Shocks and Human Capital
Evidence on Education, Health and Nutrition

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1. Introduction

Nutrition, health and human capital have played a key role in the literature on poverty traps. The nutrition wage hypothesis posited that agricultural workers could earn a wage that was so low they would be trapped in a recurring cycle of poverty in which they were unable to feed themselves sufficiently well to sustain the productivity necessary to be able to climb out of the poverty trap. The model was among the early descriptions of poverty traps and underlies the larger literature on the efficiency wage hypothesis (Liebenstein, 1957; Mazumdar, 1959). Investments in human capital, broadly defined to include nutrition, health, education and cognition, have been shown to be central mechanisms through which an individual, family, community and state may be lifted out of poverty and out of enduring poverty traps.

Shocks have been implicated in a large theoretical and empirical literature on poverty and poverty traps. The loss of resources because of an unanticipated negative shock results in a spiral down into ever greater poverty because of a loss of earnings capacity (due to, for example, a health shock), a greater debt burden (to cover the costs of the losses associated with the shock) or the loss of a productive asset that an individual simply cannot be replaced (because of a lack of liquidity or credit markets or because it is not worth the replacement cost).

The goal of this research is to evaluate some of the evidence on the links between shocks, on one hand, and health and human capital outcomes, on the other hand. We focus on the impact of shocks on human capital during early life and examine the extent to which the human capital of populations and population sub-groups display resilience in the face of an adverse economic shock.

Three main points emerge. First, it is very hard to identify shocks that are unanticipated and not likely to be correlated with other factors that affect human capital investments and outcomes. One reason is that investing in child human capital is universally recognized as important and individuals and families are likely to attempt to mitigate the impact of shocks on the human capital of the next generation. This is our second point: establishing generalizable evidence on the impact of a shock is complicated by the fact that there may be behavioral responses to mitigate the impact of the shock and the nature and effectiveness of those behaviors will vary with the context in ways that are not straightforward to predict or model. Third, even in cases that seem relatively straightforward to model, the impacts of shocks on child human capital outcomes in the short and longer-term may differ and drawing inferences about the longer-term impacts based on negative impacts in the short term can be very misleading.

2. Early life shocks and human capital in later life

Many studies have investigated impacts of early life shocks on later life outcomes. The earliest shocks an individual will experience, those that occur in utero, have been linked to health in adulthood. Building on a foundation in biology, and drawing on evidence from animal and human models, in a very creative body of research, Barker describes the impact on the fetus of nutrition insults at specific times during the pregnancy. As an example, he elucidates pathways from a shock at the time when arteries are being developed in utero to hardened arteries and coronary heart disease in mid- life (Barker, 1995). Links between in utero shocks, birthweight, adult health and premature mortality have all been widely documented.

For example, influential research has exploited the timing of ration restrictions because of the Nazi occupation of the Netherlands during the second World War to establish the impact of nutrition shocks on health and well-being in adulthood. Critically important for this research on the Dutch Hunger is that rations were restricted in areas where the resistance was active but rations were not changed in other areas so that women who were pregnant and living in the resistance areas experienced a negative nutrition shock while pregnant women in other areas served as controls. The restriction of rations in the resistance areas was not anticipated and short-lived; there was very limited scope for moving to avoid the restrictions. Assuming women who were pregnant at the time and living in the resistance areas are exchangeable with pregnant women living in other areas in the Netherlands, comparing birth and later health outcomes of those who were in utero at the time, it is possible to identify a casual effect of in utero nutrition shocks.

Moreover, as predicted by Barker, the precise timing of the nutrition insults plays a key role. Research on the impact of the Dutch Hunger finds that those who were exposed early during the fetal period, were at elevated risks of obesity, lipid dysregulation, cardio-vascular disease and premature mortality. In contrast, those who were exposed only towards the end of the gestation period were more likely to be born small but there is little evidence that adult health outcomes were affected. (Ravelli et al, 1998; Ravelli et al, 1999; Roseboom et al, 2001; Rooij et al, 2010).

This evidence is consistent with programming in utero to develop a “thrifty phenotype” as an adaptive response to nutrition insults (Hales and Barker, 1992). Specifically, nutrition deprivation early during gestation programs the fetus to adapt to the environment and extract all the nutrients possible from the placenta. Having been programmed to do this in utero, the body

continues to maximize nutrient absorption after birth which results in poor health outcomes in later life. Nutrition deprivation towards the end of gestation does not cause the same type of re-programming.

Many studies have investigated the longer-term consequences of in utero insults using, for example, variation in rainfall, famines, terrorist attacks, civil conflicts and, weather-related and other natural disasters. The key issue with any natural experiment is that the variation underlying identification of the causal effect must be outside the control of the mother, and her family. In some cases, this calls for strong assumptions. As an example, studies have exploited the timing of Ramadan, the month of fasting in the Muslim calendar, to identify a causal effect of nutrition deprivation in utero. There are at least two concerns. First, Ramadan occurs on an established calendar and, give or take a day or two, it is timing is known years in advance. In societies where contraception is widespread, pregnancy timing is, at least in part, a choice. Second, it is permissible for pregnant women not to fast if their own health or that of their baby is at risk. It is difficult to test whether the assumptions underlying the identification strategy are met in most studies.

In utero exposure and human capital in adulthood

In a widely-cited and very influential study, Almond (2006) examines the impact of in utero exposure to the 1918 influenza pandemic on education, work and income in adulthood. This is a very creative study that combines the unanticipated nature of the influenza pandemic in the fall of 1918 with census data and compares outcomes of birth cohorts exposed to the pandemic in utero with surrounding birth cohorts. Almond concludes that males who were exposed in utero completed fewer years of education, had lower income, poorer socio-economic status, received more in public transfers and were more likely to have physical disabilities. Moreover, the results are based on two, complementary identification strategies. First, the 1919 birth cohort is compared with surrounding birth cohorts and, second, exposure to the influenza pandemic is proxied with a measure of mortality infection that varies over time and across states.

The left hand panel of Figure 1 is from Almond (2006). It displays completed years of education of males by birth year as reported in the 1960 U.S. census. Males born in 1919 completed between 0.15 and 0.19 fewer years of education than expected given the secular trend. Almond reports similar results for comparisons of high school graduation, earnings, total income, poverty and socio-economic status (SES) in middle age.

A potential concern with a cohort-based identification strategy is that the birth cohort is different from surrounding birth cohorts in ways other than exposure to influenza in utero. The 1919 birth cohort was conceived as World War I was ending and it is possible that the parents of that birth cohort are different from the parents of surrounding birth cohorts. The right hand panel of Figure 1 is drawn from Brown and Thomas (2016). It displays the fraction of fathers of each birth cohort who are literate as recorded in the 1930 U.S. census. The fathers of the 1919 birth cohort are over 0.25 percentage points (s.e.=0.12) less likely to be literate than expected (given the secular rise in literacy). Some fathers might have become literate between the birth of the child and the 1930 census and the earlier birth cohorts in the figure might be selected on human capital since they would have to co-reside with the father. The paternal literacy gap is even larger if the comparisons is based on the 1920 census (which does not include post-1919 births): fathers of the 1919 birth cohort are 1.42 percentage points (s.e.=0.50) less likely to be literate. It is not possible to compare paternal education across birth cohorts of their sons because neither the 1920 nor the 1930 census records education. However there are other markers of paternal SES and they display comparable patterns.

It is not clear how much of the deficit in completed education of the 1919 birth cohort can be attributed to differences in paternal human capital. The 1960 census does not record paternal background. Brown and Thomas construct proxies for paternal characteristics using the 1920 census (and the 1930 census) and include those proxies along with state fixed effects in models that compare human capital and socio-economic outcomes of the birth cohorts in adulthood. The results for models that compare the completed years of education of the birth cohort exposed in utero to influenza with surrounding birth cohorts are in Table 1.

The first row in the table reports models that control paternal characteristics constructed using the 1920 census. This has the advantage of recording paternal information that is proximate the birth of the target (and comparison) males but the disadvantage that the analyses are restricted to the 1912 through 1919 birth cohorts. Paternal characteristics are state- and race-specific averages for each birth cohort of paternal literacy rates, occupation-income score, number of children in the household and paternal age at the birth of the index child. With no paternal controls, males in the 1919 birth cohort completed 0.188 fewer years of education. Adding these paternal controls, the 1919 birth cohort completed 0.230 more years of education.

The second row of the table repeats the analysis using the 1930 census to construct paternal controls. This has the advantage of extending the comparison cohort to after 1919 but the disadvantage that the gap between the birth of the cohorts and measurement of proxies for

paternal characteristics is longer. As a result, paternal characteristics that are unlikely to have changed over time are selected for these models: they are whether the father is white, whether the father was a World War I veteran and age of the father at the birth of the child. (The proxies for paternal characteristics constructed from the 1920 census are likely to change over time and so are not included in these models.) The results using the 1920 and 1930 census to construct proxies for background are striking in their similarity. Without paternal controls, the 1919 birth cohort completed significantly less education but with even these very crass paternal controls, the 1919 birth completes significantly more education. Brown and Thomas document similar patterns for other indicators of socio-economic status in adulthood of the 1919 birth cohort relative to surrounding cohorts.

Almond did not rely on cohort differences alone to identify the effect of in utero exposure to influenza. His second approach examines the effect of the state-specific maternal infection rate on outcomes in models that include both cohort effects and state of birth fixed effects. The estimates are identified by changes in the maternal infection rate. Almond finds that those males who were exposed to higher infection rates in utero completed fewer years of education. The effects of variation in maternal infection rates on socio-economic outcomes in adulthood are also negative but not statistically significant.

State-specific maternal infection rates are potentially correlated with other state characteristics – such as the quality of health services and infrastructure – which are likely related to state-levels of income and economic prosperity. To probe this concern, Brown and Thomas re-estimate the models adding a measure of the state-level SES, the paternal socio-economic index of children born in each state in each cohort. As shown in the third row of Table 1, the estimated effects of states-specific maternal infection rates are reduced in magnitude by over a third and are not statistically significant.

This example illustrates the difficulties that arise in identifying the impact of early life shocks on later life outcomes. Almond's study is a very creative approach to addressing an important question and he deserves much credit for developing the approach and conducting the analyses. It seems clear from the literature on the 1918 influenza pandemic that it was a shock. The flu was recorded in Kansas in March 1918 and made its way to New York and Pennsylvania, but this was not described in the popular press. In fact, it appeared a little later in Europe and it was only in Spain that it received widespread media attention and, hence, was dubbed the "Spanish flu" although it likely originated in the United States (Barry, 2004). Interpretation of

the evidence is complicated by a coincident event and potentially contaminating event: the end of World War 1.

This example is not intended to imply that there are no long-term consequences of in utero shocks. Clearly, there are. Rather, the point is that even seemingly very well-identified studies may not be as well identified as they seem. Importantly, whereas several studies have established that early life shocks have long term consequences on health, establishing that in utero shocks have long-term impacts on education and socio-economic status, and, thus on poverty, has proven to be somewhat more difficult to establish.

3. Financial shocks, human capital and behavioral responses

A second concern that arises with interpreting results of studies of the impact of shocks on human capital outcomes is that even if a shock is truly unanticipated – in the sense that it was not anticipated and that it did not coincide with other events that affected human capital – the impact of the shock may be mitigated by behavioral responses of individuals, their families and their communities. This is important since there is considerable evidence that children who are born into disadvantaged environments are not necessarily destined to have poor human capital outcomes in adulthood. (Heckman, 2006, for example.)

There are at least two concerns with inferences drawn under the assumption that there are no behavioral responses to a shock. First, if individuals and their families invest in behaviors that mitigate the impact of the shock on, say, human capital, the finding that the shock had no impact on human capital outcomes in the immediate term may be misleading if the mitigating behaviors affect future investments. For example, if a household liquidates assets when prices are relatively low in order to keep a child in school, it is possible that the reduced long-run wealth will have negative impacts on future well-being. Moreover, it is important to trace out these types of responses in order to develop a fuller understanding of the impacts of shocks on human capital.

Second, if some individuals and families are more able to smooth the impact of a shock than others, distributional impacts of the shock on human capital outcome may not be informative about the incidence of the initial impact of the shock or about the longer-term consequences of the shock.

These concerns are illustrated using results from one example of the global financial crisis that occurred at the end of the 20th century. Specifically, we focus on Indonesia, the country that experienced the most disruption as a result of the East Asian financial crisis.

The 1998 Indonesian financial crisis

After almost three decades of sustained economic growth, of all the East Asian countries Indonesia was hit the hardest by the 1997 financial crisis. As shown in Figure 2a, the Indonesian rupiah came under pressure in the last half of 1997 when the exchange rate began showing signs of weakness. The rupiah fell from around 2,400 per US$ in the middle of the year to about 4,800 per US$ by the end of 1997. In January 1998, the rupiah collapsed. Over the course of a few days, the exchange rate fell by a factor of three to Rp15,000 per US$. Although it soon recovered, by the middle of the year the rupiah had slumped back to the lows of January, 1998. By the end of 1998, the rupiah had strengthened to around Rp8,000 per US$. No other East Asian country experienced as deep or as prolonged a decline in the exchange rate.

Interest rates in Indonesia behaved much like the exchange rate: they spiked in August 1997 -- when they quadrupled -- and they remained extremely volatile through all of 1998. Chaos reigned in the banking sector and several major banks were taken over by the Indonesian Bank Restructuring Agency. All of this turmoil has wreaked havoc with both the confidence of investors and the availability of credit. Asset prices collapsed – both in the stock market and real asset including land.

Simultaneously, there was dramatic transformation in the political sector. Protests across the country that began in January 1998 culminated in May 1998 with Suharto's resignation after three decades of power. Uncertainty in the country was widespread and while the incoming president, Habibie, declared that multi-party elections would be held, it was very difficult to predict whether the elections would, in fact, be held, whether the outcome would result in further turmoil and whether there would be any fundamental change in the political sphere. It turned out that Suharto's resignation presaged the ushering in of democracy in Indonesia – an outcome that few predicted at the time. (Fisman, 2001)

The 1998 financial and political crisis was a major shock. Overall, GDP per capita in 1998 was about 15% below its level in 1997 (Figure 2b). This is the magnitude of the Great Depression in the U.S.; by contrast, GDP per capita declined by less than 5% during the 2008 Great Recession. The Indonesian crisis was not only large; it was also largely unanticipated and, certainly, its severity was a shock both inside and outside the country.

In January 1998, the IMF described Indonesia's economic situation as "worrisome" (IMF, 1998) and, at the same, the Indonesian government announced policies to address the situation, predicting economic growth for 1998 would be zero. Indeed, the president of the World Bank

summed up the situation in July 1998 saying "we were caught up in the enthusiasm of Indonesia. I am not alone in thinking that 12 months ago, Indonesia was on a very good path."

One way to summarize the impact of the crisis on the lives of Indonesians is through its impact on prices. Prices of many commodities spiraled upwards during the first three quarters of 1998. As shown in Figure 3, according to Statistics Indonesia, annual inflation was between 5 and 10% from the early 1980s to 1997. In 1998 inflation rocketed to an unprecedented 80% in a matter of months. Subsidies were removed from several key goods, most notably rice, oil and fuel, the prices of which were pegged by the government at levels below world prices. As the rupiah collapsed, those subsidies became unsustainable and were scaled back along with other public expenditures. Food prices, driven primarily by the rise in the price of the staple, rice, rose by 120%, which is about 50% more than the general price index.

Net food consumers were severely impacted by the crisis whereas net food producers either received some protection or possibly benefited from the crisis. While the financial crisis provided new opportunities in the export sector, it was a major negative shock for those on fixed incomes and those outside the tradeable sector.

Impacts on child health and education

To provide empirical evidence on the impact of the shock on human capital outcomes, we draw on data from the Indonesia Family Life Survey (IFLS) that were specifically designed to provide evidence of the immediate impact of the shock. The second wave, IFLS2, was fielded between August 1997 and February 1998 with the main fieldwork being completed by mid-December 1997 and a small number of very difficult tracking cases being completed in January and February, 1998. Because of the political and financial crisis, we decided to conduct a special rapid-response survey, IFLS2+, between August and December 1998 in order to provide a snap-shot of the immediate impact of the shock.

It was imperative to put IFLS2+ in the field without delay in order to measure the impact of the onset of the crisis. Since it was not possible to mount an entire wave of IFLS in a short time-frame, we selected a sample of one in four enumeration areas that were included in the 1993 IFLS baseline. IFLS2+ collected information from all the respondents who were living in those enumeration areas at the baseline, including those who had moved away. This is important because migration was a potential response to the crisis.

IFLS2 returned to the baseline enumeration area and interviewed every member of the baseline households in the community. Baseline respondents who had moved were tracked and

interviewed in the new location, along with the members of their new household. IFS2 re-interviewed at least one member of 94% of the baseline households.

IFLS2+ followed the same protocols, returning to the location of each household interviewed in IFLS2, interviewing all members and tracking movers to their new locations. IFLS2+ interviewed at least one member of over 99% of the households interviewed in IFLS2 and over 96% of all baseline households. (Frankenberg, Thomas and Beegle, 1999).

It is useful to use data from IFLS2 and IFLS2+ to provide a description of the magnitude of the shock that households experienced. The ILO predicted that millions would join the ranks of the unemployed because of the crisis and called for public works programs. Wage employment did decline – but only by 2% -- and it was more than offset by an increase in the number of people working in self-employment, particularly females who joined family businesses. With the dramatic increase in the price of rice, the number of people working in the agricultural sector rose and there was considerable migration from urban to rural areas. While the drama of the crisis was not reflected in employment, it was reflected in hourly earnings.

Between 1986 and 1997, in the market sector, real hourly wages grew by over 40% on average. In 1998, all of those gains were wiped out: real wages declined by 40% because nominal wages did not keep up with rising prices. In the self-employed sector, hourly earnings declined by 40% in urban areas. In rural areas, hourly earnings of females declined by 40% but those of males were little changed. To some extent, this reflects the impact of the price of rice (and food) and also the contributions of unpaid family workers (who contributed more work on the farm in 1998 on average than in 1997). Overall, household income declined by 30% between 1997 and 1998. (Smith et al, 2002).

There is some evidence that households smoothed these real income declines as real household per capita expenditure (PCE) declined 23% with the biggest declines occurring among those who were best off prior to the crisis. Using the PCE distribution from 1993, the households at the 75^{th} percentile reduced PCE by 30% whereas the decline was 15% for the households at the 25^{th} percentile of the 1993 PCE distribution. The crisis appears to have hit PCE of those who were better off the most although, to be sure, these are very large declines throughout the pre-crisis distribution of PCE. (Frankenberg, Smith and Thomasl, 2003).

What was the impact on human capital of children? We first examine anthropometric outcomes and then turn to education. Anthropometry is measured for all household respondents in IFLS. The upper panel of Figure 3 displays the z score of height for age (in the upper panel) and weight for height (in the lower panel) of all children age 9 and under measured in IFLS2, before

the onset of the financial crisis, and in IFLS2+, after the onset of the financial crisis. Height for age is a longer-run indicator of nutritional status and an important marker of health in and of itself. In addition, length during the first few years of life has been shown to be affected by health and disease-related shocks and it is thought that length by around 24 or 30 months is a very good predictor of attained adult height which, in turn, is predictive of longevity, improved health and socio-economic success. Weight-for-height is a measure of shorter-run nutritional status and is likely to reflect the impact of recent health inputs including food intake and disease insults. (Martorell and Habicht, 1986; Alderman, Hoddinott and Kinsey, 2006; Hoddinott et al, 2008).

There is no evidence that child height for age was deleteriously impacted by the financial crisis. There are no significant differences in height for age for any of the age groups although the sample size for the very youngest, and arguably the most vulnerable, is too small to be informative. The evidence on weight for height is less clear for children age under 36 months with these children having lower weight, given height, in the 1998 survey relative to the 1997 survey. The difference, however, is not statistically significant and, importantly, the deficits in weight for height for the youngest children are not evident in height for age.

Figure 4 is based on all children in both surveys, including new entrants into the 1998 survey in order to capture new births and mitigate the costs of the relatively small sample size. There were few new entrants and with 99% recontact, attrition is ignorable. The figures restricted to panel children are essentially the same. While the samples are small, separating rural from urban households does not suggest differential effects across these sectors in anthropometric outcomes.

Results for school enrollment are displayed in Table 2.Among children age 13 to 16, 25.6% were not enrolled in school in 1997 and in 1998, 25.1% of the children in that age group were not enrolled: with the onset of the crisis, there was a 2% decrease in the fraction not in school. However, among children age 7 to 12, 3.1% were not enrolled in school in 1997 and 5.9% were not enrolled in 1998: a 90% increase in non-enrollment. This gap was largest for the poorest households: for the households in the bottom quartile of PCE (in 1997), 4.9% of the children were not enrolled in school in 1997 and 10.7% were not enrolled in 1997: an almost 120% increase in non-enrollment. Further, for poor children in this age group, having an older sibling who was enrolled in school was associated with a greater chance of not being enrolled. (And, conversely, older poor children with younger siblings who were not in school in 1998 were more likely to be enrolled in 1998. We find no differences between urban and rural children or between male and female children (Thomas et al, 2004).

Why would older children in the poorest households stay in school when their younger siblings were not enrolled? There are at least two plausible reasons. First, if a 13 to 16 year old does not enroll in school in a year, it is not likely that the child will return to school in the future. However, for younger children, being out of school for a year, say, is less likely to have long-term consequences because there is enormous dispersion in the ages of children in each grade and so re-entering when the child is a year older is not difficult. This reflects the combination of a great deal of variation in the age at which children start school and the high rates of repetition of grades in primary school. Second, the return to primary school education is very low in Indonesia whereas the return to secondary school and college is substantial and thus the long-term economic costs of not completing secondary school likely weighed on family decision-making. It turned out that this was a good choice: by 1999, a scholarship program had been implemented so that the costs of enrollment were forgiven if families were unable to afford them. These included the costs of fees, uniforms, transport costs and examination charges. By 2000, enrollment rates and disparities across the income distribution had all returned to their pre-crisis levels (and disparities had been reduced).

Intra-household substitution favoring school enrollment of older children over younger children is one mechanism that families used to mitigate the impact of the shock on human capital. There is evidence that adults literally tightened their belts to assure that the growth of the young children did not falter in the face of the crisis. Figure 5 displays the body mass index (BMI), which is weight (in kgs) divided by the square of height (in m) of males (in the upper panel) and females (in the lower panel) by age in 1997 and 1998. BMI rises with age until around the mid-forties and then declines. Height does not change for prime age adults and so this reflects changes in weight. The remarkable result is the dramatic decline in BMI across the entire age distribution for males and females between 1997 and 1998. On average, the declines are slightly smaller among adults who were living in rural areas prior to the crisis. However, overall, it is the oldest females whose weight declined the most: among females age 60 and older in 1997, weight declined by 15% in one year. This is unlikely to reflect the impact of energy expenditure and so we conclude that these women reduced their food intake of favor of their grandchildren.

How did households smooth consumption? There are three key mechanisms. First, people moved. Urban dwellers moved to the rural sector where the cost of living is lower and there were opportunities to earn income from working on a farm. In addition, household members moved in together to benefit from economies of scale of living arrangements. As shown in Table 3, overall, household size increased from 4.33 to 4.53 members, with the increase being slightly greater in rural areas (because of urban to rural migration).

Second, while real per capita expenditure declined by 23%, food expenditures were protected and declined by 9%. This reflects the combination of reduced food intake and increased price of food. Households cut spending on two classes of goods. First, spending on clothing, furniture and ceremonies was reduced by 35% ; we call these items 'deferable' because delaying spending on these items for the short term is unlikely to have the same impact on welfare as reduced spending on, for example, staples. (See Browning and Crossly, 1997, for a discussion of deferred spending.) Second, spending on health and education fell by 37%. The reduction in education spending reflects the combination of young children not attending school and parents cutting back on school expenditures (including paying fees). The reduction in spending on health is reflected in a dramatic reduction in use of health care during the year after the onset of the financial crisis relative to before the crisis. Whether these cuts in spending have longer-term consequences for welfare depends on the longevity of the cut, whether delayed care will have result in poor health in the future and whether the child's schooling is affected over the longer term.

Third, households are likely to draw down savings or sell off assets to mitigate the impact of the crisis (Deaton, 1992; Browning and Lusardi, 1996). The value of that strategy was limited by the fact that many assets collapsed in value. Savings in cash lost value as inflation rocketed. The Jakarta stock exchange had lost 75% of its pre-crisis value by mid 1998. Land and housing markets dried up along with credit markets. One important asset rose in value: gold. The price of gold is set in world terms and so as the rupiah fell to below half its pre-crisis level, the value of gold more than doubled. There is an active market for gold which is sold by weight and according to the IFLS community survey, there is a gold seller within half an hour travel time of every IFLS community. In 1997, over half the households owned gold and the vast majority of this gold was in the hands of women. By 1998, a substantial fraction of the gold had been sold. In regression models of that link changes in budget shares to pre-crisis assets, we find that spending on health and education are protected the most by gold ownership (Frankenberg, Smith and Thomas, 2003).

We conclude that even in the face of a very large financial shock, Indonesian households and families sought to protect the human capital of the next generation through every means possible: they migrated, moved in together, took up work, particularly in family businesses, cut spending, especially on deferrable items, reduced spending on some members in favor of others and spent down savings, primarily gold. In so doing, the immediate impact of the financial crisis on the nutrition and schooling of children were minimized. Using later waves of IFLS, there is little evidence that the nutrition and education outcomes were deleteriously affected by the financial crisis over the longer term (Strauss et al, 2004; Thomas and Frankenberg, 2007). The

extent of resilience of child human capital outcomes after a shock that rivals the Great Depression in the United States in magnitude and occurred in a low income setting is nothing short of remarkable. This is not to argue that the shock had no welfare consequences for children and their families: it did. Poverty rates rose, wealth declined and the physical and psycho-social health of adults was worse and it took several years for Indonesia to regain the lost ground.

4. Natural disasters and human capital

In some cases, shocks are so devastating that mitigating their impact in the short-term is beyond the reach of even the most resourceful families. It is useful to contrast the immediate and longer-term impact on health and human capital of children of such shocks relative to shocks like the financial crisis. The 2004 Indian Ocean earthquake and tsunami provides a platform for this investigation.

2004 Indian Ocean earthquake and tsunami

The 2004 Sumatra-Andaman earthquake occurred at 8am on Sunday December 26, 2004, and registered 9.3on the Richter scale. One of the strongest earthquakes in recorded history, it shifted the North Pole by several centimeters, caused a 1200 km rupture along the floor of the Indian Ocean and results in a tsunami that was felt across the entire Indian Ocean. The epicenter of the earthquake was about 150 kms off the west coast of the province of Aceh on the northern tip of the island of Sumatra, Indonesia. Since the tsunami surge was greater than 800 km/hour, its impact was felt along the Acehnese coast within 15 minutes of the earthquake.

At the time, the Indian Ocean was not thought to be prone to tsunamis and there were no warning buoys or an early warning system (in contrast with the Pacific Ocean). According to archaeological data, there had not been a tsunami on the coast of Aceh for over 600 years.

The impact of the tsunami depended on a combination of the wave height, which reached up to 15 meters in some areas, the wave direction and the topography of the land. Areas that were low-lying (such as river basins) that faced the wave were devastated: in some areas water and mud reached up to 6 kms inland. Nearby areas that were higher up (because of a cliff, for example) or were protected from the wave by a promontory were not directly affected.

In the SUSENAS survey conducted by Statistics Indonesia in February/March 2004, about 10 months before the tsunami, fewer than 3% of all respondents in Aceh thought that it was likely they would experience a natural disaster such as an earthquake, tsunami or volcanic

eruption and that fraction did not differ depending on whether their community was in fact directly affected by the tsunami.) In sum, the tsunami was unanticipated; where it hit along the Acehnese coast was impossible to predict and few people living in Aceh expected a large-scale natural disaster.

The tsunami was not only unanticipated; it was also a major shock. It is estimated that in Aceh, the earthquake and tsunami killed about 170,000 people which is about 5% of the total population of the province. About three-quarters of a million people (about 20% of the population) were displaced because their homes were destroyed or damaged. In addition to the loss of home, there was massive physical destruction with roads and infrastructure being severely damaged, over 40,000 hectares of agricultural land being destroyed along with the crops and livestock. The World Bank estimated there was about $4.5 billion in property damage (Doocy et al, 2007; Frankenberg et at, 2008; Gray et al, 2012; Cossee, Hermes, and Mezhoud 2006).

There are at least three important dimensions to the shock from the perspective of examining the impact on child human capital. First, many lost assets and livelihoods as their income-earning opportunities were wiped out and as family members were killed in the tsunami. Second, the destruction to roads and bridges reduced access to markets which resulted in increases in the price of food and the loss of housing resulted in increased rents. On both counts, those who were net sellers of food or housing benefited from the shock. Third, the loss of family and friends in the tsunami and the destruction and loss of livelihoods affected the psycho-social health and well-being of the population (Frankenberg et al, 2008). It is difficult to credibly disentangle the income (and asset) shock, price shock and psycho-social (stress) shock.

The tsunami was followed by the largest influx of aid after a natural disaster in any developing country. It is estimated that combining domestic and international aid from government and non-government sources, about $7.2 billion in aid was directed to recovery efforts in Aceh and North Sumatra. Initially, aid efforts focused on providing food and temporary shelter and even that aid took several months to be deployed. Food had to be transported by air or ship to reach many of the most devastated communities as the roads were impassable. Camps were constructed and a small number of displaced moved into camps within three months of the disaster; the vast majority of camps were not constructed until later in 2005. After these needs were met, aid efforts focused on infrastructure investments – roads, bridges, public building and housing. The goal was to “build back better”.

In short, in December 2004, there was a major shock that affected the lives of those living along the coast of Aceh at the time. It was followed by one of the most successful recovery programs conducted in a developing country. How did child human capital fare?

To answer this question, we draw on data from the Study of the Tsunami Aftermath and Recovery (STAR). The baseline sample is drawn from the 2004 SUSENAS conducted 10 months before the tsunami and includes all respondents who were living at the time in a kabupaten (district) along the coast of Aceh. SUSENAS is designed to be population-representative at the kabupaten level and so the baseline is representative of the pre-tsunami population that was affected, to varying degrees, by the tsunami. There are over 25,000 respondents in more than 6,000 households in some 400 communities.

We conducted the first follow-up of the SUSENAS respondents between May 2005 and June 2006 and we refer to that survey as STARB. Thereafter, we conducted four additional annual follow-ups (STAR C, D E and F) and have completed a 10 year follow-up (STAR G). In the first follow-up, finding and interviewing every survivor was critically important and extremely difficult. We have determined the survival status of over 98% of the baseline respondents. In most cases, we have found one or more of the surviving baseline household members and obtained survival status from those respondents. In some cases, however, no one in the household survived and in those cases we used multiple reports from neighbors, community leaders and service providers (in the health and education sectors) to assure that our designations are correct.

Post-tsunami follow-up of survivors has been very challenging. On the one hand, in areas that were severely damaged, all (or in some cases almost all) the survivors were displaced with about half moving to camps and the other half moving to private homes. Lack of infrastructure and damage to roads compounded the difficulty of tracking and interviewing the survivors. Over 96% of the survivors have been interviewed in at least one of the STAR follow-ups.

SUSENAS does not collect data on height of respondents. Given the complexity of the fieldwork, identifying, finding and interviewing surviving respondents in the first follow-up, we chose to not also burden the enumerators with the equipment necessary to measure height and weight and conduct other health assessments in that follow-up. Measurement of anthropometry along with the collection of other biomarkers was added in the second follow-up, STARC, which was in the field between July 2006 and June 2007. Anthropometry was measured in all subsequent waves. Those data are used in our analyses of child nutrition. (Frankenberg, Ingwersen and Thomas, 2016).

In utero exposure to the tsunami and child height

We focus on the height for age of children who were in utero at the time of the tsunami and exclude all children born in areas that were very heavily damaged by the tsunami and where there were very high death tolls because of the tsunami. There are very few births in the nine months following the tsunami among women who were living in those areas and so the exclusion of those births has no perceptible impact on the estimates discussed below. Their exclusion assures that none of the results are contaminated by selective survival of the strongest mothers since women who were pregnant were much more likely to have died when they were swept away by the water than women who were not pregnant.

The average height given age of this cohort at the time of the second follow up when the child was, on average about 15 months old is 1.76 standard deviations below the CDC norm. We compare the height for age of this cohort with the cohort born in 2001 since, according to the nutrition literature, the growth trajectory of the height of those children is thought to be largely determined by the time of the tsunami. Conception date is not known for children in STAR and so we examine children by quarter of birth and assume that those born in the first quarter of 2005 were in the third trimester at the time of the tsunami, those born in the second quarter were in the second trimester and those born in the third quarter were in the first trimester. Each birth cohort is compared with the same quarter of birth cohort in the reference cohort.[1] The gap in the z-score of height for age of those exposed to the tsunami in utero in the second STAR follow-up (in 2006-7) is displayed in the first column of Table 4. All models also control gender of the child, month of interview and birth order. All the children in utero at the time of the tsunami are shorter than the reference cohort and the gaps are large and statistically significant for those born in the second quarter of 2005. Based on this evidence, the unanticipated shock of the tsunami resulted in reduced linear growth of those children who were in utero at the time of the tsunami by the time they were, on average, 18 months old. This likely reflects the combination of the nutrition shock (because of the rise in the price of food and access to markets), the income shock (because of the loss of livelihoods) and the stress shock (associated with exposure to the horror of the tsunami).

One year later, however, the estimated gaps are smaller and after two years none of the gaps between the exposed and reference cohort is significantly different from zero. As shown in

[1] It is important to note that it is likely that more of the children who were in utero at the time of the tsunami were born prematurely relative to the reference cohort and this effect is captured in the comparison of height for age.

column 4, five years after the tsunami, the cohort exposed to the tsunami in utero is taller than the 2002 cohort and the differences are statistically significant for those born in the first and third quarters of the year. Growth in height for age between 2006/7 and 2009/10, relative to the reference cohort, is displayed in the fifth column of Table 4: all of the exposed children have grown much faster, relative to the CDC norms than the reference cohort and the growth is greatest for those who had the largest disadvantage in 2006/7, those born in the second quarter. That group of children has grown almost 1 standard deviation more than the reference cohort.[2]

It is possible that the growth trajectory of the reference cohort was affected by the tsunami; children in the reference cohort were born in 2002 and so were, on average, age 30 months at the time of the tsunami. The 2001 birth cohort was potentially affected by the 1998 financial crisis and so comparison with that cohort may be compromised. Results using this cohort as the reference are displayed in column 6 of the table and the results are not substantially affected. The cohort that was in utero at the time of the tsunami was born with very substantial deficits in length but have more than made up those deficits by age 4.

Nutrition and stress insults in utero are likely to have resulted in some fetuses being miscarried. It is possible to investigate this issue by comparing the impact of the in utero exposure to the tsunami on males and females. Table 5 displays the height for age z scores of the exposed cohorts relative to the reference cohorts. Overall, miscarriages are more common for males largely because males are far more likely to be miscarried early in the pregnancy whereas females are more likely to be miscarried later in the pregnancy. Thus, males who were in the first or second trimester of the pregnancy at the time of the tsunami are more likely to have been miscarried, relative to males in the third trimester and so we expect the impact of the tsunami to be greatest on third trimester males who are most likely to be born in the first quarter of 2005. This is the case, as shown in the first column of panel 1 of the table. For females, those in the third trimester are more likely to be miscarried and so, among these fetuses, only the strongest are likely to have survived. Impacts of the tsunami are likely to be greatest on the fetuses exposed earlier in the pregnancy. For females born in the second quarter (and thus most likely to have been exposed during the second trimester), the impacts are very large and statistically significant (column 1 of panel 2 of the table). All of the exposed cohorts grew faster than the reference cohorts with growth being greatest for females born in the second quarter of 2005. This evidence is consistent with a biological model of miscarriage and the results suggest that the

[2] Results using the WHO norms are essentially identical. Recall that comparisons are made between standardized height for age of the exposed cohort with standardized height for age of the reference cohort.

overall impacts reported in Table 2 are unlikely to be driven by other, unobserved differences between the exposed and reference cohorts.

To address this issue more directly, Table 6 provides dose-response estimates of the effects of exposure to the tsunami. In the first post-tsunami survey, adults were asked a battery of questions about their exposure to the tsunami including, for example, whether they saw people die in the tsunami, whether they were swept away by the water, and whether they saw or heard the water. We dichotomize mothers into two groups: two-thirds who saw or heard the water and one-third who did not. As shown in column 1 of each panel of Table 6, the children of mothers who saw or heard the water and were born in the second quarter of 2005 are 0.88 standard deviations shorter than the reference cohort (panel 1) which is almost three-quarters of a standard deviation shorter than children born of other mothers (in panel 2). Growth rates between 2006/7 and 2009/10 are not statistically different between the cohorts whose mothers saw or heard the water and mothers who did not and so the gap in height for age for the second quarter births persists for 5 years after the tsunami. This evidence is consistent with a dose-response impact of exposure to the tsunami on length at birth and that the children whose mothers were most exposed have an enduring deficit in height for age.

We have also explored whether the impacts of the tsunami on child height differ with parental resources prior to the tsunami as measured by paternal education (or maternal education) and per capita expenditure. Children in households with fewer resources tend to have more negative impacts in the short run but these differences are very modest and not statistically significant. There is no evidence that growth rates differ with pre-tsunami resources.

It is not possible to distinguish between the effect of the tsunami on prices and resources (a nutrition shock), on one hand, and the impact on maternal stress, on the other. Respondents in STAR were asked the post-traumatic stress symptom checklist as they felt in the days following the tsunami. We have constructed an index of post-traumatic stress reactivity (PTSR) for each pregnant woman and estimated models that stratify the mothers into those who have high levels of PTSR symptoms, those with moderate levels and those with relatively low levels. We find that children born to mothers who reported high levels of PTSR are much shorter (in 2006/7) than children whose mothers did not report as high levels of PTSR and that these gaps persisted to 5 years after the tsunami. This suggests that maternal stress is implicated in the negative impacts of the tsunami on child height for age. However, it is important to recognize that self-reported measures of PTSR reflect a combination of the woman's exposure to the tsunami and her reactivity to it and so it is not possible to assign a causal interpretation to this evidence.

There are several factors that potentially contribute to the fact that cohort that was in utero at the time of the tsunami grow faster than the reference cohorts. First, the success of the post-tsunami recovery effort resulted in rapid economic growth across all Aceh providing new earnings opportunities for many families. These opportunities included reconstruction-related work such as construction and repair of roads, bridges and buildings; the opportunities also include the provision of services to the workers and firms involved in the reconstruction such as food preparation, logistics, housing and transportation. The fact that growth rates were no different for children whose mothers saw or heard the water and those who did not is consistent with the observation that the reconstruction effort benefited all the communities along the coast of Aceh and did not only benefit those communities that were hardest hit. This reflects two forces: on the one hand, road and bridge reconstruction was spread across most of the coast of the province; on the other hand, there was substantial migration in response to the reconstruction effort.

The second factor that potentially contributed to the rapid post-tsunami growth of the affected cohort is that as parents rebuilt their livelihoods, they invested heavily in their surviving children. This would be consistent with evidence on investments by Indonesian families in the face of the 1998 financial crisis. Third, it is possible that some of the faster growth can be attributed to post-uterine growth of premature births although much of that catch-up growth is likely to have occurred in the first few months of life.

We have investigated whether there are differences in the weight for height of the exposed cohort and the reference cohort and find none. Nor do we find any differences depending on the magnitude of exposure. These results are not surprising: there is no reason to assume that the tsunami would not impact the weight of all cohorts. In fact it is precisely because we are drawing on the biology of human growth and assume that the height of the reference cohort is not much affected by exposure to the tsunami, at least relative to the cohort exposed in utero, that it is possible to identify the causal impact of the tsunami on longer-term child nutrition. We have not examined the growth of children born after the tsunami because there was a very large fertility boom in the months of the tsunami and the mothers of those children tend to be younger and better educated than the mothers of the children conceived before the tsunami (Nobles, Frankenberg and Thomas, 2015).

In sum, the Indian Ocean tsunami was a large, unanticipated shock that caused unprecedented disruption in the lives of people living along the coast of Aceh at the time. The shock took a substantial toll on the height of children who were in utero at the time of the

tsunami. Had we not followed these children, we would have concluded that the tsunami likely had a significant longer-term impact on the health and well-being of the population. However, the deleterious impacts were for most of the children short-lived and the cohort that was in utero at the time of the tsunami appear to have substantially benefited from the reconstruction effort. Evidence from STAR documents that large scale, unanticipated shocks have consequences for child growth, there is scope for catch-up growth and that income in the first few years of life plays a key role in mitigating the consequences of these shocks.

5. Conclusions

Measurement of the casual impact of shocks on health and human capital is extremely difficult. This is not a novel insight. The goal of this research has been to provide examples of the impact of shocks on health and human capital that turn out to be much more complicated to interpret than a prime facie description of the shock would suggest.

First, it is not straightforward to identify shocks that are, in fact shocks. Many of the events that are treated as shocks in the literature are anticipated or at least potentially anticipated. Even if the exact timing may be hard to predict, the expectation that there will be, for example, a poor agricultural season periodically complicates interpretation of a poor season as a shock. It is likely that those people who are affected by a poor season will invest in behaviors that mitigate the impact of the poor season. In some cases, a shock is correlated with other changes in the environment or possibly behaviors that contaminate interpretation of estimated effects of the shock. This arises, for example, in the analysis of the 1918 influenza pandemic which occurred at the same time as WWI ended.

Second, the assumption that as shocks unfold, individuals and those in their networks, broadly defined, do not respond to the shock is not consistent with the core tenets of economics. We have shown that in the face of a large financial and political crisis in Indonesia that resulted in huge declines in resources, individuals, families and communities demonstrated extraordinary resilience and adaptability and sought to mitigate the longer-term impacts of the crisis, even as the magnitude and longevity of the crisis was being revealed. Indonesians are not unique in this regard. Stillman and Thomas (2010) establish that in the face of the collapse of oil prices and, therefore, a massive income shock in Russia, households substantially cut into food spending by switching from more expensive calories and protein to less expensive calories and protein. While

spending on food declined by about 25%, calorie and protein intake hardly changed and there is no evidence of a change in BMI.

Third, some shocks are so devastating that responses in the short-term are limited. However, as our example from the Indian Ocean earthquake and tsunami establishes, drawing conclusions from short-term impacts can be substantially misleading. Over and above evidence on the impacts on child height, we have investigated the impact of the death of one or both parents on the human capital of children who were not killed by the tsunami. Studies of parental death have established that the children of those parents tend to complete fewer years of education (Case and Paxson; Beegle et al; Evans et al). The majority of those studies are conducted in settings where parental death is due to HIV/AIDS and is treated as if it were a shock. It is not clear that assumption is correct: neither is the death likely to be a shock if there was an illness prior to the death nor are the parents who contract HIV necessarily exchangeable with those who do not. We investigate the impact of an arguably exogenous parental death (from the tsunami) on the education of children age 9 to 14 and, separately, on outcomes of children age 15 to 17 at the time of the tsunami (Cas, Frankenberg, Suriastini and Thomas, 2011). For young children (age 9-14), we find no impact of parental death of education outcomes: the evidence suggests that other family members and also members of the community rallied to the aid of these children. For older children, however, the story is completely different. Males who lost one or both parents left school earlier and started working earlier than males in the same communities who did not lose a parent. Females also left school earlier and married earlier if one or both parents died. With the new earnings opportunities that accompanied reconstruction, the longer-term consequences of these choices are not obvious; they will be investigated drawing on the 10 year follow-up of the STAR sample. We will also investigate the longer-term consequences of in utero exposure to the tsunami and subsequent catch-up growth. It is possible that catch-up growth has deleterious consequences for metabolic functioning in later life, as suggested by the thrifty gene hypothesis. In addition to testing this hypothesis using data on metabolic-related biomarkers that we are collecting, we will examine education and cognitive outcomes of the children exposed to the tsunami drawing on data from STAR follow-ups.

References

Alderman, H., J. Hoddinott and B. Kinsey. (2006). Long term consequences of early childhood malnutrition. *Oxford Economic Papers*, 58.3:450-74.

Almond, D. (2006). Is the 1918 influenza pandemic over? Long-term effect of in utero influenza exposure in the post 1949 U.S. population. *Journal of Political Economy,* 114.4:672-712.

Barker, D. J. P. (1995). "Fetal origins of coronary heart disease." *BMJ*, 311: 171-4;

Brown, R. and D. Thomas. (2016). On the long term effects of the 1918 U.S. influenza pandemic. Mimeo.

Browning, M. and T. E. Crossley. (2009). "Shocks, stocks and socks: Smoothing consumption over temporary income loss", Journal of the European Economic Association, 7.6:1169-92.

Browning, M. and A. Lusardi. (1996). "Household saving: Micro theories and micro facts", *Journal of Economic Literature*: 34.4:1797-1855.

Cas, A., E. Frankenberg, W. Suriastini and D. Thomas. (2014). The impact of parental death on child well-being: Evidence from the Indian Ocean tsunami. *Demography*, 51.2:437-57.

Cosse, O., R. Hermes and S. Mezhoud. 2006. *Real time evaluation of the FAO tsunami response*. Rome:FAO.

Deaton, A. (1992). *Understanding consumption*, Oxford: Clarendon Press.

Doocy S, Gorokhovich Y, Burnham G, Balk D, Robinson C. Tsunami mortality estimates and vulnerability mapping in Aceh, Indonesia. *American Journal of Public Health.* 2007:97: S146–151.

Frankenberg, E., J. Friedman, T. Gillespie, N. Ingwersen, R. Pynoos, I. Rifai, B. Sikoki, A. Steinberg, C. Sumantri, W. Suriastini and D. Thomas. (2008). Mental health in Sumatra after the tsunami. *American Journal of Public Health*, 98.9:1671-7.

Frankenberg, E., T. Gillespie, S. Preston, B. Sikoki and D. Thomas. (2011). Mortality, the family and the Indian Ocean tsunami. *Economic Journal,* 121.554:F162-82.

Frankenberg, E., J. P. Smith and D. Thomas. (2003). Economic shocks, wealth and welfare. *Journal of Human Resources,* 38.2:280-321.

Frankenberg, E., D. Thomas and K. Beegle. (1999). The real costs of Indonesia's economic crisis: Preliminary findings from the Indonesia Family Life Surveys.

Friedman J. and J. Levinsohn. (2002). The distributional impacts of Indonesia's financial crisis on household welfare: A rapid response methodology. *World Bank Economic Review*, 16.3:397-423.

Gray, C., E. Frankenberg, T. Gillespie, C. Sumantri and D. Thomas. (2014). Studying displacement after a disaster using large-scale survey methods: Sumatra after the 2004 Tsunami. Annals of the Association of American Geographers, 104.3:594-612.

Hales, C and D. Barker. (1992). Types 2 (non-insulin-dependent) diabetes mellitus: The thrifty phenotype hypothesis. *Diabetologia*, 35.595-601.

Heckman, J. J. (2006). Skill formation and the economics of investing in disadvantaged children. *Science.* 312.5782:1900-2.

Hoddinott, J., J. A. Maluccio, J. R. Behrman, R. Flores, and R. Martorell. (2008). Effect of a nutrition intervention during early childhood on economic productivity in Guatemalan adults. *Lancet*. 371: 411-416.

Liebenstein, H. (1957). The theory of underemployment in densely populated backward areas, in G. Akerlof and J. Yellen (eds.) *Efficiency wage models of the labor market*. New York: Cambridge University Press.

Martorell, R. and J. P. Habicht. (1986). Growth in early childhood in developing countries. In F. Falkner and J. M. Tanner, *Human growth: A comprehensive treatise*, Volume 3. New York: Plenum Press.

Mazumdar, D. (1959). The marginal productivity theory of wages and disguised unemployment. *Review of Economic Studies*, 26, 190-7.

Nobles, J., E. Frankenberg and D. Thomas. (2015). The effects of mortality on fertility: Population dynamics after a natural disaster. *Demography*, 52.1:15-38.

Ravelli, A.C.J., van der Meulen, Jan H.P., Michels, R.P.J., Osmond, C., Barker, D., Hales, C.N. and Bleker, O.P. (1998). Glucose tolerance in adults after prenatal exposure to famine. *Lancet*, 351:173-177.

Ravelli, A.C.J., van der Meulen, Jan H.P., Osmond, C., Barker, D. and Bleker, O.P. (1999). Obesity at the age of 50 years in men and women exposed to famine prenatally. *American Journal of Clinical Nutrition*, 70:811-816.

Rooij WH, Yonker JE, Painter RC, Roseboom TJ. 2010. Prenatal under-nutrition and cognitive function in late adulthood. *Proceedings of the National Academy of Sciences* 107:16881-86.

Roseboom, Tessa J., van der Meulen, Jan H.P., Osmond, Clive, Barker, David J. and Blecker, Otto P. (2001). Adult survival after prenatal exposure to the Dutch famine 1944-1945. *Paedeatric and Perinatal Epidemiology*, 15:220-225.

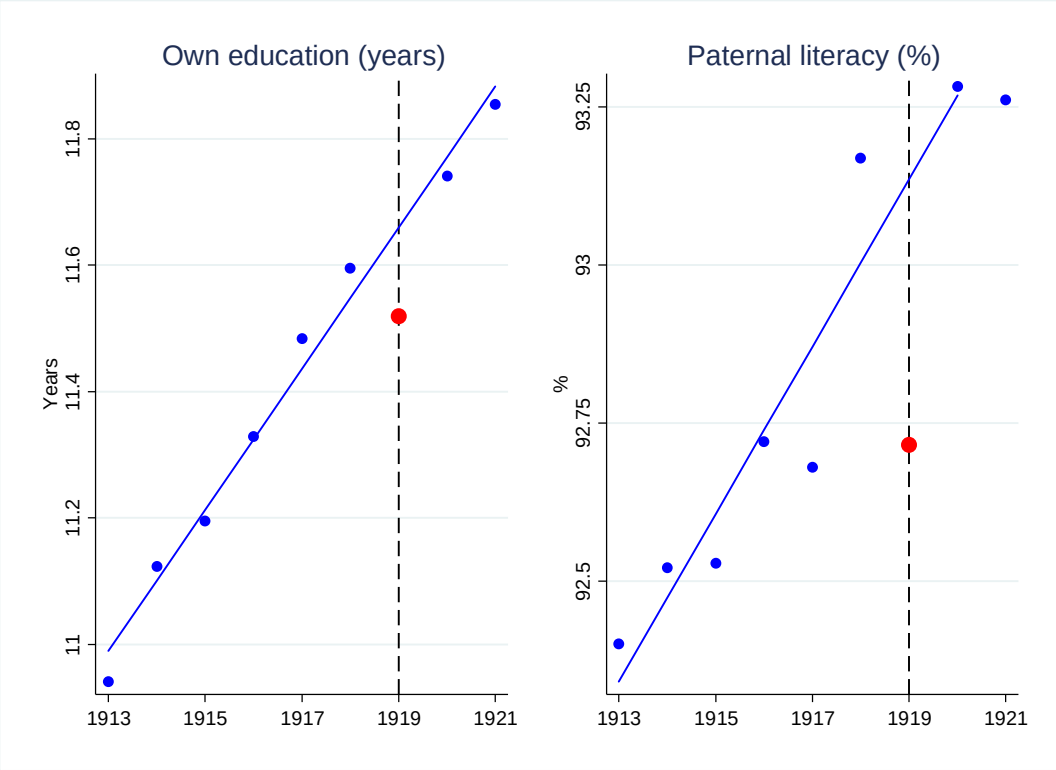
Smith, J. P., D. Thomas, K. Beegle, E. Frankenberg and G. Teruel. (2002). Wages, employment and economic shocks: Evidence from Indonesia, *Journal of Population Economics*, 15:161-93.

Strauss, J., K. Beegle, A. Dwiyanto, Y. Herawati, D. Pattinasarany, E. Setiawan, B. Sikoki, Sukamdi and F. Witoelar. *Indonesian living standards before and after the financial crisis: Evidence from the Indonesia Family Life Survey*. Singaore:Institute of Southeast Asian Studies.

Thomas, D., K. Beegle, E. Frankenberg, B. Sikoki, J. Strauss and G. Teruel. (2004). Education in a crisis. Journal of Development Economics. 74.1:53-85.

Thomas, D. and E. Frankenberg. (2007). Household responses to the financial crisis in Indonesia: Longitudinal evidence on poverty, resources and well-being. In A. Harrison (ed). *Globalization and Poverty*, Cambridge University Press.

Figure 1. Human capital and in utero health insults
Own education and paternal literacy by birth year
1913-1921 United States birth cohorts



Sources: 1960 Census (own education) and 1930 Census (paternal literacy)

Figure 2a. US$ exchange rates for Singapore, Thailand and Indonesia, 1997-2002

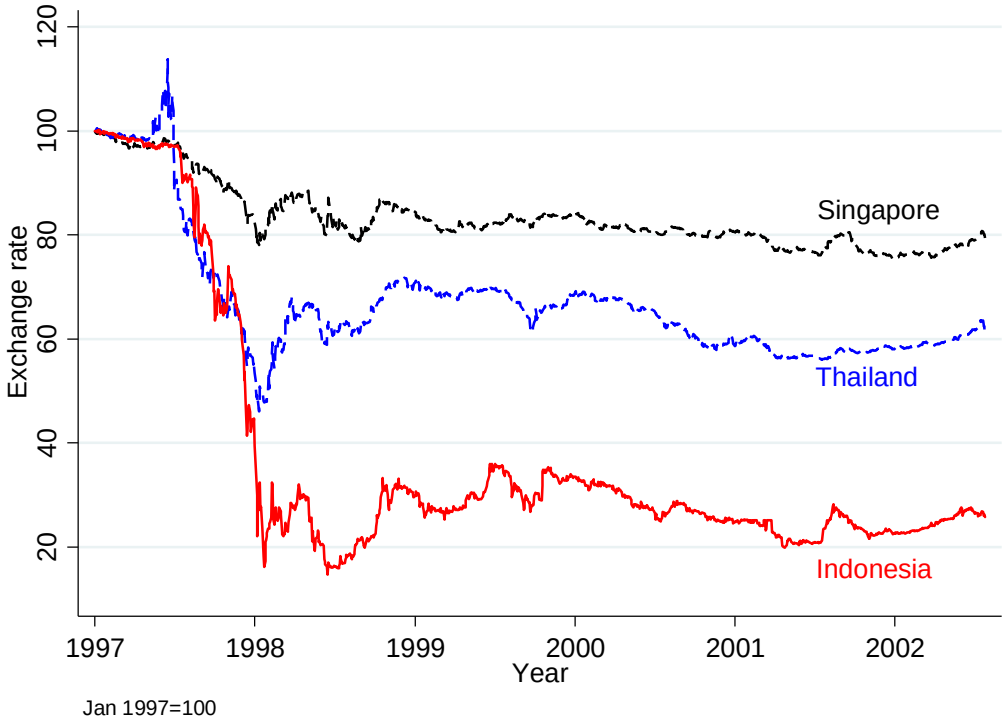


Figure 2b. %age change in per capita GDP in Indonesia, 1980-2010 and timing of IFLS

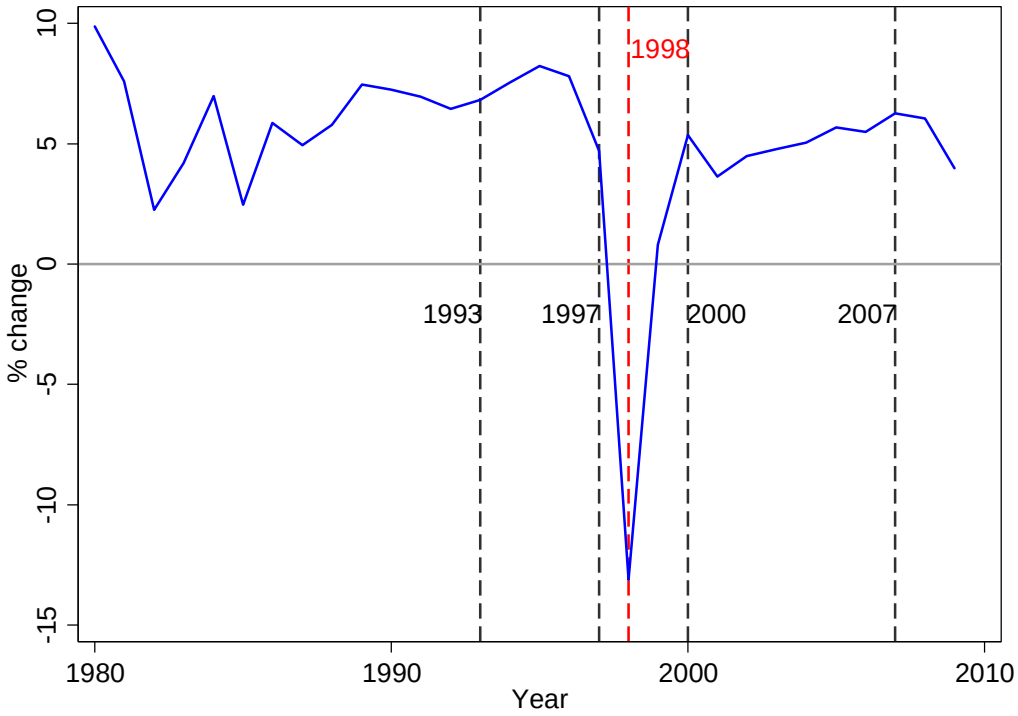


Figure 3. Annual inflation in Indonesia, 1985 to 2007

All prices (by month) and food prices (by quarter)

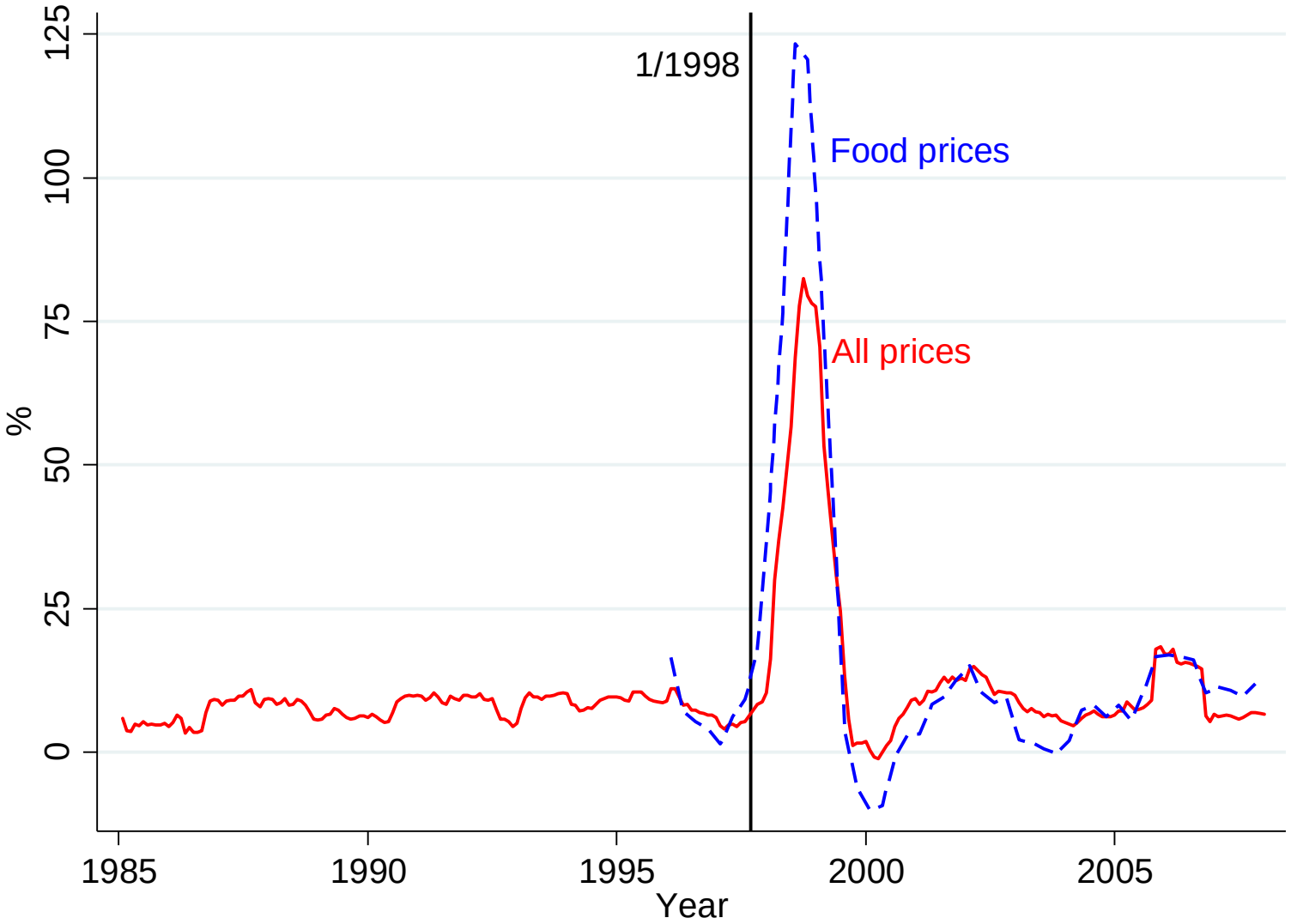
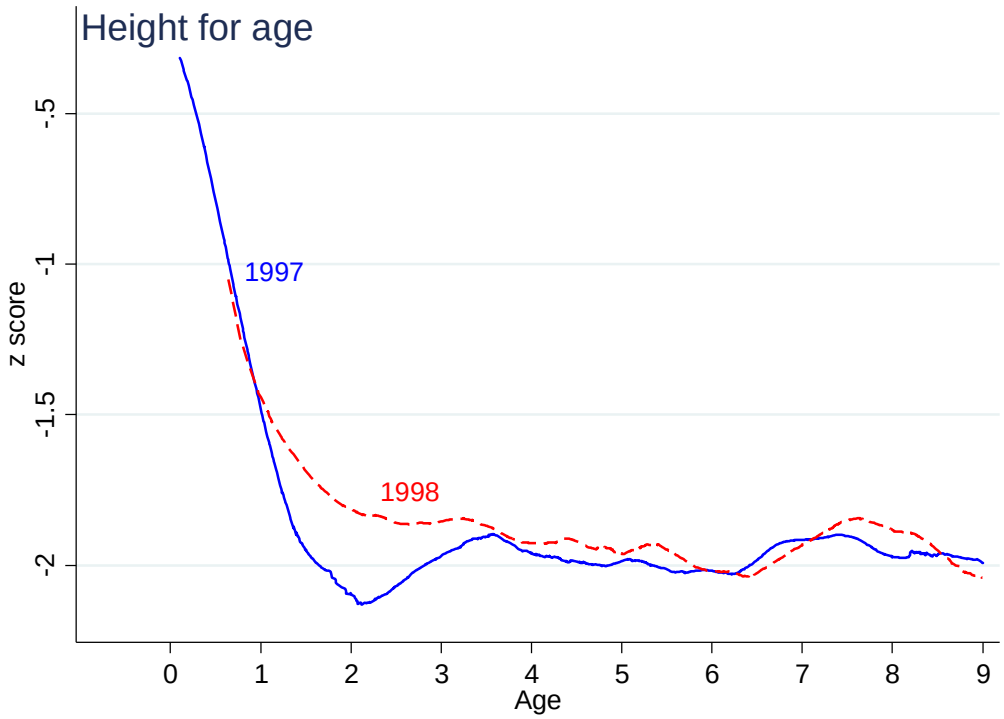


Figure 4. Child anthropometry before and after onset of the 1998 crisis



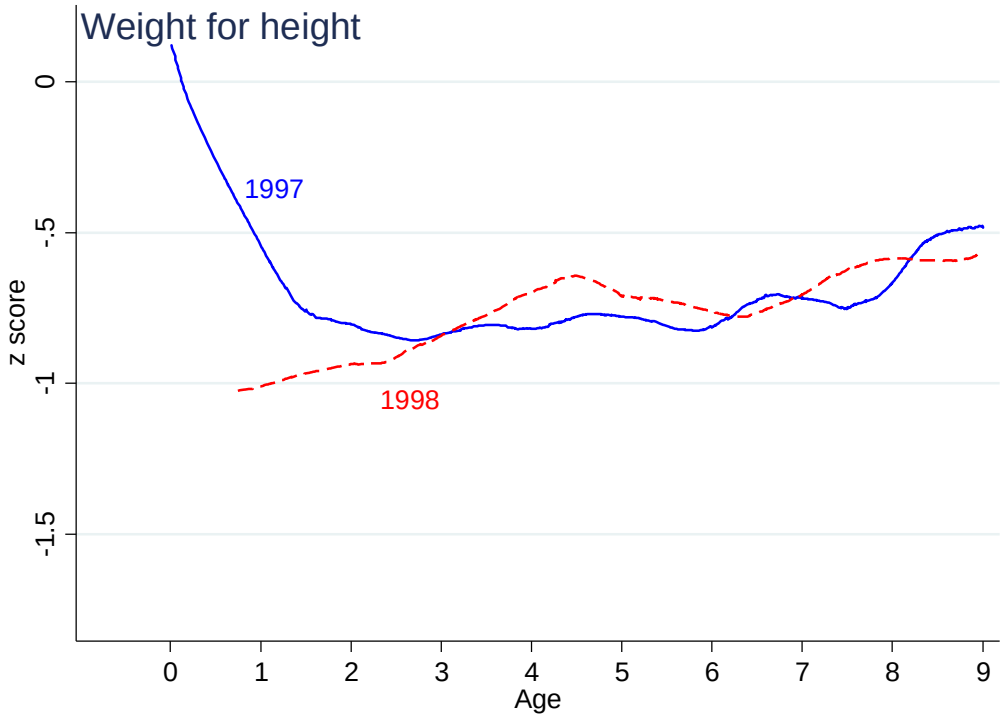


Figure 5. Adult BMI before and after onset of the 1998 crisis

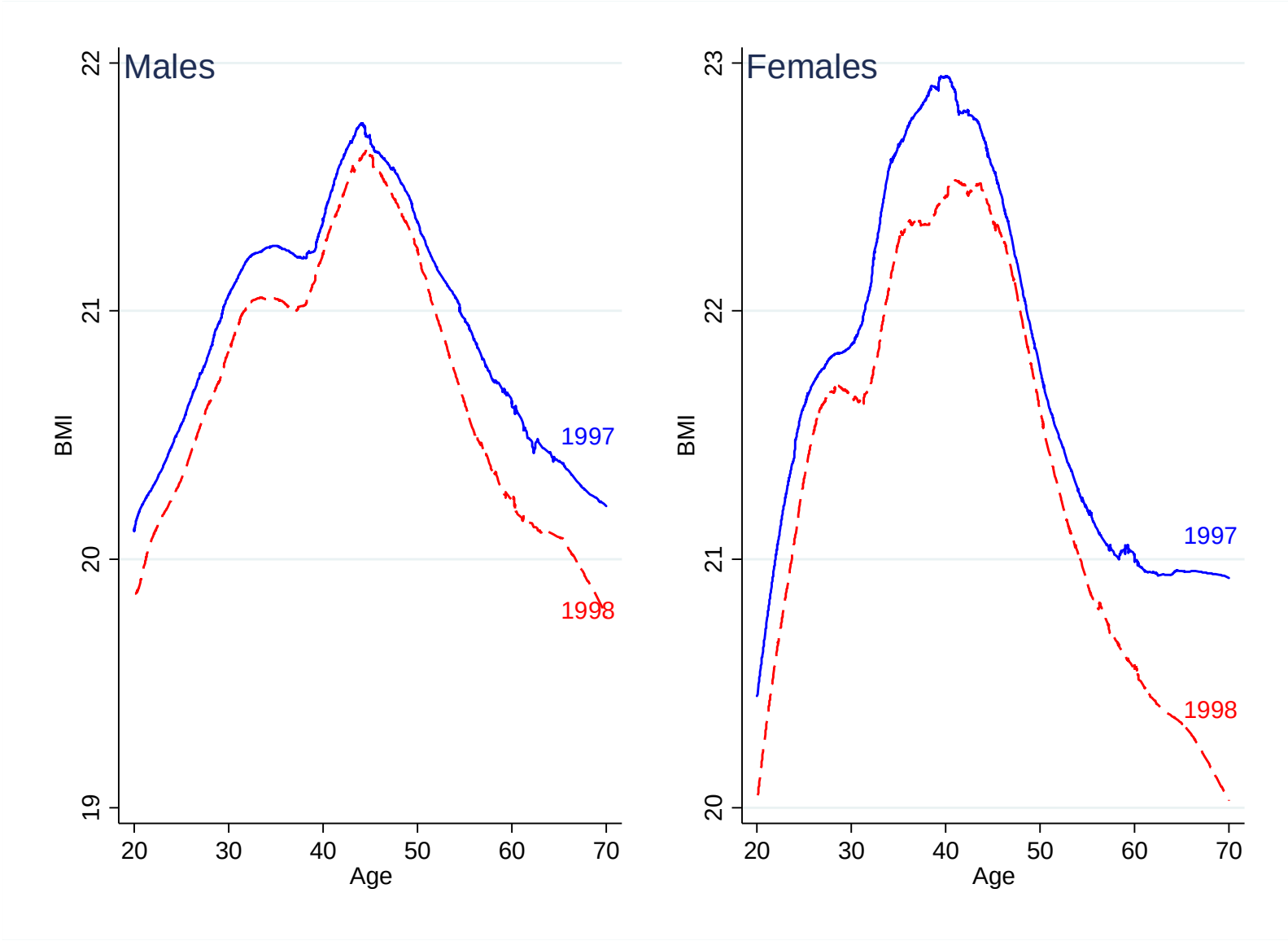


Table 1

Effect of in utero exposure to 1918 influenza pandemic on education

Dependent variable: Years of education (in 1960 U.S. census)

	Include paternal characteristics?	
	No	Yes
	[1]	[2]
A. Cohort comparisons		
Paternal characteristics from		
1920 Census	-0.188	0.230
	[0.064]	[0.062]
1930 Census	-0.148	0.083
	[0.039]	[0.037]
B. Dose-response effects		
Maternal infection rate	-0.692	-0.431
	[0.322]	[0.399]

Notes: [Robust standard errors] below coefficient estimates.
Panel A displays estimate of 1919 birth cohort relative to trend.
Panel B displays estimate of effect of maternal infection rate.
All models include birth year indicator variables and linear trend.
Sample sizes are 80,695 (Pane A, 1920 Census), 216,633 (Panel A, 1930 Census) and 114,032 (Panel B).
Source: Brown and Thomas (2016)

Table 2.
Fraction of Indonesian children not enrolled in school in 1997 and 1998 by age group

	1997	1998	%Δ
Fraction of children not enrolled in school			
Children age 13-16			
	25.6%	25.1%	-2%
Children age 7-12			
All	3.1%	5.9%	90%
Poor children	4.9%	10.7%	118%
(Bottom quartile of PCE in 1997)			

Notes: IFLS2 and IFLS2+. Source Thomas et al (2004).

Table 3.

Household composition, household consumption and composition of consumption

Indonesian households in 1997 and 1998

1997 Rp000s	1997	1998	%change
1. Household size	4.33	4.53	7
	[0.05]	[0.04]	[0.8]
2. Per capita household consumption	176	117	-23
	[12]	[9]	[2]
3. Composition of *per capita* household consumption			
A. Food	79.7	68.9	-9
	[4.9]	[7.4]	[2]
B. Non foods	95.2	48.4	-34
	[10.2]	[2.2]	[2]
C. Deferable items	9.1	6.4	-35
(Clothing, furniture and ceremonies)	[0.4]	[0.4]	[3]
E. Human capital investments	9	5.7	-37
(Health and education)	[0.6]	[0.3]	[3]

Note: Consumption measured in thousands of 1997 Rupiah.

1,971 households interviewed in 1997 and 1998 included in sample

Source: Frankenberg et al (2003)

Table 4.

Height for age z scores by year of measurement and growth in height for age z scores

	2006-07	2007-08	2008-09	2009-10	Growth	Growth
Ref cohort:			2002		2002	2001
	[1]	[2]	[3]	[4]	[5]	[6]
2005Q1	-0.14	-0.09	0.12	0.33	0.52	0.56
	[0.18]	[0.17]	[0.17]	[0.16]	[0.16]	[0.15]
2005Q2	-0.68	-0.41	0.08	0.25	0.96	0.72
	[0.17]	[0.16]	[0.15]	[0.15]	[0.17]	[0.16]
2005Q3	-0.29	-0.10	-0.03	0.39	0.64	0.66
	[0.18]	[0.17]	[0.16]	[0.16]	[0.18]	[0.19]
Sample size	4,170	4,170	4,170	4,170	4,170	4,170
R^2	0.09	0.09	0.10	0.11	0.07	0.07
F (joint signific	15.6	11.8	19.4	12.8	19.6	16.4
p-value	0.000	0.000	0.000	0.000	0.000	0.000

Notes

Dependent variable is standardized height for age z-score using CDC standards

Reference cohort : 2002 same quarter of birth for columns 1-5; 2001 cohort in column 6

Models include controls for gender, birth order, month of interview.

Standard errors take into account clustering at enumeration area level and heteroskedasticity

Source: Frankenberg et al, (2016)

Table 5.

Height for age z scores by gender, year of survey and growth in z scores

	1. Males			2. Females		
	2006-07	2009-10	Growth	2006-07	2009-10	Growth
	[1]	[2]	[3]	[1]	[2]	[3]
2005Q1	-0.56	0.01	0.67	0.18	0.53	0.36
	[0.25]	[0.26]	[0.22]	[0.28]	[0.25]	[0.28]
2005Q2	-0.36	0.26	0.65	-0.90	0.33	1.26
	[0.25]	[0.21]	[0.27]	[0.23]	[0.25]	[0.21]
2005Q3	-0.28	0.23	0.46	-0.44	0.42	0.81
	[0.31]	[0.24]	[0.30]	[0.25]	[0.25]	[0.23]
Sample size		2126			2044	
R^2	0.06	0.04	0.07	0.06	0.06	0.11

Notes: See Table 4.

Table 6.
Height for age by year and growth in height for age by exposure to tsunami
Exposure measured by whether mother saw/heard water

	1. Mother saw/heard water			2. Mother did not see/hear water		
	2006-07	2009-10	Growth	2006-07	2009-10	Growth
	[1]	[2]	[3]	[1]	[2]	[3]
2005Q1	-0.24	0.33	0.62	0.03	0.38	0.35
	[0.24]	[0.19]	[0.22]	[0.29]	[0.29]	[0.21]
2005Q2	-0.88	0.06	0.98	-0.12	0.82	0.94
	[0.21]	[0.18]	[0.22]	[0.30]	[0.26]	[0.23]
2005Q3	-0.23	0.27	0.51	-0.36	0.61	0.81
	[0.24]	[0.24]	[0.25]	[0.28]	[0.22]	[0.26]
Sample size		2703			1372	
R^2	0.11	0.11	0.08	0.12	0.17	0.1

Notes: See Table 4.