Effects of Early Childhood Health on Later Life Cognitive Functioning: Evidence from Matlab Bangladesh[1]

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Abstract: Early childhood health and nutrition interventions have been shown to improve the health status of young. It is believed that early life circumstances are crucial to success later in life. Yet causal evidence that the benefits of early childhood health interventions continue into adolescence and adulthood is sparse. This paper exploits a quasi-random placement of the Matlab Maternal and Child Health and Family Planning Program in Bangladesh and the rolling out of the program over time to determine whether children who were eligible to receive child health interventions when they were young, had better cognitive functioning at ages 8-14. I find the program lead to a 0.38 standard deviation increase in cognitive functioning. The effect remained the same after controlling for educational attainment highlighting that educational attainment is not a good proxy for cognitive functioning.

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1. Introduction

Cognitive development is a key contributor to future educational attainment, labor market outcomes, and overall well-being; but over 200 million children under five years of age are failing to reach their cognitive potential due to deprivation early in life (Grantham-McGregar et al. 2007). Many programs in developed and developing countries, such as Head Start and conditional cash transfer programs, aim to improve the early life circumstances of children through improved health and nutrition. However, little is known about the long-run effects of such interventions. Concern about fade-out is particularly pertinent in developing countries, where individuals face many competing health risks and shocks to their health and the ability to smooth consumption is often limited. A growing literature suggests that large negative shocks to a person's health or nutrition early in life, such as from flu pandemics or famines, lead to worse outcomes later in life.[2] However, causal evidence on the long-run effects of *interventions* designed to improve the health and nutrition of young children in general and on cognitive functioning in particular is limited due to the lack of well-designed programs that took place 10 or more years ago and data on cognitive functioning.[3] The few studies that do examine the effects of such programs on longer term cognitive development show mixed results.[4]

This paper exploits quasi-random variation in eligibility for a child health program in Bangladesh to estimate the causal effect of improvements in early-life health and nutrition on cognitive functioning at older ages. In 1982, the Matlab Maternal and Child Health and Family Planning (MCH-FP) Program provided the measles vaccine to children under five in the treatment area, but not in the comparison area. The program was expanded in 1985 to include other interventions such as vaccinations against DPT[5], polio, and tuberculosis. Preventing these diseases, especially measles, not only reduces the chance of cognitive impairment from the disease, but improves children's ability to absorb nutrients as well as their overall nutritional status, and the MCH-FP program has been shown to reduce the burden of these diseases.[6] The geographic separation of the treatment and comparison groups minimizes the possibility that the estimates will be biased due to the large potential spillovers associated with vaccines, an important advantage of this research design relative to randomization at the individual or village level. While the program was not randomly assigned, the rich set of pre-program data for the study site show that the treatment and comparison groups were similar on a variety of pre-program characteristics.

I estimate the intent-to-treat double difference effects on cognitive functioning for children who were born when the child health interventions were introduced, using cohorts born prior to the program to measure baseline differences. Surprisingly, to my knowledge, this is the first paper to take advantage of the program phase-in to examine the longer term effects of the

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[2] See Currie (2009), Strauss and Thomas (2008) and Glewwe and Miguel (2008) for recent reviews of this and related literature, and Garces et al. 2002 for evidence of the longer-term effects of Head Start.

[3] Evidence on short- and long-run effects of nutrition and infectious diseases on cognitive development are reviewed in Currie (2007), Walker et al. (2007) and Grantham-McGregor (1999a, 1999b).

[4] For example, a nutritional supplementation study in Jamaica found a significant positive impact on children development two years after the program, but no statistically significant effects when the children were ages 7 to 8 (Grantham-McGregor et al 2007). On the contrary, Maluccio et al. (2006) found that childhood nutritional supplementation in the well known INCAP study in Guatemala lead to improved adult nonverbal cognitive functioning.

[5] The DPT vaccine protects against diphtheria, pertussis (whooping cough), and tetanus.

[6] Koenig et al. 1991, 2001 show the program vaccinations led to reductions in infant and child mortality.

program.[7] The findings demonstrate that the program led to a large 0.38 standard deviation increase in cognitive functioning for the 8-14 year olds who were eligible for the health interventions when they were under the age of five. Impacts on the treated are over twice as large as the intent-to-treat effects. The effects remain even after controlling for educational attainment, highlighting that the effect of health on cognitive functioning cannot be captured through the effect of health on educational attainment alone. The results are robust to the inclusion of village and mother fixed-effects, and mortality and migration selection. Positive spill-over effects are found in comparison villages that neighbor treatment villages biasing the program effect downwards.

The MCH-FP program introduced a family planning and maternal health program in 1977 prior to the child health interventions. It is possible that family planning and maternal health interventions affect the cognitive function of the 8-14 year olds through a quantity-quality trade-off. I perform various analyses, including examining the group of children born when the family planning and maternal health interventions were available but before the child health interventions were introduced, that together suggest that the effect on cognitive functioning is likely to result primarily from the early child health interventions.[8] Even so, given that vaccination and family planning programs are perhaps two of the most important and widespread health programs worldwide, the combined effect of the program is of great importance.

The rest of the paper proceeds as follows. Section 2 describes the MCH-FP program and the mechanisms through which the program affects cognitive functioning; section 3 describes the data, section 4 lays out the identification and estimation strategy, the findings and robustness analysis are discussed in section 5, and section 6 concludes.

2. The Matlab MCH-FP program

2.1 The Intervention

The MCH-FP program began in 1977 in a rural area of Bangladesh called Matlab and was implemented by ICDDR,B, a non-government institution. The goal of the program was to improve children's health and reduce fertility. This study site covered approximately 200,000 people in 149 villages at program start with about half the population in the treatment area, leaving the other half as a comparison area (Figure 1). All interventions were administered in the house of the beneficiary during monthly visits made by local female health workers hired and trained by the program, and interventions were provided free of charge. Most of the interventions were not available in the government clinics in the comparison area until after 1988, providing an experimental period between 1977 and 1988 to evaluate the success of these interventions.

The health and family planning interventions were phased-in over time (Table 1) starting with family planning and maternal health interventions in 1997. The family planning and

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[7] Joshi and Schultz (2007) and Chaudhuri (2005) examine the effect of the program on various outcomes including educational attainment and height for age and weight-for-age z-scores. Neither paper pays careful attention to the phasing-in of program interventions over time in treatment or comparison groups in their designation of age groups, nor do they examine the effect on age groups who would not have been affected by the program, or look at cognitive functioning as an outcome.

[8] Joshi and Schultz (2007) argue that the family planning interventions of the program led to an increase in education for boys aged 9-14 but not for girls. Their research design does not take into account the fact that these children received significant child health interventions, nor do they provide analyses that separate out the family planning and child health interventions.

maternal health interventions mainly included provision of modern contraceptives, tetanus toxoid vaccinations for pregnant women.[9]

The child health interventions were introduced over time in the experimental period. Between 1982 and 1985 only vaccination against measles was available for children between the ages of 9 and 59 months in *half* the treatment area, referred to as the *Treatment Area 1* (Figure 1). In 1985, the measles vaccine was extended to the other half of the treatment area, referred to as the *Treatment Area 2*. In 1986, three other child health vaccinations (DPT, polio, and tuberculosis), and nutrition rehabilitation for those who were nutritionally at risk were added in the entire treatment area (Fauveau, 1994). Finally, Vitamin A supplementation was introduced in 1987 in the entire treatment area while treatment for acute respiratory infections was added in 1988 in half the treatment area. [10]

2.2 Program take-up

Figure 2 demonstrates that implementation followed the planned timeline and that uptake was rapid for two of the main program interventions (the measles vaccine and contraception). The measles take-up data for children 12-59 months are presented separately for Treatment Area 1 and 2. In both areas, the measles vaccination rate reached more than 60 percent during the first year the vaccine became available (1982 in Treatment Area 1 and 1985 in Treatment Area 2). Vaccination rates in the comparison area prior to 1989 are believed to have been near zero since the government clinic in the comparison area did not provide vaccines for children until around 1989 (Koenig et al. 1991). Nationally, measles vaccination was less than 2 percent in 1986 (Kahn and Yoder 1998), and remained below 40% for children under age five in the comparison area in 1990 (Fauveau 1994).

Figure 2 shows trends in the contraceptive prevalence rate (CPR) for married women 15-49 increased to 30 percent in the treatment area during the first year contraceptives were provided by the program. A gradual increase followed and the CPR reached almost 50 percent by 1988. Modern contraception was also available at government clinics in both the treatment and the comparison areas during the experimental period (1977-1988), however the comparison area did not receive the intensive care or access provided through home visits. As a result, the CPR in the comparison area was much lower, with rates below 20 percent by 1988.

2.3 Mechanisms linking the MCH-FP program and cognitive functioning

The child health interventions may directly and indirectly affect the cognitive development of young children. Reduction in the incidence of measles and pertussis due to the vaccinations can have a direct effect on cognitive functioning because encephalitis, a complication of both these diseases, results in long-term brain damage (Greenberg et al. 2005, Reingold and Phares 2006).

Vaccine-preventable diseases can also indirectly affect children's cognitive development because the morbidity caused by these diseases may lead to undernutrition and decreased physical activity and play. These effects are likely to be much larger in developing than

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[9] There was limited antenatal and postnatal care. The focus of the antenatal visits was to identify women with high mortality risk, but no medical care referral was provided (Faveau, 1994). The actual interventions were limited to providing nutrition, hygiene, and breastfeeding advice, instructions on how to prepare oral rehydration solution, and the distribution of iron and folic acid tablets. Safe delivery kits were provided to pregnant women starting in 1983 to help improve hygiene around delivery. In addition, eligibility for tetanus toxoid immunization was expanded to all women of reproductive age in 1982.

[10] Other child health interventions, such as control for acute respiratory infections and dysenteric diarrhea, became available at the end of the experimental period in 1988 or later.

developed countries, partly because lower levels of nutrition before infection may weaken the immune system. Infections impair the child's nutritional status through reduced appetite and food intake, malabsorption of nutrients, increased demands from the body due to fever and immune response, and in some cultures food deprivation resulting from parental beliefs about caring for the sick (Reddy 1987, Grantham-McGregor 1999a, b). Measles, in particular, is known to severely impair the child's nutritional status through secondary complications such as pneumonia and diarrhea, and prolonged illness (Reddy 1987). While children's growth may catch up once the illness has passed, in high-disease environments children may experience a number of episodes of illness or diarrhea in combination or in close succession reducing the time for catch-up growth. Indeed, measles can leave a child weakened and at increased risk of illness for a year, and pertussis for months (Greenberg et al. 2005). Nonrandomized and randomized studies show that undernutrition, especially before the age of 3, affects the cognitive development of young children (Grantham-McGregor et al. 1999a and b, Walker et al. 2007). In addition, infections and undernutrition cause general malaise and apathy, resulting in lower levels of play, and apathetic children generally receive less stimulation from adults. Lack of stimulation and learning opportunities have also been shown to hinder cognitive development (Walker et al. 2007).

The child health interventions may also have indirect effects via sibling competition. Healthier children may receive greater parental investment (in the form of quality time or resources spent on education or health care) because of the increase in their potential future returns. An increase in investment in a child who received the interventions may come from an increase in total household resources as a result of the program (i.e. time and resources gained from having fewer children or not having to care for sick children), or from a reduction in investment in the siblings who did not receive the interventions. Alternatively, parents could reduce the resources to the child who received the child health interventions and provide greater resources to a child that did not in order to compensate that child for not having received the health interventions. Given, that the first few years of life are most important for cognitive development (Grantham-McGregar et al. 2007), it is not expected that sibling competition will greatly affect the cognitive function of siblings who are more than 5 years apart in age.

The non-child health components of the MCH-FP program may also have an indirect effect on cognitive development. The family planning program could drive a quality-quantity trade-off, with low-fertility parents bringing greater resources to bear on their children. Again, because cognitive functioning is mainly set early in life, these extra resources need to be provided to the child early in life if they are to significantly impact cognitive functioning. Finally, it seems unlikely that children's cognitive functioning will increase as a result of their mother receiving the tetanus toxoid vaccination. The tetanus toxoid vaccine is given to reduce a baby's chance of contracting neonatal tetanus. If left untreated, children usually die of tetanus rather than suffering from increased morbidity, so we would expect it to lead to decreased mortality (and hence mortality selection) in the treatment area but not decreased morbidity leading to improved cognitive development.

3. Data

3.1 Data sources

This paper draws on the unusually rich data available for the Matlab area. The 1996 Matlab Health and Socioeconomic Survey (MHSS) is the main source of data used for the analyses. It is a comprehensive socio-economic survey covering a wide array of topics typical of large household surveys in developing countries. Unlike most household surveys taken before 2000, it includes a measure of cognitive functioning. These data are publicly available from the Rand website (http://rand.org/labor /FLS/MHSS/. The survey was carried out on a random sample of approximately one-third (2,687) of the baris (residential compounds, which include a number of households who live together) in the treatment and comparison areas.[11] Within each bari, a primary household was selected at random.[12] People older than 59 are excluded from the sample because less than 10 percent of these observations have the necessary mother information for the mother fixed-effects model. The sample includes 5,684 8-59 year olds, and approximately 45 percent of the observations are from the treatment area.

Because the study area is a demographic surveillance site, ICDDR,B, the organization running the demographic surveillance site, takes periodic censuses of the Matlab area and collects high quality demographic data. These data include a census taken in 1974 that provides pre-intervention information on household location, composition, assets, employment, and education, and offer the opportunity to test for pre-program similarity between the treatment and comparison areas. Birth, death, and migration histories were also collected monthly between 1966 and 1996 and are used to examine attrition from mortality or out-migration in the study area.

Finally, ICDDR,B collected data on receipt of program interventions in the treatment area. It includes information on the date and type of each childhood and tetanus toxiod vaccine received, and types of family planning methods used.

An important advantage of these various data sources is that they can all be linked together at the individual or household level.

3.2 Measuring Cognitive Functioning

Cognitive functioning is measured using the Mini Mental State Exam (MMSE). The MMSE examines five areas of cognitive functioning: orientation, attention-concentration, registration, recall, and language. The test was developed as a brief screening test to assess cognitive functioning in adults (Folstein et al. 1975). It has been widely used to assess higher mental functioning and detect cognitive impairment among adults, and modest to high correlations have been found between the total score and other tests of intelligence, memory, attention and executive functioning such as the Wechsler Adult Intelligence Scale (Rush et al 2000). The means and standard deviations of the scores are fairly steady across the various age groups in the adult population so issues of the convergence of scores for certain age groups is not an issue (Strauss et al. 2006). Adaptations of the MMSE are effective at evaluating the cognitive development of children as young as 3 years (Jain and Passi 2005, Ouvrier et al. 1993, Ruvil-Alvarez et al. 2007) and it has been shown to correlated fairly well with the Kaufman Brief Intelligence Test (Rubial-Alvarez 2006).

The MMSE used in this study was adapted so that it would not depend on literacy and would be culturally and age appropriate. Kabir and Herliz (2000) designed a very similar MMSE version, the Bangla Adaptation of the Mini-mental State Examination (BAMSE), which was also

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[11] The MMSE should have been collected for all household members age six and older. By mistake, it was collected from children aged 6 to14 in only the last quarter of households surveyed. In order to obtain a random sample of all children, the MMSE was administered to a 10 percent random sample of the unsurveyed baris.

[12] The survey also collected information on a second household in each bari that was selected purposively. In order for the sample to be representative of the study area, data on the second households are not used in the analysis. The results are similar when the secondary households are included.

adapted for an illiterate population and cultural relevance to Bangladesh. They implemented both the BAMSE and the original MMSE in a literate population in Bangladesh and found that there was a high correlation between the two, indicating that the changes made to adapt the instrument for an illiterate population do not change the ranking of scores.

The MMSE asks 33 questions and gives one point for each correct response, for a maximum score of 33. As an example, in the registration section the enumerator reads the respondent a three sentence story about a house fire and then asks the respondent to repeat the story. There a 6 main points the story makes (i.e. three children in the household, the house is on fire) and the respondent is given a point for each main point they repeat back. Unfortunately, data from the recall questions cannot be used,[13] so 30 is the maximum MMSE score possible. In order to enhance comparison to other studies, the test score for each observation is normalized into a z-score by subtracting the comparison area mean and dividing by the comparison area standard deviation.

The MMSE score is known to vary with age (Holzer et al. 1984). This issue is particularly salient for these analyses because of the wide age range being examined and does indeed vary by age as demonstrated in panel A of Table 2. Age fixed-effects are included in the regression analysis in order to control for the association between age and the MMSE score.

3.3 Intent-to-treat indicator

A variable indicating eligibility based on 1996 MHSS household location might be endogenous, since households could have moved to the treatment area to benefit from the MCH-FP program. To avoid this potential endogeneity, I use 1974 location information. The variable *Treatment Area* takes on the value 1 if the individual (or household if the individual could not be matched to the 1974 census data) resided in a treatment area in 1974, and is zero if from the comparison area.[14]

4. Estimation Strategy

4.1 Quasi-random program design

A comparison group was built into the design of the MCH-FP program; however, randomization was not used to determine which households or villages belonged to the treatment and comparison areas. Instead, the treatment and comparison area are contiguous geographic areas (Figure 1) that were chosen because they were very similar. Separation of the treatment and comparison area was important for mitigating potential spill-over effects to the comparison area from the positive externalities generated by vaccination. Research shows that the treatment and comparison areas are indeed similar with respect to a number of pre-intervention variables including rates of mortality and fertility (Koenig et al., 1990; Menken & Phillips 1990; Joshi & Schultz, 2007). This shows that the program was probably not placed first in areas that had poor child health or high fertility–potential targeting criteria for such programs.

I further test whether the areas are similar using a wider array of household and household head characteristics from the 1974 census. Table 2, Panels B and C display the means and

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[13] Many observations in the recall section had no code at all (not even missing) leading to many more missing observations than for the other areas of the MMSE. Therefore, the recall section was excluded from the MMSE total. Sensitivity analysis was performed to determine if other ways of handling the recall data problem changed the results. Not including the recall questions in the score lead to more conservative findings.

[14] 1974 individual or household information was not available for 400 observations. For these observations 1982 location information was used. The results do not change if these 400 observations are excluded.

standard deviations (SD) of each characteristic for the treatment and comparison areas. The differences in means are statistically insignificant at the five percent level for all variables except drinking water sources and household head's religion. These findings, together with previous results on fertility and mortality, strongly suggest that the two areas had very similar observable characteristics.

Before the program, the treatment area had a larger Hindu population than the comparison area and a 14 percent greater proportion of households used tubewell water for drinking. There are no a priori reasons for cognitive development to be correlated with religion, but tubewell water is often thought to be cleaner than other sources of water. Given a larger percent of treatment area households have access to this water, the program effect may be biased upwards. Unfortunately, there is widespread groundwater arsenic contamination in the tubewells in Bangladesh (Alam et al. 2002, Chowdhury et al. 2000). Arsenic is a serious health concern and has been found to lead to reduced IQ of school-aged Bangladeshi children (Wasserman et al. 2006). So greater access to tubewell water in the treatment area might actually bias the estimate of program impacts downwards. I interact the treatment effect with the source of drinking water to help determine whether such a bias exists.[15]

4.2 Identification Strategy

I seek to determine the intent-to-treat (ITT) or overall program effects of the MCH-FP program on cognitive functioning. I take advantage of the variation in the program implementation across location (treatment versus comparison areas), and the phasing-in of the interventions over time within the treatment area, which left certain age cohorts differently affected by the program, to perform a double difference analysis. Table 1 summarizes program eligibility for four age cohorts: those aged 8-14, 15-19, 20-24, and 25-59 in 1996. I use the 25-59 year old age cohort as a measure of the pre-intervention difference between treatment and comparison areas, and the other three age cohorts measure the post-intervention difference between the experimental areas.

I would like to be able to show that the level of cognitive development was similar between treatment and comparison areas before the interventions. Given the long time span between the pre- and post-intervention surveys (1974-1996), and the lack of cognitive data in the pre-intervention period, it is not possible to examine the before-after program difference in cognitive functioning for any one individual or age cohort. Instead, I examine the cognitive functioning in 1996 of a cohort born prior to the program (25-59 year olds), to measure the pre-intervention difference between the treatment and comparison areas. It is doubtful that the cognitive functioning of the 25-59 year old age cohort was affected directly by the program: they were not eligible for the child health interventions, and, since cognitive development is largely completed before childbearing age, their cognition is not likely to have been affected by their eligibility for the maternal health and family planning interventions during their reproductive year. As expected, the mean MMSE scores in 1996 for this group are exactly the same between the treatment and comparison areas, at approximately 24 out of 30 (Table 2 Panel A).

The age cohort born between 1982 and 1988 (ages 8-14 in 1996) is the only group to benefit directly from the intensive child health interventions, and is the main group of interest in seeking to determine the effect of the child health interventions on cognitive functioning. They all were eligible for the DPT, polio and tuberculosis vaccinations at the recommended age (on-time), those in Treatment Area 1 were eligible for the measles vaccine on-time while those in Treatment Area 2 were eligible for the measles vaccine past the recommended late or late. The

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[15] Interactions effects show the program effects do not differ by religion.

mothers of these children were eligible for family planning and maternal health interventions, so to the extent these interventions indirectly affect cognitive development of this age group, the program effect will be a combination of the child health and family planning and maternal health interventions.

Children aged 15-19 in 1996 were born during the time the program only provided family planning and maternal health interventions in the treatment area. Given the rapid increase of contraceptive use, this age cohort provides an early estimate of the effect of the family planning and maternal health interventions on cognitive functioning, and can be used to help partial out the effects of the family planning and maternal health interventions from the program estimate for the 8-14 year olds.[16] However, it is possible that parental investments for this group would be affected by the eligibility of their younger siblings for the child health interventions in half the treatment area (Treatment Area 1), though it is unclear if this would bias the effects up or down.

It is possible that the family planning program affected the cognitive function of children born prior to the program between 1972 and 1976 (age 20-24 in 1996) indirectly. These children would have been between the ages of 1 and 5 when the family planning program started, and their cognitive development could have been affected by changing child investment patterns as a response to the family planning program.

4.3 Empirical specification

The ITT effect is estimated using a linear double difference model. This model assumes that the treatment and the comparison group would have had the same trend in cognitive functioning in the absence of the MCH-FP program. This is not a testable assumption, but it seems likely given that the mean cognitive functioning was similar between the treatment and comparison areas for the 25-59 year old pre-intervention age cohort (Table 2 Panel A). The double difference model is estimated using the following linear regression:

(1) $C_{imv} = \beta_1 T_v + \beta_2 (T_v * AG^{8-14}_{imv}) + \beta_3 (T_v * AG^{15-19}_{imv}) + \beta_4 (T_v * AG^{20\text{-}24}_{imv}) + \alpha_a + X\,'Z_v + v_{imv}$,

where *C* is the measure of cognitive functioning, MMSE z-score, for person *i* of mother *m* in area *v*. T_v (referred to as *Treatment Area* in the tables) is a binary variable that takes on the value 1 if person *i* or *i's* household was from a treatment area before the MCH-FP program started in 1974, and 0 if from the comparison area. AG^{Y} is a binary variable used to indicate whether person *i* is or is not in age group Y. β_1 represents the difference in mean cognitive functioning between the treatment and comparison area for the 25-59 age group (the pre-intervention cohort). β_2- β_4 are the double difference ITT effects, and represents the difference in mean cognitive functioning between the treatment and the comparison area for the age groups 8-14, 15-19 and 20-24 respectively, subtracting out the pre-program differences in the two areas (using the 25-29 year old cohort). α_a are age fixed-effects to control for differences in the MMSE score due to age as well as other events that may be correlated with age and common to the study population. *X* is a vector of individual (gender and religion) and baseline household and household head characteristics, presented in Table 2. Standard errors are clustered at the village level to account

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[16] Some children born during this time period were eligible for measles vaccination past the recommended age of 9 months since the measles vaccination was available to children under the age of 5 in Treatment Area 1 in 1982. Measles is highly contagious and it takes 10-12 days before the first symptoms appear, so it is likely that many of these children would have already had measles before they became eligible for the measles vaccination. To the extent that some children did benefitted from the measles vaccine, estimates of the effect of the family planning and maternal health interventions on cognitive functioning will be biased upwards.

for the likely intracluster correlation in the error term. This model assumes that various individual, mother or household, and village unobservables are not correlated with T_v because of the quasi-random placement of the program. Models with village and mother fixed-effects will be examined as a robustness check to determine if these unobservables are biasing the results.

While β_4 is estimated, the results are not reported since this age group is not of particular interest and as expected the point estimates are small and insignificant.

5. Program impacts

5.1 Intent-to-treat program effects

Double difference ITT effects controlling only for age fixed-effects and individual characteristics are presented in column 1 of Table 3.[17] They show a statistically significant 0.33 SD increase in the MMSE score for the 8-14 year olds, the age cohort that benefited from the child health interventions. The results remain almost unchanged with the inclusion of pre-program characteristics in Table 3 column 2, 0.38 SD. This provides some confidence that the few differences in baseline characteristics are not biasing the results.[18] The effect size is similar to studies of the benefits of nutrition, in particular iron, on cognitive-language abilities (Walker et al. 2007).

As expected, but importantly, the point estimate is almost zero (-0.02) and insignificant for the variable *Treatment Area* that gives the difference in means between the treatment and comparison areas for the 25-59 year old age group (the group that represents pre-intervention differences in cognitive functioning).

The point estimates are small, negative, and insignificant with and without controlling for pre-intervention characteristics for the 15-19 group. The lack of a significant positive impact on the 15-19 year olds highlight that the family planning and maternal health interventions did not have a strong positive effect on the cognitive development of children in the first five years of the program. This provides suggestive evidence that the program effect on the 8-14 year olds is likely to be a result of the child health interventions.

To examine whether increased education is a mechanism through which the program led to improved cognitive functioning, education level fixed-effects are included (column 3). The point estimates are stable, providing evidence that the MCH-FP program effect on cognitive functioning for the 8-14 years olds is not a result of increased levels of education. An examination of the point estimates on the education level fixed-effects (estimates not reported) shows that the program effect of 0.38 is equivalent to the effect size for completing the first 3 years of primary school.

5.2 Robustness checks

It is possible that non-time varying village or mother unobservables are biasing the results. Village fixed-effects are included in column 4. Because of the inclusion of the village fixed-effects, the coefficient on *Treatment Area* cannot be identified. The age group left out is still the 25-59 year old cohort, and the interpretation of the coefficients does not change. The results show that the effect on the 8-14 year olds remains unchanged. While village fixed-effects will not be included in the other analysis in the paper, I note when their inclusion changes any results.

Introducing mother fixed-effects (MFE) reduces the sample substantially to 887

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[17] Results without the individual controls (sex and religion) are similar.

[18] Robustness analysis using propensity score weighting are not reported but results are similar.

observations.[19] In order to separate out the effect of the change in sample size from the addition of the MFE, I present the double difference estimates using the MFE sample but without the MFE in column 7 and those with the MFE in column 8. The effect of the change in sample size is determined by comparing columns 2 and 7. For the 8-14 year olds, the point estimate increases from 0.38 for the full sample to 0.55 for the MFE sample. Adding the MFE leads to a 0.14 SD increase in the point estimate for the 8-14 year olds to 0.69 and it is marginally significant at the 10 percent level. This suggests that not including maternal fixed-effects may bias the results for the 8-14 year olds downwards.

There was some concern in section 4.1 that the poor balance in access to tubewell water between the experimental groups in the pre-program period may bias the treatment effect. Interaction effects of the double difference estimator for children age 8-14 with a binary variable indicating whether the household used tubewell water for drinking in 1974 are presented in column 5. The results highlight that, if anything, cognitive functioning is lower in households that had access to tubewell water before the program. Since a higher percent of families used tubewell water for drinking in the treatment area before the program, if anything this imbalance is likely to bias the estimates of the program effect downwards. The downward bias is consistent with the presence of arsenic in some tubewell water in Bangladesh.

Finally, a control for the family planning program, a variable which indicates if the child's mother was ever eligible for the family planning program during her fertile years, is included in column 6 as an additional check that that family planning program does not affect cognitive functioning. Again the results remain unchanged providing some further suggestive evidence that the results are likely from the child health interventions.

5.2.1 Spatially correlated errors

Since the treatment and comparison areas are in contiguous geographic areas, it is possible that errors are spatially correlated in either the treatment or the comparison area. This could arise, for example, if there was a health shock such as a disease outbreak in a given year in one area but not the other. These outbreaks are likely to affect the cognitive development of younger children than of older ones, so the double difference model will not control for these unobservables. Clustering at the village level is not sufficient to correct for the resulting lack of independence. To check the possible between-village clustering in the treatment and comparison areas for the 8-14 age group I use the following test. First, I predict the errors from the base model in Table 3, column 2, and average the errors for the 8-14 year olds at the village level. To test whether these village level error terms are correlated, I use Moral's I test using the Euclidean distance between village centroids as weights. I examine whether village level error terms are correlated for the whole sample and for the age group of most interest (8-14 year olds) in the treatment and comparison areas separately. In all cases, I do not find that the errors are spatially correlated.

5.2.2 Mortality and migration attrition

Two prominent causes of attrition in this context are mortality and migration. Even if the MCH-FP program were truly randomized, the program itself is likely to cause mortality and migration to differ between treatment and comparison areas over time, potentially biasing the results. For example, if frailer individuals (or those with lower health endowments) are more likely to survive in the treatment area, then there may be a higher probability of observing someone with a

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[19] This sample excludes all observations for which the mother's identification code was missing and there was no identifiable sibling in the data.

lower level of cognitive functioning in the treatment than in the comparison area in the follow-up period, biasing the results downwards. Similarly, since migration from rural areas in Matlab tends to occur among families with few resources (Kuhn 2003, 2006), the MCH-FP program will likely encourage worse off families who might have migrated to stay in the treatment area, as the program subsidizes the cost of raising children. This type of endogenous migration may again leave a higher proportion of children who have lower human capital (since they come from worse off families) in treated areas, potentially biasing the results downwards.

To address this problem I create attrition bounds for the ITT estimates for the 8-14 year olds using the approach by Lee (2009). To determine the percent of attrition among children born in the treatment and comparison areas resulting from mortality and out-migration between 1974 and 1996 (time between the pre-intervention year and when the MHSS survey was taken), I take advantage of the monthly birth, death and migration data collected on all individuals living in the study site. Taking mortality and out-migration attrition into consideration together the point estimate is always positive and bounded between 0.18 and 0.53.

5.2.3 Spillover effects

Spillover effects occur when the program indirectly affects nonparticipants and will bias the ITT effects. Spillover effects could affect the untreated through the positive externalities of some of the interventions, such as vaccinations, or through informational spillovers. In the comparison area, spillovers are more likely to occur in those areas that border or are closer to the treated villages, since knowledge about the programs is likely to be spread by word-of-mouth in the local area, and the positive externalities of vaccination are more local.

I explore the possibility of spillovers to comparison area villages that border a treatment village using the following linear regression:

$$
\begin{aligned}
(2)\quad C_{imv} = {} & \beta_1 C_v + \beta_2 (CV_v * AG_{imv}^{8-14}) + \beta_3 (CV_v * AG_{imv}^{15-19}) + \beta_4 (CV_v * AG_{imv}^{20\text{-}24}) \\
& + \beta_5 \mathrm{B}_{\mathrm{v}} + \beta_6 (CV_v * AG_{imv}^{8-14} * B_v) + \beta_7 (CV_v * AG_{imv}^{15-19} * B_v) + \beta_8 (CV_v * AG_{imv}^{20-24} * B_v) + \alpha_a + X'Z + v_{imv}\,.
\end{aligned}
$$

CV_v indicates if the person came from a comparison area in 1974 (defined as 1-T_v, where $T_{v,}$ is defined as above). B_v takes on the value 1 if person *i* or person *i*'s household lived in a comparison village that borders a treatment village in 1974 and 0 otherwise. All other variables are defined as above. Equation 2 examines the spillover effect by splitting the comparison area into two groups, those who lived in a village that borders a treatment village in 1974 and those who did not. $\beta_2 - \beta_4$ are the double difference estimators for each age group and show how much lower the outcome variable is in the comparison area that does not border the treatment village than in the treatment area. $\beta_6 - \beta_8$ provide the difference in effect for the various age groups between those who lived (in 1974) in a comparison area village that borders and one that does not border a treatment village.

The point estimates in Table 4, column 1, indicate a positive spillover effect in control areas that border treatment areas for the 8-14 age group, but the effect is not statistically significant. Villages are of varying sizes, and it may be that on average there are no significant spillover effects because spillovers may not extend throughout the whole village, especially in a larger village. I use GIS data to determine the Euclidean distance between the centroid of a comparison village and the border of a treatment village, and create a binary variable to indicate the comparison villages that are in the first quartile of distance (closest) to a treatment village border (*Border treatment village –first quartile*). Using this specification, the MMSE z-score for the 8-

14 year olds is 0.33 SD higher and marginally significant (at the 10 percent level).[20] These findings indicate that there may have been a spill-over effect in the comparison area and the intent-to-treat effects are an underestimate.

5.2.4 Program impacts using the phasing-in of the measles vaccine in the treatment area

I exploit the phasing-in of the measles vaccination over time within the treatment area to provide an additional estimate of the ITT effects of the child health vaccinations on cognitive development, and to estimate an effect which better controls for the family planning and maternal health interventions. Unfortunately, due to small samples sizes, these estimates can only provide a robustness check.

The measles vaccine was phased-in over time in the treatment area; children under the age of five in half the treatment area (Treatment Area 1) were eligible to receive the vaccine starting in 1982 and in the other half (Treatment Area 2) in 1985. As a result, children aged 12-14 in Treatment Area 1 were eligible to receive the measles vaccination at the recommended age while those in Treatment Area 2 were only eligible to receive the measles vaccine past the recommended age. 12-14 year olds in both Treatment Area 1 and 2 were eligible for the other child health interventions at the same time and all of their mothers were eligible for the family planning and maternal health interventions. 8-11 year olds in Treatment Area 1 and 2 were eligible for the child health interventions, including measles vaccination, at the same time and again all their mothers were eligible for the family planning and maternal health interventions. Therefore, the rolling out of the measles vaccination among the 12-14 year olds in the treatment area provides an opportunity to examine if the program effect differs for children who were eligible to receive the measles vaccination on-time versus late.

I first examine whether the double difference results using the same specification as equation 1 but disaggregate the 8-14 year old treatment group to show the program impact on the 12-14 year olds in Treatment Area 1 and Treatment Area 2 separately. Table 5 panel A demonstrates that the intent-to-treat effects are large, 0.44 SD, and significant for those children who were eligible for the measles vaccine at the recommended age (12-14 year olds in Treatment Area 1) but are lower, 0.21 SD, and not statistically significant for those who were eligible for the measles vaccine past the recommended age (12-14 year olds in Treatment Area 2).

To more rigorously estimate the program effect using the rolling out of measles within the treatment area, I restrict the analysis in Table 5 panel B to observations from only the treatment area. I estimate an intent-to-treat double difference estimator using the Treatment Area 1 as the treated group and Treatment Area 2 as a comparison group. Similar to the main analysis, I use the 25-59 year old cohort to measure the pre-intervention differences in cognitive function, and the 8-11, 12-14, and 15-19 year olds as the measures of the post-intervention difference. The sample sizes for the age group of interest are small; there are 42 observations on children aged 12-14 in Treatment Area 1 and 54 in Treatment Area 2. The coefficient on *Treatment Area 1* in Panel B of Table 5 is the difference in the MMSE z-score for the 25-59 year old group between Treatment Area 1 and Treatment Area 2. The coefficient is small, negative, and not significantly different, demonstrating that the MMSE z-score was similar, and if anything lower in the treated area, in the two areas prior to the MCH-FP program.

For the 12-14 year olds, being in the group that was eligible to receive the measles vaccine on-time (Treatment Area 1) resulted in a 0.29 SD increase in cognitive functioning. The point estimate is not statistically significant likely due to the small sample size. However, this is one

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[20] This finding is significant at the 5 percent level when village fixed-effects are included.

case when inclusion of village fixed-effects changes the results. The point estimate is slightly higher, 0.34 SD, and statistically significant at the 10% level (results for all other groups remain unchanged). These estimates used a different source of variation but are similar to the main finding. The effect is slightly lower which is to be expected since it is only measuring the effect of the measles vaccine and not all the child health interventions. However, the effect size is only 9 SD lower indicating that the measles vaccine itself may have accounted for most of the increase in cognitive functioning for these children.

There continues to be a large program effect in Treatment Area 1 for the children aged 8-11, even though for this age group the two areas received the same interventions. Given that measles is highly contagious, it is possible that it is a result of positive spillovers effects to younger children in Treatment Area 1 from their healthier older siblings who received the measles vaccination on-time. An additional explanation is that the administration of the child health interventions to the 8-11 year olds was better in Treatment Area 1 due to the longer experience in that area. As expected there is no effect for the 15-19 year old born prior to the introduction of the child health interventions.

5.3 Results by sub-component of MMSE

Table 6 presents the results by the main subcomponents of the MMSE (orientation, attention-concentration, registration, and language) for the 8-14 year olds. The effects of the program by subcomponent are significant at the 5 percent level for attention-concentration (0.28) and registration (0.37), and at the 10 percent level for orientation (0.28). There are no significant impacts for language, likely because the questions in this section were too easy for this age group. The average score on the language section for 8-14 year olds is six out of seven and the standard deviation is one.

5.4 Results by gender and birth order

Patterns of mortality risk, which in part reflect differential investments (resources and time) in children, vary by family composition in Bangladesh. In particular, first-born children, girls with no sisters, and boys tend to have lower mortality risk (Muhuri and Menken 1997). There are approximately 5 percent more first-born children in the treatment area than in the comparison area for those aged 8-14, most likely because families have delayed having first or later born children due to the family planning program. If the patterns seen in mortality risk also hold for cognitive functioning and are not changed by the introduction of the program, it is possible that the positive program impact on cognitive development is in part a result of there being a higher percent of first-born children in the treatment area. To explore this possibility and the effects by gender and birth order, the ITT effects from Table 3 column 2 for the age 8-14 and 15-19 year olds are interacted with binary variables indicating whether the child is male (*Male*) and whether the child's birth order is second or higher (*Birth Order 2+*).[21] All other interactions of these variables with age group and eligibility dummies are included in the regressions but results not reported. It is important to note that birth order is endogenous so results are not causal.

Results by sex in column 1 of Table 7 show that, similarly for mortality risk, females have not benefited from the program as much as males. The program effect for girls aged 8-14 is a statistically insignificant 0.19 SD increase in the MMSE z-score, but the effect for boys is 0.38

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[21] It is not possible to examine the specific effects of second, third, or higher order births separately or the effect interacted by both sex and birth order because the sample sizes are too small.

SD higher than for girls and the difference between the genders is significant at the 10 percent level.

The sample size is greatly reduced when birth order is introduced into the model due to missing birth order information mainly for those aged 25-59. To separate out the effect due to the reduction in sample size, I replicate the results from Table 3 column 2 with the smaller sample in Table 7 column 2. Birth order interactions are presented in column 3. The program effects for the 8-14 year olds do not follow the pattern of mortality risk. The program did not have an effect on firstborn children but led to a 0.70 SD increase in cognitive functioning for second and higher birth order children. This indicates that the program effect is not likely a result of an increased number of first born children and that it may have benefited those who otherwise would have been likely to receive fewer investments.

5.5 Treatment on the treated effects

It is important to determine the impact on those who participated in the program, rather than those who were eligible. Since program take-up is likely to suffer from self-selection bias, I use an instrumental variables approach in which program take-up (or receipt) is instrumented by program eligibility. The main preventative interventions provided by the program to children were vaccinations. The indicator of receipt of child health interventions (*All Vaccinations*) is a binary variable indicating whether the child received all the program vaccinations by the age of five. *All Vaccinations* is instrumented by a variable indicating whether the child was eligible to receive the vaccinations (*Treatment Area*Age 8-14*). Two-stage least square (2SLS) is used to estimate the model and given the quasi-random program design the exclusion restrictions are likely to hold. However, because the 2SLS model assumes no spillovers in the treatment area, the effects represent an upward bound since it is possible there are spillover effects from the vaccinated to the unvaccinated among the 8-14 year olds in the treatment area.

The first stage regression in column 1 of Table 8 show that the instrument is highly correlated with a child receiving all the program vaccinations and that the analysis does not suffer from weak instruments (the f-statistic on the excluded instrument is 141). At 0.92 SD (column 2), the effect of the program on the treated is more than double the intent-to-treat effects, and demonstrates that the provision of vaccinations has a large effect on cognitive functioning.

In order to control for the possible separate effect of the family planning interventions on cognitive functioning, a control for the number of siblings born alive is included (*number of siblings)*. Because the number of siblings is endogenous, it is instrumented with a variable indicating whether the child's mother was ever eligible for the family planning program in any of her fertile years. The sample size is reduced since the number of siblings is missing for the majority of observations on those over age 22. The first stage regressions (columns 4 and 5) show that the instruments are strongly correlated with the endogenous variables and are not weak (the f-statistics are 16 and 79). The inclusion of the number of siblings does not substantially change the results, though the point estimate of the impact of vaccinations increases slightly to 1.03 SD. On the contrary, the number of siblings does not have an effect on a child's cognitive functioning providing further evidence that the family planning program did not likely have a substantial effect on cognitive functioning.

6. Conclusions

This paper examines the longer-term effect of a maternal and early childhood health and family planning program in Bangladesh on the cognitive functioning of children who were eligible to receive important early childhood health interventions, such as vaccination against measles, once they reached ages of 8-14. The advantages of evaluating this program include (i) the quasi-random placement of the program in a treatment/control design, (ii) the rolling out of the program interventions over time allowing for comparisons of age cohorts who were differentially affected or not affected by the program, (iii) passing of sufficient time to permit longer term evaluation of the effects of the interventions, (iv) available of a rich set of data allowing examining of the treatment and comparison area pre-intervention balance and estimation of mortality and migration attrition bounds, (v) examination of the effects of perhaps some of the most important health interventions, for which evaluation is now difficult due to the widespread use of these vaccinations.

The findings show that children who were eligible for the early child health interventions under the age of five experienced a 0.38 SD increase in their cognitive function, as measured by the Mini Mental State Exam, when they were 8-14 year olds. An effect size of 0.38 SD is large and similar to effect sizes in studies of the benefit of nutrition programs, and is equivalent to the effects size in the same sample for completing 3 years of primary school. The effect among children who actually received all the program vaccinations was more than twice as large at almost 1 SD. These large program effects provide needed causal evidence of the importance of child health interventions for future opportunities through better cognitive functioning, even in a high disease environment where competing health risks may have led to fading out of the effects of the interventions.

Comparison villages neighboring treatment villages experienced positive spillover effects. This highlights the advantage of this study in having geographic separation between many of the treatment and comparison villages. It also emphasizes the need for randomized or non-randomized evaluations of health and nutrition interventions, where positive spillovers can be substantial, to ensure sufficient distance between treatment and control areas, or to randomize the interventions at a level of larger units, such as a cluster of villages.

Educational attainment is often used as a measure of the longer-term effects of health and nutrition programs on human capital, and has been thought to proxy for cognitive functioning. My findings show that program impacts on cognitive functioning remained unchanged with the inclusion of education level fixed-effects, demonstrating that this is not the case. While level of education attained or test scores, are certainly correlated with cognitive ability, educational outcomes are a function many other factors, such as cost of enrollment, school quality and access, policies on automatic promotion, security, and labor market opportunities. As such, educational outcomes are unlikely to reflect cognitive functioning accurately and may fail to show effects for certain subpopulations such as girls.[22] While collection of cognitive functioning data is more difficult, determining the effects of health and nutrition programs on the various domains of cognitive functioning is important and may yield different results than educational attainment.

One limitation of this study is that the family planning and child health interventions were not randomly introduced in a factorial design, making it difficult to determine their effects separately. The total program effect is itself of great interest, since these interventions are commonly provided in combination and vaccination is such an important health intervention. An

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[22] Girls may drop out of school early because of early marriage or their higher productivity in the home, rather than lower cognitive ability.

important contribution of this paper has been to demonstrate that the program effect is likely as result of the child health interventions. To do this I perform an number of analyses. First, I separately estimate the effect of the program for children whose mothers were eligible for the family planning program but born prior to the availability of the child health interventions (15-19 year olds) and find the effect of the program is small, negative and statistically insignificant. This analysis is by itself insufficient since the family planning program can be expected to have a greater effect on later-born children since parental resource constraints may be tighter. Therefore, I include a control for the family planning program in the ITT and TOT models and find no evidence that the family planning program leads to improved cognitive function. Lastly, I exploit the phasing-in of the measles vaccine over time in the treatment area to partial out any effect of the family planning program, and find that the ITT effect for the measles intervention alone was approximately 0.3 standard deviations. While any one of these analyses is insufficient, they all point to the importance of the child health interventions themselves, and taken together suggest that is the child health interventions that were responsible for the improvement in cognitive functioning.

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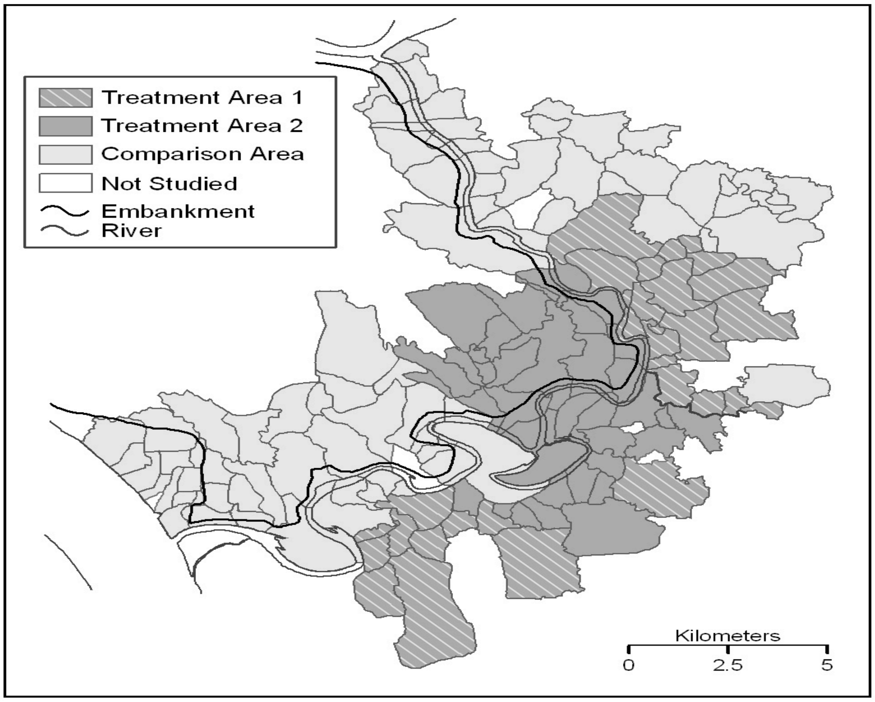
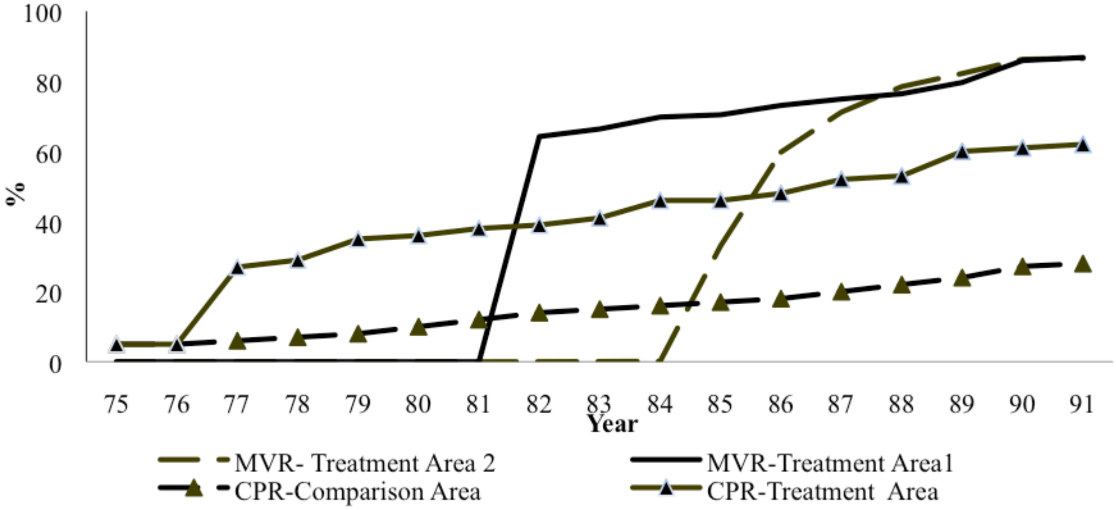
Figures

Figure 1: Map of Matlab study



area

Figure 2: Trends in measles vaccination (MVR) rates for children 12-59 months and contraceptive prevalence (CPR) and treatment area by calendar year



Source: Contraceptive use data van Ginniken et. al. 1998; Measles vaccination data is from ICEDR,B Record Keeping System.

Tables

Table 1: MCH-FP Program Eligibility

Year Born	Age in 1996	Program Eligibility
1982-1988	8-14	*Born during MCH-FP experiment - Child health, family planning and maternal health interventions* Preventive Child Health Interventions: On-time or late vaccination (measles, DPT, polio, tuberculosis) Rehabilitative Child Health Interventions: nutrition rehabilitation, vitamin A supplementation Mother eligible for family planning and maternal health interventions
1977-1981	15-19	*Born during MCH-FP experiment - Family planning and maternal health interventions* Preventive Child Health Interventions: late measles vaccination possible in half the treatment area Mother eligible for family planning and maternal health interventions
1972-1976	20-24	*Born before MCH-FP experiment - No interventions but possible sibling competition* Not eligible for child health interventions and unlikely to use family planning and maternal health interventions Potentially affected by the program through sibling competition
1938-1971	25-59	*Born before MCH-FP experiment - Pre-Intervention group* Women of reproductive age eligible for family planning and pregnant women maternal health interventions

Notes: MCH-FP experiment refers to the years 1977 to 1988, when most of the program interventions were available only in the Treatment area and not in the comparison area; DPT=Diphtheria-Pertussis-Tetanus

Table 2: Differences in Means between Treatment and Comparison Areas

	Treatment Area			Comparison Area			Difference in Means	
	Mean	SD	Obs	Mean	SD	Ob	Diff	T-stat
Panel A. Characteristics from 1996 MHSS								
MMSE for age 25 to 59	23.93	0.21	1808	23.93	0.18	2067	0.00	0.00
MMSE for age 20 to 24	24.94	0.34	237	25.21	0.27	271	-0.27	-0.64
MMSE for age 15 to 19	25.05	0.27	342	25.39	0.27	445	-0.34	-0.91
MMSE for age 8 to 14	22.67	0.55	193	20.79	0.53	321	1.89	2.49
Age	32.82	0.47	2636	32.00	0.32	3165	0.81	1.45
Female (=1)	0.57	0.01	2636	0.55	0.01	3165	0.02	2.52
Hindu (=1)	0.16	0.03	2636	0.05	0.01	3165	0.11	3.77
Years of education	3.59	0.23	2621	3.02	0.11	3149	0.58	2.78
Mother's years of education	1.15	0.14	2563	0.93	0.06	3097	0.22	1.85
Mother ever eligible for MCH-FP (=1)	0.58	0.01	2562	0.00	0.00	3081	0.58	43.12
Panel B. Household Characteristics from 1974 Census								
Family size	7.00	0.10	2481	6.82	0.10	2929	0.18	1.25
Owns a lamp (=1)	0.66	0.03	2481	0.61	0.02	2929	0.05	1.51
Owns a watch (=1)	0.17	0.02	2481	0.16	0.01	2929	0.01	0.48
Owns a radio (=1)	0.09	0.01	2481	0.09	0.01	2929	0.00	0.08
Wall made of tin (=1)	0.09	0.01	2476	0.07	0.01	2913	0.02	1.37
Wall made of tinmix (=1)	0.24	0.01	2434	0.25	0.01	2886	-0.01	-0.58
Roof made of tin (=1)	0.83	0.02	2477	0.83	0.01	2914	-0.01	-0.32
Latrine in household compound (=1)	0.83	0.02	2481	0.85	0.02	2929	-0.03	-1.22
Number of rooms per capita	0.21	0.00	2481	0.21	0.00	2929	-0.00	-0.02
Number of cows	1.52	0.08	2481	1.35	0.07	2929	0.17	1.70
Number of boats	0.69	0.04	2481	0.67	0.03	2929	0.02	0.37
Drinking water from tube well (=1)	0.30	0.03	2481	0.16	0.02	2926	0.14	3.85
Drinking water from tank (=1)	0.38	0.04	2481	0.32	0.04	2926	0.06	1.14
Drinking water other source (=1)	0.32	0.05	2440	0.52	0.04	2903	-0.20	-3.29
Panel C. Household Head Characteristics 1974 Census								
Age	47.19	0.50	2473	46.01	0.45	2927	1.18	1.78
Hindu (=1)	0.16	0.03	2440	0.05	0.01	2906	0.11	3.51
Years of education	2.61	0.16	2481	2.40	0.12	2929	0.21	1.38
Primary occupation is agriculture (=1)	0.60	0.02	2481	0.59	0.02	2929	0.01	0.38
Primary occupation fishing or boatman (=1)	0.05	0.01	2481	0.07	0.01	2929	-0.01	-0.81
Spouse's years of education	1.17	0.09	2258	1.27	0.07	2597	-0.10	-1.07
Spouse's age	36.60	0.45	2254	35.56	0.40	2597	1.04	1.71

Notes: SD = standard deviation, Obs = observation; Diff = difference, T-stat = T- statistic; Standard errors are clustered at the village level.

Table 3: Intent-to-Treat Program Effects for the MMSE Z-Score by Age Group

	Full Sample: Double Difference OLS						MFE Sample	
							DD OLS	MFE
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Treatment Area (=1)	0.02	-0.02	-0.05		-0.01	-0.01	-0.17	
	(0.05)	(0.05)	(0.05)		(0.06)	(0.05)	(0.13)	
Treatment Area*(Age 8-14)	0.33*	0.38**	0.38**	0.37**	0.40*	0.37*	0.55*	0.69+
	(0.15)	(0.14)	(0.14)	(0.14)	(0.16)	(0.15)	(0.22)	(0.40)
Treatment Area*(Age 8-14)*Tubewell drinking water					-0.31			
					(0.31)			
Treatment Area*(Age 15-19)	-0.08	-0.07	-0.07	-0.10+	-0.02	-0.08	-0.15	0.15
	(0.06)	(0.06)	(0.06)	(0.06)	(0.07)	(0.07)	(0.17)	(0.35)
Mother ever eligible for FP						0.01		
						(0.04)		
Individual characteristics	Y	Y	Y	Y	Y	Y	Y	Y
Pre-intervention characteristics	N	Y	Y	Y	Y	Y	Y	Y
Education level fixed-effects	N	N	Y	N	N	N	N	N
Village fixed-effects	N	N	N	Y	N	N	N	N
Mother fixed-effects	N	N	N	N	N	N	N	Y
Observations	5684	5684	5656	5527	5527	5295	887	887
Adjusted R-Squared	0.14	0.19	0.29	0.19	0.20	0.25	0.32	0.56

Notes: Standard errors are clustered at the village level. "**" ,"*", "+" indicates that the difference in the coefficient from zero is statistically significant at 1 percent, 5 percent, and 10 percent significance level. Individual characteristics include age fixed-effects and dummies for being female and for the Islamic religion. Pre-intervention characteristics include all household and household head characteristics from 1974 presented in Table 2. OLS=Ordinary least square, MFE = mother fixed-effects.

Table 4: Spillover Effects on MMSE Z-Score

	(1)	(2)
Comparison Area (=1)	-0.02	0.00
	(0.06)	(0.06)
Comparison Area*(Age 8-14)	-0.45**	-0.47**
	(0.15)	(0.15)
Comparison Area*(Age 15-19)	0.06	0.06
	(0.07)	(0.07)
Comparison Area*(Age8-14)*Border treatment village	0.22	
	(0.18)	
Comparison Area*(Age15-19)*Border treatment village	-0.03	
	(0.07)	
Comparison Area*(Age8-14)*Border treatment village - first quartile distance		0.33+
		(0.17)
Comparison Area*(Age15-19)*Border treatment village – first quartile distance		-0.05
		(0.07)
Observations	5462	5462
Adjusted R-Squared	0.2	0.20

Notes: Standard errors are clustered at the village level. "**" ,"*", "+" indicates that the difference in the coefficient from zero is statistically significant at 1 percent, 5 percent, and 10 percent significance level respectively. All regressions include age fixed-effects, individual controls (gender and religion) and household and household head characteristics from 1974 presented in Table 2.

Table 5: Intent-to-Treat Effects on MMSE Z-Score Disaggregated in the Treatment Area

Panel A: Full Sample DD OLS		***Panel B: Treatment Area Sample DD OLS***	
Treatment (=1)	-0.01	Treatment Area 1 (=1)	-0.10
	(0.05)		(0.07)
Treatment Area*(Age 8-11)	0.44*	Treatment Area 1*(Age 8-11)	0.30
	(0.17)		(0.24)
Treatment Area*(Age 12-14)		Treatment Area 1*(Age 12-14)	0.29
			(0.25)
Treatment Area 1*(Age 12-14)	0.44**	Treatment Area 1*(Age 15-19)	-0.00
	(0.17)		(0.09)
Treatment Area 2*(Age 12-14)	0.21		
	(0.25)		
Treatment*(Age 15-19)	-0.06		
	(0.06)		
Observations	5684		2580
Adjusted R-Squared	0.2		0.21

Notes: Standard errors are clustered at the village level. "**" ,"*", "+" indicates that the difference in the coefficient from zero is statistically significant at 1 percent, 5 percent, and 10 percent significance level respectively. All regressions include age fixed-effects, individual controls (gender and religion) and household and household head characteristics from 1974 presented in Table 2.

Table 6: Effects by Subcomponent of MMSE (Z-Scores)

	Orientation	Attention-Concentration	Registration	Language
Treatment Area*(Age 8-14)	0.28+	0.28*	0.37*	0.20
	(0.14)	(0.11)	(0.14)	(0.17)
Treatment Area (=1)	-0.01	0.02	-0.08	0.10
	(0.04)	(0.03)	(0.07)	(0.08)
Observations	5684	5684	5684	5684
Adjusted R-Squared	0.23	0.18	0.09	0.05

Notes: Standard errors are clustered at the village level. "**" ,"*", "+" indicates that the difference in the coefficient from zero is statistically significant at 1 percent, 5 percent, and 10 percent significance level respectively. All regressions include age fixed-effects, individual controls (gender and religion) and household and household head characteristics from 1974 presented in Table 2.

Table 7: Gender and Birth Order Effects on MMSE Z-Score.

	Full Sample	No Missing Birth Order Sample	
	(1)	(2)	(3)
Treatment Area (=1)	-0.00	-0.05	-0.00
	(0.06)	(0.11)	(0.17)
Treatment Area*(Age 8-14)	0.19	0.45**	-0.11
	(0.18)	(0.16)	(0.30)
Treatment Area*(Age 15-19)	-0.05	-0.08	0.10
	(0.08)	(0.11)	(0.19)
Treatment Area*Male	-0.01		
	(0.05)		
Treatment Area*(Age 8-14)*Male	0.38+		
	(0.20)		
Treatment Area*(Age 15-19)*Male	-0.03		
	(0.10)		
Treatment Area*Birth Order 2 +			-0.06
			(0.21)
Treatment Area*(Age 8-14)*Birth Order 2 +			0.70*
			(0.35)
Treatment Area*(Age 15-19)*Birth Order 2 +			-0.25
			(0.26)
Observations	5684	1695	1695
Adjusted R-Squared	0.21	0.26	0.27

Notes: Standard errors are clustered at the village level. "**" ,"*", "+" indicates that the difference in the coefficient from zero is statistically significant at 1 percent, 5 percent, and 10 percent significance level respectively. All regressions include age fixed-effects, individual controls (gender and religion) and household and household head characteristics from 1974 presented in Table 2.

Table 8: Treatment-on-the-Treated Effects

	First Stage Equation Received all MCH-FP vaccines	Second Stage Equation MMSE Z-Score		First Stage Equations		Second Stage Equation MMSE Z-Score
				Received all MCH-FP vaccines	Number of Siblings	
	OLS	2SLS	2SLS	OLS	OLS	2SLS
	(1)	(2)	(3)	(4)	(5)	(6)
Endogenous variables						
Received all MCH-FP vaccines (=1)		0.92*	0.95**			1.03**
		(0.39)	(0.34)			(0.34)
Number of siblings						0.12
						(0.11)
Instruments						
Eligible*(Age 8-14) (=1)	0.41**			0.43**	0.27	
	(0.03)			(0.3)	(.27)	
Mother Eligible for MCH-FP (=1)				0.00	-0.61**	
				(0.00)	(0.10)	
Sample with no missing number of siblings	N	N	Y	Y	Y	Y
F-statistic on excluded instruments	141			79	16	
Observations	5681	5681	1894	1894	1894	1894
Adjusted R-Squared /Partial R-squared	0.33	0.19	0.25	0.35	0.01	0.15

Notes: Standard errors are clustered at the village level. "**" ,"*", or "+" indicates that the difference in the coefficient from zero is statistically significant at the 1 percent, 5 percent, or 10 percent level, respectively. All regressions include age fixed-effects, individual controls (gender and religion), and household and household head characteristics from 1974 presented in Table 2.