Stunting later in childhood and outcomes as a young adult: Evidence from India

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Abstract

This paper looks at patterns of growth faltering and catch up of around 1000 children as they moved from 8 to 19 years of age, from middle childhood through adolescence to young adulthood, using Height for Age Difference (HAD) and the more conventional Height for age z-scores (HAZ). It also looks at what individual and household characteristics may have moved these children into or out of situations of nutritional deprivation and how their stunting profile in later childhood correlates with psychosocial outcomes at age 19 and how it may have intergenerational consequences. The paper uses 4 rounds of longitudinal data collected in 2002, 2006, 2009 and 2013 from Andhra Pradesh and Telengana, India when the children were aged 8, 12, 15 and 19. The paper finds that there are significant gender based biases in growth faltering later in childhood disfavouring girls and that becoming newly stunted as an adolescent is strongly correlated with a child reporting to have poorer relationships with peers compared to the group that were never stunted. We also find that a girl experiencing stunting in middle childhood or adolescence (even if they were not stunted at age 8 or eventually moved out of being stunted by age 19) correlates significantly with offspring being shorter and thinner than the offspring of girls never stunted. This is one of few, if any, studies that look at growth patterns in middle childhood and adolescence and outcomes as a young adult and the results are important for their implications for further research and policy.

Keywords: Stunting; Adolescence; Psychosocial; Gender; India

Highlights

- Significant biases in linear growth exist later in childhood, disfavouring girls
- Growth patterns observed depend on whether height-for-age differences or z-scores are used
- Becoming newly stunted in adolescence correlates significantly with poorer peer relationships at age 19
- Girls becoming newly stunted in middle childhood and adolescence correlates with poorer offspring health outcomes.

1. Introduction

Stunting affects around a quarter to half of children in developing countries due to poverty, nutritional deprivation and burden of diseases (Grantham-McGregor et. al 2007, Victora et. al 2010). Its consequences for outcomes later in life can be detrimental with significant negative effects on cognitive and non-cognitive development, schooling attainment and later outcomes as an adult in terms of earnings and productivity (Doyle et. al 2009, Maluccio et. al., 2009, Dercon and Sanchez 2013, Hoddinott et al., 2013). Short stature is also found to increase the chances of giving birth to smaller babies and experiencing complications during pregnancy and childbirth (Black et. al. 2008). Recent studies have also found evidence that both contemporary and childhood health of the mother correlate positively with offspring health, and these effects are likely to be persistent (Bhalotra and Rawlings 2011). Most papers on stunting, however, focus on children under 5. This is unsurprising given that stunting is argued to occur mainly within the first few years of life (Martorell et. al. 1994). Moreover children usually enter middle childhood (defined here as ages 7 to 12) and adolescence (between ages 13 to 19) with nutritional deficits accrued from earlier on in life. However, children already stunted may 'catch up' later on childhood given appropriate conditions (Golden 1994, Tanner 1986) just as much as some of those who did not enter middle childhood stunted may falter in their growth and become stunted by the time they reach young adulthood. There is little empirical research on growth patterns of those who enter middle childhood stunted: Did they remain stunted as adults or move out of being stunted during adolescence? Were those not stunted in middle childhood falter in their growth during adolescence such that they were stunted as adults? What observable individual and household characteristics drove these results? To what extent did variations in the stunting profile in middle childhood and adolescence influence

outcomes as young adults including cognitive and psychosocial outcomes, and health outcomes of the offspring of the stunted children? This paper attempts to answer these questions using 4 rounds of the *Young Lives* longitudinal data collected for children from Andhra Pradesh and Telangana from ages 7 to 19. There is some evidence in the economic and public health literature, obtained using earlier rounds of the same longitudinal data set used in this paper, that growth catch up can occur in middle childhood (Himaz 2009) and early adolescence (Outes and Porter 2013; Fink and Rockers 2014). These studies use height for age z-scores (HAZ) as the key unit of measurement to assess catch-up growth. However, a recent debate in the literature argues that HAZ is inappropriate for measuring 'catch up' in a longitudinal dataset although it is appropriate for comparing groups of children between countries at a given point in time (Leroy et. al 2015). The argument is based on the observation that the HAZ is derived by using the difference between the actual height of the child in centimetres and the expected height according to the standard (HAD), divided by the age-sex based standard deviation for the reference population. This standard deviation increases over time and is based on cross section data. Thus a gain in the HAZ value may partly be due 'true gains' in HAD (the numerator), but also due to the fact that the denominator has risen even if the numerator has not. This means that observed 'catch up' when measured using longitudinal data is a statistical artefact if driven by increases in the denominator that are higher than the numerator. Thus HAD arguably is a more suitable measure of assessing catch-up. There are significant differences in results depending on what you use. For example the 'substantial' changes in catch up growth observed by Fink and Rockers (2014) that appear 'equally likely' in middle childhood and early adolescence are not quite substantial if one uses HAD. Instead, catch up growth is more noticeable in adolescence.

Thus this paper deviates from the previous Young Lives data based studies on growth catch up and faltering by (a) analysing the latest round of data for India that includes information for the children aged 19, (b) using both HAD and HAZ to reassess faltering and catch-up effects in middle childhood and adolescence and (c) looking at differentials in growth patterns between boys and girls in the sample (d) looking at how variations in the stunting profile between ages 8 and 19 have an impact on various psychosocial outcomes as a young adult as well as offspring outcomes. Psychosocial outcomes (which refer to behavioural attributes of the individual)are measured using the rich data collected in the Young Lives survey including measures of agency (the child's sense of freedom of choice to influence own life), self-esteem (overall evaluation of self-worth), self-efficacy (coping with daily hassles as well as adaptation after experiencing all kinds of stressful life events), relationship with peers and parents, and general subjective wellbeing.

The paper is organised as follows. Section 2 describes some methodological issues and data used. Section 3 looks descriptively at growth patterns based on how the stunting profile changed among our children as a group as they moved from middle childhood to adolescence and how using HAZ and HAD indicates differences in growth patterns among boys and girls. Section 4 uses HAZ to categorise the sample based on their stunting profile as those who were never stunted, persistently stunted, moved out of being stunted as an adolescent and moved into being stunted during adolescence, to glean insights as to what individual and household characteristics may have influenced height as an adult among individuals in the different groups. Section 5 looks at how the stunting profile correlates with psychosocial outcomes as young adults and the health outcomes of offspring for the subsample who became parents. Section 6 concludes.

2. Methodological issues and data

2.1. Reference values, growth faltering and catch up

The HAZ for a child proxies accumulated investments in child health and is derived by standardizing a child's height using the expected height and standard deviation for a child of his (or her) age and sex. The expected height and standard deviation come from the mean growth trajectory of a population of healthy children from birth to 19 years of age, as constructed by the World Health Organisation, referred to as the WHO reference 2007[i]. The reference population mean growth trajectory is expected to be at the median of the growth standard. A population level deficit in height (calculated as the average of the individual height-for-age differences- HADs), is reflective of growth impairment caused by a deficient environment that may include poor diet, inadequate care and attitudes to health, as faced by the population of children under study.

A child is deemed 'stunted' if the HAZ is below -2 standard deviations of the mean. The -2 Z-score cut off is used by the World Health Organisation (WHO) Global Database on Child Growth and Malnutrition implying that 2.3% of the reference population will be classified as being stunted even if they have no growth impairment and are not unhealthy.

Catch up growth can be defined as partial recovery from a linear growth deficit accumulated in the past. For recovery to happen children should grow faster than the expected velocity for their age and gender, making up for lost growth in height. But as Chrestani et.al.(2013) observe in a systematic review of articles in the medical and public health literature as found in Medline/PubMed databases on catch up growth among children under 12, there is no uniformity in the operational definition of the concept of catch up. In recent econometric literature such 'catch-up' has been identified by looking at the slope of the lagged HAZ in a

dynamic model of nutritional status. But as discussed in the introduction, the use of HAZ to measure catch up when using longitudinal data, is debated. Leroy et. al. (2015) suggest that when using longitudinal data, true catch up can be measured only using the HAD absolute values. HAD uses an expected growth trajectory based on a reference population of children unlike HAZ changes that do not compare against an “expected HAZ trajectory". Thus this paper avoids measuring catch up growth using regression analysis and instead investigates it in descriptive terms using changes to HAD in section 3, which is compared to trends in HAZ.

2.2 Data

The data for much of this paper comes from the older cohort of the Young Lives longitudinal survey data for children, households and their communities collected in 2002, 2006, 2009 and 2013 from two regions in Andhra Pradesh (Coastal Andhra and Rayalaseema), and Telangana, India, when the 'index child', was aged 8, 12, 15 and 19 years on average, respectively[ii]. The original sample contained 1000 children which dropped to 994 and 976 in the second and third rounds. By the fourth round the number was 951. Still, the overall attrition rate of 4.8 per cent over 11 years (averaging 0.4 per cent per year) is one of the lowest in longitudinal surveys of this nature (Barnett et. al. 2013)[iii].

The sample is largely pro-poor, as the aim of the Young Lives project is to look at the causes and consequences of childhood poverty. However, a careful analysis of the distribution of child characteristics included in the sample suggests that the data covers a wide variety of children in terms of wealth, consumption, similar to nationally representative datasets. Therefore, while not suited for simple monitoring of child outcome indicators (as the mean characteristics will be different), the Young Lives sample is appropriate for analysing

correlates, causal relations and dynamics (Kumra 2008). The Young Lives project also collected information for 2000 children over the 4 rounds from a younger cohort, aged 12 months on average in 2002. This data is used occasionally in this paper for descriptive purposes, as acknowledged where appropriate.

Appendix Table A1 provides descriptive statistics (means, standard deviation and range) for the key variables used in this paper, as well as providing definitions for these variable. As the table shows, roughly half the sample of children are female. Overall, the average HAD across the four rounds indicate a worsening although not necessarily in a linear manner, moving from -8.93 in 2002 when the child was aged around 8 years to -11.72 at age 15 to -10.57 as a young adult aged around 19. In contrast, average HAZ seems to fluctuate around the -1.56 standard deviation mark that was noted in the first round. However, these averages hide significant gender and other variations, as discussed in the analysis to come.

3. Growth Faltering and Catch up

Around 30 per cent of the children in our sample were stunted at age 8. However, less than half of these children remained stunted at age 19. As Figure 1 shows, some children moved into being stunted later in childhood and adolescence while others moved out at various points. By age 19, around 10 per cent of the sample were persistently stunted since age 8, while 20 per cent were newly stunted in middle childhood or adolescence. So there seem to be considerable dynamics in terms of growth faltering between ages 8 and 19.

FIGURE 1 HERE

Table 1 provides some descriptive statistics that indicate how child health outcomes varied among our groups at age 19 compared to the reference group of children that experienced no stunting[iv]. The table shows that children who experienced some stunting in middle childhood remain significantly shorter than the reference group even if they are no longer stunted. However, the Body Mass Index for age is significantly lower only for those children persistently stunted and for those that were stunted in middle-childhood but moved out of this category in adolescence. Those persistently stunted also have significantly higher long term health issues and a significantly lower perception about their own health compared to those never stunted. Quite noticeably, a significantly higher proportion of those who moved into being stunted in middle childhood are girls (76%) as are those who moved into being stunted during adolescent years (62%).

TABLE 1 HERE

Were patterns of growth different depending on the measurement used i.e., HAZ or HAD? As Figure 2 shows, conventional HAZ measures for both boys and girls show a fall between 12 to 60 months. But there is an improvement in these averages between ages 5 and 8. Patterns observed for boys and girls deviate from this point onwards in our sample of children. The average HAZ seems to improve for boys relative to the reference population during middle childhood (ages 8 and 12). Then there is a dip between ages 13 to 15 followed by improvements later in adolescence between aged 16 and 19. This 'dip and rise' of HAZ among boys may be indicative of a slightly later onset of the pubertal growth spurt in our sample compared to the WHO reference population. This is unsurprising given that age at puberty differs considerably between populations with later puberty occurring in

populations with a poor nutritional status, as is our sample which is drawn from children from poorer backgrounds. Unlike for boys, girls' HAZ scores continue to deviate from the reference from age 8 onwards until about age 15. The patterns emerging from the HAD analysis are different. Here growth deviations do not show any improvement in middle childhood and continue falling until age 12 or so for both boys and girls. For boys, however, there seems to be some catching up occurring later in adolescence from age 15 onwards. For girls, HAD seem to stop widening compared to the reference population in adolescence with marginal improvements observable, as for boys, in later adolescence. Overall, growth faltering for girls seems worse than that for boys, after age 5.

FIGURE 2 HERE

One implication of the patterns in HAD and HAZ measurements is that the onset of puberty is perhaps slightly later for our sample of poorer children giving rise to a higher growth velocity later on in adolescence compared to the reference population. The older cohort data asks children directly about the onset of puberty. Roughly 27 per cent of the girls have started menstruating by age 12. They have a mean HAZ of -0.93. Only 11 per cent of this group are stunted. Among boys 22 per cent have experienced a deepening of voice and less than 3 per cent have any visible hair growth on their chin by age 12. Just like the girls who have reached puberty, a majority of these boys (95 per cent) are not stunted, with an average HAZ of -1.05. In the reference population the growth velocity for girls seem to peak around ages 11 to 12 and boys around 13 to 14. Thus it seems that the pubescent growth spurt occurs slightly later especially for children who are stunted in our sample than that in the reference population, similar to discussions in Kulin et. al (1982) or Parent et. al. (2003).

Does this mean that the apparent 'catch up' in the graph above is simply the effect of later timing of the pubescent growth spurt between the sample and the reference group especially for stunted children rather than true catch up? This is a difficult question to answer but what can be noted is that although average HAD indicates a rise later in adolescence there are substantial differences among different groups of children, with some indicating substantial faltering rather than catch up. To illustrate, Table 2 shows that the highest improvement of HAD in adolescence is amongst those who were stunted in middle childhood but moved out during adolescence; 5.46cm for boys and 8.01cm for girls. The second highest improvement is seen in those who were persistently stunted; 2.04cm for boys and 1.76cm for girls. So these are the groups that seem to have had some amount of growth 'catch up'. But for those children who did not enter middle childhood stunted but went on to becoming stunted later on in childhood -roughly 15 per cent on the sample- growth faltered substantially between ages 12 and 19. For boys in this group, HAD widened by 10.92cm over adolescence while for girls it widened by 6.58cm. In contrast HAD widened by less than 1cm during the adolescent years for boys and girls who had never been stunted. .

Overall, therefore, the improvement in average HAD deficits in adolescence is driven by those that moved out of being stunted and those persistently stunted (32 per cent of the sample in total), reflective perhaps of delayed pubertal growth spurts. But the average improvement conceals the fact that for around 15 per cent of the sample substantial growth faltering has occurred.

TABLE 2 HERE

4. What drove growth dynamics in adolescence?

Himaz (2009) argues that even if a child starts middle childhood with significant shortfalls in height accrued from earlier on in life, nutritional interventions and adult female education may have a positive impact on linear growth and perhaps mitigate consequences of early age stunting by the time the child reaches age 12, using Young Lives data for 2002 and 2006. That analysis is extended here using further rounds of data collected in 2009 and 2013 to ascertain what individual and household characteristics may be helping children move in to stunting and out of stunting in *adolescence*. The model estimated is specified as follows:

$$y_{it} = \alpha + \beta_1 X_i + \beta_2 \delta_{i,j,t-1} + \beta_3 \theta_{c,t-1} + \epsilon \qquad (1)$$

where *y* refers to the stunting profile of child *i* at time *t* (when the child is a young adult aged 19) with *y*=1 if the child had never been stunted (i.e., not stunted at age 8, 12 or 19), *y*=2 if the child moved out of being stunted during adolescence (i.e., stunted at age 8 or 12 but not stunted at 19 indicating stunting in middle childhood but moving out during adolescent ages 13-19), *y*=3 if the child moved into being stunted during adolescence (i.e., not stunted at age 8 and 12 but is stunted by age 19) and y=4 if the child has been persistently stunted through middle childhood, adolescence and is stunted as a young adult (i.e., stunted at ages 8, 12 and 19); *X* is a vector of child-specific fixed characteristics such as gender, birth order, caste (Scheduled Tribe, Scheduled Caste, Backward Caste with Other omitted), mother's height in centimetres (log), and a proxy for the quality of the child's diet; δ is a vector of household characteristics for child *i* in house hold *j*, at time period *t-1*, which refers to the time when the child was 12 years old at the start of adolescence (round 2). The

household characteristics include the wealth index, mother's education, demographic composition of children in the household by age and gender (proportion of females in the household aged 0-5, 6-12, 13-17, 18+ and males aged 0-5, 6-12, 13-17, 18+ with females 18+ omitted), region of residence (Coastal Andhra, Rayalaseema, Telangana with Telangana omitted), and the area of residence (rural or urban). Finally, θ refers to community level characteristics such as whether the sewerage disposal facilities are considered 'good' as opposed to being bad or 'so-so', and if access to electricity was considered to be good, at round 2. These are both dichotomous variables.

The inclusion of these control variables have been based mainly on the theoretical and empirical literature based on child health in India. For example, gender, birth order and demographic composition of household are included as there is evidence to suggest that household resource allocation and attitudes to investment in health may depend on these factors (Behrman (1988), Jayachandran and Kuziemko (2011), Jayachandran and Pande(2015), Pande (2003)), while mother's height is meant to capture as far as possible the influence of genetic factors especially during adolescent years (Addo et. al. 2013). Caste is a proxy for access to health related services as well as attitudes and practices related to health inputs[v]. In order to account for the possibility that diet and food security may have an impact on adolescent linear growth (as argued in Rogol et. al 2000, Belachew 2013) I create a dietary diversity index based on Swindale and Bilinsky (2006). The index is mainly a proxy for the diversity in a child's diet in terms of the various food groups consumed, but also food security[vi]. The community level variable regarding access to good sewerage facilities is included as there is evidence to suggest that sanitation can play a strong role in child health

(Spears et. al 2013) while access to electricity proxies the level of 'development' in the community the child comes from, and thereby accounting, at least partially, for any community-level factors that maybe affecting adolescent health. The region of residence controls for distinct agro-climatic and other variations in the three regions the children come from.

Specification 1 is estimated using multinomial logit estimation with results reported in Table 3, columns 1-3 below. Column 4 reports Ordinary Least Squares (OLS) regression results with HAZ at age 19 being the dependent variable.

TABLE 3 HERE

The results in column 1 show that one of the most significant factors associated with moving a child out of being stunted relative to the never stunted category is region of residence (coming from Rayalaseema compared to Telangana) while influencing negatively are belonging to the scheduled caste compared to other casts and rather counter intuitively, household wealth. Even when the wealth index is substituted with real expenditure per capita in round 2[vii], the result is the same: increases in wealth (or household consumption proxied by per capita total spending) seems to contribute negatively towards moving a child out of being stunted in adolescence compared to the never stunted category. One reason maybe that per capita increase in wealth (or spending) does not necessarily increase child calorie intake. This may be due a change in household tastes as incomes increase resulting in a change to dietary composition, with better quality cheaper food such as coarse grain substituted for poorer quality often more expensive refined food (Deaton and Dreze

2009:56-58) or due to the increase in spending on non-food items (Basole and Basu 2015)[viii]. The signs for other control variables are as expected, although not statistically significant.

The results from column(2) shows that the most significant factor that is associated with moving a child into being stunted during adolescence is gender: the relative log odds of moving *into being stunted* in adolescence compared to never being stunted falls by 0.57 when moving from being female to male. Similarly, belonging to the Scheduled Caste as opposed to Other, positively affects moving into being stunted compared to never being stunted. In contrast, increased dietary diversity and wealth both reduce the relative log odds of moving into being stunted compared to never being stunted. Thus being female, having a diet that is not varied, household level wealth deprivation and belonging to the scheduled caste are all factors that significantly influence moving into being stunted in adolescence.

Column 3 shows that the relative log odds of being persistently stunted compared to never being stunted is increased when coming from Scheduled Caste, Backward Castes or Scheduled Tribes as opposed to Other castes. The strong correlation persistent stunting has with certain castes suggest that attitudes to health and nutrition, health inputs as well as access to services may affect investments in linear growth in adolescence significantly. Persistent stunting compared to never being stunted is associated negatively by mother's education and positively with coming from Rayalaseema as opposed to Telangana. The Wald test at the bottom of the table tells us that our model as a whole fits significantly better than an empty model (i.e., a model with no predictors)[ix].

Column 4 shows that the key characteristics at age 12 that correlate significantly positively a child's final linear growth outcome are being male, genetic effects, dietary diversity, mother's education, household wealth and being from 'other' caste.

The robustness of these results are checked for by changing the specification to be more parsimonious. I exclude mother's height (since this value was not available for 43 observations and thus limited the number of observations I used in the previous estimation) and household demographic composition. All the results discussed previously remain robust to this change in specification (Appendix Table A2).[x] It is acknowledged here that although the issue of reverse causality is minimal under the specifications above since the explanatory variables are lagged, it does not preclude the possibility that there may be unobservables that may have not been captured in the specifications (in spite of the efforts to include suitable proxies). This means the relationships I estimate are largely correlations rather than being causal.

5. Stunting profile and outcomes

5.1. Psychosocial outcomes

In this section I investigate how changes in the stunting profile in middle childhood and adolescence are associated with cognitive and psychosocial outcomes as a young adult of age 19. This focus is different to that of most papers in the empirical literature that look at stunting in early childhood and subsequent psychosocial outcomes. For example, poor early investment in health is hypothesised to have significant negative impacts on psychosocial outcomes later on (Chang et. al 2002, Walker et. al. 2007). Supporting this, Dercon and Sanchez (2013) find empirically a correlation between height-for-age at age 7-8 (a proxy for

early investments in child health) and psychosocial competences ('non-cognitive' skills such as school aspiration, self esteem and self efficacy) at age 11-12. They stipulate that this provides evidence for an underlying relationship between undernutrition earlier on in childhood and the formation of non-cognitive skills. But psychosocial skills -especially later on in childhood- can also be shaped by height relative to peers rather than by long-term effects of early investments in nutrition. For example, short stature may be correlated with increased experience of being teased, juvanilisation (i.e., socialisation that can happen according to height-age rather than chronological age) or overprotection by parents as stunted children are (or are perceived to be) physically weaker than peers that may itself cause victimisation by peers, all contributing to poorer psychosocial outcomes (Stabler et. al. 1994, Steinhausen 2000). Although compelling especially in clinical data based studies, this view is not always supported empirically in non-clinical settings (Voss and Sandberg 2001, Voss et. al. 2004). For example, the longitudinal community-based Wessex Growth Study compared psychosocial outcomes for a sample of very short (less than the 3rd centile) but otherwise healthy children at school entry (around age 5) with those of controls with average height, at ages 7-8, 11-12 and 18-20 to find no significant differences in terms of Adolescent to Adult Personality Functioning Assessment (ADAPFA) scores between the groups (Downie1997, Ulph 2004). The ADAPFA measures functioning in the domains of education and employment, love relationships, friendships, coping, social contacts, and negotiations. Thus the empirical evidence found in the literature on whether stunting in early childhood has a causal link to psychosocial competencies -via the nutrition link or the relative height link- is both sparse and not particularly strong in terms of evidence. But almost no work has been done on what impact becoming stunted later in childhood has on psychosocial outcomes and this is what we turn to next.

The model I estimate is as follows:

$$y_{it} = \alpha + \beta_1 X_i + \beta_3 \delta_{i,j,t-1} + \beta_4 \theta_c + \epsilon \qquad (2)$$

where y refers to the psychosocial outcome of child *i* at age 19 (time *t*), which is a continuous variable reflecting standardized values for measured psychosocial outcomes. These outcomes are agency (i.e., power or ability to influence one's life and related to the concept of 'locus of control' as discussed in Rotter (1966)), self-esteem(i.e., an evaluation of self worth which follows an adapted version of the self-esteem Scale in Rosenberg 1965, self efficacy (coping with daily hassles as well as adaptation after experiencing all kinds of stressful life events, see Schwarzer and Jerusalem 2010) and relationships with peers, parents and general self perception as effective, capable individuals taken from the Self Description Questionnaires as discussed in Marsh (1984)[xi]. The full list of survey questions included to compute these scales, tailored to suit a context of child poverty and specific dimensions of the child's living circumstances such as housing, clothing, work, school, are reported in Table A3 in the Appendix. Vector *X* refers to child specific characteristics such as stunting profile (i.e., five categorical variables as described previously, with never stunted being the omitted group), gender, birth order, caste; vector δ reflects household characteristics for child *i* in house hold *j*, at time period *t-1*(when the child was 8 years old). The household characteristics include household size, wealth, father's and mother's education in years, region of residence, area of residence (rural/urban). Finally, θ refers to community level fixed effects used to control for unobserved heterogeneity among the communities and ε is the random error term. Equation 2 is estimated using OLS regression analysis.

TABLE 4 HERE

The results reported in Table 4, columns 3 and 4 show that moving into the 'stunted' category in adolescence (between ages 12 and 19) is strongly correlated with a child reporting to have poorer relationships with peers (at the 1 per cent level of significance) and lower self efficacy (at the 10 per cent level) compared to the group that were never stunted. Our previous discussion suggests that this group of children seem to be ones that experienced substantial growth faltering between ages 12 and 19. Apart from this, persistent stunting is positively significantly correlated at the 10 per cent level with having strong relationships with parents. What these correlations suggest is that i there is no support for the hypothesis that early inadequacies in health inputs have long term negative implications on psychosocial outcomes as a young adult. If it did have an impact, then the profiles where the children were stunted at age 8 (such as persistently stunted) should have shown a strong negative correlation between being stunted at age 8 (a proxy for poor inputs in child health in early childhood) and psychosocial competencies as we measure them[xii]. We will return to discussing the significant correlations we *do* find shortly, after noting several other significant explanatory variables in the results. Gender, for instance is significant, with being male exerting a positive effect on agency and self efficacy but a negative impact on most other measures of psychosocial wellbeing compared to being female. The father's and mother's education is significant for agency and self efficacy while household wealth is significantly positive for agency, self efficacy and peer relationships. Caste does not have a systematic significant influence on outcomes, but coming from Coastal Andhra and Rayalaseema seem to exert a positive impact compared on all outcomes

apart from relationship with parents, compared to coming from Telengana (the omitted category). A higher birth order significantly increases a child's general self perception as an effective, capable individual at age 19, possibly due to the responsibilities and respect culturally placed upon an older sibling (Cicirelli 1994).

Returning to the results regarding stunting profile and psychosocial outcomes, why would growth faltering in adolescence(among children not previously stunted) have significant impacts on peer relationships and self efficacy while becoming stunted at other ages, including persistent stunting, do not? One explanation may lie in the relative height hypothesis that being shorter in stature compares to others of the same age imposes significant challenges. Were these children who moved into being stunted in adolescence bullied or teased more by peers, for instance? Descriptive statistics provided in Table 5 show that children who experience stunting in childhood, *regardless of when they became stunted*, report higher incidence of being physically bullied by peers in adolescence (between ages 13 and 15) compared to those never stunted. Table A4 in the appendix describes how the indices for bullying are constructed. Stunted children also feel they are treated less well by adults than their peers. But once controlled for confounding factors in the baseline analysis, children who were newly stunted in adolescence indicate a strong correlation between their stunting profile and psychosocial outcomes such as relationships with peers and self efficacy, unlike other categories of stunted children.

TABLE 5 HERE

Could this be because of omitted variable bias since unobserved determinants of a child's psychosocial development may also be correlated with what may have driven a child into being stunted in adolescence? Such determinants could be, for example, unobserved parental or household inputs. Such inputs and attitudes may be shaped in part by poverty, which we account for some what imperfectly in our baseline specification. But it could also be because children in this group who are largely female (62%), experience different contemporaneous life circumstances to others that affect their relationship with peers in particular and self efficacy. They could have married, for instance, and experienced in consequence less mobility and freedoms in the homes of their husbands' affecting these outcomes. Or the boys and girls may have different time use patterns due to having dropped out of school or spending more time in paid work, both instances that presumably reduce opportunities for positive experiences in socialising with peers and adults. For example Pells at. al (2016:34) quote from the young lives qualitative surveys to argue that caregivers and teachers describe children out of school as being undisciplined and a bad influence on those that attend school and so, encouraged to be disassociated by those who attend school. The latter rows of Table 5 compare the proportions married, enrolled at school and time spent on income earning activity among children by stunting profile. For almost all these variables, apart from 'married', the differences between the average values for all the stunted groups are statistically significantly different to the non-stunted group. For the variable 'married', differences are significant only among those that became stunted in adolescence and those never stunted. Thus 30 per cent of those who experienced growth faltering in adolescence are married compared to around 17 per cent in the other groups. It is possible then that 'married' is correlated to some unobservable (such as parental attitudes perhaps) that affects this group but not other groups of children and I therefore

reestimate equation (2) including married as an explanatory variable. The results reported in Table A5 panel A, show that being married does exert a significant negative impact on agency, relationships with peers and parents but does not change the previously found significant association between becoming stunted during adolescence and peer relationships. There is no longer a statistically significant association between being newly stunted and self efficacy at age 19.

The baseline specification is then re-estimated with further controls that indicate other contemporaneous circumstances that might have impacts on psychosocial competencies. One such variable is when the child is currently attending school. Another is contemporaneous household wealth and controls for household vulnerability to a variety of exogenous economic shocks (natural disasters such as droughts, floods, etc., and income shocks arising out of job loss, stolen crop, etc) that occurred during adolescence. We also include the child's BMI at age 19. Finally we include proxies for a child's psychosocial outcomes at age 8, where available, to remove as far as possible, child specific fixed effects. The results are shown in Table A5 panel B. As with the previous robustness check, becoming stunted during adolescence remains significantly correlated with peer relationships but not self efficacy[xiii].

The baseline results corroborated by the robustness checks suggest that there is a relationship between becoming newly stunted as an adolescent and peer relationships at age 19. One reason for this could be that children in this group are more sensitive to the

negative effects of short-stature (such as bullying and differential treatment) as they have had less time to build adequate coping strategies, unlike those persistently stunted or those who became stunted in middle childhood. Moreover, the baseline results also showed that those persistently stunted enjoy better relationships with their parents, possibly arising out of parents being more protective towards these children who on average have more long term health issues and significantly lower BMI for age (see Table 1). These closer relationship with parents can help reduce sensitivity to negative peer behaviour and improved resilience. Several studies support this contention including Dekovic and Meeus (1997) who argue that the quality of the parent–child relationship influences an adolescent's self-concept which in turn affects his or her integration into the world of peers.

5.2. Offspring health outcomes

I now move onto looking at how changes in the stunting profile in middle childhood and adolescence are associated with the outcomes associated with the offspring of these children. Out of the 951 children in our round 4 sample, 107 (101 girls and 6 boys) went onto have children themselves. In order to ensure comparability with the literature that focuses mainly on the transmission of poor health from mothers to their offspring, only offspring born to female index children are included in the discussion and analysis below. As shown in Appendix Table A6, the 101 girls in our sample who had offspring by age 19 were slightly older on average than those girls who did not (19.05 years as opposed to 18.98), with a higher proportion (44% as opposed to 24%) reporting to have reached menarche at the time of round 2 (age 12). The girls who had offspring were statistically significantly taller than those who did not at ages 8 and 12, but these differences reduced

over time such that by age 19, there was no statistically significant differences in height. Although the rough proxies we have used for health outcomes between the two groups are not significantly different, girls who already have offspring came from predominantly rural areas, from households that were significantly poorer in all four rounds and had attained levels of formal education that were significantly lower than those who did not have children. There were no differences in the representation of castes between the two groups.

Out of the 101 girls who went on to have offspring by age 19, twenty three had two children each, and thus the total number of offspring in the sample is 124. However, 6 of the offspring had died by the time round 4 interviews occurred. Also excluded are a few observations, where appropriate, if data is missing or recorded values are outside the reasonable range (for example HAZ is outside range ± 6, WAZ outside range -6 and +5). In Table 6 columns (1)- (5) below, I look at how the stunting profile of the Young Lives children (reduced to just a dichotomous variable that equals one if a child experienced some stunting or persistent stunting at ages 8, 12 and 19 and zero if the child experienced no stunting), offspring age, gender and household characteristics in round 4, namely, wealth, region and rural residence, correlate with offspring HAZ, weight for age z-scores (WAZ), incidence of offspring still being alive, pre-term birth and the mother having had a difficult labour.

The sample size is small and the results are indicative rather than being definitive. Moreover, as noted earlier, the sub sample of girls who are already mothers is selective, in that they come from relatively more disadvantaged backgrounds in terms of wealth, for

example, although not necessarily more disadvantaged in terms of accumulated investments in health as captured by HAZ or stunting profile. Nonetheless, the fact that the group is selective in terms of wealth suggests that there could be unobservable such as attitudes to health that are correlated with the mother's stunting profile as well as the error term, that may bias the coefficient on the stunting profile. Unless appropriately corrected for this sample selection bias the results produce at best some indication of correlation rather than causation. Bearing these caveats in mind it can be noticed from the regression results that there seems to be evidence of the intergenerational transfer of poor health outcomes, with those girls that experienced stunting in middle childhood or adolescence more likely to have children with lower HAZ, lower WAZ and be more likely to have offspring who died in infancy. In order to glean some insight as to whether these results are driven mainly by mothers who were persistently stunted, columns (6) to (10) present results where the stunting profile is split into three categories rather than two: those persistently stunted (i.e., stunted at age 8, 12 and 19), those that experienced some stunting (i.e., stunted at age 8 or age 12 or age 19 but not persistently) and those that experienced no stunting at all (reference category). The results indicate that persistent stunting in mothers correlates significantly with offspring mortality (column 8). Mothers who experienced some (but not persistent) stunting in middle childhood and adolescence seem to have offspring who are significantly shorter and thinner than mothers never stunted (columns 6 and 7). It is not clear why those persistently stunted do not have surviving children with poorer health outcomes compared to the never stunted mothers, but it is likely that the fittest survived and are possibly have health inputs that compensate for the loss of their siblings. The latter is of course conjecture that needs further investigation using the qualitative data the Young Lives project collects.

Mirroring patterns for the parents, columns (1), (2) (6) and (7) also indicate that growth faltering seems to happen with the offspring as well, with age having a significant negative impact on both WAZ and HAZ. Moreover, sharp deviations to HAD starts within the first few years of life for offspring, similar to trends observed for index children in Figure 2. With regard to the other control variables used, it can be seen that household wealth and living in Coastal Andhra compared to Telangana exert a positive influence on WAZ. Column (4), (5), (9) and (10) indicate that stunting profile does not have a significant effect on premature birth or having a difficult labour.

TABLE 6 HERE

6. Conclusions

This paper used longitudinal data from four rounds of the Young Lives older cohort survey for Andhra Pradesh and Telangana, India, to investigate how growth faltering beyond early childhood can have an impact on outcomes as young adults and of the offspring of these young adults. One of the first things noted was that although around 30 per cent of the children in our sample enter middle childhood stunted, there is considerable fluidity in the children's stunting profile as they move through middle childhood and adolescence to reach young adulthood. Adolescence in particular is a period where children -especially boys- who were previously stunted seem to catch up in terms of growth, at least partially, and move out of being stunted. More than half the individuals who are stunted at age 19 are those that moved into being stunted during middle childhood and adolescence. The incidence is much higher among girls, possibly linked to inadequate nutrition, wealth deprivation and factors associated with attitudes. It could also be related to health practices, given that

caste is associated significantly with moving into being stunted. This is an area that requires further investigation. Persistency in stunting through childhood and adolescence was positively correlated with mother's education level as well as caste.

Growth faltering and catch-up patterns are different depending on whether height for age difference (HAD) or the more conventional (HAZ) are used. When HAZ is used as the measure of growth changes, it shows that for boys, the average HAZ improves relative to the reference population mainly in middle childhood and then again later in adolescence. For girls the trend is a continued fall in average HAZ from middle childhood onwards until about age 15. However, when HAD is used, growth deviations do not show any improvement in middle childhood for either boys or girls. Instead, it continues falling throughout middle childhood until age 12 or so with the deviation from the heights of the reference population higher for girls than boys. After this point, HAD stops widening for girls with marginal improvements observable later adolescence. For boys, HAD widens during the early adolescent years before improving later in adolescence. Overall, trends in HAD show that growth faltering in girls is higher than that for boys, after age 5, particularly during middle-childhood and pre-adolescent years. In the text it was argued that some but not all of these patterns in growth may be explained perhaps by delayed pubertal growth spurts among children in our sample compared to those of the reference population.

The results also show that moving into the 'stunted' category in adolescence (between ages 13 and 19) is strongly correlated with a child reporting to have poorer relationships with peers compared to the group that were never stunted. This may be due to these children being more sensitive and less resilient to the effects of peer bullying and other difficulties

arising out of short relative stature as they have had less time to build effective coping strategies unlike those persistently stunted. The results for those newly stunted also do not indicate significantly positive relationships with parents unlike those persistently stunted, which also reduces their resilience to the negative effects associated with short stature. Psychosocial outcomes at age 19 were also significantly influenced by factors such as a child's gender, parental education levels, household wealth at age 8, birth order and region of residence. Interestingly, there is no evidence that poor investments in early nutrition (as proxied by being stunted at age 8) has had long term negative impacts on non-cognitive outcomes at age 19.

Although the sample of offspring is small and therefore results tenuous, there seems to be some evidence of the intergenerational transfer of poor investments in health between mothers and their offspring, as those who experience some or persistent stunting in middle childhood/adolescence have offspring who are significantly shorter and thinner than those who were never stunted as well as having a higher chance of their offspring dying in infancy. The strong correlation between offspring mortality and stunting seems to be driven by mothers who were persistently stunted. So growth fluctuations during the pre-adolescent and adolescent years -especially if such fluctuations cause growth faltering and stunting in a girl child- is associated significantly with offspring health and mortality outcomes. This area requires further research and can possibly shed some light on how to reduce the inter-generational persistence of poor health outcomes. For example, stronger evidence in this area, perhaps through the use of further rounds of Young Lives data that will become available in the future, can lend support to policy recommendations that target female nutrition during middle childhood and adolescence to reduce the intergenerational

persistence of stunting. Other policy recommendations can include family planning, and if pregnancy does occur during teenage years, maternal and child health interventions.

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Table 1: Gender and health outcomes at 19 for selected groups of stunted versus never stunted children

	Never Stunted	Stunted at 8,12,19 (persistently stunted)	Stunted at 12, 19 (moved to being stunted in mid. childhood)	Newly Stunted at 19 (moved into being stunted in adolsc.)	Stunted at 8 or 12(moved out of being stunted in mid ch.hood/adolsc)
	(1)	(2)	(3)	(4)	(5)
Girls (%)	0.48	0.5	0.76***	0.62***	0.56
Health Outcomes at age 19 (round 4)					
Height for age z-score (HAZ)	-0.94	-2.64***	-2.71***	-2.44***	-1.36***
Height for age difference (HAD) cm	-6.6	-18.23**	-18.23***	-16.62***	-9.56***
Body Mass Index for age	19.89	18.93***	20.49	19.58	18.8***
Own perception of general health (very poor=1, poor=2, average=3, good=4, very good=5)	3.89	3.75**	3.43***	3.87	3.92
Permanent disability affecting work/school	0.01	0.05***	0.03	0.02	0.01
Nr of Obs. at age 19 (round 4)	459	112	37	103	193

Note: Asterisks ** and *** mark that the mean outcome for that group was significantly different to the corresponding mean outcome in the reference group (never stunted, col. 1) at the 5 and 1 per cent levels, respectively.

Table 2: Catch up or further faltering of growth in adolescence?

	Disaggregated Sub-Sample				Full Sample
	Never Stunted	Persistently stunted	Moved into being stunted in mid. ch.hood/adols.	Moved out of being stunted in mid ch.hood/adols.	
Boys					
HAD in round 1 (a)	-4.87	-16.26	-6.35	-13.34	-8.67
HAD in round 2 (b)	-5.95	-20.14	-6.91	-15.53	-10.41
HAD in round 4 (c)	-6.67	-18.1	-17.83	-10.07	-10.32
Change in HAD ages 8-12: (b)-(a)	-1.08	-3.88	-0.56	-2.19	-1.74
Change in HAD ages 12-19: (c)-(b)	-0.72	2.04	-10.92	5.46	0.09
Girls					
HAD in round 1 (a)	-5.76	-16.8	-8.75	-13.03	-9.21
HAD in round 2 (b)	-6.39	-20.12	-9.82	-16.91	-11.29
HAD in round 4 (c)	-6.53	-18.36	-16.4	-8.9	-10.81
Change in HAD ages 8-12: (b)-(a)	-0.63	-3.32	-1.07	-3.88	-2.08
Change in HAD ages 12-19: (c)-(b)	-0.14	1.76	-6.58	8.01	0.48
Number. of observations	459	112	137	193	901

Table 3: Determinants of persistent stunting, moving in and out of being stunted in adolescence and HAZ at age 19 (full model)

Dependent variable	Multinomial Logit			OLS
	Moved out of being stunted in adolesc.	Moved into being stunted in adolescence	Persistently Stunted	HAZ at age 19
	(1)	(2)	(3)	(4)
Gender (1 = male)	0.21	-0.57***	-0.06	0.20***
	(0.18)	(0.22)	(0.19)	(0.06)
Mother's height	0.30	-1.08	-0.94	0.85**
	(1.07)	(1.16)	(1.26)	(0.40)
Dietary diversity	-0.09	-0.18*	0.06	0.07**
	(0.11)	(0.11)	(0.14)	(0.03)
Mother's education	-0.02	-0.01	-0.09**	0.03***
	(0.03)	(0.03)	(0.04)	(0.01)
wealth index, 2006	-1.85***	-1.80**	-0.70	0.47**
	(0.62)	(0.73)	(0.79)	(0.21)
Scheduled Caste	-0.88***	0.71*	1.65***	-0.52***
	(0.34)	(0.43)	(0.49)	(0.11)
Scheduled Tribe	0.03	0.76	1.64***	-0.49***
	(0.41)	(0.51)	(0.59)	(0.15)
Backward Caste	0.16	0.47	1.36***	-0.28***
	(0.23)	(0.35)	(0.47)	(0.07)
Coastal Andhra	-0.31	0.01	-0.05	0.10
	(0.26)	(0.31)	(0.33)	(0.08)
Rayalaseema	0.49**	0.35	0.56**	-0.06
	(0.24)	(0.29)	(0.28)	(0.08)
Constant	-1.34	4.11	1.38	-5.99***
	(5.44)	(5.90)	(6.24)	(2.02)
Observations	858	858	858	893
R-squared				0.18
Wald test	$\chi^2\,(60) = 345.44\; Prob > \chi^2 \;= 0.00$			

Notes: All regressions include the following variables apart from those reported: birth order, household size, household demographic composition, rural residence, good access to electricity and sewerage disposal facilities at the community level. Robust standard errors (adjusted for 100 community clusters) in parentheses *** p<0.01, ** p<0.05, * p<0.1

Table 4: Stunting profile, characteristics at age 8 and psychosocial outcomes as a young adult aged 19

Dependent variable	Agency	Self esteem	Self efficacy	Peer relationship	Parent relationship	General
	(1)	(2)	(3)	(4)	(5)	(6)
Stunting profile (reference group: never stunted)						
Moved in adolescence	-0.07	-0.07	-0.13*	-0.25***	-0.06	-0.03
	(0.07)	(0.11)	(0.07)	(0.08)	(0.08)	(0.06)
Moved in mid ch.hood	-0.01	-0.09	-0.02	-0.04	0.06	-0.02
	(0.08)	(0.10)	(0.10)	(0.08)	(0.09)	(0.07)
Persistently stunted	-0.03	-0.09	0.09	0.08	0.13*	0.04
	(0.06)	(0.09)	(0.07)	(0.08)	(0.08)	(0.06)
Moved out mid ch.hood	0.06	-0.05	-0.01	-0.01	0.05	0.01
/adolsc.	(0.04)	(0.06)	(0.05)	(0.05)	(0.07)	(0.04)
Gender (1=male)	0.20***	-0.26***	0.18***	-0.09**	-0.22***	-0.03
	(0.04)	(0.06)	(0.04)	(0.04)	(0.06)	(0.04)
Birth order	0.00	0.00	0.01	0.01	0.00	0.03**
	(0.01)	(0.02)	(0.02)	(0.02)	(0.02)	(0.01)
Father's education	0.01**	0.01	-0.01	-0.00	-0.00	0.00
	(0.01)	(0.01)	(0.01)	(0.00)	(0.01)	(0.00)
Mother's education	0.01*	0.01	0.02***	0.01	0.01	0.01
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Wealth Index, 2002	0.26**	0.24	0.37**	0.47**	0.26	0.30**
	(0.13)	(0.21)	(0.16)	(0.19)	(0.17)	(0.13)
Scheduled Caste	0.03	0.06	0.02	-0.06	-0.18**	-0.01
	(0.08)	(0.11)	(0.07)	(0.08)	(0.08)	(0.07)
Scheduled Tribe	-0.04	0.21*	0.01	0.08	-0.10	0.03

	(0.07)	(0.12)	(0.07)	(0.12)	(0.11)	(0.10)
Backward Caste	-0.02	0.02	0.00	-0.03	0.01	0.04
	(0.06)	(0.09)	(0.06)	(0.06)	(0.06)	(0.06)
Rural	-0.00	-0.14	-0.06	0.26*	-0.04	0.14
	(0.13)	(0.21)	(0.15)	(0.15)	(0.28)	(0.17)
Coastal	1.26***	1.78***	0.68***	-0.32***	0.19	0.23*
	(0.36)	(0.38)	(0.16)	(0.10)	(0.40)	(0.12)
Rayalaseema	1.10***	1.36***	0.40***	-0.45***	0.10	0.03
	(0.04)	(0.08)	(0.05)	(0.06)	(0.06)	(0.05)
Constant	-1.03***	-1.02***	-0.55***	-0.05	-0.10	-0.42**
	(0.19)	(0.23)	(0.17)	(0.19)	(0.29)	(0.17)
Observations	932	932	931	932	932	932
R-squared	0.26	0.22	0.27	0.19	0.20	0.20

Notes: All regressions also controlled for household size and community fixed effects. Robust standard errors (adjusted for 100 community clusters) in parentheses *** p<0.01, ** p<0.05, * p<0.1

Table 5: Experience of being bullied, treatment by adults and selected circumstances during adolescence[1]

	Never stunted	Moved into being stunted in adolescence	Moved into being stunted in middle childhood	Moved out of being stunted in adolescence	Persistently Stunted
	(1)	(2)	(3)	(4)	(5)
Experience of being bullied by peers between ages 12-15 (proportion)[2]					
Verbal	0.24	0.26	0.15	0.33***	0.31
Physical	0.15	0.27***	0.25***	0.31***	0.26***
Other types	0.24	0.16	0.25	0.27	0.30
Adults treat child as well as they do others of same age (proportion)[3]	0.90	0.82***	0.83***	0.87	0.80***
Married (proportion), 2013	0.18	0.30***	0.19	0.15	0.17
Enrolled at school (proportion), 2013	0.55	0.32***	0.24***	0.50	0.46*
Activities for pay/money (hours a day), 2013	0.72	1.20**	1.35**	1.19**	1.10
Observations	459	103	37	193	112

[1] Mean outcomes for the respective values in column 1 compared with corresponding mean values in columns 2-5, to test for statistically significant differences, * 10 per cent, ** 5 per cent and *** 1 per cent.

[2] See Table A4 for details on how these indices are calculated.[3] Based on round 3 survey question 'Adults in my community treat me as well as they do other children at my age'. Experience coded as 1 if response was agree or strongly agree, 0 otherwise.

Table 6. Offspring outcomes regressed against parent stunting profile

Dependent Variable	HAZ (1)	WAZ (2)	Still Alive (3)	Born early (4)	Difficult Labour (5)	HAZ (6)	WAZ (7)	Still Alive (8)	Born early (9)	Difficult Labour (10)
Stunting Profile										
Some/Pers	-0.96**	-0.71**	-0.04**	0.08	-0.01					
	(0.43)	(0.27)	(0.03)	(0.07)	(0.10)					
Some stunting						-1.11**	-0.79***	-0.01	0.11	-0.05
						(0.46)	(0.29)	(0.01)	(0.08)	(0.10)
Persistent stunting						-0.27	-0.40	-0.16***	-0.04	0.15
						(0.75)	(0.57)	(0.13)	(0.13)	(0.18)
Offspring age	-0.42***	-0.19**				-0.39***	-0.17*			
	(0.12)	(0.09)				(0.12)	(0.09)			
Offspring gender	-0.31	0.16	-0.03	0.00	0.16*	-0.28	0.18	-0.03	-0.00	0.17*
	(0.33)	(0.24)	(0.02)	(0.07)	(0.09)	(0.35)	(0.25)	(0.02)	(0.06)	(0.09)
Wealth index, 2013	2.00	2.25**	-0.12*	0.16	0.22	2.07	2.26**	-0.07	0.18	0.20
	(1.24)	(0.97)	(0.07)	(0.24)	(0.33)	(1.25)	(1.00)	(0.05)	(0.24)	(0.33)
Coastal Andhra	0.38	1.03**	-0.01	0.19	-0.14	0.32	1.01**	-0.00	0.20	-0.15
	(0.66)	(0.42)	(0.03)	(0.12)	(0.12)	(0.66)	(0.42)	(0.01)	(0.12)	(0.12)

Rayalaseema	-0.03	0.13	0.02	-0.01	-0.32***	-0.01	0.13	0.02	-0.01	-0.33***
	(0.45)	(0.28)	(0.02)	(0.08)	(0.09)	(0.45)	(0.28)	(0.02)	(0.08)	(0.09)
Rural	0.49	-0.24	0.02	-0.04	-0.01	0.49	-0.25	0.02	-0.03	-0.01
	(0.47)	(0.31)	(0.02)	(0.09)	(0.12)	(0.47)	(0.32)	(0.02)	(0.09)	(0.12)
Constant	-2.92***	-2.94***				-2.06**	-2.25***			
	(1.05)	(0.81)				(1.02)	(0.79)			
Observations	108	113	124	118	118	108	113	124	118	118
R-squared	0.20	0.26				0.21	0.26			

Notes: For results in col. (1) - (5) reference category for some stunting and persistent stunting is never stunted. Regressions (1), (2), (6) and (7) are OLS estimations while the rest are probit estimations with marginal effects reported. . Standard errors adjusted for clusters in same mother. *** p<0.01, ** p<0.05, * p<0.1

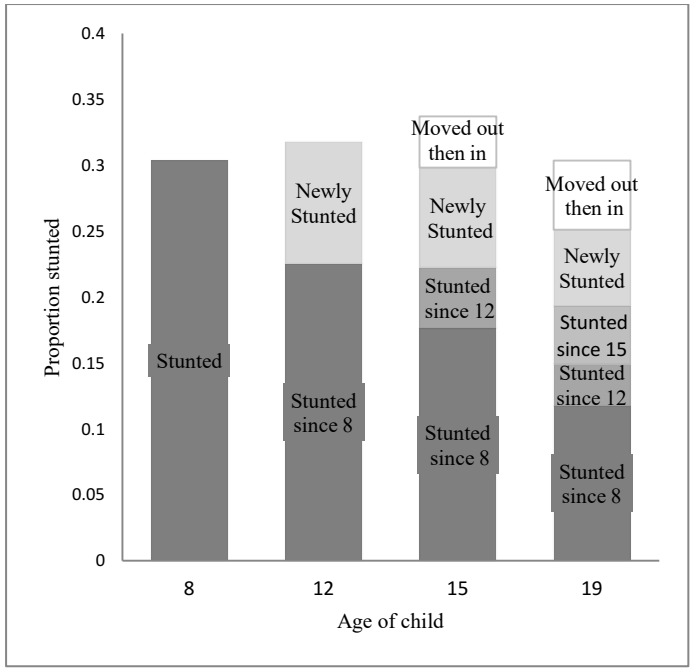

Figure 1: Stunting though middle childhood and adolescence

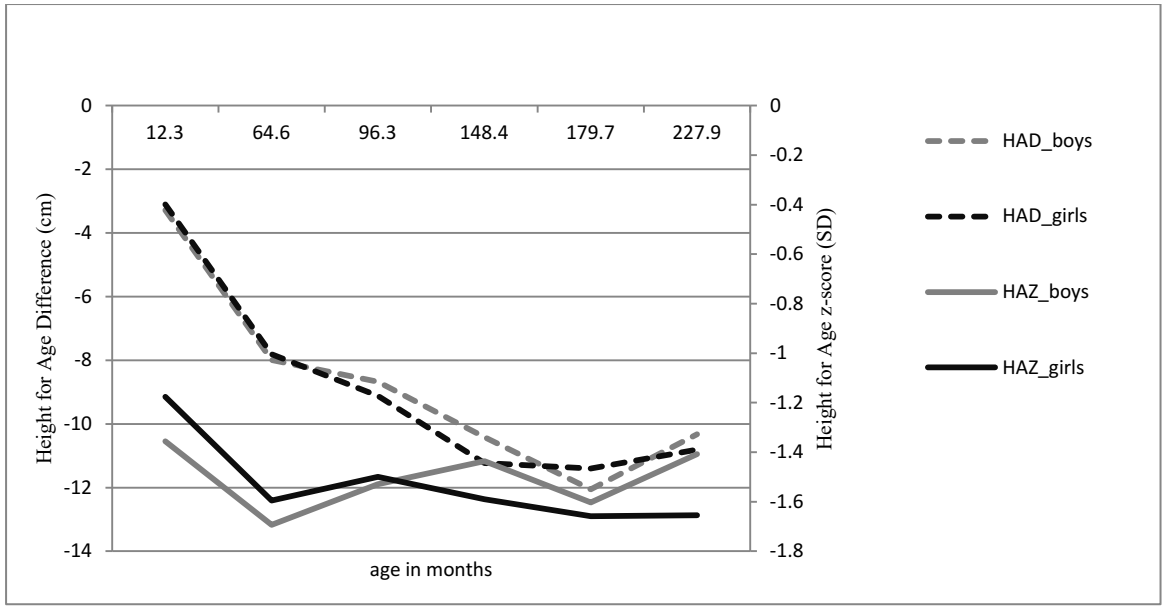

Figure 2: Mean height-for-age z-scores (HAZ) and height-for-age difference (HAD) at ages 1, 5, 8, 12, 15 and 19 years for boys and girls separately

Note: Data points for ages 1 and 5 are based on the Young Lives younger cohort round 1 and round 2 data collected in 2002 and 2006, for 2000 children. The rest of the data points are based on the Older cohort data.

APPENDIX

Table A1 Summary statistics for selected variables for index children present in all 4 rounds of the survey

Variable	Obs.	Mean	Std. Dev.	Min	Max	Variable Description
Index Child Characteristics						
HAD 2002	948	-8.93	5.91	-26.21	11.75	HAD: Height for Age Difference calculated as the difference
HAD 2006	935	-10.83	7.4	-44.24	17.98	between the actual height of the child in centimetres and the
HAD 2009	949	-11.72	7.32	-34.79	16.2	expected height according to the WHO 2007 standard.
HAD 2013	937	-10.57	6.31	-32.78	8.85	
HAZ 2002	950	-1.56	1.03	-4.61	2	HAZ: Height for Age z-score calculated as HAD divided by the age,
HAZ 2006	934	-1.53	1.04	-4.5	2.41	sex based standard deviation for the WHO 2007 reference
HAZ 2009	950	-1.63	0.99	-4.84	2.05	population. Observations with -6<HAZ<6 are excluded.
HAZ 2013	937	-1.53	0.92	-4.97	1.2	
Sex	951	0.49	0.5	0	1	Sex of child =1 if male, 0 if female.
Birth order	951	2.32	1.4	1	13	Birth order of the index child compared to siblings born to the same mother who survived for more than 24 hours after birth.
Dietary diversity, 2009	951	4.73	1	0	7	Index can range from 0 (least diverse) to 8 (most diverse) counts the number of food items in the following groups consumed by the index in the last 24 hours: (i) grains, roots or tubers (ii) vitamin A rich vegetables, fruit and organ meat (iii) other fruits and vegetables; (iv) meat (non organ) and fish (v) eggs; (vi) pulses and legumes; (vii) milk and dairy products; (viii) food cooked in oil or fat. Method follows Swindale and Bilinsky (2006).
Household Characteristics						
Mother's education	946	2.8	3.94	0	14	Highest school-grade obtained excluding pre-primary
Father's education	947	4.64	4.79	0	14	Highest school- grade obtained excluding pre-primary
wealth index, 2002	951	0.41	0.21	0.01	0.9	Index ranges from 0 (least wealthy) to 1 (most wealthy)

						constructed using the simple average of 3 sub indices for housing quality (crowding and materials used to build roof, wall and floor), access to services(electricity, safe drinking water, sanitation and adequate fuel) and consumer durables (owns at least one asset out of 9 such as TV, bicycle). See Espinoza (2014), p.3-4 for further details.
Mother's Height	943	5.00	0.12	3.5	5.1	Maternal height to the nearest 0.1 cm in logs.
Household size, 2002	5.20	1.832	994	2	22	The total number of members in the households
Caste						
SC	951	0.21	0.41	0	1	The caste a child belongs to: Scheduled caste (SC), Backward
ST	951	0.10	0.30	0	1	caste(BC), Scheduled Tribe (ST) or Other (other castes as well as
BC	951	0.48	0.49	0	1	not belonging to a caste, as is common with several non-Hindu
Other	951	0.21	0.41	0	1	households).

Table A2: Determinants of moving out and in to being stunted in adolescence, persistent stunting, and HAZ in round 4 (Parsimonious model)

	Multinomial Logit			OLS
Dependent variable	Moved out of being stunted in adolescence	Moved into being stunted in adolescence	Persistently Stunted	HAZ in round 4
	(1)	(2)	(3)	(4)
Gender (=1	0.27	-0.53**	-0.14	0.21***
If male)	(0.18)	(0.21)	(0.18)	(0.05)
dietary diversity	-0.08	-0.19*	0.07	0.07**
	(0.10)	(0.11)	(0.12)	(0.03)
Mother's	-0.03	-0.02	-0.10**	0.03***
education	(0.03)	(0.03)	(0.04)	(0.01)
Wealth index, 2006	-1.66**	-1.93***	-0.67	0.52**
	(0.66)	(0.68)	(0.77)	(0.21)
Scheduled Caste	-0.71**	0.78*	1.59***	-0.54***
	(0.31)	(0.42)	(0.53)	(0.11)
Scheduled Tribe	0.28	0.84*	1.67***	-0.52***
	(0.38)	(0.49)	(0.63)	(0.15)
Backward Caste	0.22	0.59*	1.27**	-0.30***
	(0.24)	(0.34)	(0.51)	(0.07)
Constant	0.03	-1.07	-2.96***	-1.86***
	(0.87)	(0.91)	(0.92)	(0.23)
Observations	895	895	895	932
R-squared	-	-	-	0.16
Wald Test	Wald $\chi^2\,(39) = 230.6\ Prob > \chi^2\ = 0.00.$			

Note: Regressions include household size, type (urban/rural) and region of residence, as well as two dummies indicating if the community the child lived in round 2 had access to good electricity and good water. Robust standard errors in parentheses, adjusted for clustering at community level,*** p<0.01, ** p<0.05, * p<0.1

Table A3: Young Lives Round 4 questions used to build psychosocial outcome indices.

Index Name	Round 4 survey questions used
Self Esteem, compatible with questions in previous rounds of data Scale:1-5	I am proud of my clothes; I am proud of the work I have to do; I feel my clothing is right for all occasions; I am proud of my shoes or of having shoes
Agency, compatible with questions in previous rounds of data Scale:1-5	Other people in my family make all the decisions about how I spend my time; I have no choice about the type of work I do- I must do this sort of work; If I try hard I can improve my situation in life; I like to make plans for my future studies and work; If I study hard at school I will be rewarded by a better job in the future
Self Efficacy Scale:1-4	If someone opposes me, I can find the means and ways to get what I want; When I am confronted with a problem I can usually find several solutions; If I am in trouble, I can usually think of a solution; I am confident that I can deal efficiently with unexpected events; I can always manage to solve difficult problems if I try hard enough; It is easy for me to stick to my aims and accomplish my goals; I can remain calm when facing difficulties because I can rely on my own coping abilities; I can usually handle whatever comes my way; Thanks to my resourcefulness I can handle unforeseen situations; I can solve most problems if I invest the necessary effort
Peer Relations Scale:1-4	I make friends easily; I am popular with kids of my own age; Most other kids like me; Other kids want me to be their friend; I have more friends than other kids; I have lots of friends; I am easy to like; I get along with other kids easily
Parent Relations	I like my parents; My parents like me; My parents and I spend a lot of time together; I get along

Scale:1-4	well with my parents; My parents understand me; If I have children of my own, I want to bring them up like my parents raised me; My parents are easy to talk to; My parents and I have a lot of fun together
General Scale 1-4	I am as good as most other people; Overall I have a lot to be proud of; I can do things as well as most other people; Other people think I am a good person; I do lots of important things, In general I like being the way I am; A lot of things about me are good; When I do something I do it well.

The procedure adopted to compute the indices is to first recode all relevant questions are recoded to be positive outcomes, second, normalise all responses to z-scores (subtract mean and divide by SD) and third, take an average of the relevant z-scores across the questions that have no missing values. Questions follow Likert type scales ranging from 1 to 4 or 5.

Table A4: Young Lives Round 3 questions used to build indices to account for being bullied or teased.

Index	Round 3 survey questions used
Verbal Bullying	Called you names or sworn at you; Made fun of you for some reason,
Physical Bullying	Punched, kicked or beaten you up; Hurt you physically in any other way,
Other types of Bullying	Tried to get you in trouble with friends; Made you uncomfortable by staring at you for a long time; Refused to talk to you or made other people not talk to you; Tried to break or damage something of yours; Took something without your permission or stole from you.

The relevant survey questions are based on the standardized Social and Health Assessment Peer Victimisation scale (Ruchkin, Schwab-Stone and Vermeiren 2004). Children were asked to if they had experienced the 9 items of bullying above with responses coded as never, 1 time, 2-3 times, 4 or more. For each child, the procedure adopted to compute the indices was to first construct a dummy variable that equalled 1 for each of the survey questions above if answers are '2-3 times' or '4 or more times'. Second, if at least one of the question relating to the Index to be computed equals 1, then the corresponding index is assigned 1 (and 0 otherwise).

Table A5: Robustness check for results reported in Table 4, with additional controls.

Dependent variable	Agency	Self esteem	Self efficacy	Peer relationship	Parent relationship	General
Extended Specification A, selected controls						
Stunting profile (reference group: never stunted)						
Moved in adolescence	-0.07	-0.07	-0.13	-0.25***	-0.06	-0.03
	(0.06)	(0.10)	(0.08)	(0.08)	(0.07)	(0.06)
Moved in mid ch.hood	-0.05	-0.06	-0.03	-0.06	0.01	-0.01
	(0.08)	(0.10)	(0.10)	(0.08)	(0.09)	(0.07)
Persistently stunted	-0.08	-0.07	0.06	0.06	0.08	0.04
	(0.06)	(0.08)	(0.06)	(0.07)	(0.08)	(0.06)
Moved out mid ch.hood /adolsc.	0.04	-0.04	-0.02	-0.02	0.03	0.01
	(0.04)	(0.06)	(0.05)	(0.05)	(0.07)	(0.04)
Married	-0.39***	0.09	-0.09	-0.20***	-0.24***	0.03
	(0.06)	(0.08)	(0.07)	(0.07)	(0.08)	(0.05)
Extended Specification B, selected controls						
Stunting profile (reference group: never stunted)						
Moved in adolescence	-0.05	-0.07	-0.11	-0.24***	-0.05	0.04
	(0.07)	(0.11)	(0.07)	(0.07)	(0.08)	(0.07)
Moved in mid	-0.00	-0.07	-0.03	-0.05	0.03	-0.02

ch.hood	(0.07)	(0.10)	(0.10)	(0.08)	(0.08)	(0.09)
Persistently stunted	-0.05	-0.07	0.07	0.07	0.10	0.02
	(0.06)	(0.08)	(0.06)	(0.07)	(0.08)	(0.07)
Moved out mid	0.05	-0.04	-0.02	-0.01	0.04	0.04
ch.hood/adolsc.	(0.04)	(0.06)	(0.05)	(0.05)	(0.07)	(0.05)
Enrolled at	0.41***	-0.01	0.17***	0.13**	0.18***	-0.01
School, 2013	(0.04)	(0.05)	(0.05)	(0.06)	(0.06)	(0.05)
R1_efficacy	-0.01		0.07*			
	(0.04)		(0.04)			
R1_liked				0.02		
				(0.05)		
R1_not bullied				-0.03		
				(0.04)		
R1_nofears						0.10**
						(0.05)
Constant	-0.95***	-1.13***	-0.37*	-0.11	-0.09	-0.51*
	(0.20)	(0.26)	(0.19)	(0.20)	(0.28)	(0.28)
Observations	931	931	930	931	931	931
R-squared	0.35	0.22	0.28	0.20	0.21	0.18

In addition to reported variables, both extended specifications include all controls used in baseline specification. The signs and significance of these unreported variables are the same as for the baseline specification. Specification B also includes wealth 2013, two dummy variables controlling for household level shocks experienced during adolescence and BMI for age 2013. All regressions controlled for community fixed effects. Robust standard errors in parentheses adjusted for clustering *** p<0.01, ** p<0.05, * p<0.1.

Table A6: Comparing characteristics between girls who already have offspring by age 19 with girls those who do not.

	Has offspring by age 19 (1)	No offspring (2)	t-Ratio (p-value)testing for equality in means in (1) and (2)
Age	19.05	18.98	-1.85 (0.06)*
Menarche by age 12 (round 2)?	0.44	0.24	-4.15 (0.00)***
HAZ at age 8	-1.28	-1.57	-2.68(0.00)
HAZ at age 12	-1.44	-1.66	-1.72 (0.08)*
HAZ at age 19	-1.73	-1.64	0.80 (0.42)
Some stunting	0.38	0.45	0.67(0.50)
Persistent stunting	0.10	0.05	-0.79(0.43)
Never stunted	0.52	0.50	-0.21(0.83)
Wealth index, age 8	0.35	0.41	2.66(0.00)***
Wealth index, age 12	0.40	0.47	3.22(0.00)***
Wealth index, age 15	0.47	0.52	2.87(0.00)***
Wealth index, age 19	0.57	0.60	1.87(0.06)*
Caste			
Scheduled Caste	0.23	0.19	0.97 (0.33)
Backward Caste	0.52	0.46	-1.31 (0.25)
Scheduled Tribe	0.11	0.11	0.02(0.98)
Rural residence	0.76	0.63	-2.40(0.00)***
Highest educational qualification[1]	1.9	3.10	8.3(0.00)***
Number of observations	101	407	

[1] Highest qualification: 0=none, 1=primary (class 5), 2=upper primary (class 7), 3=matriculation certificate (class 10), 4=Senior Secondary School certificate, 5= ITI certificate, 6=Diploma in technical education

[i] http://www.who.int/childgrowth/standards/height_for_age/en/. See also Onis et.al (2007) for methods and detail pertaining to the WHO 2007 standard. The young Lives Round 4 sample has children between ages 18.5 years and 19.5 years. In order to include the children over 19 years of age in the sample and thus avoid loss of data, the ages of those over 19 are rounded down to 19, under the assumption that 'growth virtually ceases because of epiphyseal fusion, typically at a skeletal age of 15 years in girls and 17 years in boys' (Rogol et.al 2000:524, Tanner 1989).

[ii] See Huttly, S., Jones, N. (2014), Boyden (2014a), Boyden (2014b), Boyden(2016), Boyden et. al. (2016).

[iii] See Hill (2004) or Alderman et. al (2001) for some examples of developing country longitudinal datasets and their attrition rates that can be as high as 50 per cent in some cases.

[iv] Ages 13-19 has been grouped as adolescence for the sake of clarity instead of breaking up the group as 13-15 and 15-19 as the latter provides too much information that is better presented in a format other than a table. In any case, given the volatility associated with growth during puberty and adolescence the differentiation here between early and later adolescence is not necessarily meaningful.

[v] India's caste system is a form of social stratification that is around 3000 years old. It divides Hindus into rigid hierarchical groups based on their *karma* and *dharma,* which mean work and duty, respectively. At the top of

the hierarchy are the teachers and intellectuals (Brahmins), followed by warriors and rulers (Kshatriyas), traders and money lenders (Vaishyas) and at the bottom those who did menial jobs (Shudras). The main castes are further divided into around 25000 occupation-specific sub castes. Outside the traditional castes were the Dalits or untouchables (scheduled caste), undertaking the lowest of the menial jobs. The caste system dictated almost every aspect of Hindu religious and social life for many centuries, favouring upper castes over lower castes. The Indian constitution banned caste-based discrimination in 1947 while wider access to education and social mobility have reduced somewhat the influence of castes. However, significant caste-based differences still remain in various spheres (Deshapande 2000, Borooah 2005).

[vi] Aurino (2016) using the same dataset as this paper (but without the fourth round when children were 19) argues that a significant pro-male bias emerges in dietary quality with 15 year old girls much less likely to consume foods that contain most of the protein necessary for healthy development such as eggs, legumes and meat as well as root vegetables and fruit. An analysis of round 4 dietary patterns at age 19 indicate that girls continue to consume significantly less vegetables, greens and legumes than do boys. As dietary diversity between boys and girls is statistically significantly different only in rounds 3 and 4 (but not 2) the baseline specification uses the round 3 dietary diversity index. In an alternative specification we include the round 2 index as a control variable to find that it is not significant. Note that the index does not account for *quantities* of the various nutrient groups consumed, but counts the range of food types consumed.

[vii] Real household monthly spending per capita on both food and non-food items with regional variations in prices accounted for, as calculated in Espinoza (2014)..

[viii] The puzzle as to why cross section data may indicate a positive correlation between calorie consumption and income but why there is a negative correlation when looking at data over time in India is discussed more broadly in Deaton and Dreze (2009).

[ix] I also conduct a Wald test to see if the dependent categories we identified as (1) never stunted, (2) moved out, (3)moved in and (4)persistent can be combined to provide more efficient estimates. The tests find that none of the pairs of categories are indistinguishable with respect to variables in the models at the 1 per cent level. Thus there is little statistical support to combine any of the dependent categories. Hausman tests of

Independence of Irrelevant Alternatives (IIA) assumption, a stringent assumption of the model, indicate no significant evidence against Ho: Odds(Outcome-J vs Outcome-K) are independent of other alternatives).

[x] Diagnostic tests done on the full model versus nested model, based on Long and Freese (2014) indicate that the Difference in Bayesian Information Criterion (BIC) between the models provides support for the more parsimonious model. The BIC test tends to favor more parsimonious models when sample size gets larger. However, an alternative criteria such as the difference in chi-squared statistics between parsimonious model and full model or Akaike Information Criterion, favor the fuller model.

[xi] See Dercon and Krishnan (2009) for more details on the development of the agency and self esteem measures using Young Lives data. For the other measures as well as a discussion about validity, issues faced in piloting these instruments and caveats see Yorke and Orgando-Portela (forthcoming).

[xii] To check the robustness of this claim we run an alternative specification of (2) excluding the stunting profile dummies but including instead HAZ at age 8. The variable is not significant in any of the regressions (unreported).

[xiii] An issue with the result from this extended robustness check is that the specification may suffer from reverse causality as current BMI and household wealth could affect psychosocial outcomes and vice versa. Thus the results indicate correlation rather than causality. Reverse causality is less of an issue in the baseline specification as lagged explanatory variables are used.