, and provide further analysis of heterogeneities across urban and rural-born mothers.

Second, I will expand the limited evidence on the potential dynamic effects of transmission. Using data from a rich longitudinal cohort study, I am able to identify how the transmission of effects to the second generation presents at specific ages, rather than at a single snapshot (Hyland & Russ, 2019; Lumey, 1992), or using a pooled sample of respondents of different ages (Fung & Ha, 2010; Khan, 2021; Venkataramani, 2011). This allows me to assess how exposure impacts postnatal growth trajectories and if effects grow or diminish as offspring age. Third, I exploit rich longitudinal data to conduct an in-depth analysis of potential mediator variables (Acharya et al., 2016), providing evidence for a primary mechanism through which effects of shock exposure are transmitted from the zero to second generation. These potential mechanisms are discussed further in the following section.

2.2 Transmission Mechanisms

In estimating the multigenerational effects of early life shock exposure, I also explore the potential mechanism channels through which these effects are transmitted across generations. Following Doyle and Jernström (2023), I consider two broad channels for which shock exposure during pregnancy can transmit to future generations: i) directly via a "biological" pathway, for example through epigenetic inheritance or by affecting germ-lines; and ii) indirectly, through an "environmental" pathway, by impacting the household environment experienced by the second generation.

Within these broad channels, the exact mechanism through which the effects are primarily transmitted may vary. For the environmental channel, it is possible that in-utero shock exposure impacts the physical and cognitive development of the mother, leading to lower educational attainment and labour market outcomes (Almond et al., 2018; Black & Devereux, 2011). This could directly affect the mother's capacity for child care through reduced parenting knowledge/ability (Mani et al., 2013), or by limiting the resources available to invest in her offspring's development (Cunha & Heckman, 2007; Del Bono et al., 2016; Todd & Wolpin, 2007). Additionally, if an in-utero shock impacts maternal adulthood health, educational attainment, or socio-economic status (SES), then it may impact the quality of her chosen partner's human capital (e.g. her partner may also have poorer health or lower skills/parenting knowledge), further limiting resources available to invest in subsequent generations. For example, Behrman and Rosenzweig (2002) and Akresh et al. (2023) attribute a significant portion of the relationship between mother and child

[7] In Peru rural-to-urban migration is generally associated with a lower prevalence of stunting and improved HAZ (Escobal & Flores, 2009) but greater overweight prevalence (Rougeaux et al., 2022) for offspring, however it is unclear how migration interacts with early life shock exposure.

schooling outcomes to be driven by assortative matching. Furthermore, evidence from the economics literature shows a clear association between the attainment and SES of parents and the health and cognitive outcomes of their children (Almond et al., 2012; Behrman et al., 2017; Bevis & Barrett, 2015; Bhalotra & Rawlings, 2013; Black & Devereux, 2011; Black et al., 2005; Currie & Moretti, 2003, 2007; Royer, 2009). If this mechanism acts as the primary pathway for transmission then it is likely that effects persist across generations, by perpetuating economic or environmental disadvantage, suggesting that post-shock interventions are likely an effective way to mitigate intergenerational effects.

For the biological channel, it is possible that an in-utero shock can have a permanent effect on maternal physiology and metabolism, by altering or "programming" gene expression (Bale, 2015; Skinner, 2014). This could impact maternal health during pregnancy, by either creating an abnormal intra-uterine environment and/or altering her ability to transfer vital nutrients to her offspring (Gluckman & Hanson, 2004; Godfrey & Barker, 2000). Alternatively, it is possible that exposure could directly impact the germ cells (the gametes/reproductive cells), present within the first generation as a foetus while in-utero, and from which the second generation will be formed (Drake & Liu, 2010; Skinner, 2008).[8]

In support of this mechanism, a large body of research within epidemiology based on animal studies finds a persistence of in-utero nutritional shocks that last several generations, even if all subsequent generations are fed a normal diet and the mother is returned to a normal diet after the birth of her offspring (see Aiken & Ozanne, 2014; Drake & Liu, 2010, for details). For observational studies of humans, while it is hard to disentangle biological factors from environmental, an emerging body of work also provides suggestive evidence that the biological mechanism explains a large proportion of the transmission of health between generations (Bevis & Villa, 2022; Dabelea et al., 2000; Hyland & Russ, 2019; Ibáñez et al., 2000; Klebanoff et al., 1999; Van Den Berg & Pinger, 2016; Venkataramani, 2011). Therefore if this mechanism acts as the primary pathway for transmission then it suggests that post-shock interventions may have a limited ability to mitigate multigenerational effects, and that interventions that protect the zero and first generation individuals from the initial exposure to shocks should be prioritised.

3 Data

3.1 Young Lives

I use data from Young Lives (YL), a longitudinal cohort study of around 12,000 children and their families in four low- and middle-income countries (Ethiopia, India, Peru, and Vietnam) examining the causes and consequences of poverty (Favara et al., 2022). It consists of two cohorts: the younger cohort, born in 2000-2002, and the older cohort,

[8] See Figure A1 for a visual representation.

born in 1994-1996. This analysis focuses on the younger cohort of the Peru survey, who were first interviewed in 2002 and revisited in 2006, 2009, 2013, and 2016 – at ages 1, 5, 8, 12, and 15 respectively.[9] The Young Lives Peru study employs a multi-stage, cluster-stratified, random sampling technique, and was evaluated to be suitable for analysing the causal determinants of child welfare and their longitudinal dynamics (Escobal & Flores, 2008).

The younger cohort consists of 2052 respondent children and their households in the first round. Attrition is low given extensive tracking: by round 5 (2016) attrition due to respondent refusal, moving abroad, death, or being untraceable was 9.36%, with 1860 respondents present in round 5. My analytical sample is restricted further as information on the place of birth and birth month of the mother, required to identify her exposure to early life drought, was collected in round 4 only if the mother is still alive and residing in the same household as the child. The place and date of birth was derived for a total of 1734 mothers. I focus only on the drought exposure of maternal grandmothers, rather than paternal grandmothers, due to the practical limitation that birth location is only available for a subsample of fathers present during interview and therefore is missing for significant number of respondents in a likely non-random pattern, which could produce biased estimates. However, current evidence from the literature indicates that matrilineal transmission is generally more important for the effects of early life shock exposure (Caruso, 2015; Fung & Ha, 2010; Painter et al., 2008; Venkataramani, 2011). A further theoretical argument is that there are clearer potential transmission channels between maternal and offspring health than for paternal health if transmission occurs predominantly through a biological pathway.

Once missing outcomes and covariates are accounted for and singleton observations are dropped the analytical sample consists of 1670 mother-child pairings present in round 1 (R1). Age 1 sample summary statistics for those included in round 1 and those omitted are provided in columns (1) and (2) of Table 1. The difference in means are reported with standard errors from two-sample t-tests in brackets in column (3), and indicates that those omitted from the R1 sample do not differ significantly in terms of baseline child, mother, or household characteristics, with exception that mothers in my sample are roughly 1 year older on average than those omitted.

Notably, my sample size varies slightly across rounds as some respondents were absent during one or more interim survey rounds. Additionally, observations are set to missing in a round if their measure for either height-for-age or weight-for-age are flagged as biologically implausible following WHO standards (See Briones, 2018, for details). In my sample this explains all differences between round 1 and round 4 given data for mother's birth

[9]Additionally, 5 rounds of phone surveys were conducted throughout 2020-2021 during the global COVID-19 pandemic when younger cohort respondents were aged between 18-20 (Favara et al., 2022). As no physical health measures or cognitive ability tests could be administered, these survey waves are not considered in this analysis.

Table 1: Summary Statistics: Comparison of Baseline Characteristics

	Omitted			Attrition/unbalanced observation		
	(1)	(2)	(3)	(4)	(5)	(6)
	R1 Sample	Omitted	Diff. (2)-(1)	Balanced	Omitted	Diff. (5)-(4)
Child outcomes						
HAZ	-1.29	-1.34	-0.05	-1.26	-1.76	-0.50***
	(1.27)	(1.42)	[0.07]	(1.26)	(1.35)	[0.12]
Stunted	0.28	0.31	0.04	0.27	0.40	0.13***
	(0.45)	(0.46)	[0.03]	(0.44)	(0.49)	[0.04]
WAZ	-0.21	-0.16	0.05	-0.19	-0.51	-0.32***
	(1.17)	(1.32)	[0.07]	(1.16)	(1.28)	[0.11]
Underweight	0.07	0.09	0.02	0.06	0.15	0.09***
	(0.25)	(0.29)	[0.01]	(0.24)	(0.36)	[0.02]
Female	0.49	0.53	0.03	0.49	0.47	-0.02
	(0.50)	(0.50)	[0.03]	(0.50)	(0.50)	[0.05]
Mother outcomes						
Height (in cm)	149.96	150.29	0.33	150.01	149.08	-0.94
	(5.56)	(5.51)	[0.37]	(5.50)	(6.35)	[0.57]
Weight (in Kg)	58.70	58.35	-0.36	58.77	57.50	-1.28
	(9.94)	(9.99)	[0.66]	(9.99)	(9.17)	[1.08]
Grade attainment	7.13	7.17	0.04	7.21	6.01	-1.20***
	(4.51)	(4.73)	[0.29]	(4.52)	(4.29)	[0.45]
Age in years	27.02	25.98	-1.04***	27.08	26.10	-0.98
	(6.70)	(7.00)	[0.39]	(6.65)	(7.41)	[0.67]
HH outcomes						
HH size	5.70	5.74	0.05	5.69	5.72	0.02
	(2.36)	(2.23)	[0.13]	(2.34)	(2.57)	[0.23]
Wealth index	0.43	0.41	-0.01	0.43	0.35	-0.08***
	(0.24)	(0.24)	[0.01]	(0.23)	(0.24)	[0.02]
N	1670	382	2052	1560	110	1670

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Columns (1)-(2) and (4)-(5) provide mean values, with standard deviations in parentheses. (1) Provides baseline summary statistics for the analytical sample at round 1. column (2) presents values for those observations present in the full cohort that are omitted from the analytical sample. columns (3) and (6) provide the difference in means from a 2-sample t-test, with standard errors in square brackets.

location and month are first collected in round 4, as discussed above. therefore further attrition from the sample occurs only between round 4 and round 5. Column (4) and (5) provide age 1 summary statistics for those who appear in all rounds (balanced panel) compared with those observations which are either missing in at least one interim round or attrited between rounds 4 and 5. Column (6) indicates that there are large difference between those in the balanced panel and those dropped, in particular respondents are significantly more likely to be stunted and underweight, with significantly lower height- and weight-for age. Households have lower wealth scores and mothers have lower educational attainment (highest grade achieved). Additionally, while the difference is not statistically different from zero, mothers are also almost a 1cm shorter, 1.28Kg lighter, and 1 year younger than those in the balanced panel. If shock exposure is negatively related to second generation outcomes and to either mothers health or socioeconomic outcomes, which may play an important role as potential pathways for transmission, this suggests that excluding these observations from this analysis may downwards bias estimates of the effect of shock exposure on the outcomes of interest. Therefore for my primary specification I use the full unbalanced panel in each round. This decision is consistent with other work which provides estimates of second generation effects at different ages (Bevis & Villa, 2022; Khan, 2021). However, as shown in subsection 5.4, results remain robust to using the balanced panel.

3.2 Outcomes

3.2.1 Second Generation

The impact of exposure of mothers (first generation) to drought while in-utero during the grandmother's (zero generation) pregnancy is measured on the outcomes of the second generation children in two dimensions of human capital: health and cognitive ability.

Health is measured using anthropometric outcomes related to an individual's height and weight. Health stock is measured using child height-for-age z-scores (HAZ).[10] Child growth is seen as a high-quality indicator of child health, capturing the cumulative effect of health shocks/investments, nutrition, and environmental factors (Case & Paxson, 2008; De Onis, 2017; Martorell & Habicht, 1986). Child health flow in early years is captured by weight-for-age (WAZ), which is more susceptible to current health inputs (Bevis & Villa, 2022; WHO, 1995) than health stock. WHO reference tables for weight-for-age are provided only up to age 10, as it is considered inadequate for monitoring growth beyond childhood (De Onis et al., 2007). As such, Young Lives provide weight-for-age scores only up to round 3, when children were aged 7.5-8.5 years old (Briones, 2018). For rounds 4-5 (roughly ages 12 and 15 respectively) health flow is therefore measured as BMI-for-age z-scores (BMIAZ). However, this is an imperfect measure as it is constructed using

[10]For age 1 this is measured as length-for-age using a board.

both weight and height, and therefore may mask changes in weight if accompanied by changes in height in the same direction. Z-scores for child growth are preferred to using raw measures as they provide an indication of how a child's growth compares with that of a healthy individual of the same age and gender. Observations which are flagged as biologically implausible based on WHO standards are dropped.

Mean height-for-age (age 1-15), weight-for-age (1-8), and BMI-for-age (available for ages 1-15) are presented in Table 2. Indicator variables are also defined for a child being classed as stunted, underweight, or wasted if their age-specific z-score is less than 2 standard deviations (S.D.) from the mean for HAZ, WAZ, and BMIAZ respectively, based on WHO reference tables (De Onis, 2017; De Onis & Habicht, 1996). The sample is relatively short, with a mean score of less than -1 S.D. below the reference average height for a child of the same gender at all ages. The incidence of stunting (HAZ $\leq$ -2 S.D.) in early years is relatively high, with approximately 28% stunted at age 1. Stunting peaks at age 5 at 33%, before falling to 16% by age 15. In contrast, the incidence of underweight (WAZ $\leq$ -2 S.D.) is relatively low, at only 7% at age 1, decreasing slightly to 5% for ages 5 and 8, with average WAZ remaining around -0.20 to -0.53 S.D. from reference values. Interestingly, the rate of wasting is very low (0.3% to 2%), with the average BMI-for-age being positive. This potentially reflects the well documented "double burden" of malnutrition in Peru (and amongst many other middle-income countries), a recent trend which has seen the simultaneous coexistence of high levels of childhood stunting (or under-nutrition), and an increasingly high prevalence of child and teenage overweight/obesity, particularly amongst girls and women in rural, poor areas (Perez-Escamilla et al., 2018; Santos et al., 2021; WHO, 2017). As such, adolescents with short stature and relatively high weight will display relatively higher BMI scores (calculated as weight in kilograms divided by height in metres squared, kg/m^2), however may still exhibit poor health. This highlights a potential limitation of using anthropometrics, particularly BMI, as a measure of overall health.

Cognitive ability is measured across two sub-dimensions. First, using the Spanish version of the revised Peabody Picture Vocubulary Test (PPVT) (Dunn et al., 1986), a widely-used and well-validated assessment of vocabulary acquisition.[11] The test is administered orally, is un-timed, and norm-referenced. While the 125 items in the test are the same for all ages, not all are administered in each test, with only a subset of questions administered after a basal item and ceiling item are established, depending on the number of consecutive correct/incorrect responses (Espinoza Revollo & Scott, 2022; Leon, 2020). This measure is first made available in round 2, when younger cohort respondents were aged 5, and was administered until round 5 when respondents are aged 15.

[11] *Test de Vocabulario en Imagenes Peabody* (TVIP) in Spanish. This test is adapted and validated for use in Latin America. Additionally, it has been further translated and validated by the Young Lives team to make it available for children whose primary language is Quechua.

Table 2: Summary Statistics: Time-Varying Child Outcomes

	Age 1	Age 5	Age 8	Age 12	Age15
Anthropometrics					
Height-for-age	-1.28	-1.53	-1.15	-1.02	-1.15
	(1.27)	(1.11)	(1.04)	(1.10)	(0.88)
Stunted	0.28	0.33	0.20	0.18	0.16
	(0.45)	(0.47)	(0.40)	(0.39)	(0.37)
Weight-for-age	-0.20	-0.53	-0.33		
	(1.18)	(1.01)	(1.17)		
Underweight	0.07	0.05	0.05		
	(0.25)	(0.22)	(0.23)		
BMI-for-age	0.73	0.66	0.51	0.55	0.42
	(1.18)	(0.95)	(1.04)	(1.07)	(0.97)
Wasted	0.02	0.00	0.01	0.01	0.01
	(0.15)	(0.05)	(0.09)	(0.10)	(0.09)
Cogntive scores					
Vocabulary z-score		0.01	0.03	0.01	0.01
		(1.00)	(0.99)	(1.00)	(1.00)
Mathematics z-score		0.03	0.03	0.03	0.02
		(0.99)	(1.00)	(0.99)	(1.00)

Notes: Sample mean values, with standard deviations in parentheses.

Second, respondents' quantitative skills are assessed. In round 2, the Cognitive Development Assessment (CDA), developed by the International Evaluation Association to study the effect of preschool attendance on cognitive development in children, was administered to the younger cohort. Given the long administration time for the full assessment, only the quantity subscale (15 items) was administered (Espinoza Revollo & Scott, 2022). Beginning in round 3, mathematics tests were administered to respondents, based on previously validated items from the Trends in International Mathematics and Science Study (TIMSS) and the Programme for International Student Assessment (PISA). The contents of maths tests differ slightly across rounds, and differ from the CDA administered in round 2. Therefore to provide a more relevant measure, raw scores are age-standardised, to provide a measure of relative performance within cohort.[12] For further details, see Espinoza Revollo and Scott (2022) and Leon (2020).

3.2.2 First Generation

While the primary focus of this study is the impact on grandchildren's outcomes, I also assess the relevancy of the shock on the adult age outcomes of mothers who are exposed while in-utero. In doing so, this may provide an insight in to the potential channels

[12]Leon (2020) derive cross-round comparable scores for PPVT and Math tests (not including the CDA subscale) based on item response theory (IRT) – Results remain unchanged when using IRT scores rather than age-standardised scores.

through which effects are transmitted to the second generation, however it does not provide information on the relevance or importance of this variable as a mediator for the multigenerational effect, and I cannot rule out that there are unobserved variables which determine both mother and grandchild outcomes. Therefore, to address the potential mechanism channels formally, I conduct a mediation analysis in section 6. Following from subsection 2.2, I hypothesis two potential channels, the biological and environmental mechanism. If effects are predominantly transmitted to grandchildren through the biological channel, then it could be expected that mother's exposed to a shock in-utero would display signs of poorer adult health. I use height (cm) and weight (kg) as proxies of mothers' health stock and flow, respectively. Alternatively, if effects are transmitted through the environmental channel by directly impacting maternal cognitive development, then it could be expected that shock exposure has some impact on measures of adult human capital accumulation or socioeconomic status. I use mother's educational attainment, measured as years of completed schooling, as well as her household wealth index to capture these dimensions.[13] Finally, if effects are transmitted indirectly through the environmental channel by impacting mothers' mating/marriage prospects, then it is expected that they may match with lower quality partners. To capture this potential indirect effect, I also regress the educational attainment and height of the father of the child, where available, on mother's shock exposure.

3.3 Standardised Precipitation-Evapotranspiration Index

The first generation mothers were born between 1950 and 1988 in 381 districts across Peru, with median year of birth being 1976, and the youngest mother being aged 13 at the birth of the YL child (second generation).[14] To identify the exposure of the grandmother (zero generation) to drought shocks during her pregnancy, I match data on historical drought exposure from the Standardised Precipitation-Evapotranspiration Index (SPEI) (Begueria et al., 2010; Vicente-Serrano et al., 2010). It provides a multi-scalar drought measure, which accounts for the effects of temperature and potential-evapotranspiration (PET, i.e. the amount of water that is used by plants, or evaporates from the surface, under local normal conditions) on the intensity and duration of droughts. It has been shown to perform better in predicting changes in crop yields and local weather conditions over other common drought indices (Vicente-Serrano et al., 2012), while retaining the simplicity of calculation and multi-temporal nature of probabilistic measures such as the Standardised Precipitation Index (McKee et al., 1993). It has seen increased use in recent economics literature, most notably Harari and Ferrara (2018).

[13] This is a country-specific composite measure of household socioeconomic status, measuring households' access to services such as water and sanitation, their ownership of consumer durables, and the quality of materials used in their dwelling. See Briones (2017) for details.

[14] See Figure A2.

I use monthly gridded data (0.5° resolution) derived as the across a 12-month rolling window time-scale from the SPEIbase (v2.9) database (Begueria et al., 2023), which covers the period 1901-2022. Cell values are aggregated to the district level (3rd level administrative area) as the area-weighted mean value of all overlapping grid cells, to provide a monthly district-level time series. The index is normalised with mean zero and standard deviation (S.D.) one. Following the drought classification system of McKee et al. (1993), I define a drought shock experienced during the grandmother's pregnancy:

$$Drought_{m,d,t} = SPEI_{d,t} \leq -1 S.D. \tag{1}$$

Where $SPEI_{d,t}$ is the SPEI value for the 12 months preceding the month of birth t of mother m, in the district of birth d, such that $Drought_{m,d,t}$ takes a value of one when conditions are less than or equal to one standard deviation worse than the long-run location specific mean conditions, and zero otherwise.[15]

4 Empirical Strategy

4.1 Second Generation Effects

To examine the multigenerational effect of exposure of the grandmother while pregnant to drought on the outcomes of her grandchildren, I estimate the following equation using OLS:

$$Y^a_{c,m,v} = \beta_0 + \beta^a_1 Drought_{m,d,t} + \beta_2 female_{c,m} + \gamma_y + \delta_p + \rho_0 + \sigma^a_v + \varepsilon_p \tag{2}$$

Where $Y^a_{c,m,v}$ are the outcomes of child c, of mother m, at each age a. Health outcomes are estimated for each age $a \in \{1, 5, 8, 12, 15\}$ separately. As discussed above, health stock is proxied by height-for-age z-scores from ages 1-15. Similarly, health flow is measured using weight-for-age and BMI-for-age z-scores for ages 1-8 and 12-15, respectively. Additionally, I assess the effects on cognitive ability, estimating for ages $a \in \{5, 8, 12, 15\}$ the impact on age-standardised PPVT and maths scores (age 5 maths is measured using the CDA quantity subscale, while age 8-15 is measured using YL mathematics tests (Espinoza Revollo & Scott, 2022)).

An indicator that the child c is female is included, as well as a fixed effect (σ^a_v) for child cluster of residence v at age a (for $a = 1$, this is their cluster of birth) and month of birth cohort (ρ_0). Fixed effects for mothers' year-of-birth (γ_y) and province-of-birth (δ_p) are also included. Standard errors are clustered at the level of mother's province of birth (ϵ_p), to account for localised spatial correlation in shock exposure, which varies at

[15] That is, if the mother was born in July 1979, the SPEI value refers to the deviation in conditions between August 1978 and July 1979 from the long-term average.

the district level.[16]

4.2 Heterogeneous Treatment Effects

A common finding within the literature is that the effects of early life investments or shocks can be sex-specific (Almond & Currie, 2011; Almond et al., 2018), and previous evidence suggests this may extend to multigenerational effects (Fung & Ha, 2010; Venkataramani, 2011). To explore whether the multigenerational effect is different for male and female grandchildren, I expand on Equation 2, including an interaction between grandmothers exposure to drought during pregnancy with if their grandchild is female.[17]

Additionally, it is unclear *a priori* if effects found in childhood and early adolescence represent a permanent impact on growth, or simply represent slow growth during childhood, with a subsequent catch-up once children enter into puberty. Therefore I assess how the effects of grandmother's exposure to drought interact with respondents entering into pubertal growth, as measured using reported physical indicators of puberty, discussed further below.

It is expected that drought shocks impact the zero and first generation either directly, through impacting local crop yields or food prices, impacting food availability, or by impacting agricultural income and hence resources or nutrition available to the grandmother during pregnancy or for the mother immediately after her birth. As such it is expected that the effect is either driven exclusively by, or is at least strongest in, rural areas, where a higher proportion of households are likely to be reliant on local food sources and agriculture-related income. Therefore, I also interact the drought exposure indicator with an indicator of if a mother was born in an urban or rural district. Finally, it is common within the early life shocks literature that in-utero shock exposure in a specific trimester may have a stronger effect than in other periods (Almond & Mazumder, 2013). Indeed, within the second generation literature Khan (2021) and Stein and Lumey (2000) find exposure earlier in the pregnancy during the first and second trimester has the largest effect on second generation outcomes. I therefore assess if effects of drought exposure differ by exposure in specific trimesters of pregnancy.

4.3 First Generation Effects

I also assess the direct impact of in-utero shock exposure on the adult age outcomes for the first generation (the mother), estimating the following equation:

[16]Results are also robust to clustering standard errors at the cluster of birth, which is the sampling level. Results are presented in Table A1.

[17]While previous work has noted effects of extreme famine on sex ratios of the second generation (Almond et al., 2012), which would suggest the sex of the grandchild may be endogenous, it is unclear if this effect extends to this context, where drought exposure is less intense and is less likely to present issues of selective mortality. While not directly testable in this context, drought exposure does not predict the sex of grandchildren within the sample, as shown in column 6 of Table A12.

$$M_m = \alpha_0 + \alpha_1 Drought_{m,d,t} + \gamma_y + \gamma_p + \nu_p \quad (3)$$

Where M_m are the health and human capital outcomes for the mother m, measured by her adult height and weight, educational attainment, and household socioeconomic status. Additionally, to capture potential effects on mother's mating/marriage market prospects, I regress the height and educational attainment of the father of the child on mother's shock exposure. Maternal fixed effects for year and province of birth are as above. While this exercise may provide suggestive evidence of potential mechanisms, it does not provide information on the relevance or importance of this variable as a mediator for the multigenerational effect on grandchildren, therefore I conduct a formal mediation analysis in section 6.

5 Results

5.1 First Generation Effects: Mother Outcomes

Before addressing the impacts of drought on the outcomes of the grandchildren, I first assess the evidence of effects being present in the first generation, who were in-utero during the shock exposure. Panel A of Table 3 provides estimates of the impact of an in-utero exposure to drought on their individual adult age outcomes, while Panel B provides estimates on the potential indirect effect of shock exposure on household and partner outcomes, as described in subsubsection 3.2.2. The only dimension for which a significant effect is estimated is in mother's adult height, my measure of long-run health stock. Exposure to drought in-utero is associated with a -0.752cm lower height in adulthood (-0.5% decrease compared to the sample mean). There are no significant effects found for measures of educational attainment, household socio-economic status, or on the outcomes of their partner. Additionally, there is no significant effect found on adult age health flow, which is more susceptible to current inputs. This is suggestive that shock exposure may have an effect on mother's long-run physical health, and with no evidence of an effect on household socio-economic status, attainment, or selective pair-bonding, could indicate that any transmitted effects may operate predominantly through a biological channel. However, while much of the variation in adult height in developing countries is thought to be due to negative shocks experienced in early life, rather than genetic potential (Beard & Blaser, 2002; Silventoinen, 2003; WHO, 1995), it is an imperfect measure. It is likely the result of all unobserved cumulative health shocks and investments experienced in childhood and early adulthood, which may be positively or negatively correlated with the prenatal shock of interest, potentially biasing estimates of the effect. As such, with this method I cannot rule out that there is some unobserved mediator which acts as an intermediate confounder between in-utero shock exposure, maternal height, and second

generation outcomes. Therefore inferences about the causal mechanisms for the zero to second generation effects are restricted to those found using formal mediation analysis in section 6.

Table 3: Effect of Shock Exposure on First Generation Outcomes

	Height	Weight	Education
Panel A: Mother outcomes			
In-utero shock	-0.752	-0.590	0.201
	(0.324)**	(0.580)	(0.247)
Mean	149.97	58.78	7.15
N	1656	1632	1671
	Father		Household
	Height	Education	Wealth
Panel B: Father/Household outcomes			
In-utero shock	0.325	-0.067	-0.000
	(0.315)	(0.294)	(0.010)
Mean	162.18	8.25	0.43
N	1168	1425	1671

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Cluster robust standard errors in parentheses. Fixed effects for mother year- and province- of birth are suppressed. Sample mean values for dependent variables are reported in the foot of each panel.

5.2 Second Generation Effects: Grandchild Outcomes

This section presents the estimated multigenerational effects of a grandmother's exposure to a drought shock while pregnant on the outcomes of her grandchild. I first present the impact on anthropometric outcomes, followed by cognitive outcomes.

5.2.1 Anthropometry

Panel A of Table 4 and Figure 1 present estimates the impact on second gerneration health stock, measured as height-for-age z-scores for ages 1-15. Panel B of Table 4 and Figure 2 provides estimates of the effect on health flow, measured as weight-for-age for ages 1-8, BMI-for-age for ages 12-15.

A persistent negative effect is estimated for HAZ at each age (-0.076 to -0.173 S.D.). While I cannot reject the null hypothesis $H_0 : \hat{\beta}_2^{a=1} = 0$, for age 1 HAZ, estimated effects beginning age 5 and persisting to age 15 are statistically significant at conventional levels, with the largest difference measured at age 12. Although there is a peak at age 12, given overlapping confidence intervals, it is unclear if this effect is increasing in age until early adolescence, or simply persistent.[18] The result of a persistent effect on child height

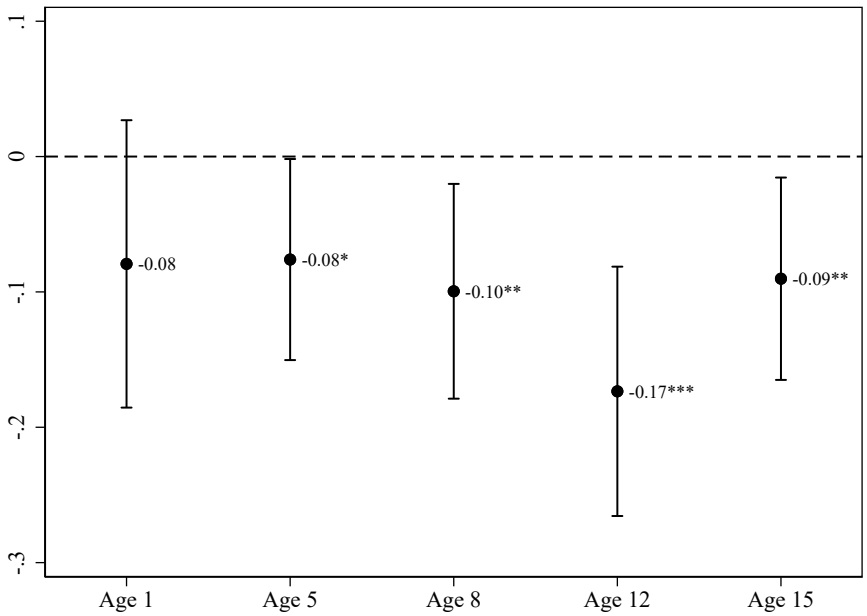
[18] Additionally, confidence intervals are widest for age 1 HAZ, for which measurement was conducted

Table 4: Effect of Shock Exposure on Second Generation Outcomes: Anthropometric Z-scores

	Age 1	Age 5	Age 8	Age 12	Age 15
Panel A: Height-for-age					
In-utero shock	-0.079	-0.076	-0.100	-0.173	-0.090
	(0.064)	(0.045)*	(0.048)**	(0.055)***	(0.045)**
Controls	Yes	Yes	Yes	Yes	Yes
N	1670	1657	1665	1671	1620
	Weight-for-age			BMI-for-age	
	Age 1	Age 5	Age 8	Age 12	Age 15
Panel B: Weight-/BMI-for-age					
In-utero shock	-0.179	-0.109	-0.059	0.045	-0.021
	(0.063)***	(0.047)**	(0.056)	(0.049)	(0.059)
Controls	Yes	Yes	Yes	Yes	Yes
N	1670	1657	1665	1671	1620

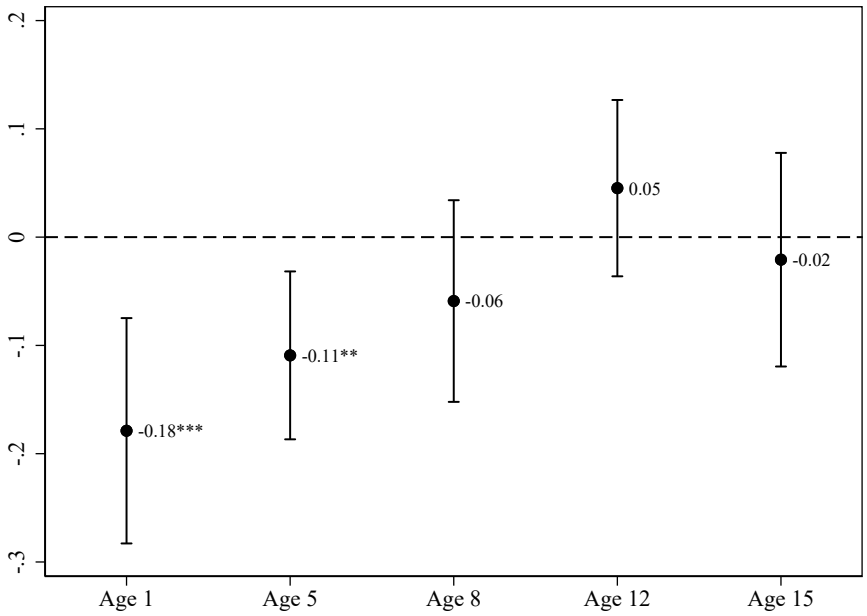
Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Estimates are reported in standard deviations from the age- and gender-specific mean value. Cluster robust standard errors in parentheses. Controls include an indicator of if the child is female. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Figure 1: Effect of Shock Exposure on Second Generation Outcomes: HAZ



Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. 90% Confidence intervals. Coefficients reported in Table 4. P-values calculated using cluster robust standard errors. Controls include an indicator for if the child is female and fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Figure 2: Effect of Shock Exposure on Second Generation Outcomes: WAZ/BMIAZ



Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. 90% Confidence intervals. Coefficients reported in Table 4. P-values calculated using cluster robust standard errors. Controls include an indicator for if the child is female and fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

are consistent with the findings of Bevis and Villa (2022) who find a lasting relationship between a 1cm increase in mother's height due to early life weather shocks (using an novel instrumental variable approach) and child height-for-age measured across their whole childhood. However, this contrasts with the findings of Khan (2021) in India, who finds that while in-utero shock exposure is associated with a negative impact on HAZ for a pooled sample aged 0-5, there is no significant effect on HAZ in the age 8-11 sample.

A large negative effect is estimated for weight-for-age at ages 1 and 5 (-0.179 and -0.109 S.D.), significant at the 1% and 5% level respectively. At older ages the estimated effect size diminishes towards zero, with no significant effect estimated for weight-for-age at age 8, or for BMI-for-age at ages 12 and 15. This finding, and the effect on maternal health stock noted above, complements the literature indicating the strong association between maternal health and children's weight in early years (Currie & Moretti, 2007; Hyland & Russ, 2019). However, if the impact of shock is transmitted between mother and child through its effect on the biological channel, then it also provides a similar conclusion to that of Bevis and Villa (2022), that while (early-life shock-induced) variation in mother's health is important for child health flow in early years, the significance and magnitude diminishes as children age. This is intuitive, given that health flow is more variable with current period inputs, the importance of maternal health endowment becomes less

using a length board rather than height scale. This may indicate greater measurement error, as is common with length measurements where it can be difficult and stressful to ensure a struggling/crying infant is laying fully stretched out for measurement (WHO, 2006).

important as children age. Alternatively, the estimated impact on health stock suggests that the transmitted effect of the shock seems to have a more permanent effect on long-term cumulative health. I explore this possibility further in section 6.

The average effect sizes are relatively small (-0.076 to -0.179 S.D.) compared to the large deficits that categorise stunting and wasting (z-scores $\leq$ -2 S.D.), however as previously documented in Table 2, the sample mean is generally below average height and weight at each age (with the exception of having a slightly positive BMI in adolescent years). Given the importance of these categories for global health targets and policy decisions (De Onis, 2017), I consider the multigenerational effect of shock exposure on the extensive margins of growth. Estimating a linear probability model by OLS, Table 5 provides estimates of the effect of grandmother's shock exposure on probability of stunting (HAZ $\leq$ -2 S.D.) in Panel A, and underweight/wasting (WAZ and BMIAZ $\leq$ -2 S.D., respectively) in Panel B. Coefficients are reported in percentage points (p.p.). While some estimates are not significant (notably age 12 stunting and age 1 underweight) the pattern remains similar to that seen in the intensive margin, with a positive impact of the shock on the probability of stunting of 6.4 p.p. (a 32.0% increase relative to the sample mean) and 5.5 p.p. (34.4% relative increase) for ages 8 and 15, respectively. For health flow, shock exposure is associated with a 3.4 p.p. (68%) increase in probability of being underweight at age 5, all significant at the 5% level. This suggests that while the magnitude of the effect on the intensive margin appears relatively small, there is a notable multigenerational effect of drought exposure on commonly used health targets, adding relevance to these findings for future health policy. I now turn to the results for child cognitive outcomes.

5.2.2 Cognitive Ability

Considering the implications of shock exposure on grandchild cognitive ability, Table 6 shows the estimated effect of exposure on age-standardised scores for receptive vocabulary (Panel A) and quantitative skills (Panel B). Receptive vocabulary is measured using the Spanish-version of the PPVT (Dunn et al., 1986) for all ages, while quantitative skills are measured by the CDA quantity sub-scale for age 5 and using Young Lives mathematics tests for ages 8-15. All scores are age standardised. There is little evidence of an effect on cognitive ability as measured by these indicators, with exception of a negative impact of shock exposure on performance in the CDA quantity sub-scale at age 5, however this effect is only marginally significant at 10%. Given limited evidence of an effect on cognitive outcomes, the remainder of this chapter will focus on the multigenerational impacts on health. Next I assess the potential for heterogeneous effects.

Table 5: Effect of Shock Exposure on Second Generation Outcomes: Stunting & Wasting

	Age 1	Age 5	Age 8	Age 12	Age 15
Panel A: Stunted					
In-utero shock	0.031	0.014	0.064	0.033	0.055
	(0.028)	(0.023)	(0.026)**	(0.027)	(0.023)**
Controls	Yes	Yes	Yes	Yes	Yes
Mean	0.28	0.33	0.20	0.18	0.16
N	1670	1657	1665	1671	1620
	Underweight			Wasted	
	Age 1	Age 5	Age 8	Age 12	Age 15
Panel B: Underweight/Wasted					
In-utero shock	0.004	0.034	0.026	-0.001	0.001
	(0.016)	(0.015)**	(0.016)	(0.006)	(0.007)
Controls	Yes	Yes	Yes	Yes	Yes
Mean	0.07	0.05	0.05	0.01	0.01
N	1670	1657	1665	1671	1620

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Estimates are reported in percentage points. Cluster robust standard errors in parentheses. Controls include an indicator of if the child is female. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed. Sample mean values for dependent variables are reported at the foot of each panel.

Table 6: Effect of Shock Exposure on Second Generation Outcomes: Cognitive Scores

	Age 5	Age 8	Age 12	Age 15
Panel A: Vocabulary score				
In-utero shock	-0.041	0.027	-0.011	-0.017
	(0.046)	(0.057)	(0.060)	(0.057)
Controls	Yes	Yes	Yes	Yes
N	1620	1562	1624	1580
	CDA	Young Lives tests		
	Age 5	Age 8	Age 12	Age 15
Panel B: Maths score				
In-utero shock	-0.115	-0.045	0.015	0.006
	(0.058)*	(0.050)	(0.062)	(0.051)
Controls	Yes	Yes	Yes	Yes
N	1620	1562	1624	1580

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Estimates are reported in standard deviations from the age-standardised sample mean. Cluster robust standard errors in parentheses. Controls include an indicator of if the child is female. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

5.3 Heterogeneous Effects

5.3.1 Sex Specific Differences

Panel A of Table 7 and Figure 3 show the estimated average marginal effects of grandmothers' shock exposure on HAZ of male and female grandchildren. Coefficients plotted are the average marginal effects for a discrete change in the indicator of grandchild sex. Regression coefficients for the level and interaction terms are reported in Table A2. Results suggest that the majority of the impact of shock exposure on HAZ is driven by the effect on boys. This is consistent with Venkataramani (2011), who finds that shock-induced variation in maternal height is most strongly associated with boys height against a null effect for girls, and Fung and Ha (2010), who find that the second generation effects on HAZ and WAZ of maternal in-utero exposure to the great Chinese famine is isolated to boys. Additionally for age 1 length-for-age, there is a large negative effect of shock exposure for boys, compared with a small insignificant positive effect on girls. This could suggest that, at least for boys, the effect on height could be present across their whole childhood from age 1, however this effect remains imprecisely estimated with wide confidence intervals, and is only significant at the 10% level.[19] That the effect of shock exposure on health stock is isolated to boys means that it is not clear whether the multigenerational shock could persist beyond the second generation. Given limited information on the birth location of fathers in my sample, and that I do not currently observe the third generation in this dataset, I am not able to assess how effects may persists patrilineally, however evidence from the wider literature provides evidence that matrilineal transmission is more important for second generation effects (Caruso, 2015; Fung & Ha, 2010; Painter et al., 2008; Venkataramani, 2011).

Panel B of Table 7 and Figure 4 show estimates for WAZ and BMIAZ. While effects are consistently more negative for boys, in contrast to HAZ, infant WAZ is significantly different from zero for both boys and girls. Overall results indicate that there is a sex-specific difference in multigenerational effects, with the largest effects found for boys.

5.3.2 Urban/Rural Differences

Panel A of Table 8 and Figure 5 show the marginal effects for HAZ of the second generation child for mothers who were born in urban areas compared with rural areas. While a large and significant effect is estimated for rural-born mothers (urban-born = 0) for ages 5-15, the marginal effect for the children of urban-born mothers is insignificant across all rounds, with inconsistent sign and diminished magnitude. Similarly, Panel B of the same table and Figure 6 show large and statistically significant effects for age 1-5 WAZ for a baseline effect, with an insignificant marginal effect estimated for the children of urban-

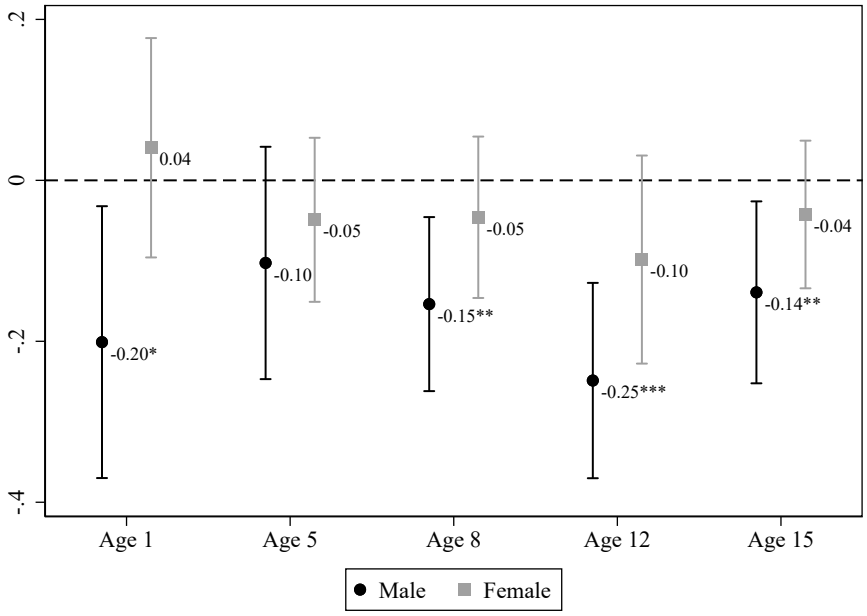
[19] This imprecision may reflect greater measurement error for infant length-for-age, given reported difficulties with keeping infants still and stretched out.

Table 7: Marginal Effect of Shock Exposure on Second Generation Outcomes: By Sex

	Age 1	Age 5	Age 8	Age 12	Age 15
Panel A: Height-for-age					
In-utero shock × Female = 0	-0.201	-0.103	-0.154	-0.249	-0.139
	(0.103)*	(0.088)	(0.066)**	(0.074)***	(0.069)**
In-utero shock × Female = 1	0.040	-0.049	-0.046	-0.098	-0.042
	(0.083)	(0.062)	(0.061)	(0.079)	(0.056)
N	1670	1657	1665	1671	1620
	Weight-for-age			BMI-for-age	
	Age 1	Age 5	Age 8	Age 12	Age 15
Panel B: Weight-/BMI-for-age					
In-utero shock × Female = 0	-0.230	-0.167	-0.113	0.010	-0.093
	(0.083)***	(0.058)***	(0.077)	(0.063)	(0.075)
In-utero shock × Female = 1	-0.128	-0.050	-0.006	0.080	0.050
	(0.071)*	(0.067)	(0.077)	(0.087)	(0.092)
N	1670	1657	1665	1671	1620

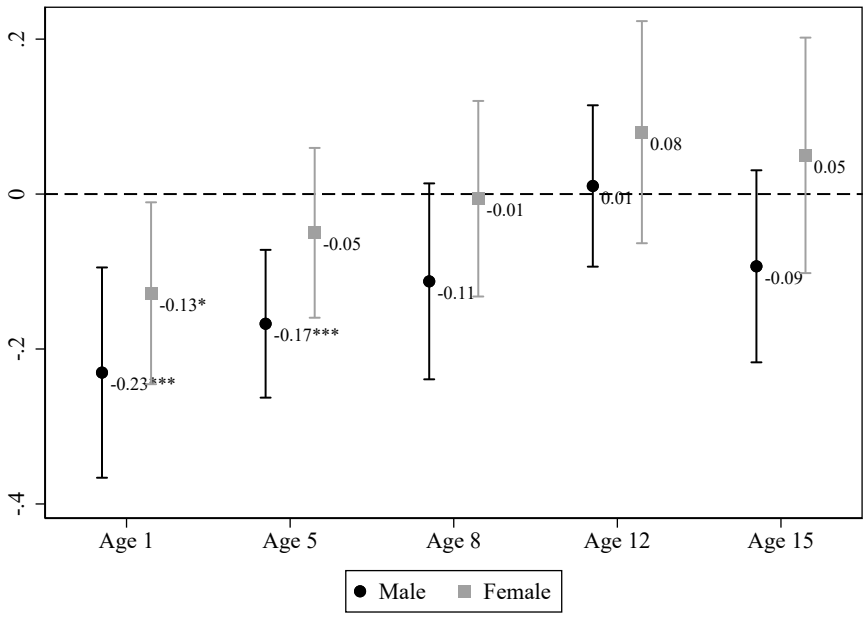
Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Table reports the marginal effects of shock exposure on second generation HAZ (Panel A) and WAZ/BMIAZ (Panel B). Relevant regression coefficients are reported in Table A2. Cluster robust standard errors in parentheses. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Figure 3: Marginal Effect of Shock Exposure on Second Generation Outcomes: HAZ, by Sex



Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. 90% Confidence intervals. Marginal effects reported in Table 7. P-values calculated using cluster robust standard errors. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Figure 4: Marginal Effect of Shock Exposure on Second Generation Outcomes: WAZ/BMIAZ, by Sex



Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. 90% Confidence intervals. Marginal effects reported in Table 7. P-values calculated using cluster robust standard errors. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

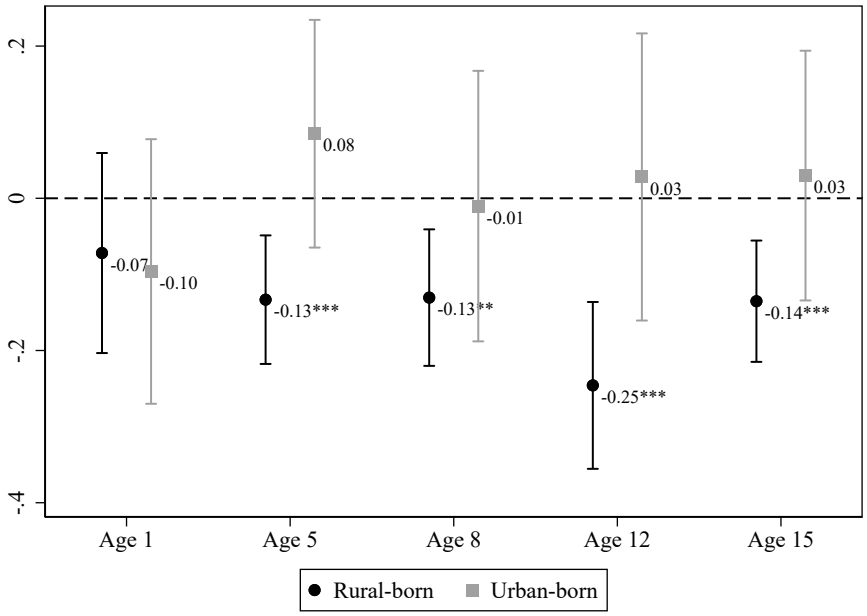
born mothers. This evidence suggests that the overall effect seems to be driven by the large and significant effect for the children of mothers born outside of urban areas.

Table 8: Marginal Effect of Shock Exposure on Second Generation Outcomes: By Mother Birth-Location

	Age 1	Age 5	Age 8	Age 12	Age 15
Panel A: Height-for-age					
In-utero shock × Urban-born = 0	-0.072	-0.133	-0.131	-0.246	-0.135
	(0.080)	(0.051)***	(0.055)**	(0.067)***	(0.048)***
In-utero shock × Urban-born = 1	-0.096	0.085	-0.010	0.028	0.030
	(0.106)	(0.091)	(0.108)	(0.115)	(0.100)
N	1670	1657	1665	1671	1620
	Weight-for-age			BMI-for-age	
	Age 1	Age 5	Age 8	Age 12	Age 15
Panel B: Weight-/BMI-for-age					
In-utero shock × Urban-born = 0	-0.213	-0.144	-0.094	0.061	-0.036
	(0.067)***	(0.054)***	(0.069)	(0.063)	(0.064)
In-utero shock × Urban-born = 1	-0.084	-0.010	0.041	0.002	0.023
	(0.127)	(0.108)	(0.116)	(0.070)	(0.098)
N	1670	1657	1665	1671	1620

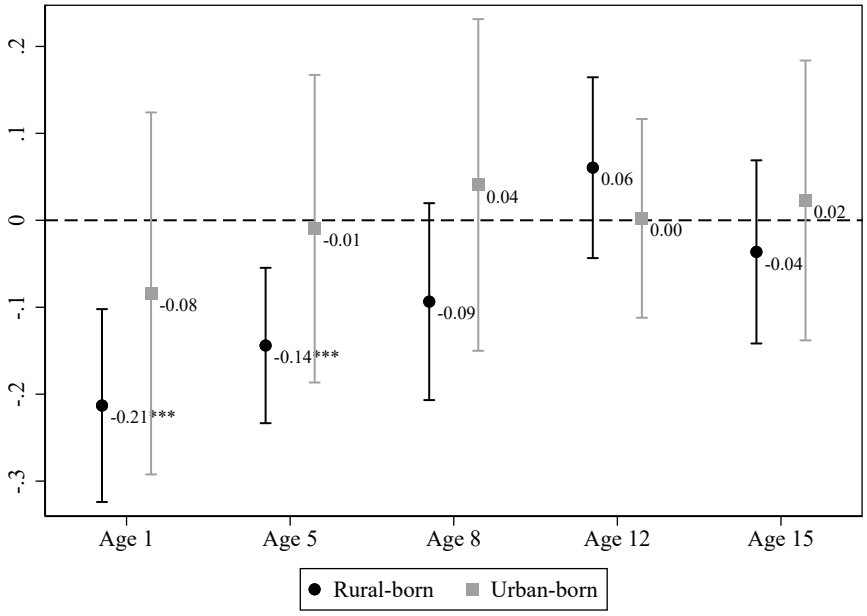
Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Table reports the marginal effects of shock exposure on second generation HAZ (Panel A) and WAZ/BMIAZ (Panel B). Relevant regression coefficients are reported in Table A3. Cluster robust standard errors in parentheses. Controls include an indicator of if the child is female. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Figure 5: Marginal Effect of Shock Exposure on Second Generation Outcomes: HAZ, by Mother Birth-Location



Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. 90% Confidence intervals. Marginal effects reported in Table 8. P-values calculated using cluster robust standard errors. Indicator for if the child is female and fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Figure 6: Marginal Effect of Shock Exposure on Second Generation Outcomes: WAZ/BMIAZ, by Mother Birth-Location



Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. 90% Confidence intervals. Marginal effects reported in Table 8. P-values calculated using cluster robust standard errors. Indicator for if the child is female and fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

5.3.3 Growth Stage Differences

Notably, the results in Table 4 suggest that second generation effects for height-for-age are most pronounced between age 8 and 15, with the largest effect at age 12. This peak may suggest that impacts are largest around when grandchildren may be transitioning between childhood growth and pubertal growth, but it is unclear if the gap between exposed and unexposed individuals remains stable once they enter into pubertal growth. It is possible the height differential widens once in puberty, providing at least suggestive evidence that effects may impact final adulthood growth potential and indicating a persistent multigenerational gap that is potentially difficult to remediate. Alternatively, if the gap narrows in puberty, it could suggest that the multigenerational effect of drought exposure may impact only on second generation childhood growth velocity, but not necessarily final adult height potential, indicating a greater opportunity for catch up or remediation with post-exposure intervention.

To assess potential heterogeneity in the transmission of effects by adolescent or childhood growth stage, I use information on the timing of physical signs of pubertal onset to construct an indicator for likely pubertal growth. beginning in around 4 (age 12) respondents are asked about when they first noticed certain physical traits associated with puberty. For girls, respondents are asked if they experience menstruation, and at what age did they experience their first period. Similarly for boys, respondents were asked if they have noticed hair on their chin, and at what age they first noticed hair growing on their chin. In round 5 (age 15), girls who had not yet experienced their period and boys who had not reported/had visible facial hair were asked these questions again.[20] Pooling information between rounds 4 and 5, I construct an indicators of if a respondent has reported first experiencing their period or having noticed hair on their chin by age 12 or 15. For a small number of observations, this information is missing across both rounds, due to refusal or non-response.[21] A limitation of this data is that it is self reported, however previous research using the Young Lives dataset has shown their reliability as an indicator, as the most important determinant of growth velocity in adolescence (Duc & Tam, 2015).[22]

Table 9 and Figure 7 show the estimated average marginal effects of grandmother's shock exposure on HAZ and BMIAZ of grandchildren, interacted with the likely pubertal growth indicator for ages 12 and 15, respectively. Interestingly, the marginal effects

[20] Notably, this question was incorrectly coded in round 5 for the Peru survey, but was subsequently collected either by in-person follow up or via an additional phone survey. This data is not currently available in the public release and must be requested directly from Young Lives.

[21] A concern is that the age of pubertal onset may be endogenous to shock exposure. I do not observe the eventual age of onset for respondents who do not report signs, therefore I cannot directly test this relationship, however shock exposure is not predictive of onset of puberty by age 12.

[22] A further limitation is that these measures, in particular for boys are not directly relatable to well established measures of pubertal growth, such as the Tanner stages/Sexual Maturity Rating scale (Marshall & Tanner, 1969, 1970).

estimated for HAZ at ages 12 and 15 for those reporting signs of puberty (-0.244 S.D. and -0.099 S.D.) are larger than those found in the main specification, significant at the 1% and 10% level respectively. While negative effects are estimated for those not yet reporting signs of puberty, these effects are not statistically different from zero. This indicates that effects estimated in adolescence are primarily driven by those who have reported signs of puberty, suggesting that the gap between those exposed and unexposed may widen as respondents begin pubertal growth. In contrast, there is no significant effect estimated for BMI-for-age, regardless of growth stage, consistent with my primary findings. Compared to a large effect estimated for HAZ at age 12, the effect at age 15 is diminished. This could suggest that the effect, while initially wider in early puberty, narrows as children reach young adulthood, however the effect is imprecisely estimated, with overlapping confidence intervals, therefore it is not possible to draw clear conclusions in this aspect.

These effects are in contrast with those found by Bevis and Villa (2022), who provide suggestive evidence that the transmitted effect of early life weather induced height variation between first and second generation peaks at the average age of puberty onset for boys (age 11) and girls (age 8), however they do not find a statistical difference in transmission effects between those likely in pubertal growth and child growth at the next round (age 15 and age 11 for boys and girls, respectively).

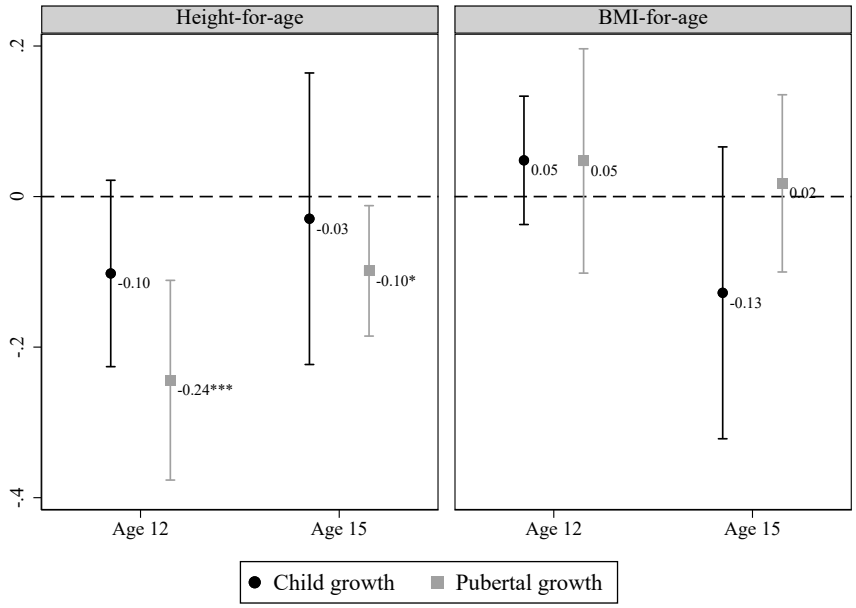
Relevant regression coefficients are reported in Table A4, with large positive coefficients estimated for the level term for puberty for both HAZ and BMIAZ as the outcome, providing suggestive evidence that indicator does seem to explain growth associated with actual puberty. However, given this indicator is constructed using self-reported data, it is likely there is significant measurement error arising from recall error and misreporting. Therefore results are interpreted with caution.

Table 9: Marginal Effect of Shock Exposure on Second Generation Outcomes: By Growth Stage

	Height-for-age		BMI-for-age	
	Age 12	Age 15	Age 12	Age 15
pubertal growth = 0 × In-utero shock	-0.102	-0.029	0.048	-0.128
	(0.075)	(0.118)	(0.052)	(0.118)
pubertal growth = 1 × In-utero shock	-0.244	-0.099	0.047	0.018
	(0.081)***	(0.053)*	(0.091)	(0.072)
N	1665	1617	1665	1617

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Table reports the marginal effects of shock exposure on second generation adolescent HAZ and WAZ/BMIAZ. Relevant regression coefficients are reported in Table A4. Cluster robust standard errors in parentheses. Controls include an indicator of if the child is female. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Figure 7: Marginal Effect of Shock Exposure on Second Generation Outcomes: HAZ & BMIAZ, by Growth Stage



Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Marginal effects reported in Table 9. 90% Confidence intervals. P-values calculated using cluster robust standard errors. Controls include an indicator for if the child is female. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

5.3.4 Trimester of Exposure

Finally, I assess if exposure to drought within a specific trimester of the grandmother's pregnancy is important for grandchild effects. Estimates for the second generation effects of exposure to a SPEI shock ≤ -1 S.D. in each of the approximate trimesters of the grandmother's pregnancy are shown in Table 10. Results indicate that both the effects on HAZ and early years WAZ are strongest for exposure to a shock in the first trimester. A significant effect on second generation HAZ of 1st trimester exposure is estimated between ages 5 and 12, although the effect found at age 15 for the main analysis is no longer significant at conventional levels. These results are consistent with those in other multigenerational studies, which find exposure to negative shocks earlier in the pregnancy during the first and second trimester has the largest effect on second generation outcomes (Khan, 2021; Stein & Lumey, 2000). A limitation however, given that birth date is only available at the month and year level, is that it is not possible to precisely define if a respondent was exposed to a shock in a specific trimester, or if the defined intervals include periods prior to conception or post-birth, therefore these results may be subject to some measurement error.[23]

[23]Additionally, SPEI shocks for trimesters are defined over a different time period than the main specification shock, therefore they may not represent the same intensity of drought as a shock defined over a longer period, which indicate a more sustained period of below average rainfall.

Table 10: Effect of Shock Exposure on Second Generation Outcomes: By Trimester

	Age 1	Age 5	Age 8	Age 12	Age 15
Panel A: Height-for-age					
1st trimester	-0.048	-0.151	-0.103	-0.125	-0.077
	(0.065)	(0.056)***	(0.046)**	(0.054)**	(0.047)
2nd trimester	-0.027	-0.013	-0.016	-0.008	0.019
	(0.067)	(0.051)	(0.052)	(0.057)	(0.048)
3rd trimester	-0.062	-0.023	-0.001	-0.069	-0.042
	(0.063)	(0.053)	(0.053)	(0.060)	(0.058)
Controls & FEs	Yes	Yes	Yes	Yes	Yes
Observations	1670	1657	1665	1671	1620
	Weight-for-age			BMI-for-age	
	Age 1	Age 5	Age 8	Age 12	Age 15
Panel B: Weight-/BMI-for-age					
1st trimester	-0.145	-0.124	-0.103	-0.056	-0.045
	(0.071)**	(0.060)**	(0.054)*	(0.052)	(0.051)
2nd trimester	-0.070	-0.050	-0.013	-0.013	-0.034
	(0.063)	(0.058)	(0.071)	(0.053)	(0.074)
3rd trimester	-0.068	-0.026	0.009	0.006	0.004
	(0.059)	(0.057)	(0.064)	(0.057)	(0.058)
Controls & FEs	Yes	Yes	Yes	Yes	Yes
Observations	1670	1657	1665	1671	1620

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Estimates are reported in standard deviations from the age- and gender-specific mean value. Cluster robust standard errors in parentheses. Controls include an indicator of if the child is female. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

5.4 Robustness Checks

To assess the sensitivity of results to shock definition, in Table A5 and Table A6 I re-estimate results for height and weight outcomes respectively using alternative exposure indicators. Panel A uses a lower cutoff point of less than or equal -0.8 S.D. for 12-month SPEI values proceeding the month of mother's birth. Panel B defines a drought shock as an average monthly SPEI value $\leq$ -1 S.D. throughout the growing season prior to the mother's month of birth of the primary crop in each department. The primary crop for each department of Peru is defined based on the annual sown area in hectares, using data from the Peruvian Ministry of Agriculture (MINAGRI).[24] The crop-growing season for each primary crop is defined based on a global gridded crop calendar data from the University of Wisconsin-Madison's Nelson Institute (Sacks et al., 2010), which provides estimates of planting and harvesting days for 19 crops on a 0.5x0.5° global grid, based on national and sub-national agricultural censuses.[25] I aggregate this grid-level data to the department level to obtain the mean planting and harvesting date, rounded to the month-of-year level. The pattern of results remains for both specifications.

The sample size varies slightly across rounds due to both attrition and interrupted observations, as discussed in subsection 3.1. Table A7 re-estimates Equation 2 for anthropometric outcomes on a balanced sample with results remaining robust (small variations in sample size remain due to singleton observations, which are dropped during estimation).

This analysis focuses on in-utero shock exposure, however it is possible that the multi-generational effects of early life shocks are not exclusive to exposure in the prenatal period. To test this, in Table A8 and Table A9 I estimate the impact of shock exposure in each year from 3 years prior to birth until 5 years after birth on HAZ and WAZ/BMIAZ, respectively. Shock exposure in any 12 month period outwith the 12 months prior to birth (the in-utero shock) has no significant impact on HAZ at any age. Interestingly for BMIAZ at age 15, a positive effect is estimated for shock exposure two or three years before birth, as well as 5 years after birth, although only the effect at 2 years before birth is significant at the 5% level or above. Otherwise There is no significant impact for any other outcome outside of the year prior to birth.[26]

While there is no evidence of an effect on the socioeconomic outcomes of the mother, a potential threat is if there is non-random selection in to treatment, with poorer families being more likely to be exposed. To check if there is an endogenous relationship between socioeconomic status of the grandparents' household and shock exposure, I regress in-utero shock exposure on the education level of the grandparents, measured by an in-

[24] This data is extracted from the *cropdatape* R package, available: https://github.com/omarbenites/cropdatape. Crops included are rice, quinoa, potato, sweet potato, tomato and wheat. Tomato was excluded as it is a perennial crop.

[25] Available from: https://sage.nelson.wisc.edu/data-and-models/datasets/crop-calendar-dataset/

[26] Additionally, I estimate all these periods separately, with similar results reported in Table A10 and Table A11.

dicator of at least one grandparent completing secondary education, finding no evidence of grandparent educational attainment as being predictive of shock exposure. However, data on grandparent education is only available for a sub-sample of households where at least one grandparent is present in the household roster at any point between round 1 and 5, likely leading to a selection bias. Therefore I use as an alternative measure an indicator of whether the maternal grandmother's mother tongue was Spanish, which is available for the majority of the sample. This is a rough proxy of long-term SES, with large inequalities present in education and SES between Spanish and indigenous language speaking households (Leon et al., 2021). I find no significant relationship with shock exposure. Results are reported in Table A12.

Alternatively a further threat to identification is if shock exposure leads to selection into the sample, for example by influencing the subsequent migration choices of the mother's family after her birth. To assess this, I use an indicator of if the mother moved prior to age 5, estimating an insignificant null effect of exposure to shock on migration choices, as reported in column 1 of Table A13. Shock exposure is also not predictive of ever-migrating (prior to birth of the YL child), migration to a departmental capital, or to Lima/Callao, as shown in columns 2-4. Finally, shock exposure may impact the health of mothers born if shock exposure has an effect on infant mortality, leading to survivorship bias amongst exposed mothers. Alternatively, if some grandmothers can react to shock exposure by choosing to delay having children, then shock exposure may also affect the composition of cohorts.[27] To attempt to address the latter, in column 5 of Table A13 I regress shock exposure on the month of birth (January to December) of the mother, finding no significant relationship.[28] For the former, I am unable to assess the impact of shock exposure on mortality within my dataset. However, if selective mortality occurred it would be expected that the surviving mothers are on average healthier, therefore my estimates of the negative effect of shock exposure on maternal height would likely represent a lower bound of the effect of shock exposure. In the next section I explore the potential transmission channels for multigenerational effects using mediation analysis.

6 Mechanisms

6.1 Mediation Analysis

To explore the potential mechanism channels I conduct a mediation analysis, estimating the average controlled direct effect (ACDE)(Joffe & Greene, 2009; VanderWeele, 2009), which is the effect of changing treatment status with the mediator held at a fixed value

[27]Notably, in the 1986 DHS survey 65.5% of married women aged 15-49 reported having ever used contraception, although 86.7% of those only reported using traditional methods, e.g. withdrawal and rhythm (Goldman et al., 1989).

[28]the distribution of month of birth of the mother is shown in Figure A3.

for all units. The ACDE therefore provides an estimate of the direct effect of treatment that does not operate through the specified mediator (Acharya et al., 2016). If the effect of treatment is completely mediated by some variable M and a set of other mediator variables W, then a non-zero ACDE for mediator M implies that the effect of treatment does not exclusively operate through that channel M, allowing alternative mechanisms to be ruled out (VanderWeele, 2011). Additionally, if the null hypothesis that the ACDE is not different from zero cannot be rejected at conventional levels, then that mediator M is likely the main mechanism through which the treatment causes the outcome (Bellemare et al., 2021), provided identifying assumptions hold. See Appendix B for greater detail.

To measure socioeconomic status of the household, therefore capturing the home environment and ability of parents to invest in children (Khan, 2021), I use the Young Lives wealth index (Briones, 2017). To measure mother's human capital, I use mother's educational attainment (highest grade/level achieved), reflecting her cognitive ability, parenting skills, and earnings potential, which are important determinants of child health and human capital (Van Den Berg & Pinger, 2016). Finally, to capture maternal health I use mother's adult height (cm). As discussed above, adult height is a measure of cumulative health and a good proxy of morbidity risk (Case & Paxson, 2008).

If multigenerational impacts are transmitted predominantly through the environmental channel then it is expected that the ACDE for measures of SES and parent human capital will be close to zero (Acharya et al., 2016). Alternatively, if effects are transmitted predominantly through the biological channel then it is expected that the ACDE for environmental mediators are non-zero and do not differ significantly from the baseline estimate, while the ACDE for maternal health will be close to zero.

Under strong assumptions, the use of standard regression analysis using a single equation with the mediator as an additional regressor could be a valid way of testing a mechanism only if there is no omitted variables for the effect of treatment on both mediator and outcome, nor for the effect of mediator on outcome, and importantly, only if all relevant confounders are pre-treatment (Bellemare et al., 2021). However, this is insufficient if there exists some post-treatment covariate Z, which is influenced by treatment D, influences the mediator M, and is independently associated with the outcome Y (Robins, 1986). The exclusion of these "intermediate" confounders if they exist could induce a spurious relationships between treatment and the outcome when including mediators in the regression equation (Rosenbaum, 1984), while conversely including them as regressors could introduce intermediate variable bias to the estimate of the direct effect (Acharya et al., 2016).

An important advantage therefore of the ACDE for mediators is that it can be identified in the the face of these intermediate confounders when estimated using "sequential g-estimation" (or reverse sequential two-stage (RS2S) parametric estimation), as set out by VanderWeele (2009) and Joffe and Greene (2009). Full details of identifying assump-

tions and implementation are provided in Appendix B. I identify a number of socioeconomic controls and measures of parental investments in the child which are likely related to the mediator and outcome, and could potentially be caused by the treatment. These vary across rounds due to relevance for that stage of development, and are listed in Table A14. Furthermore, I condition on further "pretreatment" variables, that is variables which can affect the treatment, outcome or even the mediator, but are not determined by the treatment and do not come between the mediator and outcome. For this, I include an indicator for if the child is female, as well as all the fixed effects included in the baseline specification.[29]

Finally, Acharya et al. (2016) note that the demediation function generally identifies the ACDE when the mediator is set to zero, which may be nonsensical in context, and that in these cases it is suitable to recentre the mediator around a specific value. For my analysis, a maternal height of 0 cm is not plausible, therefore I recentre the height at the sample mean, creating a normalised index of mean 0 and standard deviation 1, however results are robust to use of the raw value in centimetres.

6.2 Mediation Results

Table 11 and Figure 8 provide comparisons of baseline results for HAZ with the ACDE estimated separately after accounting for three different potential mediators: i) mother's attainment (highest grade/qualification achieved) and ii) household wealth index, measures representing the hypothesised environmental channel, through which the shock impacts the household environment, parenting ability, or resource constraint for investments in children; and iii) mother's height, representing the biological channel, wherein exposure to an in-utero shock has permanent effects on maternal physiology, metabolism and ability to transfer nutrients to her offspring in-utero.

Estimates of the ACDE net of the indirect effect of maternal attainment and household wealth are of similar magnitude as the baseline estimated effect, and remain significantly different from zero, with exception of the ACDE for age 5 HAZ net of household wealth, for which the same effect size is only marginally significant under the baseline specification. This suggests that a significant effect of shock exposure that is either transmitted directly or through an alternative set of mediators remains, and that these environmental channel measures do not seem to play a role in the causal pathway between shock exposure and grandchildren outcomes.

In contrast, all estimates of the ACDE net of the indirect effect of mother's health stock, measured by her adult height, are considerably diminished. The majority of estimates are also not significantly different from zero, with exception of the age 12 coefficient, which remains significantly different from zero above the 5% level. A similar

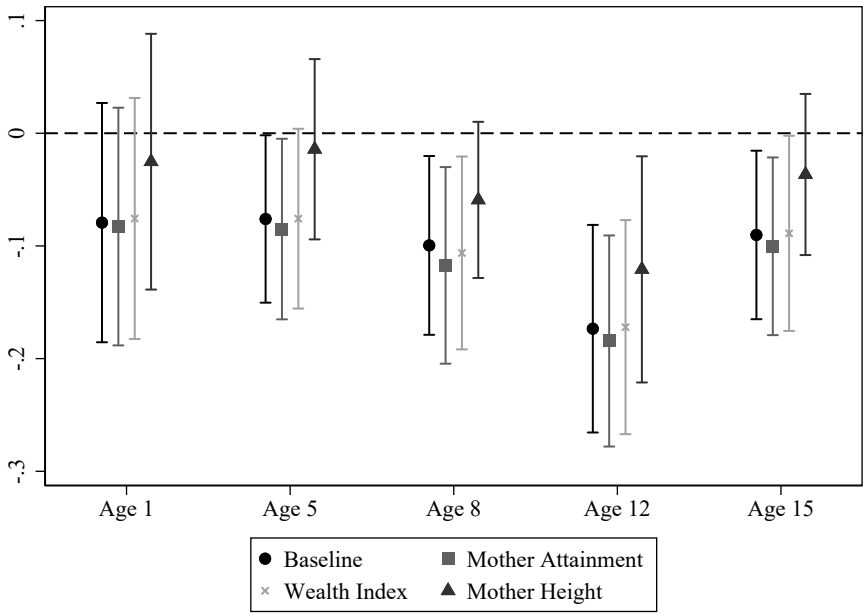
[29] Within my sample grandmother's shock exposure during pregnancy does not predict the sex of her grandchild, as shown in column 6 of Table A12.

Table 11: Mediation Analysis: Comparison of Baseline Results with ACDE, HAZ

		ACDE		
	Baseline Result	Mother Attainment	HH Wealth Index	Mother Height
Panel A: Age 1				
In-utero shock	-0.079	-0.083	-0.076	-0.025
	(0.064)	(0.064)	(0.065)	(0.069)
Observations	1670	1670	1670	1655
Panel B: Age 5				
In-utero shock	-0.076	-0.085	-0.076	-0.014
	(0.045)*	(0.049)*	(0.048)	(0.049)
Observations	1657	1657	1657	1649
Panel C: Age 8				
In-utero shock	-0.100	-0.117	-0.106	-0.059
	(0.048)**	(0.053)**	(0.052)**	(0.042)
Observations	1665	1665	1665	1650
Panel D: Age 12				
In-utero shock	-0.173	-0.184	-0.172	-0.121
	(0.055)***	(0.057)***	(0.058)***	(0.061)**
Observations	1671	1671	1671	1655
Panel E: Age 15				
In-utero shock	-0.090	-0.100	-0.089	-0.037
	(0.045)**	(0.048)**	(0.053)*	(0.043)
Observations	1620	1620	1620	1609

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Estimates are reported in standard deviations from the age- and gender-specific mean value. Cluster robust standard errors in parentheses. Pretreatment controls include an indicator of if the child is female. Intermediate confounders for each age are listed in Table A14. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Figure 8: Mediation Analysis: Comparison of Baseline Results with ACDE, HAZ



Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Coefficients reported in Table 11. 90% Confidence intervals. P-values calculated using cluster robust standard errors. Pretreatment controls include an indicator for if the child is female. Intermediate confounders for each age are listed in Table A14. Fixed effects include child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth. Controls and fixed effects are suppressed.

pattern emerges for health flow, as shown in Table 12 and Figure 9, where estimates for the ACDE net of environmental mediators do not differ from the baseline results. The direct effect with mother's height as the mediator is diminished and is not significantly different from zero at age 5, however while diminished for age 1, the ACDE remains non-zero (significant at the 5% level), suggesting that mother's health is not the sole mediator for the effect on infant WAZ.

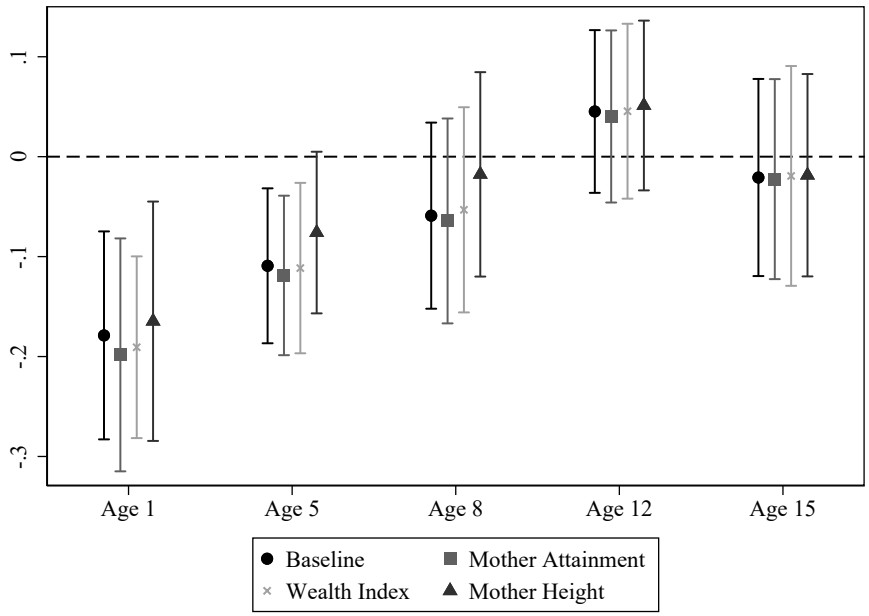
While the evidence above suggests that maternal health, as measured by height, is not the sole mediator for the effect, several issues which may impact the estimated ACDE must be considered. First, direct effects are likely biased if the mediator is measured with error, or the measure of a mediator does not capture fully all the effect of the treatment on all dimensions of the mediating variable (VanderWeele, 2012). To illustrate, consider an example presented by Huber (2019), where there exists a measure of a mediator that only captures the extensive margin (e.g. a dummy indicator of employment) but not the intensive margin (actual hours worked). If the effect of treatment on the mediator induces change in both the intensive and extensive margins, the estimate of the indirect effect will only account for the proportion of the actual indirect effect related to treatment-induced changes at the extensive margin, while the treatment-induced changes to the mediator at the intensive margin will be wrongly attributed to the direct effect (that is, as not operating through that mediator). Given adult height represents a relatively imperfect

Table 12: Mediation Analysis: Comparison of Baseline Results with ACDE, WAZ/ BMIAZ

		ACDE		
	Baseline Result	Mother Attainment	HH Wealth Index	Mother Height
Panel A: Age 1				
In-utero shock	-0.179	-0.198	-0.191	-0.165
	(0.063)***	(0.071)***	(0.055)***	(0.073)**
Observations	1670	1670	1670	1655
Panel B: Age 5				
In-utero shock	-0.109	-0.119	-0.111	-0.076
	(0.047)**	(0.049)**	(0.052)**	(0.049)
Observations	1657	1657	1657	1649
Panel C: Age 8				
In-utero shock	-0.059	-0.064	-0.053	-0.018
	(0.056)	(0.062)	(0.062)	(0.062)
Observations	1665	1665	1665	1650
Panel D: Age 12				
In-utero shock	0.045	0.040	0.046	0.051
	(0.049)	(0.052)	(0.053)	(0.052)
Observations	1671	1671	1671	1655
Panel E: Age 15				
In-utero shock	-0.021	-0.022	-0.019	-0.019
	(0.059)	(0.061)	(0.067)	(0.062)
Observations	1620	1620	1620	1609

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Estimates are reported in standard deviations from the age- and gender-specific mean value. Cluster robust standard errors in parentheses. Predetermined controls include an indicator of if the child is female. Intermediate confounders for each age are listed in Table A14. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Figure 9: Mediation Analysis: Comparison of Baseline Results with ACDE, WAZ/ BMIAZ



Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Coefficients reported in Table 12. 90% Confidence intervals. P-values calculated using cluster robust standard errors. Pretreatment controls include an indicator for if the child is female. Intermediate confounders for each age are listed in Table A14. Fixed effects include child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth. Controls and fixed effects are suppressed.

measure of maternal health, which may not capture all the dimensions of health which are affected by in-utero shock exposure (alterations to maternal physiology which impact maternal prenatal health but not stature), it is likely that the indirect effect transmitted through the biological channel is underestimated, biasing the ACDE estimate upwards from the true population value.

Second, there could exist an alternative biological transmission channel which operates not through impacting maternal health of the first generation, but is transmitted directly to the second generation if shock exposure in-utero impacts the germ cells (the gametes/reproductive cells), present within the first generation as a foetus while in-utero, and from which the second generation will be formed (so-called "gametic epigenetic effects" (Youngson & Whitelaw, 2008)). However, while this potential channel has been discussed in epidemiology, the evidence from animal studies is limited and how exactly this potential channel operates is not well understood. Therefore it is outside the scope of this study to attempt to address it directly here. For a discussion of the theory and some evidence from studies of rats, see Drake and Liu (2010).

Overall the results suggest that maternal health seems to play a considerable role in explaining the mechanism of transmission, providing support to the biological channel as the primary mechanisms for the multigenerational effects, however I cannot conclusively rule out the potential role of other mediating variables.

7 Conclusions

In this chapter, I contribute to the growing "second generation" literature on early childhood shocks. Using high-quality data from the Young Lives Peru study I estimate the multigenerational effects of prenatal shock exposure on the outcomes of the first and second generations. To identify exposure to exogenous variation in drought experienced by a grandmother while pregnant, I link gridded time series data from SPEIbase (Begueria et al., 2023) to the date and location of birth of the mother of the Young Lives child. Using longitudinal data tracking a cohort of children and their family from birth into adulthood allows for a detailed assessment of how effects in the second generation manifest and vary as the child grows into adulthood, rather than providing just a snapshot at one age.

This chapter provides three contributions to the literature. First, I present evidence that the exposure of the grandmother to drought while pregnant has a negative multigenerational effect on the long-term health stock of both her child and her grandchildren, with the first generation being shorter on average in adulthood, and with significant impacts on the height and weight of the second generation. Exposure to drought in the first trimester of pregnancy in particular is associated with a negative impact on the growth outcomes of her grandchildren. Furthermore, evidence suggests that effects are isolated to those grandmothers located in rural areas, where households may have been more reliant

on local food supply and agricultural income.

Second, exploring the dynamics of this multigenerational effect in the second generation shows this impact becomes clear early in life, with a negative impact on infant and early childhood weight-for-age. However while impacts on early life health flow diminish, a persistent effect on height-for-age is evident from early/mid-childhood and remains significant into adolescence, suggesting a permanent effect on long-term health stock. Using self-reported data on signs of pubertal growth, I find evidence that this height differential remains significant and appears to widen for those reporting pubertal growth at either age 12 or 15. Additionally, I find evidence of sex-specific effects, with a disproportionate impact of grandmother's exposure on the height-for-age of boys, consistent with other work in this literature (Fung & Ha, 2010; Venkataramani, 2011). That the effects for grandchildren are primarily seen in boys makes it unclear whether this multigenerational effect would persist beyond the second generation, a limitation which cannot be addressed within this current study. Furthermore, current data limitations means it is not yet possible with this dataset to fully assess if effects persist into adulthood, affecting final adult height potential. In contrast to significant impacts on physical growth, there is little evidence of an effect on measures of cognitive ability. Results remain robust to alternative shock definitions, and I provide evidence that the multigenerational effect of shock exposure is limited to in-utero exposure of the first generation only.

Third, results from mediation analysis suggest the primary channel which facilitates this transmission of health effects across generations is biological, with the majority of the baseline effect operating indirectly through an impact on the maternal health of the first generation, while there is little to no indirect effect operating through the environmental pathway, capturing maternal human capital accumulation and socioeconomic status. However for some outcomes a non-zero direct effect remains after accounting for maternal health as well as a range of potential intermediate confounders, suggesting that the causal effect on the first generation is not fully captured by my measure of mothers' health, or alternatively that there potentially exists another unobserved transmission channel, for example through "gametic epigenetic effects".

These findings provide some policy implications. First, in-utero exposure to a negative drought shock has a lasting impact across generations. As such, if policy is designed without accounting for the potential of multigenerational consequences, it will likely underestimate i) the full cost of exposure of individuals to a shock; and ii) the true cost-benefit ratio of any policy aimed at mitigation (Doyle & Jernström, 2023). Second, that effects remain persistent in to adulthood for the second generation and are not mediated by the human capital accumulation or socioeconomic status of the first generation also suggests that effects are not easily remediated after exposure. Therefore an emphasis should be placed on the importance of early intervention for the timing and targeting of future policy.

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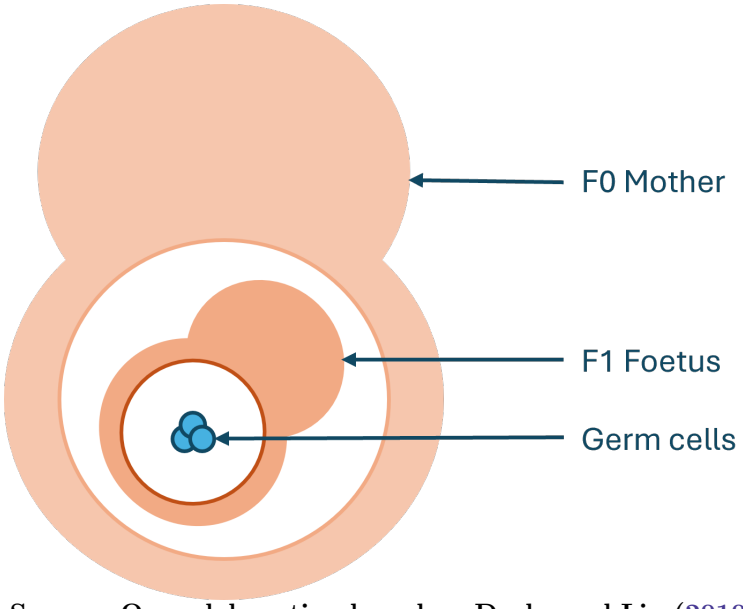
Appendices

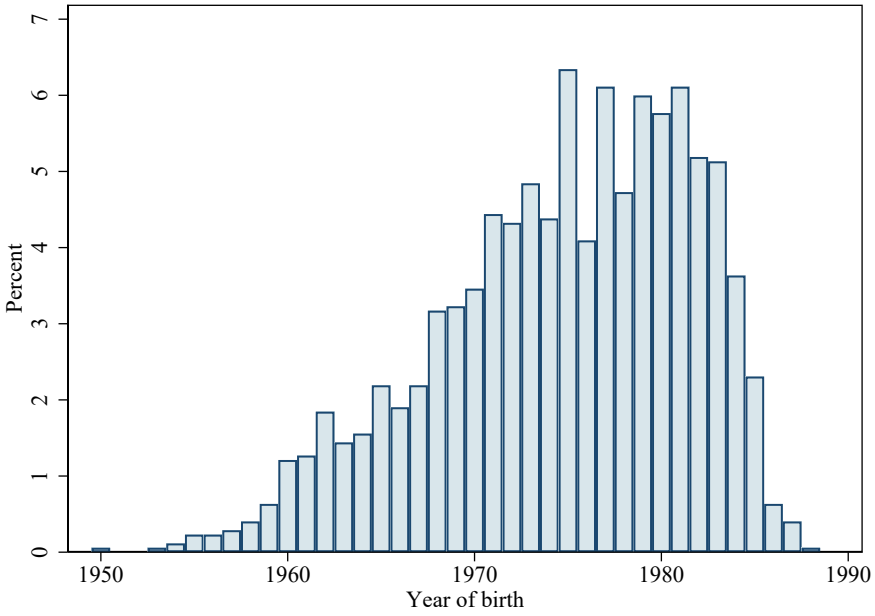
A Additional tables and figures

Figure A1: Multigenerational exposure to an environmental effect in-utero



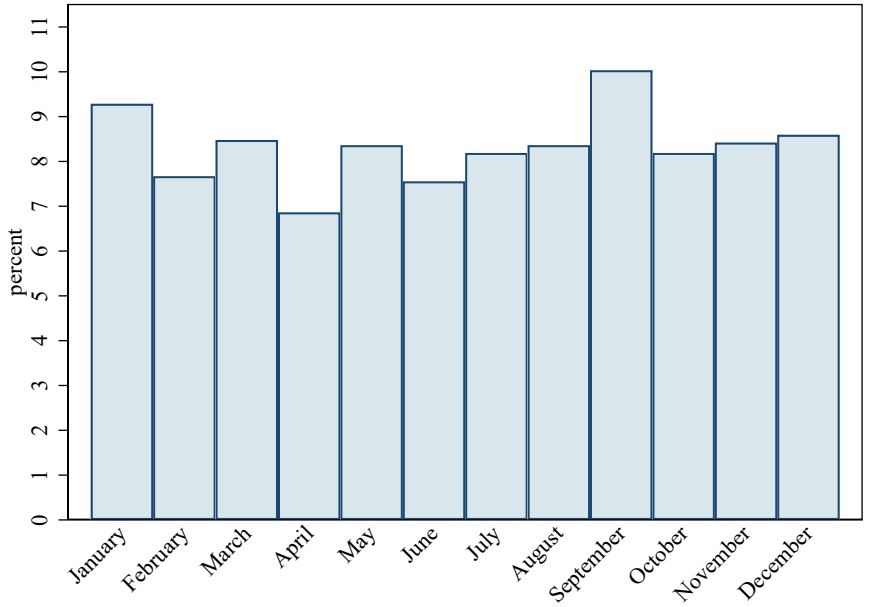
Source: Own elaboration based on Drake and Liu (2010).
Notes: An environmental insult during pregnancy to a mother (F0 generation) might affect not only the developing foetus (F1 generation) but also the germ cells which will go on to form the F2 generation.

Figure A2: Distribution of mother year of birth



Notes: Year of birth of mother based on reported age in years in R4 household roster. Bins are discrete, representing one year.

Figure A3: Distribution of mother month of birth



Notes: Month of birth of mother as reported in R4 household roster. Bins are discrete, representing one month-of-year.

Table A1: Effect of shock exposure on second generation outcomes: Alternative cluster group

	Age 1	Age 5	Age 8	Age 12	Age 15
Panel A: Height-for-age					
In-utero shock	-0.079	-0.076	-0.100	-0.173	-0.090
	[0.076]	[0.040]*	[0.047]**	[0.058]***	[0.051]*
Controls	Yes	Yes	Yes	Yes	Yes
N	1670	1657	1665	1671	1620
	Weight-for-age			BMI-for-age	
	Age 1	Age 5	Age 8	Age 12	Age 15
Panel B: Weight-/BMI-for-age					
In-utero shock	-0.179	-0.109	-0.059	0.045	-0.021
	[0.064]**	[0.043]**	[0.058]	[0.064]	[0.059]
Controls	Yes	Yes	Yes	Yes	Yes
N	1670	1657	1665	1671	1620

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Estimates are reported in standard deviations from the age- and gender-specific mean value. Cluster robust standard errors at the child cluster of birth presented in square brackets. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Table A2: Effects of shock exposure on second generation outcomes: By sex, regression coefficients

	Age 1	Age 5	Age 8	Age 12	Age 15
Panel A: Height-for-age					
In-utero shock	-0.201	-0.103	-0.154	-0.249	-0.139
	(0.103)*	(0.088)	(0.066)**	(0.074)***	(0.069)**
Female	0.131	-0.039	0.020	-0.102	-0.259
	(0.091)	(0.048)	(0.051)	(0.061)*	(0.070)***
Shock*Female	0.242	0.054	0.108	0.150	0.097
	(0.134)*	(0.123)	(0.084)	(0.104)	(0.087)
N	1670	1657	1665	1671	1620
	Weight-for-age			BMI-for-age	
	Age 1	Age 5	Age 8	Age 12	Age 15
Panel B: Weight-/BMI-for-age					
In-utero shock	-0.230	-0.167	-0.113	0.010	-0.093
	(0.083)***	(0.058)***	(0.077)	(0.063)	(0.075)
Female	0.152	-0.195	-0.113	-0.256	0.115
	(0.068)**	(0.053)***	(0.053)**	(0.053)***	(0.068)*
Shock*Female	0.102	0.117	0.107	0.069	0.143
	(0.088)	(0.081)	(0.104)	(0.116)	(0.117)
N	1670	1657	1665	1671	1620

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Estimates are reported in standard deviations from the age- and gender-specific mean value. Cluster robust standard errors in parentheses. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Table A3: Effects of shock exposure on second generation outcomes: By mother birth-location, regression coefficients

	Age 1	Age 5	Age 8	Age 12	Age 15
Panel A: Height-for-age					
In-utero shock	-0.072	-0.133	-0.131	-0.246	-0.135
	(0.080)	(0.051)**	(0.055)**	(0.067)***	(0.048)***
Urban-born	-0.102	-0.197	-0.161	-0.301	-0.110
	(0.206)	(0.147)	(0.137)	(0.161)*	(0.145)
Shock*Urban-born	-0.024	0.218	0.120	0.274	0.165
	(0.132)	(0.115)*	(0.125)	(0.142)*	(0.109)
Controls	Yes	Yes	Yes	Yes	Yes
N	1670	1657	1665	1671	1620
	Weight-for-age			BMI-for-age	
	Age 1	Age 5	Age 8	Age 12	Age 15
Panel B: Weight-/BMI-for-age					
In-utero shock	-0.213	-0.144	-0.094	0.061	-0.036
	(0.067)***	(0.054)***	(0.069)	(0.063)	(0.064)
Urban-born	-0.073	-0.165	-0.186	0.076	-0.168
	(0.156)	(0.124)	(0.141)	(0.130)	(0.119)
Shock*Urban-born	0.129	0.134	0.134	-0.058	0.059
	(0.142)	(0.130)	(0.148)	(0.094)	(0.107)
Controls	Yes	Yes	Yes	Yes	Yes
N	1670	1657	1665	1671	1620

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Estimates are reported in standard deviations from the age- and gender-specific mean value. Cluster robust standard errors in parentheses. Controls include an indicator of if the child is female. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Table A4: Effects of shock exposure on second generation outcomes: By growth stage, regression coefficients

	Height-for-age		BMI-for-age	
	Age 12	Age 15	Age 12	Age 15
In-utero shock	-0.102	-0.029	0.048	-0.128
	(0.075)	(0.118)	(0.052)	(0.118)
Pubertal growth	0.639	0.152	0.396	0.092
	(0.065)***	(0.069)**	(0.063)***	(0.076)
Pubertal growth = 1 × In-utero shock	-0.142	-0.069	-0.001	0.145
	(0.112)	(0.139)	(0.105)	(0.142)
Controls	Yes	Yes	Yes	Yes
N	1665	1617	1665	1617

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Estimates are reported in standard deviations from the age- and gender-specific mean value. Cluster robust standard errors in parentheses. Controls include an indicator of if the child is female. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Table A5: Effects of shock exposure on second generation outcomes: HAZ, Alternative specifications

	Age 1	Age 5	Age 8	Age 12	Age 15
Panel A: SPEI ≤ -0.8 S.D.					
SPEI ≤ -0.8 S.D.	-0.082	-0.039	-0.086	-0.127	-0.066
	(0.074)	(0.046)	(0.048)*	(0.057)**	(0.045)
Controls & FEs	Yes	Yes	Yes	Yes	Yes
N	1670	1657	1665	1671	1620
Panel B: Growing Season SPEI ≤ -1 S.D.					
Growing SPEI ≤ -1 S.D.	-0.126	-0.143	-0.145	-0.207	-0.156
	(0.087)	(0.078)*	(0.099)	(0.098)**	(0.088)*
Controls & FEs	Yes	Yes	Yes	Yes	Yes
N	1670	1657	1665	1671	1620

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Estimates are reported in standard deviations from the age- and gender-specific mean value. Cluster robust standard errors in parentheses. Controls include an indicator of if the child is female. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Table A6: Effects of shock exposure on second generation outcomes: WAZ/BMIAZ, Alternative specifications

	WAZ			BMIAZ	
	Age 1	Age 5	Age 8	Age 12	Age 15
Panel A: SPEI ≤ -0.8 S.D.					
SPEI ≤ -0.8 S.D.	-0.180	-0.091	-0.090	0.080	-0.009
	(0.072)**	(0.044)**	(0.056)	(0.053)	(0.071)
Controls & FEs	Yes	Yes	Yes	Yes	Yes
N	1670	1657	1665	1671	1620
Panel B: Growing Season SPEI ≤ -1 S.D.					
Growing SPEI ≤ -1 S.D.	-0.126	-0.123	-0.150	-0.007	-0.049
	(0.070)*	(0.081)	(0.081)*	(0.068)	(0.085)
Controls & FEs	Yes	Yes	Yes	Yes	Yes
N	1670	1657	1665	1671	1620

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Estimates are reported in standard deviations from the age- and gender-specific mean value. Cluster robust standard errors in parentheses. Controls include an indicator of if the child is female. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Table A7: Effects of shock exposure on second generation outcomes: Balanced panel sample

	Age 1	Age 5	Age 8	Age 12	Age 15
Panel A: Height-for-age					
In-utero shock	-0.067	-0.057	-0.080	-0.147	-0.077
	(0.070)	(0.045)	(0.045)*	(0.053)***	(0.046)*
Controls & FEs	Yes	Yes	Yes	Yes	Yes
N	1563	1563	1563	1561	1562
	Weight-for-age			BMI-for-age	
	Age 1	Age 5	Age 8	Age 12	Age 15
Panel B: Weight-/BMI-for-age					
In-utero shock	-0.179	-0.099	-0.027	0.094	-0.003
	(0.066)***	(0.043)**	(0.052)	(0.046)**	(0.060)
Controls & FEs	Yes	Yes	Yes	Yes	Yes
N	1568	1568	1568	1565	1568

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Estimates are reported in standard deviations from the age- and gender-specific mean value. Cluster robust standard errors in parentheses. Controls include an indicator of if the child is female. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Table A8: Effects of early-life shock exposure on second generation HAZ: Joint estimation

	Age 1	Age 5	Age 8	Age 12	Age 15
3 years before birth	-0.002	0.016	0.027	0.012	-0.061
	(0.086)	(0.080)	(0.072)	(0.077)	(0.060)
2 years before birth	0.066	0.055	0.033	0.028	0.037
	(0.057)	(0.059)	(0.058)	(0.066)	(0.065)
In-utero shock	-0.067	-0.072	-0.095	-0.161	-0.088
	(0.062)	(0.048)	(0.049)*	(0.055)***	(0.046)*
1 years after birth	0.097	0.080	0.094	0.103	0.035
	(0.081)	(0.077)	(0.062)	(0.066)	(0.079)
2 years after birth	-0.093	-0.025	-0.064	-0.048	-0.038
	(0.081)	(0.048)	(0.052)	(0.051)	(0.043)
3 years after birth	-0.012	-0.021	-0.015	0.019	-0.008
	(0.087)	(0.067)	(0.057)	(0.059)	(0.061)
4 years after birth	-0.011	0.018	-0.040	0.071	0.014
	(0.068)	(0.056)	(0.054)	(0.065)	(0.065)
5 years after birth	0.016	-0.064	-0.019	-0.002	-0.034
	(0.056)	(0.055)	(0.048)	(0.059)	(0.046)
Controls & FEs	Yes	Yes	Yes	Yes	Yes
N	1670	1657	1665	1671	1620

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Estimates are reported in standard deviations from the age- and gender-specific mean value. Cluster robust standard errors in parentheses. Controls include an indicator of if the child is female. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Table A9: Effects of early-life shock exposure on second generation WAZ/BMIAZ: Joint estimation

	Age 1	Age 5	Age 8	Age 12	Age 15
3 years before birth	0.012	-0.028	0.028	0.058	0.099
	(0.067)	(0.067)	(0.081)	(0.053)	(0.056)*
2 years before birth	0.022	0.088	0.121	0.080	0.152
	(0.083)	(0.061)	(0.081)	(0.077)	(0.056)***
In-utero shock	-0.181	-0.089	-0.039	0.053	0.005
	(0.066)***	(0.050)*	(0.063)	(0.052)	(0.063)
1 years after birth	-0.003	0.071	0.095	-0.022	0.049
	(0.062)	(0.069)	(0.073)	(0.052)	(0.060)
2 years after birth	-0.062	-0.070	-0.012	-0.025	-0.037
	(0.060)	(0.046)	(0.051)	(0.059)	(0.066)
3 years after birth	0.037	0.026	0.029	0.044	0.027
	(0.087)	(0.056)	(0.054)	(0.062)	(0.063)
4 years after birth	0.100	0.049	-0.000	0.052	0.059
	(0.088)	(0.060)	(0.066)	(0.073)	(0.082)
5 years after birth	-0.042	0.050	0.008	0.025	0.081
	(0.050)	(0.041)	(0.049)	(0.052)	(0.044)*
Controls & FEs	Yes	Yes	Yes	Yes	Yes
N	1670	1657	1665	1671	1620

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Estimates are reported in standard deviations from the age- and gender-specific mean value. Cluster robust standard errors in parentheses. Controls include an indicator of if the child is female. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Table A10: Effects of early-life shock exposure on second generation HAZ: Estimating separately

	Age 1	Age 5	Age 8	Age 12	Age 15
Panel A: 3 years before birth					
SPEI ≤ -1 S.D.	-0.010	0.007	0.020	-0.005	-0.067
	(0.082)	(0.074)	(0.071)	(0.075)	(0.055)
Panel B: 2 years before birth					
SPEI ≤ -1 S.D.	0.063	0.057	0.036	0.027	0.043
	(0.059)	(0.059)	(0.061)	(0.068)	(0.065)
Panel C: In-utero					
In-utero shock	-0.079	-0.076	-0.100	-0.173	-0.090
	(0.064)	(0.045)*	(0.048)**	(0.055)***	(0.045)**
Panel D: 1 year after birth					
SPEI ≤ -1 S.D.	0.110	0.086	0.105	0.121	0.050
	(0.078)	(0.073)	(0.062)*	(0.063)*	(0.079)
Panel E: 2 years after birth					
SPEI ≤ -1 S.D.	-0.093	-0.023	-0.061	-0.056	-0.034
	(0.075)	(0.050)	(0.054)	(0.049)	(0.042)
Panel F: 3 years after birth					
SPEI ≤ -1 S.D.	-0.002	-0.020	-0.007	0.016	0.004
	(0.079)	(0.059)	(0.056)	(0.060)	(0.058)
Panel G: 4 years after birth					
SPEI ≤ -1 S.D.	-0.008	0.027	-0.032	0.077	0.023
	(0.074)	(0.053)	(0.056)	(0.064)	(0.063)
Panel H: 5 years after birth					
SPEI ≤ -1 S.D.	0.027	-0.055	-0.003	0.009	-0.025
	(0.056)	(0.053)	(0.047)	(0.057)	(0.044)
Controls & FEs	Yes	Yes	Yes	Yes	Yes
Observations	1670	1657	1665	1671	1620

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Estimates are reported in standard deviations from the age- and gender-specific mean value. Cluster robust standard errors in parentheses. Controls include an indicator of if the child is female. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Table A11: Effects of early-life shock exposure on second generation WAZ/BMIAZ: Estimating separately

	Age 1	Age 5	Age 8	Age 12	Age 15
Panel A: 3 years before birth					
SPEI $\leq$ -1 S.D.	0.001	-0.043	0.010	0.049	0.082
	(0.058)	(0.064)	(0.083)	(0.055)	(0.056)
Panel B: 2 years before birth					
SPEI $\leq$ -1 S.D.	0.023	0.084	0.119	0.066	0.135
	(0.077)	(0.064)	(0.081)	(0.074)	(0.055)**
Panel C: In-utero					
In-utero shock	-0.179	-0.109	-0.059	0.045	-0.021
	(0.063)***	(0.047)**	(0.056)	(0.049)	(0.059)
Panel D: 1 year after birth					
SPEI $\leq$ -1 S.D.	0.017	0.088	0.094	-0.033	0.041
	(0.058)	(0.065)	(0.072)	(0.050)	(0.064)
Panel E: 2 years after birth					
SPEI $\leq$ -1 S.D.	-0.066	-0.074	-0.015	-0.031	-0.041
	(0.055)	(0.045)	(0.053)	(0.056)	(0.061)
Panel F: 3 years after birth					
SPEI $\leq$ -1 S.D.	0.041	0.029	0.023	0.034	0.008
	(0.078)	(0.053)	(0.056)	(0.062)	(0.062)
Panel G: 4 years after birth					
SPEI $\leq$ -1 S.D.	0.108	0.043	-0.010	0.037	0.037
	(0.085)	(0.061)	(0.068)	(0.071)	(0.077)
Panel H: 5 years after birth					
SPEI $\leq$ -1 S.D.	-0.036	0.053	0.010	0.010	0.073
	(0.047)	(0.039)	(0.047)	(0.048)	(0.045)
Controls & FEs	Yes	Yes	Yes	Yes	Yes
Observations	1670	1657	1665	1671	1620

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Estimates are reported in standard deviations from the age- and gender-specific mean value. Cluster robust standard errors in parentheses. Controls include an indicator of if the child is female. Fixed effects for child cluster-of-residence, year-month birth cohort, and mother year- and province-of-birth are suppressed.

Table A12: Effect of zero generation socioeconomic status on probability of shock exposure

	In-utero shock	In-utero shock
Grandmother speaks Spanish	-0.072	
	(0.046)	
Grandparent completed secondary		0.044
		(0.063)
Controls	No	Yes
N	1670	522

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Cluster robust standard errors in parentheses. Controls include if the grandparent reporting educational attainment if female, and their age in years. Fixed effects for mother year- and province-of-birth are suppressed.

Table A13: Effect of shock exposure on zero generation migration/fertility choices

	Migration				Fertility	
	Before age 5	Ever migrate	Rural-urban	Lima/ Callao	Mother birth month	Grand-child gender
In-utero shock	-0.004	0.013	0.021	-0.009	-0.410	0.028
	(0.008)	(0.028)	(0.021)	(0.016)	(0.277)	(0.032)
N	1632	1632	1632	1632	1670	1670

Notes: * p< 0.10, ** p< 0.05, *** p< 0.01. Cluster robust standard errors in parentheses. Fixed effects for mother year- and province-of-birth are suppressed.

Table A14: Socieoeconomic status controls and parent investments at each survey round

Age	**Intermediate confounders**
1	HH size, family own house, attended antenatal classes, attended birth, hospital birth, parenting skills index
5	HH size, family own house, p/c food expenditure, child food diversity/frequency, HH food security, health and education expenditure, preschool
8	HH size, family own house, p/c food expenditure, child food diversity/frequency, HH food security, health and education expenditure, # of books in HH, caregiver involvement index
12	HH size, family own house, p/c food expenditure, child food diversity/frequency, HH food security, health and education expenditure, # of books in HH, caregiver involvement index
15	HH size, family own house, p/c food expenditure, child food diversity/frequency, HH food security, health and education expenditure, # of books in HH

Notes: The list of intermediate variables varies over rounds as not all questions are asked in each round. *Abbreviations:* HH - household; p/c - per capita.

B Estimating ACDE using sequential g-estimation

B.1 Average controlled direct effect

Let Y_i be the observed outcome for unit i and $Y_i(a)$ the potential outcome if treatment was set to a. Following the potential outcomes framework (Rubin, 1974), the causal effect of treatment is the difference between the two potential outcomes in which unit i switched from treatment level a' and a:

$$\tau_i(a, a') = Y_i(a) - Y_i(a').$$

Given that we only observe one of these potential outcomes, we focus on the average treatment effect (or total effect), defined as the difference in means between two potential different outcomes:

$$ATE(a, a') \equiv \tau(a, a') = E[Y_i(a) - Y_i(a)],$$

where $E[\cdot]$ is the expectation over units in the population of interest. Given some mediator M for the effect of treatment on the outcome, the controlled direct effect can be defined as the effect of changing treatment while holding fixed the value of the mediator. $Y_i(a, m)$ is the outcome for unit i for a set level of treatment a and mediator m. The potential value of the mediator may also be defined similarly to potential outcomes as $M_i(a)$, the level the mediator takes on given treatment level a. The controlled direct effect is therefore expressed:

$$CDE_i(a, a', m) = Y_i(a, m) - Y_i(a', m).$$

As above, defining based on the expectation the average controlled direct effect ACDE is given by:

$$ACDE(a, a', m) = E[Y_i(a, m) - Y_i(a', m),] \tag{B1}$$

which describes the average direct effect of treatment if the mediator is fixed at value m for all units in the population. See Acharya et al. (2016), Joffe and Greene (2009), and VanderWeele (2009) for in-depth discussion.

B.2 Assumptions

Following Acharya et al. (2016) I estimate the average controlled direct effect (ACDE) using "sequential g-estimation" (or reverse sequential two stage (RS2S) parametric estimation), as set out by VanderWeele (2009) and Joffe and Greene (2009). The ACDE is identified under the following assumptions: 1) Sequential unconfoundedness; and 2) No

intermediate interactions.

Assumption 1: Sequential unconfoundedness:

$$\{Y_i(a,m), M_i(a)\} \perp\!\!\!\perp A_i \mid X_i = x, \tag{B2}$$

$$Y_i(a,m) \perp\!\!\!\perp M_i \mid A_i = a, X_i = x, Z_i = z, \tag{B3}$$

For which the following conditional probabilities must be non-zero:

$$P(A_i = a \mid X_i = x) > 0,$$

$$P(M_i = m \mid A_i = a, X_i = x, Z_i = z) > 0,$$

For all possible treatment values $a \in \mathcal{A}$, mediator values $m \in \mathcal{M}$, covariates $x \in \mathcal{X}$, and intermediate confounders $z \in \mathcal{Z}$.

Equation B2 states that the potential outcome $Y_i(a,m)$ (that unit i takes if treatment is set at value a and mediator at value m) and potential mediator value $M_i(a)$ (that the mediator would take under treatment level a) are conditionally independent of the observed treatment status A_i given covariates $X_i = x$. That is there is no omitted relevant variables (U_{i1}) for the effect of treatment on the outcome or mediator conditional on pretreatment covariates. This is assumed to hold in this study given exogenous and random exposure climate shocks in-utero. Equation B3 further states that conditional on set levels of treatment, covariates and post-treatment (intermediate) confounders, the potential outcome is independent of observed value of mediator, e.g. there are no omitted relevant variables (U_{i2}) for the effect of the mediator on outcomes.

Under strong assumptions, these conditions could justify the use of standard regression analysis using a single equation, however this unlikely to be sufficient if there exists some post-treatment covariate Z which is influenced by treatment A, influences the mediator M, and is independently associated with the outcome Y (Robins, 1986). The ACDE can still be identified non-parametrically under the above assumption alone in the presence of these intermediate confounders z_i, however this requires this requires the distribution of these confounders (conditional on A_i and X_i) to be known and correctly specified (Acharya et al., 2016; Joffe & Greene, 2009), therefore a further assumption is made.

Asumption 2: No intermediate interactions

$$E[Y_i(a,m) - Y_i(a,m') \mid A_i = a, X_i = x, Z_i = z] = E[Y_i(a,m) - Y_i(a,m') \mid A_i = a, X_i = x], \tag{B4}$$

For all values $a \in \mathcal{A}$, $m, m' \in \mathcal{M}$, $z \in \mathcal{Z}$, and $x \in \mathcal{X}$.

This states that the effect of the mediator on the outcome must be conditionally independent of any intermediate confounders. This assumption can in fact be false, in which case the estimated effects will be weighted averages of the ACDE within levels the

intermediate confounders (Acharya et al., 2016).

B.3 Identification

To derive the ACDE of treatment on outcome, we define a demediation function:

$$\gamma(a, m, x) = E[Y_i(a, m) - Y_i(a, 0) \mid X_i = x]. \tag{B5}$$

This function describes the difference between outcomes with mediator set at level m and zero, and does not depend on the levels of intermediate confounders if Equation B4 holds. By subtracting the demediation function from the observed outcome $Y_i = Y_i(A_i, M_i)$, variation in the outcome due to the mediator is removed:

$$E[Y_i - \gamma(a, M_i, x) \mid A_i = a, X_i = x] == E[Y_i(a, 0) \mid X_i], \tag{B6}$$

provided **assumption 1** is met, the effect of the mediator on the outcome is identified. The ACDE,

$$E[Y_i(a, 0) - Y_i(0, 0) \mid X_i = x],$$

conditional on pretreatment covariates X_i, is therefore identified as the difference in means of the demediated outcome:

$$E[Y_i - \gamma(a, M_i, x) \mid A_i = a, X_i = x] - E[Y_i - \gamma(0, M_i, x) \mid A_i = 0, X_i = x]. \tag{B7}$$

B.4 Estimation

The ACDE is estimated parametrically using sequential g-estimation in a two-stage process.

1st stage: Under **assumption 1**, the demediation function (Equation B5) can be estimated from the data as the difference in means estimator, conditioning on both the pretreatment covariates X_i and intermediate confounders Z_i. I therefore first regress the outcome on the treatment, mediator, and all covariates to obtain an estimate of the effect of the mediator on the outcome, from which I can derive the demediation function. In the simplest specification:

$$Y_i = \alpha_0 + \alpha_1 A_i + \alpha_2 M_i + \alpha_i X_i + \alpha_i Z_i + \nu_i, \tag{B8}$$

where there is no interaction between the mediator and outcome or covariates (and by **assumption 2** no interaction with intermediate confounders), the coefficient of interest is α_2. The sample version of the demediation function is expressed as:

$$\hat{\gamma}(A_i, M_i, X_i; \hat{\alpha}) = \hat{\alpha_2} M_i. \tag{B9}$$

2nd stage: First, the outcome is adjusted using the estimated demediation function:

$$\tilde{Y_i} = Y_i - \hat{\alpha_2} M_i. \tag{B10}$$

The demediated outcome is then regressed on the treatment (A_i) and pretreatment covariates (X_i), as outlined in Equation B7:

$$\tilde{Y_i} = \beta_0 + \beta_1 A_i + \beta_2 X_i + \varepsilon_i, \tag{B11}$$

where the least squares estimator $\hat{\beta_1}$ is the consistent estimate of the ACDE. Given this is a two-step process, standard errors on $\hat{\beta_1}$ are biased, therefore bootstrap standard errors are obtained.