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Parental Education and Child Health – Understanding the Pathways of Impact in Pakistan

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This study investigates the relationship between parental schooling on the one hand, and child health outcomes (height and weight) and parental health-seeking behaviour (immunisation status of children), on the other. While establishing a correlational link between parental schooling and child health is relatively straightforward, confirming a causal relationship is more complex. Using unique data from Pakistan, we aim to understand the mechanisms through which parental schooling promotes better child health and health-seeking behaviour. The following 'pathways' are investigated: educated parents' greater household income, exposure to media, literacy, labour market participation, health knowledge and the extent of maternal empowerment within the home. We find that while father's education is positively associated with the 'one-off' immunisation decision, mother's education is more critically associated with longer term health outcomes in OLS equations. Instrumental variable (IV) estimates suggest that father's health knowledge is most positively associated with immunisation decisions while mother's health knowledge and her empowerment within the home are the channels through which her education impacts her child's height and weight respectively.

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Introduction

While the significance of establishing good health during infancy and childhood is evident from the documented link between childhood health and later economic and life outcomes such as education, learning, health and earnings (Grossman 2005; Currie and Madrian 1999; Alderman, Behrman, Levy and Menon, 2001; Case, Fertig and Paxson 2003; Oreopoulous et al. 2006) there is a curious absence of evidence for Pakistan. This is surprising because Pakistan ranks very poorly in terms of child health indicatorswith 38 per cent and 42 per cent children aged less than 5 being under the requisite weight and height-for-age (UNDP, 2007-08)[1]. A factor that holds promise for improving child health levels is parental education. Thus, it is useful to understand the relation between parental education with child health status in Pakistan. This is the key objective of the paper. Firstly, we seek to document the association between parental education and child health in Pakistan Secondly, and more interestingly, we attempt to identify the 'causal' impact of parental education (if any) on child health. In doing the latter we probe the pathways and mechanisms through which parental schooling impacts child health.

The importance of parental education in the production of child health is well-established (Behrman and Deolalikar, 1988; Strauss and Thomas, 1995). Indeed, it has even been argued that education has contributed more to mortality decline than the provision of health services (Mosley, 1985 cited in Sandiford, Cassel, Montenegro and Sanchez, 1995). The association of parental education with child health may arise because educated parents are more efficient 'producers' of child health ('productive efficiency') through adopting better child-care practices or superior hygiene standards. Alternatively, it may be because they choose health input mixes that generate more health output ('allocative efficiency') than selected by less-educated parents. This may be because education instils greater knowledge of the health production function or the ability to respond to new knowledge more rapidly (Grossman, 2005, pp. 12-13).

Since Caldwell's (1979) seminal work it has been generally maintained that mother's education is the more critical determinant of child health. This is consistent with a division of labour within the household in which child-care is the larger responsibility of the mother (Grossman, 2005). Indeed, studies in several developing countries demonstrate that there is no 'threshold' level of maternal education that needs to be reached before the benefits of maternal education on child health materialise and even small levels of education improve child survival (Hobcraft, McDonald and Rutstein, 1984; Mensch, Lentzner and Preston, 1985). While a major body of evidence confirms the larger association of mother's than father's education with child health, some recent studies find otherwise. Breievrova and Duflo (2002) find that mother's and

[1] Between 1996-2005.

father's education is equally important in reducing child mortality in Indonesia. In Bangladesh, father's education is found to be a more consistent determinant of childhood stunting than maternal education (Semba, de Pee, Sun, Sari, Akhter and Bloem, 2008). This finding corroborates past evidence from Bangladesh and the Philippines (Rahman and Chowdhury 2006; Ricci and Becker 1996). Fewer studies have focused on the role of father's education in determining health largely because fathers play a less obvious role in care-giving to children. However, as Chen and Li (2009) note, father's education may be important because fathers are often more educated than mothers in developing countries. In Pakistan, for instance, the average father in our sample has 3 more years of education than the average mother and if the highest level of education matters in a household, father's education may be an important determinant of child health. Another explanation for the role of father's education rests on low social status and empowerment of mothers that potentially limits the influence they have in decision-making regarding child health (Semba *et al.,* 2008). Alternatively, it may be that fathers play a more active role in certain *kinds* of health decisions such as 'one-off' immunisation decisions particularly if they require travel to a health clinic. Mothers, on the other hand, may be involved in the day-to-day decisions on general hygiene and nutritional intake of a child. If this hypothesis is true, one would expect father's education to have a greater association with 'one-off' health seeking behaviour and mother's education to impact more on longer-term measures of health such as height and weight. Regardless of the reason, further insight is needed into the role of parent's education in children's health as formal education may be critical in breaking the intergenerational cycle of poor health (Semba *et al.,* 2008).

While the positive association between parental schooling and child health is largely undisputed, the mechanisms through which this relationship works are not as well understood and therefore a causal relationship is harder to justify[2]. The problem is largely methodological and linked to difficulties in the estimation of child health production functions. This is because the underlying structural equation relates health outputs to endogenous inputs. For example, while higher parental schooling is expected to have a positive effect on child health outcomes, parental schooling is endogenous if unobserved characteristics of the parents (such as tastes, values and preferences) are correlated with both parental education and the child's health status.

Parental education in child health functions may therefore be proxying for different factors (at the level of the individual, household or even the community in which the child resides). For example, sceptics wonder whether the association between parental schooling and child health

[2] See Hobcraft 1993 for a summary of evidence up-till the early 1990s.

merely picks up differences in socioeconomic status of households. It is well known that credit constraints in developing countries are a major factor hindering access to health services and potentially translating into inferior child nutrition and health. The evidence from past studies explicitly controlling for household socioeconomic status is somewhat mixed. For instance, Alderman and Garcia's (1994) study (the only quality study on child health outcomes in Pakistan we are aware of) discovers significant positive effects of maternal education on children's heights and weights even after controlling for income. Likewise, a study by Thomas, Strauss and Henrique (1990) confirms both parents' education to have large, independent and significant positive associations with child height in Brazil. The effect of maternal education in their study doesn't operate through income augmenting effects. Similar findings are reported by Glewwe (1999) in Morocco. However, a study by Desai and Alva (1998) on a sample of 22 developing countries finds to the contrary – that mother's education proxies for a household's socioeconomic status and the family's area of residence.

Some critics maintain that mother's education encapsulates unobserved maternal characteristics (such as the values or beliefs they inherited from their own families when they were young) that may in turn be correlated with the health and nutritional status of their children. In this case, a positive coefficient on mother's schooling could be fully or partially 'picking up' the effect of the intergenerational transfer of values rather than a causal impact of maternal schooling. Behrman and Wolfe (1987) are the strongest proponents of this critique and use data from Nicaragua to test their concern. Their findings suggest that when measures of 'maternal childhood endowments' are excluded, mother's schooling has strong positive effects on child health and nutrition but that inclusion of maternal endowments causes the effect of maternal schooling to disappear suggesting that, at least in their sample, it is picking up the effect of intergenerational transfer of values and 'cultural capital'. Handa (1999) also finds that using household fixed-effects in Jamaica causes the positive association between maternal schooling and child height to disappear. Conversely, Strauss (1990) finds that mother's schooling has a positive effect on child weight and height in the Cote d' Ivoire even after using family fixed-effects estimators.

Unsurprisingly, the literature on the relationship between maternal schooling and child health has moved towards underpinning the 'pathways' through which mother's education translates into improved child health. While a majority of the evidence hasn't directly controlled for the endogeneity of maternal schooling, introducing different 'pathways' is one way of isolating the 'true' impact of maternal education from the effect of confounding factors.

One such pathway that has received little attention (largely because of unavailability of data) is the impact of mother's education on mother's empowerment[3]. The only two studies we are aware of that use mother's empowerment as a pathway are by Strauss (1990) in the Cote d' Ivoire and Handa (1999) in Jamaica[4]. Both studies find some evidence to suggest that maternal education has a direct effect on child height but also find that maternal education does not reflect maternal bargaining power (or empowerment) within the household.

Another channel through which maternal education may act on child health is via increasing the probability of maternal labour force participation. This relationship is complex because on the one hand a child may suffer through lack of attention (in the case of infants this may mean they forgo the benefits of breast feeding, for example) while on the other hand, participating in the labour force may augment family income and lead mothers to gain external information on healthy practices enhancing their propensity to use preventive and curative medicines and treat childhood illnesses. The evidence, Tulasidhar (1993) argues, reflects this conflict. A majority of the studies cited in Dwyer and Bruce (1988), however, indicate an inverse relationship between maternal labour force participation and child health. Tulasidhar (1993) in his study in India notes that female labour force participation has a significant inverse relationship with excess female child mortality but that the direct effect of mother's education on reducing excess female child mortality is stronger than her labour force participation.

Several studies have attempted to identify more direct pathways through which maternal education may translate into improved child health. A study by Thomas, Strauss and Henriques (1990) in Brazil analyses the role of income, mother's literacy and information processing and the interaction of maternal schooling with community services. The authors find that almost all the impact of maternal schooling on child height can be explained through mother's access to information (i.e. exposure to media). In a more recent study in Morocco, Glewwe (1999) identifies three channels: 1) direct acquisition of basic health knowledge in school, 2) literacy and numeracy skills learned in school and 3) exposure to modern society. The study finds that mother's health knowledge alone impacts child health outcomes. A study by Handa (1999) in Jamaica also investigates several mechanisms including income effects, interaction of maternal

[3] Cleland (1990) identifies three components of this empowerment: 1) instrumentality (ability to feel control over the outside world), 2) social identification (engaging with modern institutions) and 3) confidence (cited in Hobcraft, 1993, pp. 161).

[4]Strauss uses whether individual is child of a senior or junior wife as a measure of empowerment while Handa uses a dummy variable measuring whether child's mother actually resides in the household and conditional on living in the household whether she is the household head.

schooling with household characteristics and community services, information processing, unobserved heterogeneity and maternal bargaining power. The evidence suggests that maternal education is correlated with unobserved heterogeneity and that maternal empowerment has positive implications for child health within households. Alderman and Christiansen (2004) in Ethiopia also find that maternal nutrition knowledge is an important determinant of child height. Another recent study by Block (2007) uses data from Indonesia to investigate the impact of maternal nutrition knowledge and schooling on child micronutrient intake and finds that the effects of maternal education are partially mediated through nutrition knowledge and household expenditure[5].

A major factor contributing to limited research in Pakistan is the lack of quality data with the indicators needed for investigating the aforementioned issues. The availability of rich recent data from Pakistan allows us to overcome this impasse in the literature. The data come from a unique purpose-designed survey of more than 1000 households. The data were collected in 2006-2007 from nine districts in Punjab and the-then North West Frontier Province (NWFP) of Pakistan (now known as Khyber-Pakhtunkhwah, KP). As well as containing standard information needed for the estimation of child health functions (anthropometric information such as height and weight, child age and gender and maternal and paternal education), the data also uniquely include measures of adult cognitive skills (scores on tests of literacy and numeracy), health knowledge scores, information on labour force participation, exposure to media and measures of female empowerment within households. Importantly, the availability of child immunisation scores also allows us to assess the impact of parental education and the proposed pathways on parental health-seeking behaviour and in doing so differentiate between any potentially important differences between 'one-off' and longer-term health decisions We use a sample of children aged 0-5 in urban and rural Punjab and the KP and estimate child health functions (discussed later).

There are some striking findings. Baseline estimates reveal that only mother's education is positively associated with children's height and weight while father's education matters only for health-seeking behaviour measured through immunisation status of the child. The introduction of several 'pathways' through which father's education may translate into greater health-seeking behaviour causes the direct effect of father's education to disappear and only father's health knowledge remains significant. In child height and weight equations, the direct effect of mother's education disappears when mother's 'pathways' are introduced. Mother's

[5] Another pathway sometimes studied in the literature is the role of education in determining use of health infrastructure (Barrera, 1990 and Thomas, Strauss and Henriques, 1990).

exposure to media, maternal health knowledge and her participation in the labour market appear to be the key channels through which her education impacts her child's height while mother's empowerment within the household matters for child weight. However, all these 'pathways' are potentially endogenous and only estimates explicitly controlling for the endogeneity of these variables are credible. Instrumental Variable (IV) estimates find that father's health knowledge is key in determining immunisation status while mother's health knowledge and her empowerment within the home have large positive effects on children's health and weight outcomes.

The paper is organised as follows. Section 2 describes the empirical methodology used. Section 3 discusses the data and some key descriptive statistics. Section 4 presents the empirical findings and Section 5 concludes.

1. Estimation Methodology

The underlying model of child health is derived from the standard paradigm of parental utility maximisation. This yields reduced form health functions[6] of the following form:

$$H_i = f(x_i, x_h, x_c, \varepsilon_i) \qquad (1)$$

where H_i is the health outcome of child i, x_i is a vector of child characteristics (such as age and gender) and parental characteristics such as mother's education and father's education, x_h is a vector of household-level characteristics such household size, x_c is a vector of community characteristics such as access to/quality of health services and ε_i is a composite error term of unobserved child, household and community-level heterogeneity.

One of the problems in estimating equation (1) is that to call it a reduced form function assumes that health inputs (including parental schooling) are exogenous. This can be a strong assumption if unobserved parental/household characteristics correlated with parental schooling (such as greater motivation or ability or certain values or traits) also influence child health directly – standard endogeneity through 'omitted variable bias'. If this is the case, then a positive coefficient on say maternal schooling in the health function may reflect the cross-section correlation between unobserved maternal traits on the one hand and both maternal

[6] Estimating the child health production function (rather than the reduced form) requires detailed information on prices and the quality of health services provision to deal with the endogeneity of health inputs. In the absence of such price data most studies include information on distance to health services or travel time variables as crude measures of the cost of services and hence prices. An alternative is to introduce community fixed effects.

schooling and child health on the other, rather than representing a causal effect of maternal schooling on the health outcome being measured.

Much of the past literature estimating the impact of parental schooling on child health has ignored the endogeneity of this variable (see for instance Thomas, Strauss and Henrique, 1990, Barrera, 1990, Alderman and Garcia, 1994, Desai and Alva, 1998, Christiansen and Alderman, 2004, and Block, 2007). One approach to addressing endogeneity is Instrumental Variables (IV). This methodology identifies variables (instruments, W_i) that are correlated with the endogenous variable (say mother's education) and uncorrelated with the unobservables (such as maternal values, motivation, ability etc.) relegated to ε_i. Glewwe (1999) recognises the potential endogeneity of maternal schooling and uses IV techniques to identify the causal impact of maternal education on child health outcomes. The set of instruments used include: education level of both the mother's parents as well as the number of married sisters she has. Glewwe reports (pp. 137) that these instruments are good predictors of mother's schooling and that the impact of mother's schooling on child health using IV was substantially lower and not significantly different from zero..

While it is possible to quibble with the set of instruments used by Glewwe (1999), finding truly exogenous sources of variation in maternal schooling is challenging and often impossible. Ideally, one needs natural experiments or quasi-experimental data similar in vein to those used in treating the endogeneity of schooling in earnings functions (summarised in Card, 2001). The paucity of such data in developing countries limits the extent to which the more credible approaches can be employed..

In the absence of data that allow identification of the truly exogenous impact of maternal schooling (if any), an alternative is to introduce 'controls' in child health functions that proxy for the unobservables (such as parental ability or motivation). This is the approach adopted in this study. One can obtain a better understanding of the 'true' impact of parental schooling by replacing equation (1) with the following:

$$H_i = f(x_i, x_h, x_c, \text{CONTROLS}_i, \varepsilon) \qquad (2)$$

where CONTROLS_i is a vector of control variables proxying for unobserved variables correlated with parent's schooling and H_i. The vector CONTROLS_i here includes (though it is not restricted to) variables that represent the 'pathways' through which parental education impacts child health. For instance, whether the mother is a labour force participant, her family's per capita income, whether she has exposure to the media, her extent of autonomy within the

household – these are all likely to proxy for the mother's unobserved traits such as the independence, attitudes, values, preferences etc.. These variables also constitute the pathways through which mother's schooling may influence child health. By including 'pathways' that are likely correlated with parents' schooling and also proxy for 'unobservables' in the error term we are likely to reduce the bias in the coefficient on parental schooling. The vector $CONTROL_i$ = [$LNPCE_i$, MTV_i, $MSLIT_i$, $MLFP_i$, MHK_i, $MEMP_i$] where LNPCE is the log of household per-capita expenditure, MTV is mother's exposure to media, MSLIT is mother's literacy score, MLFP is labour market participation, MHK is health knowledge and MEMP is a measure of mother's empowerment within the household (see Table 1 for detailed description of variables). A more restricted vector of control variables hypothesizing father's pathways includes LNPCE, FTV, FSLIT and FHK (where LNPCE is as before, FTV is father's exposure to media, FSLIT is father's literacy and FHK is father's health knowledge)[7].

The 'pathways' identified above, however, are themselves potentially endogenous. For instance, household per capita expenditure should be treated as endogenous in child health functions since time, leisure and consumption are all jointly determined with child health. Parental health knowledge is clearly endogenous because childhood illnesses cause parents to acquire more knowledge. Thus, health knowledge is expected to be negatively correlated with children's initial health endowments as parents with inherently healthier children may not need to acquire as much health knowledge as those with more sickly offspring. Equally, parents with more 'health-producing values' may have healthier children and may also actively acquire more health knowledge. Because 'values' are unobserved, this generates a bias in the health knowledge variable. Using analogous logic, mother's 'empowerment' measure may also be similarly endogenous. Literacy scores may be endogenous as actions to acquire more health knowledge to treat sick children may lead to polishing of any existing literacy skills (reading labels on medicine bottles or leaflets about how to treat childhood illnesses for instance) and so on (Glewwe 1999, pp???). Literacy scores may also be endogenous if mother's inherent health endowments lead them to be more literate and mother's with greater health genetically pass on this health benefit to their children. In this scenario, mother's health endowment would be unobserved and correlated with mother's literacy and with child health. However, we are not particularly concerned about this potential source of endogeneity because our data allows us to include mother's height as a proxy for mother's health endowment.

[7] Father's labour force participation rate is not included in the controls vector as more than 95% father's actively participate in the labour market. Similarly, in Pakistan's highly patriarchal society, the issue of 'father's empowerment' is largely redundant.

By introducing the above controls in child health functions we are unable to give a causal interpretation to the 'pathways' themselves (unless their endogeneity is explicitly controlled for). Nevertheless, we may be somewhat closer in giving a causal interpretation to parental schooling if the 'pathways' proxy for unobservables often relegated to the error term. However, as mentioned in the introduction, one of the objectives of this study is to ascertain the (causal) 'pathways' through which parental education impacts child health. To do so, endogeneity of the relevant channels will be addressed using IVs (see Section 4 for details)[8].

Several other issues arise in the estimation of equation (2). Numerous extant studies note the importance of the health environment and community infrastructure on child anthropometry (see Barrera 1990, Strauss 1990, Strauss, Thomas and Henriques 1991 and Thomas and Strauss, 1992). The consensus from these studies is that the provision of a healthier environment to children yields substantial benefits through improved child health. While the RECOUP (2007) data used in this study collected in-depth community-level information on several 'environmental' indicators, information on key variables is missing for many communities. However, as the households were drawn from a sample of 27 communities, we are able to use a community fixed-effects procedure to control for community level unobservables which may otherwise be biasing the estimated impact of the included regressors. To some extent, this also controls for differences in the 'quality' of health services and infrastructure available to a child.

[8] Another alternative to both the IV technique and the 'proxy' methodology is to use observations from different individuals within the same family to estimate 'household fixed effects' health equations. The 'true' causal effect of say maternal education on child health can be identified if information is available on children of different mother's within a given household. This is not completely implausible in Pakistan where social norms dictate large 'extended' family households where several members of the extended family live together. The idea behind the household fixed effects approach rests on the belief that to the extent that unobserved traits are shared within the family, their effect will be netted out in a family differenced model. If the sources of heterogeneity are at the level of the household – such as food preparation methods, different levels of hygiene, knowledge on how to treat illnesses etc – household fixed-effects methods can control for these unobservables to some extent. While it is unlikely to be the case that unobserved traits are identical across family members (and especially across children's mothers who are most likely from different families) it is likely that they are much more similar within a family than across families and, as such, family fixed effects estimation reduces endogeneity bias without necessarily eliminating it entirely. Household fixed effects estimates were computed in this study based on sub-samples of children within households for whom different mothers could be identified. However, the results did not have any power in picking up the effect of maternal education and this could either be due to attenuation bias or because health seeking behaviour and health outcomes differ very little within households. The results were also very imprecise possibly due to very small sample sizes and are not reported (see Wolfe and Behrman, 1987, Strauss 1990 and Handa 1999 for studies using the fixed-effects methodology).

Finally, data on initial child health endowments is often not available even in the best of data sets. However, a strong positive correlation between parental heights and child health (often child height) has been empirically proven. Although part of this correlation can be attributed to genetics, some of it can also be seen to proxy for unobserved family background and we include measures of parental height to capture both genetics as well as the impact of unobserved family background on child health outcomes.

Anthropometric status is often used to determine the extent of malnourishment among children. The following measures are frequently used: stunting (or insufficient height-for-age), being underweight (or insufficient weight-for-age) and wasting (or having insufficient weight-for-height, indicating acute malnutrition). Since children are growing and their anthropometric measures depend on age and gender, heights and weights are standardised by age and sex. Standardisation is achieved by fitting a standard normal distribution to the growth curves of a healthy population of children using an age and gender specific distribution of heights/weights. In past literature, the z-score of the health measure is computed by subtracting the sample average (of the measure available from NCHS (National Center for Health Statistics) tables referring to a healthy population of children from the US) from the measure of the index child's health, and then dividing this difference by the standard deviation of the health outcome.Because the population of NCHS children is based on a sample of children of European ancestry from a single community in the United States, the choice of these older standards has sometimes been criticised (especially when used for comparisons in developing countries). In recent years, newer WHO growth standards have become available based on a sample of children from cities from the following developed and developing countries: Davis (California, USA), Muscat (Oman), Oslo (Norway), Pelotas (Brazil) and from selected affluent neighbourhoods of Accra (Ghana) and South Delhi (India). The WHO growth standards from this Multicentre Growth Reference Study (MGRS) from July 1997-December 2003 are used to standardise the heights and weights of children from the Pakistan sample[9]. In the absence of an internationally accepted Pakistani reference population, we believe the WHO growth reference provides the best population to standardise our sample against.

The z-score of any given measure is calculated by subtracting the sample average (in a given age-range and of a given gender) from the index child's health measure, and dividing the

[9] Onis and Yip (1996) suggest that the use of a common reference population has some advantages largely because the populations can then be compared locally and with other countries. They argue that it is not appropriate to compute a local reference as children from less developed areas may have poorer health (cited in Chen and Li, 2009).

difference by the standard deviation of the health outcome. A child with a z-score of zero is exactly at the mean in terms of the measure being used (such as height-for-age) while one with a negative z-score is below the mean (for instance shorter than average) and one with a positive z-score is above the mean (for instance taller than average) of the distribution. Stunting prevalence among children is then calculated as the percentage of children under 5 that fall below minus two standard deviations from the median/mean height-for-age of the standard WHO reference population. Similarly, underweight prevalence can be calculated as the percentage of children under 5 who fall below minus two standard deviations of the median/mean weight-for-age of the reference population

Among all the different measures of child nutrition and health status, height-for-age is used most often as it is perceived as a more long-term measure of chronic malnutrition over a child's lifetime and is unlikely to be affected by temporary shocks (unlike weight which can be quite severely affected by even short durations of morbidity and ill health). As an indicator of cumulative deficient growth, it is seen to be associated most with diet, hygiene, feeding practices and exposure to infection over an extended period of time. The weight of a child, on the other hand, is a composite measure of stunting and wasting and can be useful in describing overall malnutrition as well as changes over time. In this study, we compute z-scores for the conventional measures – height-for-age (henceforth HAZ) and weight-for-age (henceforth WAZ) in the way described above, to measure children's health outcomes. We also distinguish between child health outcomes (HAZ and WAZ) and 'parental health seeking behaviour' measured by child *i*'s immunisation score (henceforth IMMU).

The choice of covariates is guided by the conceptual framework adopted as well as the previous literature on the subject. The reduced form equations of child health outcomes and immunisation status include child age and gender. Children's initial health endowments are proxied by measures of parental heights[10]. The effect of parental schooling is captured through continuous variables measuring mother's and father's completed years of schooling. The effect of family size is captured through household size. Regional and provincial fixed effects in all regressions allow for any differences in rural-urban regions or between Punjab and NWFP to be captured. Finally, community fixed effects models are estimated which account for all village/ward level factors such as the quality of public health care and other amenities in the

[10] Father's height is missing for about 22 per cent of the sample of children aged 0-5 while mother's height is missing for only about 1 per cent of the sample. Rather than restrict the sample to only those children for whom data on both parents height is available, a dummy variable has been included to represent missing values in mother and fathers heights.

village. Moreover, we allow for several 'pathways' through which maternal and paternal education may impact child health. These 'controls' also proxy for unobserved values and traits of parents. These 'pathways' include household per capita expenditure, exposure to modern media (how frequently the parent reports viewing television), parent's score on a literacy test, and parent's health knowledge. In addition, we include whether the mother participates in the labour market and how empowered she is within the household. If the effect of parental education on child health outcomes or on parental health-seeking behaviour operates exclusively through any or either of these 'channels', including them in standard regression analysis should cause the 'direct' effect of parent's education to disappear (i.e. the coefficient on parent's education should collapse to zero). However, if despite including this impressive list of 'pathways', education continues to exert a direct influence on the dependent variables, one can argue that it potentially captures unmeasured and unobserved 'values' that either schooling instils in the parents or that were acquired through their own parents and have been transferred across generations (Behrman and Wolfe, 1987).

2. Data and Descriptive Statistics

The data for this study come from the first wave of a purpose-designed household survey administered to 1194 urban and rural households between November 2006 and March 2007. Households were selected randomly through stratified sampling from 9 districts in two provinces – Punjab and the North West Frontier Province (NWFP) - in Pakistan[11]. The data were collected under the auspices of the Research Consortium on Educational Outcomes and Poverty (RECOUP).

The survey gathered rich information on several individual, family and community-level variables. While the roster noted basic demographic, education and labour market status information on *all* resident household members in the sampled households (more than 8000 individuals), detailed individual-level questionnaires were administered only to those aged between 15 and 60 years. 4907 individual-level questionnaires were filled. These individuals were also administered tests of literacy, numeracy, health knowledge, English language and the Ravens Progressive Matrices test (to assess innate ability). The first three of these – literacy, numeracy and the health knowledge test – were translated into Urdu, the National language. The literacy and numeracy instruments were designed to capture 'basic order' skills and 'higher

[11] Rahimyar Khan, Khanewal, Sargodha, Kasur, Attock and Chakwal districts were chosen from Punjab while Swaat, Charsadda and Haripur were sampled from KP. Comparable data were collected in Ghana and India in 2006 and 2007-2008 respectively.

order' skills. For example, the first half of the literacy test consisted of a small passage followed by a few questions testing reading comprehension. Only if a person could answer three out of the total of five questions correctly in the short test was he/she administered the 'long literacy test' which tested more advanced reading and comprehension skills[12]. The numeracy test was also designed similarly. The 'health knowledge' test was composed of a total of 10 questions testing an individual's knowledge pertaining to basic health and hygiene issues. Enumerators asked the respondent a question (such as 'how does one get diarrhoea?') and waited for them to respond (say either: by eating contaminated food, by drinking dirty/contaminated water and/or by eating from dirty hands or dirty utensils). A score of one was given to each correctly-coded response and a zero for each missed response. The maximum score a person could achieve on the health knowledge test was 26 and the minimum a zero (see Appendix 1 to view the test).

Anthropometric information was collected on all available residents in a household. This was done by physically measuring each person's height (in centimetres) and weight (in kilograms). Moreover, for each household resident, an immunisation 'score' was computed by enumerators by giving a score of 1 (0) for each of the following diseases an individual was reported being (not being) immunised/treated against: Polio, Tuberculosis, Diphtheria, Whooping cough, Measles, Mumps, Rubella, Hepatitis or Goiter. The maximum score achievable was nine[13]. These rich variables are often missing from developing country datasets.

Among the 'empowerment' indicators, several variables were tested as potential candidates; these included: a woman's ability to visit the natal home[14] (including distance to natal home), role in spouse selection, whether the woman wears *dopatta* or covers her body completely and perceived role in decision-making about family size. None of these variables is a perfect

[12] In this study we use the short literacy test with the view that even very basic literacy skills should help parents make healthy choices for children. We experimented with including both the long literacy test and the total literacy score (short + long) but due to *a priori* reasoning decided to include short test scores for both parents in the equations.

[13] Ideally, this measure should have been computed by viewing an 'immunisation' card by enumerators. However, initial pilot-tests revealed that many people didn't keep records of cards for the younger children while the mothers were able to reveal with some confidence whether a child had been immunised against a certain illness or not. Moreover, since this 'score' was computed for *all* resident persons in a household, it would have been impossible to compute a score for adults who were more likely not to have kept records of any cards (if they existed at all to begin with).

[14] Jeffery and Jeffery (1988) argue that a woman's ability to visit the natal home is certainly a resource and can be viewed as a reasonably good measure of female empowerment.

measure of female empowerment. The parsimonious model is based on empowerment measured through a woman's perceived role in decision-making about family size[15].

Most studies restrict their analysis of child health outcomes to children aged 5 or less. This is often guided by paucity of data (most household datasets provide anthropometric measures only for children in this age range) or by the fact that WHO growth standards are often available only for children in this age group. We restrict our sample to children aged 0-5 primarily because younger children are more dependent on mothers both in terms of the choice as well as the use of health inputs, compared to older children.

The final sample of children aged 0-5 consists of about 1000 observations on whom complete information on all variables was available[16]. Table 1 describes the variables used and Table 2 reports means and standard deviations. Of particular interest are the 'pathways' variables. All the variables show substantial variation. In particular, literacy, numeracy and ability test scores vary reasonably, which is important in identifying their effect as pathways in child health functions.

Figures 1 and 2 show epanechnikov kernel density estimates of HAZ and WAZ for children aged 0-5 years. It is clear that the health status of Pakistani children is poor when compared to the reference population. The average z-score of height-for-age is -1.65 suggesting that Pakistani children are more than one and a half standard deviations shorter on average than healthy children from the rest of the world. The average weight-for-age z-score is -1.04 implying that Pakistani children weigh on average one standard deviation less than healthy children from the reference population. Moreover, about 46.7 per cent children in our sample show stunted growth (i.e. they are more than 2 standard deviations below the mean of the reference group) and 30.4 per cent of the sample are underweight (i.e. more than 2 standard deviations below the average weight of the reference group)[17].

Table 3 reports some descriptive statistics of the relationship between maternal and paternal education, child health outcomes and immunisation status and some key variables

[15] We gratefully acknowledge the contribution made by discussions with Roger Jeffery and Patricia Jeffery on appropriate measures of female empowerment.

[16] Depending on the variables of interest, the observations range from 903 to about 1073 children.

[17] The Human Development Report (HDR, 2008) reported roughly 38% children aged 0-5 to be underweight and 42% stunted. Our figures reveal a smaller incidence of underweight prevalence (30%) and a higher prevalence of stunting (47%). However, our estimates are based on calculations only from two provinces (Punjab and KP) and past figures reported in 'Earth Trends' www.wri.org show that the proportion of underweight children in Pakistan was greatest in Balochistan and Sindh in 1991, the two provinces not part of our sample.

(including the hypothesised 'pathways' in this study). Three categories of educational attainment are considered for both parents' schooling and are guided by the proportions reporting completing different education levels in the data set[18] – mother/father is uneducated (has 0 years of schooling); has between 1 and 5 years of schooling (inclusive); or has completed more than 5 years (primary) schooling. It is clear from Table 3 that higher schooling of both parents is associated with superior health-seeking behaviour (higher immunisation scores of children). However, while maternal education is unmistakably positively associated with improved child health outcomes (a lower incidence of both stunting and underweight prevalence), such a clear pattern does not emerge with respect to father's education. Table 3 also depicts strong correlations between higher maternal schooling and the 'pathways' through which the effect of education is hypothesised to influence child health; better educated mothers reside in richer families, have greater exposure to media, are more literate and empowered and also have substantially greater health knowledge compared to mothers with no schooling. This is also true of more educated fathers - they are more literate, have greater health knowledge and report greater exposure to media, compared to illiterate fathers.

3. Empirical Results

We begin by estimating reduced-form functions of child health outcomes and parental health-seeking behaviour. Equations are estimated using Ordinary Least Squares (OLS) and Community Fixed Effects (henceforth CFE). To give parental education a more causal interpretation, we progressivley introduce more and more of the variables that may be correlated with parental education and may be causing omitted variable bias. If the introduction of a particular 'pathway' causes either the coefficient on FEDU/MEDU to decline significantly (compared to the base outcome without any proxy controls), this pathway (rather than parental education *per se*) has a direct effect on child health. Conditional health functions will be estimated controlling for the potential endogeneity of this channel (or channels) to determine the causal impact (if any) of the pathways through which parental education impacts child health. The latter tests for the second hypothesis proposed in the study: what are the channels

[18] A simple tabulation of MEDU and FEDU in our sample revealed that for 63 (30) per cent of the children aged 0-5, mothers (fathers) reported having acquired no education while for 16 (20) per cent of the children mothers/fathers had acquired education between 1-5 years (inclusive).

through which father's and mother's education contributes to child health in the absence of precise information about health-seeking behaviour and health input practices?

3.1 *Does Parental Schooling Affect Child Health?*

This sub-section addresses the first hypothesis posed in this study: does parental education affect child health outcomes and health-seeking behaviour? In particular, we do not impose any priors on whether mother's education is the more important determinant compared to father's education and allow the data to speak. Health-seeking behaviour (IMMU) and child health (HAZ and WAZ) equations are estimated on the sample of children aged 0-5[19]. Table 4 presents reduced-form ordinary least squares (OLS) estimates.

The variables of most interest are MEDU and FEDU[20]. Clearly, mother's schooling is positively associated with child immunisation scores and HAZ and WAZ. The size of the coefficient appears greatest for IMMU. Interestingly, however, father's education appears to be positively associated only with parental health-seeking behaviour. One cannot place much credence on these results as unobservables at the level of the community may be biasing the coefficients and we turn next to Table 5 which estimates the IMMU, HAZ and WAZ equations controlling for community fixed-effects[21]. It is now clear that while MEDU is positive and significant for height and weight outcomes, only father's education remains significant and positive in the IMMU equation. This is the headline story emerging from Table 5 - while fathers appear to play a role in 'one-off' immunisation decisions, mothers are more involved in the day-to-day health decisions that are hence reflected in height and weight outcomes. Indeed,

[19] Because it is well documented that Pakistan's society is highly segregated by gender across a range of individual economic and life outcomes (see for instance Aslam (2009) and Aslam, Kingdon and Söderbom (2008) for gender differences in the labour market, Aslam (2009) for gender differences in access to quality schooling and Aslam and Kingdon (2008) for gender differentials in intra-household allocation of education expenditure), we also allowed for the possibility that similar divides exist in the choice and use of health inputs for boys and girls. It was also hypothesised that the impact of parental schooling may differ for boys and girls as may the effect of various pathways through which parent's education impacts child health and immunisation status. The vector of coefficients in child health/immunisation functions was allowed to vary by gender by estimating separate functions for boys and girls. However, the results did not differ significantly and 'pooled' estimates of boys and girls are reported with the MALE dummy capturing any intercept differentials.

[20] The relationship between parental education and child health outcomes is linear. We also estimated identical regressions including the quadratic in mother's and father's education but in most cases, the quadratic was not significant.

[21] Household-size is not included in any of the regressions in Table 5 thereon to ensure parsimonious models. As a robustness check, estimates including household-size were estimated and the results were no different from those reported.

the effect of father's schooling on immunisation scores is not small - a father who has completed primary schooling (5 years) will have a child whose immunisation score is 0.2 more than the child of an uneducated father. More intuitively, a child whose father's education is within one standard deviation higher than mean schooling of all fathers will have an immunisation score about 0.43 more.

Comparing the coefficient and significance of MEDU in IMMU regressions across OLS (Table 4) and CFE (Table 5), it would seem that more educated mothers live in communities where health clinics offer immunisations, suggesting that MEDU in Table 4 was picking up this 'community' effect. The coefficient in MEDU (in immunisation functions) is upwardly biased because community factors that are correlated with maternal schooling are also likely to affect child immunisation status. For instance, in communities that are more progressive (e.g. where a large number of mothers are educated), the immunisation score of the index child is also likely to be higher, since even uneducated mothers are likely to take their children for immunisation because they observe other mothers doing so i.e. knowledge about the importance of immunisation diffuses well and the community spill-over/externality effects of immunisation appear to be large. In which case, an important beneficial effect of mothers' education is its positive externality benefits on immunisation. However, other health behaviours of educated mothers in the community – such as healthier diet, better hygiene at home etc. – are less visible to the uneducated mothers, so there is less community-level diffusion of these behaviours. The coefficient on FEDU also declines from 0.069 in Table 4 to 0.043 in Table 5 suggesting that while some of the apparent positive association of father's education with health-seeking behaviour is a community-effect, a large remaining part appears to be a direct positive effect of father's schooling itself.

Mother's education has positive 'effects' on child height and weight in the CFE regressions in Table 5[22]. In our study, an additional year of schooling of the mother increases HAZ by 0.038 standard deviations of the height for children of the same age and gender and WAZ by 0.030 standard deviations of the weight for children of the same reference group. Intuitively, this means that compared to children of an illiterate mother, those whose mothers have completed say middle schooling (8 years) are 0.3 standard deviations taller and 0.2 standard deviations heavier on average – a large effect.

[22] Arif (2004) also notes a positive effect of mother's schooling on child height and weight outcomes using data from Pakistan from 2001 although their estimates are simple OLS estimates.

In terms of the remaining variables in Table 5, while boys have a greater likelihood of being immunised compared to girls, there is no evidence of gender differentiated treatment in child health outcomes. Once again, this could reflect the nature of the decision - differential treatment may be more visible in 'one-off' immunisation decisions rather than more long-term health-input decisions. The absence of a gender effect in height and weight outcomes is consistent with other studies in Pakistan (World Bank, 2002 and Arif, 2004). The signs on child age and its square imply that immunisation scores increase at a decreasing rate as the child becomes older which is consistent with normal immunisation behaviour. In the HAZ and WAZ equations, there is a convex relationship between child height/weight and age. HAZ/WAZ decrease with age though with a decreasing slope, implying that HAZ/WAZ are worse for older children. This could be because the health disadvantage of children increases as they become older or because older birth cohorts had poorer health outcomes (Chen and Li, 2009). Finally, mother's and father's heights are important determinants of child height and weight suggesting they are capturing at least some of the typically unobserved health endowment of the child.

The positive association between parental schooling and health outcomes cannot be interpreted as causal because of the potential endogeneity of parent's schooling. The approach used here to overcome this bias is to introduce control variables to proxy for the unobserved variables generating endogeneity in the variable of interest. As mentioned before, these control variables are the hypothesised ‘pathways’ through which maternal education is expected to impact child health.

Tables 6, 7 and 8 respectively present the immunization, HAZ and WAZ equations. In each of these tables, the controls are introduced one-by-one. Because father's schooling only appears important in IMMU equations, 'pathways' through which father's education could impact health-seeking behaviour are introduced in the IMMU table (Table 6). Similarly, because only mother's schooling looks important in HAZ and WAZ equations, mother's pathways of impact are added in Tables 7 and 8. All estimates control for community fixed effects.

Focus first on Table 6 which estimates immunisation equations and introduces pathways through which father's education potentially impacts health-seeking behaviour. The base-line CFE estimate (without any controls) in column (1) report a coefficient of 0.043 on father’s education (FEDU). The introduction of household per capita expenditure (LNPCE) and father’s exposure to media (FTV) doesn’t cause the size of the FEDU coefficient to change and indeed

there is no direct effect of either variable on immunisation[23]. While the introduction of father's literacy (FSLIT) reduces the size of FEDU and causes it to become insignificant, this is largely due to the high correlation between education and literacy which prevents inference of any effect of the two independently. Notably, the introduction of father's health knowledge (FHK) causes FEDU to collapse completely to zero. Father's health knowledge appears to have a large direct, positive and significant effect on immunisation scores – a unit increase in the health knowledge score of fathers is associated with a 0.057 unit increase in a child's immunisation score. This suggests that it is fathers' health knowledge rather than their education *per se* that is positively associated with better health-seeking behaviour, as reflected in immunization against common childhood illnesses. Of course, we not know if health knowledge is acquired in school, or whether schooling assists in the gathering of health knowledge after schooling is completed. In general, health knowledge is not part of the school curriculum so it is more likely that schooling increases a person's ability to gather/assimilate/absorb health knowledge.

Tables 7 and 8 introduce pathways through which mother's education (MEDU) may impact child height (HAZ) and weight (WAZ) outcomes respectively. In Table 7, the introduction of mother's labour force participation (MLF) causes a slight decrease in the coefficient on MEDU though it is not a statistically significant reduction. This suggests that while mother's education acts partly through MLF, mother's participation in the labour force has a large independent beneficial effect on child height. This could be because mothers who are involved in the labour market are more autonomous or have higher earnings which they control which may be reflected in better nutritional status of their children. We note a similar finding when mother's exposure to media (MTV) is added as a channel: while part of the effect of mother's education operates through her exposure to media, watching television appears to have a large independent effect on her child's height and hence long-term nourishment. This could be because exposure to media increases maternal health knowledge or allows women to view female role-models whom they imitate in implementing healthier practices within their households. Finally, mother's health knowledge has a large negative coefficient which is relatively precisely determined. This suggests reverse causation in health knowledge acquisition, i.e. uneducated mothers appear to have more health knowledge possibly because of bitter experience in dealing with childhood ailments. In Table 8, the introduction of MSLIT

[23]At first glance the lack of a relationship between household income and childhood health/immunisation seems surprising. However, recent work from the World Bank (2002) suggests strong externality effects within communities in Pakistan so that there is no effect of household expenditure on child health after controlling for community per capita expenditure. This finding is consistent with the results in our study.

causes the coefficient on MEDU to collapse completely suggesting that it is not mother's schooling *per se* but the literacy acquired through schooling that positively impacts her child's weight. Finally, while part of the effect of being more empowered operates through more schooling, higher empowerment in decision-making seems to have a direct independent association with her child's weight

The introduction of each of the pathways independently is premised on there being no inter-relationships between the pathways. However, the pathways themselves may be interlinked – for instance, women's labour market participation may be a consequence of media exposure. Table 9 reports CFE estimates with all pathways added simultaneously for immunisation scores and HAZ and WAZ outcomes. In column (1), the introduction of all pathways causes the coefficient on FEDU to collapse to 0 and the effect is now fully captured in FHK. Similarly, in column (2), MEDU collapses to 0 and only MLF, MTV and MHK remain significant while in column (3) only MEMP remains significant. These results suggest that fathers' education seems to translate into higher immunisation of children solely through their health knowledge while mothers' education operates through mother's participation in the labour market, exposure to media and health knowledge in determining child height and through mother's empowerment in decision-making in determining her child's weight.

The introduction of 'pathways' through which parental education may translate into improved health-seeking behaviour or better child health status allows us to give a 'causal' interpretation to FEDU/MEDU. This is premised on the view that hypothesised that pathways proxy for unobservables correlated with parental education which confound the true effect of parent's schooling in health functions. However, as mentioned before, these pathways are themselves potentially endogenous and determining their causal impact on child health requires controlling for their endogeneity. We turn to this in the next section.

3.2 Through which pathways does parental education impact child health?

The objective of this sub-section is to identify the causal impact of the variables identified as possible 'pathways' – father's health knowledge (FHK) in immunisation equations, mother's participation in the labour force (MLF), her exposure to media (MTV) and health knowledge (MHK) in height-for-age equation and mother's relative bargaining position within the household (MEMP) in weight-for-age equations. One approach to dealing with the endogeneity of these variables is to use instrumental variables (IVs) but the challenge lies in finding

plausible instruments[24]. Glewwe (1999) instruments maternal health knowledge through three different variables: existence of close relatives who could act as sources of health knowledge, exposure to mass media and mother's education (with the view that if mother's education can be credibly excluded from child health equations, it will be a plausible instrument). None of these instruments is free from criticism. For instance, the existence of close relatives could also directly raise child health if mothers choose to take sick children to their natal homes (or husband's families' homes) for better care. To our knowledge, only Strauss (1990) and Handa (1999) use measures of 'female empowerment' in child health functions and the endogeneity of their variables is treated by using household fixed effects estimators. However, this is based on the notion that the sources of heterogeneity are at the level of the household which may not be entirely convincing for female empowerment variables where the source of heterogeneity is most likely to be at the level of the individual rather than at the household.

However, it is extremely difficult to find suitable instruments or use other convincing methodologies to control for unobserved heterogeneity. Given this constraint, we also use variables available in the dataset which we deem plausible instruments. More importantly, because mother's and father's own schooling are not directly determining either health-seeking behaviour (IMMU) or health outcomes (HAZ and WAZ), they are included as instruments in final regressions. Theoretically, this is plausible because we argue that parental education translates into better child health *through* the channels of impact. Father's health knowledge in immunisation equations is instrumented using father's schooling, mother's schooling and father's score on the ravens test. The use of the latter variable as an instrument is based on the belief that more 'able' fathers are also more likely to actively acquire health knowledge. Mother's participation in the labour market, media exposure and health knowledge are instrumented using father's and mother's own schooling, mother's ravens score and four additional variables: mother's own mother's completed years of schooling, mother's grandmother's schooling, mother's sister's schooling and mother's brother's schooling[25]. The latter set of variables is reasonably exogenous and reflects inter- and intra-generational transmission of knowledge[26]. For instance, mothers with sick children may turn to their

[24] Among the three empirical methods used to address endogeneity - including past measures of health, exploiting sibling/twins differences and the IV method - Grossman (2005) argues that the IV method imposes the fewest assumptions and has produced the most reliable estimates.
[25] The questionnaire asked the individual to report the completed years of education of the sister and brother closest in age to the individual.
[26] However, these instruments assume no intergenerational transmission of ability.

maternal homes seeking health advice. The same vector of instruments is used to instrument mother's empowerment in weight-for-age equations.

It is worthwhile to note a further point regarding the endogeneity of health knowledge. Endogeneity bias will arise from two possible sources – omitted variables bias or simultaneity bias. As an example of the latter consider the following scenario: suppose one child died or suffered a major health shock/illness because the parents had failed to immunise the child. Once the child became ill, a parent was told (by whatever source) that they should have immunised the child so they 'learnt' this and this knowledge was used in immunising the next child. Thus, the endogeneity of FHK arises because FHK causes immunisation (of the second child) but immunisation (or the lack thereof of the first child) generated learning and hence an increase in FHK. We note that our list of instruments may not be convincingly exogenous as far as learning and endogeneity arising from simultaneity is concerned.

Tables 10, 11 and 12 report CFE and IV estimates (controlling for CFE) on the following dependent variables: immunisation score, HAZ and WAZ respectively. As before, all estimates are robust and control for clustering at the community level. Focus first on the findings in Table 10[27]. The first stage regression for FHK shows that two of the three instruments have the predicted signs and are significant and very precisely determined. Father's own schooling is a large positive determinant of his health knowledge. Similarly, father's ravens score has almost the same size of coefficient as father's schooling, and is a very precise determinant of health knowledge confirming our a priori belief that more able fathers also have more health knowledge. The p-value of the F-test of excluded instruments indicates that the instruments satisfy the 'relevance' condition well. Turn now to the second stage results. The p-value of the over-id test comfortably confirms the validity of the instruments used. Finally, in terms of the key findings, a comparison across column (1) and (2) shows that instrumenting FHK causes the coefficient to become even larger though the precision decreases marginally. The FHK estimate may have been biased downwards in the CFE equation for the following reason: If there is indeed some element of reverse causation (i.e. if fathers who are less likely to immunize end up getting higher health knowledge, meaning there is negative relationship between IMMU and FHK) then in an OLS/CFE estimation, any positive coefficient of FHK on IMMU will be dampened downwards due to the negative feedback effect from IMMU to FHK (those who immunize are ones who had lower health knowledge in the first place). This is why when using IV, one prevents this reverse causation effect and is able to identify the true positive

[27] Mother's height and the dummy variable indicating missing height are not included in the list of regressors to make the final model more parsimonious.

effect of FHK on IMMU.As before, the inference remains unchanged - father's health knowledge is positively associated with children's immunisation scores and indeed, more educated fathers have more immunised children because these fathers appear to have more health knowledge.

Turn now to the findings in Table 11. MLF, MTV and MHK are treated as endogenous and instrumented using the vector specified above. In first stage regressions, only in MHK regressions do the instruments very precisely determine health knowledge and have the expected signs. For instance, mother's own schooling, her ravens score, her mother's schooling and maternal grandfather's schooling all have large positive coefficients that are significant at the 5% level or better[28]. In terms of the second stage results, among the three endogenous variables, only mother's health knowledge is significant (at the 10% level) and in fact the coefficient is now a large positive suggesting that treating the health knowledge variable as exogenous greatly underestimates it's impact on child height (Glewwe, 1999 reports similar findings using Moroccan data). Finally, Table 12 treats MEMP as endogenous in the weight-for-age equations. Only FEDU and MEDU have any power in determining a woman's empowerment within her home - indeed her own higher schooling is a slightly larger determinant of her empowerment than her husband's schooling. As before, we note that treating MEMP as exogenous underestimates its effect on child weight - the coefficient increases by almost 50 per cent when treated as endogenous (from 0.379 to 0.776)[29]. This suggests that female autonomy is a critical pathway determining child health in Pakistan. Increased maternal education seems to help change the traditional balance of power within homes which is reflected in better health outcomes of children.

Summarising, several critical findings emerge from this analysis. Firstly, we note that it is father's health knowledge acquired through schooling rather than father's schooling *per se* that is positively associated with child immunisation. In a similar vein, it is mother's health knowledge and empowerment within the home acquired through schooling rather than schooling that impacts her child's height and weight. This is akin to the finding by Glewwe

[28] As a small digression, note the importance of intergenerational transmission of knowledge – mother's maternal grandfather's education is a crucial determinant of her own health knowledge. Exposure to media is positively determined by father's education (i.e. the woman's husband's education) and mother's own education. There is also a small positive effect of mother's brother's education on her exposure to media

[29] If women's empowerment/autonomy leads to greater conflict within the household, i.e. if empowerment and conflict are positively correlated and if conflict is detrimental to child health, correcting for the endogeneity of MEMP would lead to an increase in the corresponding IV coefficient. These results are fairly robust to the choice of instruments.

(1999) where it is mother's health knowledge rather than schooling per se that matters to child health. Secondly, if we believe the results, the size of effects is not small.

4. Conclusion

This study investigates the relationship between parental schooling on the one hand and both child health outcomes (measured as child height and weight) and parental health-seeking behaviour (child immunisation status) on the other. This study aimed to understand the mechanisms through which parents' schooling translates into better child health and improved parental health-seeking behaviour. The proposed 'pathways' through which parental education may impact child health outcomes/immunisation scores are: through higher household income, greater exposure to media, literacy, better health knowledge, mother's participation in the labour market and the extent of maternal empowerment within her husband's home.

Latest data from two provinces (Punjab and NWFP) from Pakistan were used. Child health/immunisation score functions were estimated using OLS and community-fixed effects. Estimates were based on a sample of children aged 0-5 years. The potential endogeneity of parental schooling was controlled through the addition of the aforementioned 'pathways' with the view that some or all of these could proxy for unobservables correlated with parental schooling and child health. The endogeneity of the 'pathways' that appear to determine child health was dealt with using instrumental variables.

There are several interesting findings. Baseline estimates reveal that while father's education alone is positively associated with immunisation, mother's education alone positively determines child health outcomes. The introduction of 'pathways' reveals that (a) father's health knowledge acquired through schooling impacts immunisation; (b) educated mothers' greater labour force participation, higher exposure to media and better health knowledge are all potential channels of impact from mother's education onto child height; and (c) education improves women's empowerment within their homes which ultimately impacts her child's weight. However, these channels of impact are all potentially endogenous and only estimates explicitly controlling for the endogeneity of these variables are credible. IV estimates show that father's health knowledge is an even larger positive determinant of child immunization (than in OLS estimation), while only mother's health knowledge is a large and positive determinant of child height once endogeneity is explicitly controlled for. Mother's empowerment within the home is an important positive channel through which mother's education translates into better weight-for-age outcomes for children.

Three key points must be noted. Firstly, controlling for the endogeneity of the channels is crucial as we have found that their effect is largely underestimated when we do not explicitly take their endogeneity into account. Secondly, perhaps the most striking finding emerging from the analysis is how the nature of the decision regarding child health seems to be clearly demarcated within Pakistani households – while fathers clearly play a role in 'one-off' child health decisions (namely the immunization decision), mothers' health related decisions have an effect on longer term child health outcomes (height and weight). Finally, health knowledge emerges as a crucial channel through which both parents' education translates into better health outcomes for children. While we are wary of giving it a causal interpretation, it is clear that parental health knowledge is highly positively associated with both better health-seeking behaviour and better child health in Pakistan.

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Figures

Figure 1: Kernel density estimate of HAZ (aged 0-5 years)

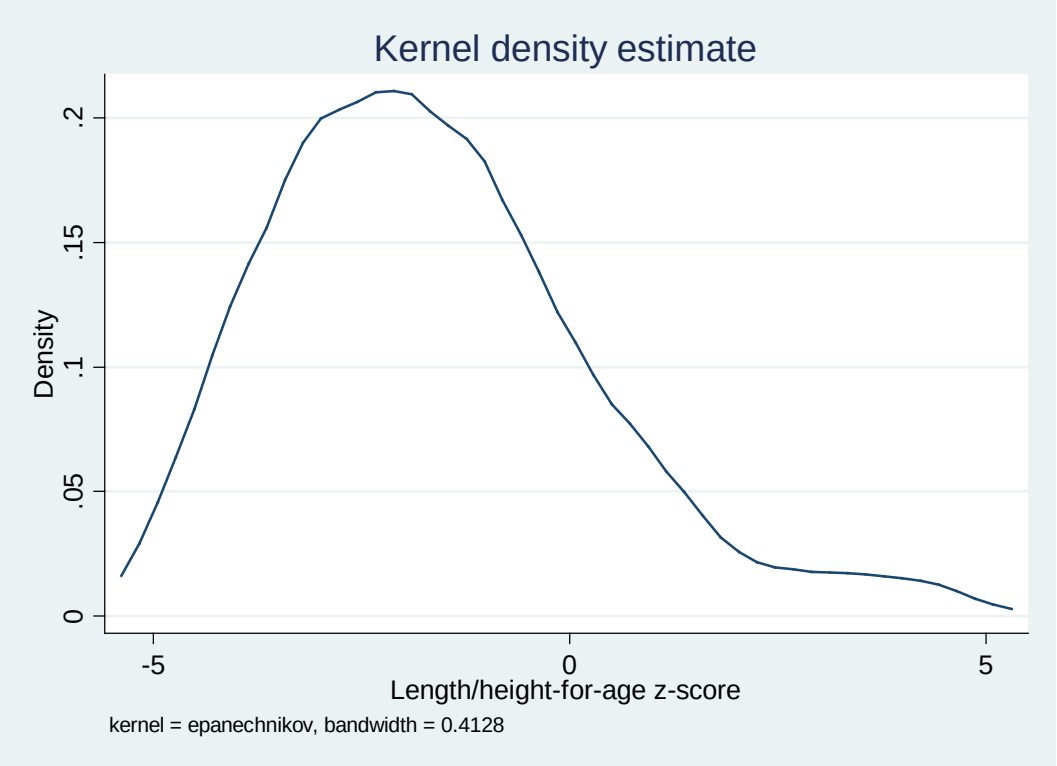
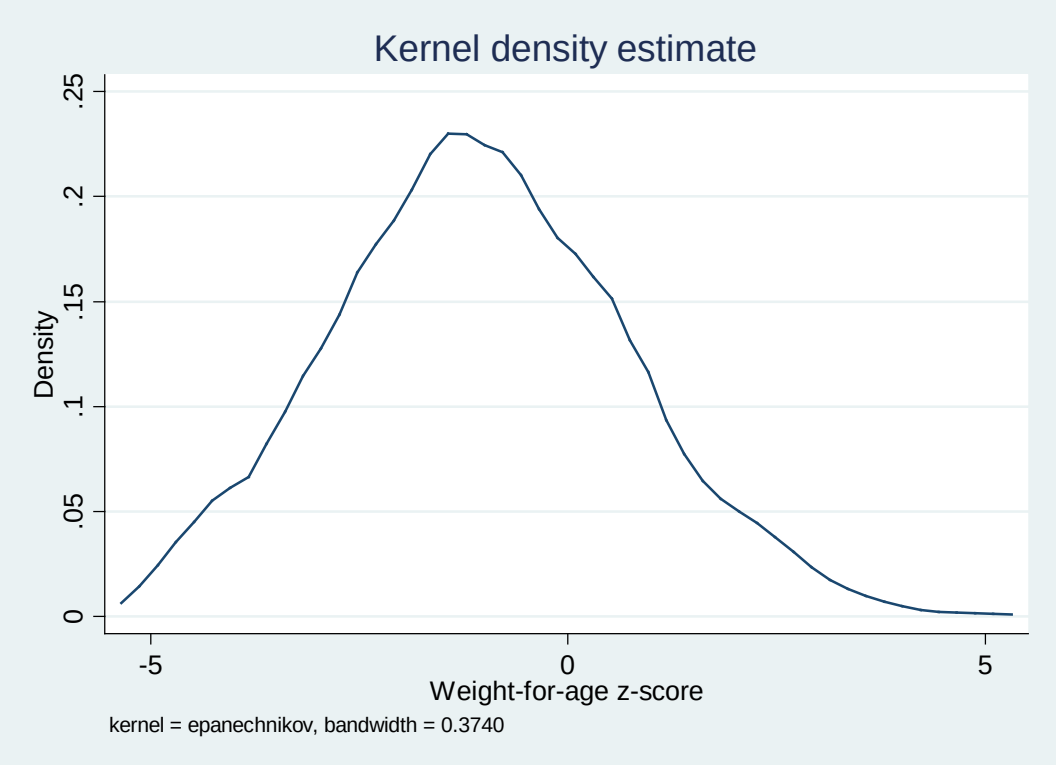


Figure 2: Kernel density estimates of WAZ(ages 0-5 years)



Tables

Table 1 – Description of Variables Used

Variable	Description
IMMU	Immunisation score (giving a score of 1 if individual is immunised/treated against any of

	the following: Polio, TB, Diptheria, Whooping Cough, Measles, Mumps, Rubella, Hepatitis or Goiter, 0 otherwise;
HAZ	Height-for-age z scores;
WAZ	Weight-for-age z scores;
MALE	Dummy equals 1 if male, 0 otherwise;
AGE	Age of child in months;
AGE2	Age squared;
HHSIZE	Household size;
FHGT	Father's height (cm);
FHGTMISS	Dummy equals 1 if father's height is missing, 0 otherwise;
MHGT	Mother's height (cm);
MHGTMISS	Dummy equals 1 if mother's height is missing, 0 otherwise;
MEDU	Mother's completed years of schooling;
FEDU	Father's completed years of schooling;
RURAL	Dummy equals 1 if in rural area, 0 otherwise;
PUNJAB	Dummy equals 1 if in Punjab province, 0 otherwise;
LNPCE	Log of per capita expenditure;
MTV	Dummy equals 1 if mother reports watching television, 0 if she reports never watching tv;
MSLIT	Mother's literacy score on short literacy test ranges from 0-?;
MLFP	Dummy equals 1 if mother participates in the labour market, 0 otherwise;
MHK	Mother's score on the health knowledge test, ranges from 0-26;
MEMP	Dummy equals 1 if mother's preferences about number of children to have taken into account when deciding on how many children couple will/has had, 0 otherwise;
FTV	Dummy equals 1 if father reports watching television, 0 if he reports never watching tv;
FSLIT	Father's literacy score on short literacy test ranges from 0-?;
FHK	Father's score on the health knowledge test, ranges from 0-26;
MEMEDU	Mother's mother's completed years of schooling;
MEMGRAND	Mother's maternal grandfather's completed years of schooling;
MEMSISEDU	Mother's sister's completed years of schooling;
MEMBROEDU	Mother's brother's completed years of schooling;
MRAVENS	Mother's score on ravens test, ranges from 0-20;
FRAVENS	Father's score on ravens test, ranges from 0-20;

Table 2: Summary Statistics of Variables Used

Variable	Mean	SD
IMMU	4.719	2.032
HAZ	-1.649	1.930
WAZ	-1.048	1.745
MALE	0.500	.500
AGE in months	33.039	19.101
AGE2	1456.148	1284.516
FHGT	130.103	69.656
FHGTMISS	0.221	0.415
MHGT	153.796	13.938
MHGTMISS	0.010	0.077
MEDU	2.731	4.240
FEDU	5.782	4.670
RURAL	0.735	0.441
PUNJAB	0.708	0.455
LNPCE	9.431	0.500
MTV	0.620	0.485
MSLIT	1.191	1.942
MLFP	0.334	0.472
MHK	9.895	4.657
MEMP	0.433	0.496
MEMEDU	0.436	1.742
MEMGRAND	0.419	1.863
MESISEDU	4.439	12.584
MEBROEDU	8.634	13.660
MRAVENS	7.334	13.658
FTV	0.684	0.465
FSLIT	2.596	2.319
FHK	10.025	4.013
FRAVENS	8.309	2.760

Table 3: Means of Key Variables by Parental Education Level

Variable	Mother			Father		
	no Education	1-5 years education	more than primary	no Education	1-5 years education	more than primary
IMMU	4.442	5.144	5.248	4.238	4.476	5.056
HAZ	-1.756	-1.554	-1.403	-1.612	-1.811	-1.616
WAZ	-1.176	-0.887	-0.781	-1.122	-1.173	-0.969
% stunted (<-2sd)	31.534	6.913	8.333	13.542	8.712	24.527
% unwgt (<-2 sd)	21.196	4.222	5.013	9.147	5.717	15.567
LNPCE	9.289	9.480	9.833	9.205	9.378	9.569
MTV	0.497	0.743	0.909	0.394	0.568	0.758
MSLIT	0.066	1.952	4.091	0.276	0.524	1.897
MLFP	0.369	0.219	0.315	0.472	0.359	0.253
MHK	8.938	10.235	12.600	9.068	9.252	10.546
MEMP	0.325	0.513	0.705	0.326	0.393	0.503
FTV	0.590	0.806	0.914	0.477	0.657	0.819
FSLIT	1.914	3.194	4.494	0.138	1.899	4.339
FHK	9.296	10.470	12.209	7.888	9.171	11.635

Table 4 - Reduced form OLS estimates of determinants of IMMU, HAZ and WAZ (0-5 years)

	(1) IMMU	(2) HAZ	(3) WAZ
MALE	0.222	-0.156	-0.253
	(2.84)***	(1.40)	(2.51)**
AGEM	0.094	-0.064	-0.008
	(9.17)***	(3.61)***	(0.66)
AGEM2	-0.001	0.001	0.000
	(6.42)***	(3.71)***	(0.61)
HHSIZE	-0.045	0.012	-0.007
	(3.38)***	(0.84)	(0.79)
FHGT	-0.016	0.010	0.016
	(1.37)	(1.31)	(2.06)**
FHGTMISS	-1.751	1.439	2.574
	(0.87)	(1.10)	(2.02)**
MHGT	0.014	0.024	0.019
	(1.25)	(1.89)*	(2.03)**
MHGTMISS	1.614	4.724	3.575
	(0.90)	(2.53)**	(2.26)**
MEDU	0.051	0.040	0.022
	(2.28)**	(3.28)***	(1.67)*
FEDU	0.069	-0.012	-0.009
	(3.01)***	(0.82)	(0.46)
RURAL	0.108	-0.216	-0.364
	(0.32)	(1.00)	(2.10)**
PUNJAB	0.364	0.259	-0.202
	(0.83)	(1.53)	(1.12)
CONSTANT	3.144	-6.294	-5.856
	(1.14)	(2.77)**	(2.88)***
N	903	995	1073
R^2	0.16	0.05	0.04

Notes: Robust t-statistics are in parentheses and correct for clustering at the community level; * denotes significance at 10%, ** at 5% and *** at 1% or more.

Table 5 - Reduced form Community Fixed Effects (CFE) estimates of determinants of IMMU, HAZ and WAZ (0-5 years)

	(1) IMMU	(2) HAZ	(3) WAZ
MALE	0.167	-0.166	-0.236
	(2.37)**	(1.50)	(2.30)**
AGEM	0.093	-0.067	-0.010
	(8.75)***	(3.72)***	(0.80)
AGEM2	-0.001	0.001	0.000
	(6.41)***	(3.90)***	(0.86)
MHGT	0.005	0.024	0.021
	(0.49)	(1.91)*	(2.34)**
MHGTMISS	-1.585	5.149	4.279
	(0.92)	(2.67)**	(2.52)**
FHGT	-0.007	0.014	0.017
	(0.58)	(1.85)*	(2.11)**
FHGTMISS	-0.143	2.143	2.913
	(0.07)	(1.72)*	(2.14)**
MEDU	0.010	0.038	0.030
	(0.49)	(2.73)**	(2.18)**
FEDU	0.043	-0.011	-0.005
	(2.16)**	(0.70)	(0.29)
CONSTANT	3.169	-6.844	-7.044
	(1.27)	(3.13)***	(3.52)***
N	903	995	1073
NO. COMMUNITY	27	27	27
R^2	0.11	0.05	0.02

Notes: Robust t-statistics are in parentheses and correct for clustering at the community level; * denotes significance at 10%, ** at 5% and *** at 1% or more.

Table 6 - Reduced form estimates (Community FE) of determinants of IMMU (0-5 years) , 'pathways' added one-by-one.

	(1) BASE CFE	(2) LNPCE	(3) FTV	(4) FSLIT	(5) FHK
MALE	0.167	0.168	0.171	0.168	0.180
	(2.37)**	(2.37)**	(2.38)**	(2.49)**	(2.59)**
AGEM	0.093	0.093	0.093	0.093	0.093
	(8.75)***	(8.85)***	(8.42)***	(8.72)***	(8.55)***
AGEM2	-0.001	-0.001	-0.001	-0.001	-0.001
	(6.41)***	(6.51)***	(6.22)***	(6.40)***	(6.39)***
MHGT	0.005	0.005	0.005	0.005	0.003
	(0.49)	(0.49)	(0.47)	(0.47)	(0.28)
MHGTMISS	-1.585	-1.578	-1.636	-1.609	-1.817
	(0.92)	(0.91)	(0.93)	(0.92)	(1.07)
FHGT	-0.007	-0.007	-0.007	-0.007	-0.012
	(0.58)	(0.58)	(0.58)	(0.58)	(1.01)
FHGTMISS	-0.143	-0.125	-0.160	-0.120	-1.011
	(0.07)	(0.06)	(0.07)	(0.05)	(0.49)
MEDU	0.010	0.011	0.010	0.011	0.009
	(0.49)	(0.48)	(0.49)	(0.51)	(0.44)
FEDU	0.043	0.043	0.043	0.034	0.023
	(2.16)**	(2.14)**	(2.07)**	(0.83)	(1.00)
LNPCE		-0.026			
		(0.14)			
FTV			0.018		
			(0.08)		
FSLIT				0.021	
				(0.33)	
FHK					0.057
					(3.51)***
CONSTANT	3.169	3.377	3.212	3.202	3.926
	(1.27)	(1.32)	(1.26)	(1.24)	(1.56)
N	903	903	901	902	903
NO. COMM	27	27	27	27	27
R^2	0.11	0.12	0.12	0.11	0.13

Notes: Robust t-statistics are in parentheses and correct for clustering at the community level; * denotes significance at 10%, ** at 5% and *** at 1% or more.

Table 7 - Reduced form estimates (Community FE) of determinants of HAZ (0-5 years) , 'pathways' added one-by-one.

	(1) BASE CFE	(2) LNPCE	(3) MLF	(4) MTV	(5) MSLIT	(6) MHK	(7) MEMP
MALE	-0.166	-0.170	-0.156	-0.166	-0.166	-0.160	-0.173
	(1.50)	(1.58)	(1.40)	(1.50)	(1.51)	(1.48)	(1.61)
AGEM	-0.067	-0.068	-0.069	-0.067	-0.067	-0.065	-0.067
	(3.72)***	(3.85)***	(3.83)***	(3.73)***	(3.71)***	(3.65)***	(3.72)***
AGEM2	0.001	0.001	0.001	0.001	0.001	0.001	0.001
	(3.90)***	(4.05)***	(3.98)***	(3.87)***	(3.89)***	(3.83)***	(3.92)***
MHGT	0.024	0.024	0.023	0.024	0.024	0.027	0.024
	(1.91)*	(1.88)*	(1.85)*	(1.93)*	(1.91)*	(2.05)**	(1.92)*
MHGTMISS	5.149	5.076	4.751	5.054	5.146	5.629	5.193
	(2.67)**	(2.65)**	(2.62)**	(2.63)**	(2.67)**	(2.78)**	(2.71)**
FHGT	0.014	0.013	0.012	0.014	0.014	0.015	0.014
	(1.85)*	(1.80)*	(1.62)	(2.00)**	(1.83)*	(2.06)**	(1.80)*
FHGTMISS	2.143	2.099	1.927	2.186	2.129	2.220	2.104
	(1.72)*	(1.69)*	(1.51)	(1.86)*	(1.71)*	(1.88)*	(1.67)*
MEDU	0.038	0.034	0.033	0.031	0.035	0.054	0.035
	(2.73)**	(2.19)**	(2.05)**	(2.08)**	(1.51)	(3.88)***	(2.25)**
FEDU	-0.011	-0.012	-0.006	-0.020	-0.011	-0.009	-0.011
	(0.70)	(0.80)	(0.41)	(1.35)	(0.71)	(0.55)	(0.74)
LNPCE		0.125					
		(0.63)					
MLF			0.353				
			(1.67)*				
MTV				0.421			
				(2.47)**			
MSLIT					0.009		
					(0.15)		
MHK						-0.051	
						(2.60)*	
MEMP							0.122
							(0.70)
CONSTANT	-6.844	-7.872	-6.502	-7.053	-6.829	-6.937	-6.851
	(3.13)***	(3.04)***	(3.05)***	(3.23)***	(3.11)***	(3.16)***	(3.11)***
N	995	995	995	995	995	995	995
NO. COMM	27	27	27	27	27	27	27
R^2	0.05	0.05	0.05	0.05	0.05	0.06	0.05

Notes: Robust t-statistics are in parentheses and correct for clustering at the community level; * denotes significance at 10%, ** at 5% and *** at 1% or more

Table 8 - Reduced form estimates (Community FE) of determinants of WAZ (0-5 years) , 'pathways' added one-by-one.

	(1) BASE CFE	(2) LNPCE	(3) MLF	(4) MTV	(5) MSLIT	(6) MHK	(7) MEMP
MALE	-0.236	-0.240	-0.234	-0.235	-0.240	-0.236	-0.253
	(2.30)**	(2.34)**	(2.28)**	(2.28)**	(2.29)**	(2.29)**	(2.43)**
AGEM	-0.010	-0.010	-0.010	-0.010	-0.009	-0.010	-0.010
	(0.80)	(0.83)	(0.84)	(0.79)	(0.75)	(0.80)	(0.84)
AGEM2	0.000	0.000	0.000	0.000	0.000	0.000	0.000
	(0.86)	(0.91)	(0.89)	(0.84)	(0.80)	(0.86)	(0.92)
MHGT	0.021	0.021	0.021	0.021	0.021	0.021	0.021
	(2.34)**	(2.27)**	(2.32)**	(2.35)**	(2.33)**	(2.30)**	(2.43)**
MHGTMISS	4.279	4.203	4.174	4.274	4.253	4.268	4.405
	(2.52)**	(2.46)**	(2.43)**	(2.51)**	(2.49)**	(2.49)**	(2.66)**
FHGT	0.017	0.017	0.017	0.017	0.016	0.017	0.016
	(2.11)**	(2.13)**	(2.07)**	(2.12)**	(1.94)*	(2.10)**	(2.04)**
FHGTMISS	2.913	2.882	2.889	2.900	2.777	2.912	2.744
	(2.14)**	(2.17)**	(2.11)**	(2.15)**	(1.98)*	(2.14)**	(2.06)**
MEDU	0.030	0.026	0.028	0.027	-0.006	0.029	0.021
	(2.18)**	(1.82)*	(2.11)**	(2.15)**	(0.27)	(2.06)**	(1.69)*
FEDU	-0.005	-0.007	-0.004	-0.009	-0.006	-0.005	-0.009
	(0.29)	(0.39)	(0.23)	(0.42)	(0.33)	(0.30)	(0.46)
LNPCE		0.107					
		(0.67)					
MLF			0.094				
			(0.84)				
MTV				0.142			
				(0.60)			
MSLIT					0.091		
					(1.82)*		
MHK						0.001	
						(0.09)	
MEMP							0.355
							(2.26)*
CONSTANT	-7.044	-7.916	-6.987	-7.111	-6.897	-7.041	-6.987
	(3.52)***	(3.23)***	(3.48)***	(3.57)***	(3.37)***	(3.51)***	(3.48)***
N	1073	1073	1073	1073	1073	1073	1073
NO. COMM	27	27	27	27	27	27	27
R-squared	0.02	0.02	0.02	0.02	0.03	0.02	0.03

Notes: Robust t-statistics are in parentheses and correct for clustering at the community level; * denotes significance at 10%, ** at 5% and *** at 1% or more

Table 9 - Reduced form estimates (Community FE) of determinants of IMMU, HAZ and WAZ (0-5 years) , 'pathways' added simultaneously.

	(1) IMMU	(2) HAZ	(3) WAZ
MALE	0.188	-0.163	-0.255
	(2.70)**	(1.51)	(2.41)**
AGEM	0.093	-0.068	-0.010
	(8.15)***	(3.86)***	(0.85)
AGEM2	-0.001	0.001	0.000
	(6.15)***	(4.02)***	(0.91)
MHGT	0.003	0.025	0.021
	(0.28)	(1.98)*	(2.34)**
MHGTMISS	-1.861	5.068	4.254
	(1.06)	(2.68)**	(2.49)**
FHGT	-0.012	0.012	0.015
	(0.97)	(1.71)*	(1.88)*
FHGTMISS	-0.989	1.856	2.578
	(0.47)	(1.58)	(1.93)*
MEDU	0.013	0.017	-0.015
	(0.53)	(0.63)	(0.73)
FEDU	0.025	-0.016	-0.012
	(0.58)	(0.98)	(0.54)
LNPCE	-0.126	0.137	0.074
	(0.68)	(0.67)	(0.44)
FTV	0.041	-	-
	(0.17)		
FSLIT	-0.005	-	-
	(0.09)		
FHK	0.061	-	-
	(4.18)***		
MLF	-	0.436	0.122
		(2.15)**	(1.07)
MTV	-	0.421	0.115
		(2.44)**	(0.50)
MSLIT	-	0.049	0.084
		(0.76)	(1.62)
MHK	-	-0.061	-0.007
		(3.07)***	(0.62)
MEMP	-	0.090	0.323
		(0.51)	(2.01)**
CONSTANT	5.004	-7.789	-7.464
	(1.81)*	(3.04)***	(2.85)***
N	900	995	1073
NO. COMM	27	27	27
R^2	0.13	0.07	0.04

Notes: Robust t-statistics are in parentheses and correct for clustering at the community level; * denotes significance at 10%, ** at 5% and *** at 1% or more

Table 10 - Reduced form CFE and Conditional Demand Estimates (Instrumental Variables with Community Fixed Effects) of Immunisation Score (0-5 years)

	CFE		IV Regressions with Community FE			
			Second Stage		First Stage (FHK)	
	(1)		(2)		(3)	
FHK	0.070	***	0.113	**	-	
	(6.04)		(2.63)			
MALE	0.171	**	0.186	**	-0.334	
	(2.46)		(2.46)		(-1.16)	
AGEM	0.095	***	0.094	***	0.020	
	(8.70)		(8.74)		(0.86)	
AGEM2	-0.001	***	-0.001	***	-0.000	
	(-6.64)		(-6.77)		(-0.41)	
FEDU	-		-		0.289	***
					(6.91)	
MEDU	-		-		0.011	
					(0.20)	
FRAVENS	-		-		0.281	***
					(4.17)	
N	903		903		903	
R^2	0.13		0.11		0.23	
No. Comm	27		27		27	
P-value (F test excluded instruments)	-		-		0.000	
p-value (Overid)	-		0.723			

Notes: Robust t-statistics are in parentheses and correct for clustering at the community level; * denotes significance at 10%, ** at 5% and *** at 1% or more; FHGT and FHGTMISS included as controls for but shown.

Table 11 - Reduced form Community Fixed Effects and Conditional Demand Estimates (Instrumental Variables with Community Fixed Effects) of HAZ (0-5 years)

	CFE		IV Regressions with Community Fixed Effects							
			Second Stage		First Stage (MLF)		First Stage (MTV)		First Stage (MHK)	
MLF	0.454	**	-0.229		-		-		-	
	(2.24)		(-0.28)							
MTV	0.480	***	-0.809		-		-		-	
	(2.95)		(-1.10)							
MHK	-0.050	**	0.176	*	-		-		-	
	(-2.41)		(1.77)							
FEDU	-		-		-0.012	**	0.022	***	0.013	
					(-2.57)		(5.30)		(0.28)	
MEDU	-		-		0.019	***	0.017	***	0.204	***
					(2.96)		(4.30)		(3.52)	
MRAVENS	-		-		-0.001		0.003		0.207	**
					(-0.14)		(0.41)		(2.14)	
MEMEDU	-		-		-0.008		-0.007		0.254	**
					(-0.61)		(-0.14)		(2.80)	
MEMGRAND	-		-		-0.001		0.008		0.149	**
					(-0.07)		(1.05)		(2.25)	
MEBROEDU	-		-		-0.001		0.003	**	0.036	***
					(-0.83)		(2.30)		(3.30)	
MESISEDU	-		-		-0.003	***	-0.001		0.005	
					(-3.11)		(-0.66)		(0.22)	
N	995		995		995		995		995	
R^2	0.06		-0.20		0.05		0.11		0.15	
No. Comm	27		27		27		27		27	
P-value (F-test excluded instruments)	-		-		0.000		0.000		0.000	
P-value (overid)	-		0.560		-		-			

Notes: Robust t-statistics are in parentheses and correct for clustering at the community level; * denotes significance at 10%, ** at 5% and *** at 1% or more; The following controls included in regressions but not shown: MALE, AGEM, AGEM2, FHGT, FHGTMISS and MHGT and MHGTMISS.

Table 12 - Reduced form Community Fixed Effects and Conditional Demand Estimates (Instrumental Variables with Community Fixed Effects) of WAZ (0-5 years)

	CFE		IV Regressions with Community Fixed Effects			
			Second Stage		First Stage (MEMP)	
MEMP	0.379	***	0.776	**	-	
	(2.60)		(2.17)			
FEDU	-		-		0.011	**
					(2.27)	
MEDU	-		-		0.019	**
					(2.52)	
MRAVENS	-		-		0.016	
					(1.47)	
MEMEDU	-		-		-0.014	
					(-1.29)	
MEMGRAND	-		-		0.020	
					(1.60)	
MEBROEDU	-		-		0.002	
					(1.58)	
MESISEDU	-		-		0.002	
					(0.96)	
N	1073		1073		1073	
R^2	0.06		0.02		0.09	
No. Comm	27		27		27	
P-value (F test excluded instruments)	-		-		0.000	
P-value (overid)	-		0.404		-	

Notes: Robust t-statistics are in parentheses and correct for clustering at the community level; * denotes significance at 10%, ** at 5% and *** at 1% or more; The following controls included in regressions but not shown: MALE, AGEM, AGEM2, FHGT, FHGTMISS and MHGT and MHGTMISS.

Appendices

Appendix I

Health Knowledge Questions

For each question, give a score of 1 for everything the respondent mentions

1. **How does one get diarrhoea?**
    - By eating contaminated food
    - By drinking dirty/contaminated water
    - By eating from dirty hands or dirty utensils

2. **What is the best way to prevent diarrhoea?**
    - Boil water before drinking
    - Eat fresh food/ avoid stale food
    - Keep food covered/cool
    - Wash hands before eating

3. **If child develops diarrhoea, what should one do if there is no doctor available?**
    - Use boiled water
    - Feed soft foods
    - Avoid milk and fat
    - Give salts/ORS

4. **If your child falls and gets a small wound, what should you do?**
    - Wash it well
    - Apply antiseptic
    - Cover it with a cloth/band-aid

5. **How can one get malaria?**
    - Mosquito bite (by an infected mosquito)

6. **If you want to protect your child against polio, what should you do?**
    - Polio vaccinations/drops

7. **If your child develops fever, what should you do?**
    - Apply cold swabs
    - Take off child's extra clothes
    - Give plenty of fluids
    - Give paracetamol

8. **Which mineral is most important for healthy bones?**
    - Calcium

9. **What is the best source of calcium?**
    - Milk

10. **What are the main signs of heat stroke?**
    - High fever
    - Listlessness
    - Dehydration (no urination by children for a long time, no tears while crying)
    - Dry mouth/tongue