Effects of Family Planning and Child Health Interventions on Adolescent Cognitive Functioning: Evidence from Matlab in Bangladesh.[1]

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Abstract: Early childhood health interventions, such as vaccinations, improve the health status of young children in developing countries. It is believed that improvements in health and nutrition early on may lead to improved cognitive development, health, educational achievements, and labor market opportunities in the future. It is unclear, however, whether the benefits of these interventions are long-term, continuing into adolescence and adulthood, especially in environments where there are many competing health risks. This paper exploits a quasi-random placement and the phasing in of the Matlab Maternal and Child Health Project in Bangladesh to determine if typical family planning and early childhood health interventions received in childhood have lasting effects on cognitive functioning in late childhood and adolescence. Single and double difference with propensity score weighting as well as mother fixed-effects models are used to determine the intent-to-treat effects of the program. We estimate the intent-to-treat effects for those children whose mother was eligible for the family planning program when they were born, but born before most of the child health interventions were available, and for those children who were born when both components of the program were available. Eligibility for both aspects of the program in childhood leads to 0.3 standard deviation increase in cognitive functioning as measured by the Mini Mental State Exam when these children are 8-14 years of age. We find no program effect on the 15-19 year olds who may have benefited from the family planning program but not the child health interventions.

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1. Introduction

The health of young children is of importance not only for the immediate improvement in their well-being but also because of the longer-term impacts on children's physical and cognitive development. It is estimated that more than 200 million children under the age of five in developing countries fail to reach their cognitive potential due to poor health, nutrition and poverty (Grantham-McGregor et al. 2007). It is believed that improvements in health and nutrition early on may lead to improved cognitive development, health, educational achievements, and labor market opportunities in the future (Barker 1995, Mendez and Adair 1999, Currie and Thomas 1999, Painter et al. 2005, Case and Paxson 2006). Economic theories of human capital development and reduction of inter-generational transmission of poverty rely on this link. Yet, in developing countries, where there are many competing health risks, it is unclear if the benefits of a limited number of health interventions early on in life will continue through into adolescence and adulthood. This paper takes advantage of a quasi-random placement of the Matlab Child Health and Family Planning Program (MCH-FP) to evaluate the impact of important early childhood health interventions (such as vaccinations) coupled with a family planning program in Matlab Bangladesh on the cognitive functioning of those same children in late childhood and adolescence.

Rigorous examination of effects of early child-health interventions on later human capital attainment is challenging. Health interventions are usually not placed randomly and it is difficult to find a similar control area, so program placement bias may affect the results (Rosenzweig & Wolpin, 1986; Pitt et al. 1995). If a program targets worse-off areas first, results are biased downward, making it difficult to find positive program effects. Studies that use program uptake (e.g., vaccination receipt) to measure program effect suffer from selection bias since factors that influence uptake likely influence human capital. In addition, estimating long-term effects of early childhood health programs is challenging owing to a lack of detailed, longitudinal data from well-designed programs that took place 10 or more years ago.

A handful of papers examine the long-term effects of health on human capital using longitudinal data and econometric techniques, such as instrumental variables, to address the methodological issues discussed above (Glewwe & Jacoby 1995; Glewwe et al. 2001, Alderman et al. 2001, Bloom et al. 2005, Alderman et al. 2006, Chen & Zhou 2007, Fields et al. 2008). Many of these papers use height as an indicator of past health status and most use education outcomes as a measure of human capital. Educational outcomes such as level of education attained or scores on math or reading exams are correlated with cognitive ability. However, these educational outcomes are also a function of many other factors including the cost of enrollment,[2] school quality and access, child's record or attendance, or parent's level of education (Connolly and Kvalsvig, 1993). Paxson and Case (2008) use data from the United Kingdom to provide evidence that the link between height and test scores is likely to be a result of cognitive ability rather than factors such as self-esteem, social dominance, or discrimination. Research on cognitive functioning as an example of human capital formation is limited in developing countries and focuses on effects of nutrition or conditional cash transfers as apposed to infection from disease (Paxson and Schady 2007; Grantham-McGregor 1999a, 1999b; Walker, 2007; Fernald et al., 2008; Martorell, 2005).

To contribute to this needed body of research, this study takes advantage of the quasi-random placement of the Bangladesh Maternal and Child Health and Family Planning (MCH-FP) in a

[2] Girls may drop out of school early not due to cognitive ability but due to their higher productivity in the home or early marriage.

treatment and comparison area. Pre-program census data shows that these two areas are indeed similar. The program started in 1977 with a family planning and maternal health interventions. In 1982 and 1985 vaccinations (measles, DPT[3], and polio) for children under five were introduced and other health interventions followed in later years. Using the phasing in of the program over time and geography, we examine the intent-to-treat effects by age groups using single and double difference estimators as well as a mother fixed-effect models and propensity score weighting to control for potential unobservables. Depending on the model, the program led to a 6-13 percent increase in cognition functioning as measured by the Mini Mental State Exam. Given the lack of effects for children born in the treatment area when the family planning program was being implemented but before the child health intervention were available, the effects are likely from the child health interventions and not the family planning program. Lastly, these findings are over and above the effect that education may have on cognition.

The rest of the paper proceeds as follows. In section 2, we describe the MCH-FP program and the mechanisms through which this program may affect cognitive functioning. Section 3, describes the various data sources and variables used in the analysis. We present the identification strategy in section 4 and results and conclusions in sections 5 and 6 respectively.

2. Background

2.1. The Matlab Child Health and Family Planning Program

In the early 1960s, ICDDR,B (formerly known as International Center for Diarrhoeal Disease Research, Bangladesh) began the Matlab Health and Demographic Surveillance System (HDSS) in the a rural region of Bangladesh called Matlab. This study site covered approximately 200,000 people and provided at least monthly data on important demographic events such as births, deaths and migration (Faveau, 1994). In 1977, ICDDR,B started a Maternal and Child Health and Family Planning (MCH-FP) program in approximately half of the HDSS site, leaving the other half as a comparison area. The main goal of the MCH-FP program was to reduce fertility rates by hiring local female community health workers (CHW) to visit each household regularly in the treatment area. During these visits CHWs provided women with a variety of contraceptives and regularly followed up to encourage continued use. In addition, CHWs provided some simple health interventions such as administering tetanus toxiod shots to pregnant women, providing iron and folic acid tables, and providing advice on nutrition and breastfeeding (Fauveau, 1994).

Starting in 1982, ICCDR,B phased-in a number of child health interventions in all or some of the treatment area in order to reduce mortality and morbidity among children. The treatment area was further spilt into four blocks (A, B, C, and D). The comparison area is block E. In particular, in blocks A and C in 1982 measles vaccination was provided to all children between the ages of 9 months and five years, tetanus toxoid immunization was expanded to all women of reproductive age (as apposed to just pregnant women), and antenatal care was provided in blocks A and C. In 1986, all interventions were expanded to blocks B and D. At the same time, vaccination against diphtheria, pertussis, and tetanus (DPT), polio, tuberculosis, and vitamin A supplementation and nutrition rehabilitation were introduced in the treatment area for children aged 5 and under. In 1988, control for acute respiratory infections and dysenteric diarrhea was also introduced in the treatment area (Fauveau, 1994). While government health services were available in treatment and comparison areas, the comparison area did not receive the intensive

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

care provided by the home visits, nor did they have access to the vaccinations in government clinics until after 1988. Data on coverage for measles from the HDSS Record Keeping System (RKS) displayed in Figure 1 demonstrates that the program was implemented according to planned time line. The RKS data is only available for the treatment area, and no data is available from the comparison area before 1991.

The analysis will take advantage of both the geographic variation of the program (treatment versus comparison area) as well as the phasing-in of the MCH-FP over time within the treatment area. Table 1 summarizes program eligibility by year of birth for the various interventions and provides the age of each group in the 1996 MHSS survey. Children who were eligible for the child health interventions are split into two groups; the "non-intensive" and "intensive" child health groups. Children in the "non-intensive" child health group are aged age 15-19 in the 1996 MHSS survey. Mother's of these children were eligible for the family planning interventions and these children became eligible for the measles vaccine past the recommended age of one, but were not eligible for the other vaccines or health interventions.[4] In particular, children in this group were only eligible for the measles vaccine between the ages of 2 and 5. The children in the "intensive" child health group are less than age 15. These children were eligible to receive all the vaccines at the recommended age or by age 3 or 4. Those aged 10 or younger were eligible for all their vaccinations by the recommended age and may have benefited from the non-vaccination child health interventions. For the analysis we examine the effects on the 6 to 7 year olds separate since some of the same child health interventions were available in the comparison area a this time so the effect of the MCH program may be smaller.

2.2. Mechanisms through which the MCH-FP affect cognition

There are number of mechanisms through which the program interventions could effect cognitive development. Reduction in the incidence of measles and pertussis due to the vaccinations can have a direct effect on cognitive functioning because encephalitis is a complication of both these disease and results in long-term brain damage (Greenberg et al. 2005, Reingold and Phares 2006).

Vaccine preventable diseases can also indirectly affect children's development because the morbidity resulting from the disease leads to undernutrition and decreased physical activity and play. These effects are likely to be much larger in developing than developed countries partly due to the lower levels of nutrition prior to infection which results in a weakened immune system. Infections from disease adversely affect the nutritional status of the child due to reduced food intake while sick,[5] malabsorption of nutrients, increased demands from the body due to fever and immune response, and in some cultures food deprivation resulting from parental beliefs for caring for the sick (Reddy 1987, Grantham-McGregor 1999a, b). Measles, in particular, is known to have a severe effect on the child's nutritional status due to 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 diaharrea in combination or in close succession not providing enough 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). Reviews of non-randomized and randomized studies show that undernutrition in developing countries, especially

[4] Some children in blocks A and C may have been eligible for the other vaccines at age 5 depending on their birth date and when the program reached their area during the year.

before the age of 3, can have long lasting effects on cognitive functioning (Grantham-McGregor et al. 1999a,b , Mortell et al. 2005,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 or learning opportunities have also been shown to have long-term effects on cognitive development in developing countries (Walker et al. 2007).

Each element of the MCH-FP program may also have indirect effects of cognitive development through changes in parental investment in children. For instance, children that are healthier as a result of the child health interventions may receive greater parental investment (in the form of quality time or money) because of the increase in the potential future returns of this child. The family planning program could drive a quality/quantity tradeoff (Becker 1960, Becker and Lewis 1973, Becker and Thomas 1976), with low fertility parents bringing greater resources to bear on the remaining children. Both family planning and maternal health interventions could lead to improved maternal survival, which could in turn lead to greater nurturing from mothers as well as a more favorable allocation of resources towards children.

This increase in investment in the child who received the interventions may come from the increase in total household resources as a result of the program (i.e. time and money gained from children not being sick or having less children), or from a reduction in investment in the siblings that did not receive the interventions (what we refer to as sibling competition). If sibling competition is present, it is possible that the program could have a negative impact on the cognition of children who received no or less intensive child health interventions (i.e. those aged 15 or older according to the age groups in Table 1).

3. Data

3.1. Data Sources

Our paper draws on a rich set of data available on the Matlab area and benefits from the ability to link each of the datasets. The 1996 Matlab Health and Socioeconomic Survey (MHSS) provides data on cognition and large number of other demographic and socio-economic variables. These data are publicly available from the Rand website (http://rand.org/labor/FLS/MHSS/). Census data is available from ICCDR,B for all HDSS households (i.e. all treatment and comparison households) for 1974, 1982 and 1993. Finally, the ICCDR,B Record Keeping System (RKS) provides data on actual receipt of the interventions provided under the MCH-FP program in the treatment area. At present, data is available only on the date of vaccination receipt for each type of vaccine in the treatment area. No data is available of vaccination receipt before 1991. A great advantage of these datasets is that they all contain unique identifiers that allow individuals and households to be linked across each of the datasets.

The 1996 Matlab Health and Socioeconomic Survey (MHSS) is a comprehensive household survey covering a wide array of topics typical of large households surveys in developing countries. Unlike most household surveys before 2000, it also includes a measure of cognition, the Mini Mental State Exam (MMSE). It is a random sample of approximately one third (2,687) of the baris (residential compound which include a number of households who live together) in the treatment and comparison areas. Within each bari, a primary household was selected at random. A second household from each bari was also selected purposively. We use both the households in each bari to provide as large a sample size as possible.

The MMSE should have been collected for household members greater than age 6 in the MHSS. Due to a mistake in the field, the MMSE was not collected from children ages 6 to 14, except from the last quarter of households surveyed. In order to obtain a random sample of children for the cognitive test, a 10 percent random sample of the bari's was selected and the MMSE was administered to the appropriate respondents in those baris. As a result, there are a total of 11,218 observations for those aged 6 and older with some cognitive data and that have unique individual identifiers used for matching to other dataset. We further restrict the sample to those less than aged 60 because there is no mother information available for 91 percent of those older than age 59 leaving few observations in this age group when the mother fixed effects are included. This leaves a sample of 9,598 6 to 59 year olds however only 9,164 of these have non-missing information on all parts of cognition (see Section 3.3). Approximately 47 percent (4,333) of these observations are from the treatment area.

The MHSS data was designed to be linked to the census data collected by ICCDR,B for demographic surveillance. Census data are available for the pre-intervention period (1974) and during the start of the child health interventions (1982). These data include information on household location, composition, assets, employment, and education. They provide the opportunity to test the similarity of the treatment and comparison areas at baseline, to control for pre-program household characteristics in the analysis, and to determine program eligibility based on the individual's household location prior to the interventions since individuals 1996 location may be endogenous.

3.2. Dependent Variable: The Mini Mental State Exam (MMSE)

The measure of cognition used for the analysis is the MMSE. It examines five areas of cognitive functioning: orientation, attention-concentration, registration, recall, and language. The results present data for the total score as well as for each area of cognitive functioning separately. The MMSE was originally developed to assess the cognitive status of geriatric patients. However, research has shown adaptations of the MMSE for children are effective at evaluating the cognitive development of children as young as 3 years (Jain and Passi 2005, Ouvrier et al. 1993). The MMSE was adapted for the study so that it would not depend on literacy skills and would be culturally- and age-appropriate.

The MMSE in the MHSS asks 33 questions and each question is given one point if the respondent correctly answers the question for a maximum score of 33. For example the recall section asks respondent remember three words (orange, house, cat) and then asks the respondent to repeat those words a few minutes later. They would get one point for each word remembered. In order to enhance comparison to other studies the test score is normalized for each observation by subtracting the mean for the sample and dividing by the standard deviation.

There are a total of 9,598 observations in which at least one of the 33 questions was answered. In some cases one or more responses to the cognitive questions were coded as don't know or missing. All don't knows are coded as zeros since they did not correctly answer the question. However, it is unclear how to handle the missing observations. If the data is missing because the respondent refused to answer the questions this is likely because the respondent did not know the answer in which case they should be coded as zeros. However, the missing could reflect a mistake by an enumerator who accidentally skipped some of the questions. Each area of cognitive function had less than 200 missing observations with the exception of recall which had more than a 1,000 missing observations. This is likely a result of lack of clarity in the survey instruments and documentation since the majority of the missing observations have no code at all (not even a missing code) in the public data. Our best guess is that they indicate the respondent could not remember any of the three objects so should be coded as zero. Since it is unclear how to code the recall section, the MMSE score used for the analysis excludes the recall section so the total score is out of 30. For the final analysis, missing observations for any part of the test were not recoded, leaving a final sample of 9164 observations. As a robustness check we also recoded the missing to zeros and include the recall section. The results were similar but slightly larger so we present the more conservative results.

Tests to determine the break point indicating cognitive impairment by age were not performed on the version of the MMSE used in the MHSS survey. However, higher scores clearly represent enhanced cognitive ability. Kabir and Herliz (2000) designed a different MMSE for Bangladesh which is referred to as the Bangla Adaptation of the Mini-mental State Examination (BAMSE). The BAMSE was also adapted for an illiteracy population and cultural relevance to Bangladesh. The BAMSE is very similar to the MMSE used in the MHSS. Kabir and Herliz implemented both the BAMSE and the MMSE used by a literate population in Bangladesh and found that the correlation between the MMSE and the BAMSE was good, indicating that the changes made to adapt the instrument for an illiterate population do not change the ranking of scores.

3.3. Intent-to-Treat Indicator

An individual is eligible for the MCH-FP interventions if the household resides in a treatment village and they meet the age requirement outlined in section 2.1. Creating a variable to indicate which households are eligible for the MCH-FP program based on 1996 MHSS household village location may be endogenous since households could have moved to the treatment area to benefit from the MCH-FP program. To avoid this potential endogeneity, 1974 location information is used to create the intent-to-treat variable*, Eligible*, which takes on the value 1 if the individual (or household if the individual was not born) resided in a treatment area in 1974, and zero otherwise.[6]

4. Identification Strategy and Empirical Specifications

We seek to determine the intent-to-treat (ITT) or overall program effects of the Matlab MCH-FP program on cognition. We take advantage of the variation in the program implementation across location (treatment versus comparison areas) and by age in the treatment area. We first use 1974 census data to show that the treatment and comparison areas are similar with respect to many observable characteristics and may indeed mimic a randomized intervention. We then present a number of different models to estimate the ITT effect and discuss the possible biases of each of these models. It is important to note that since the family planning and child health interventions were not randomly introduced in a factorial design, it is difficult to determine their effects separately. Nevertheless, the analysis tries to better isolate the effect of the child health interventions by controlling for the percent of reproductive years the child's mother was eligible for the family planning and maternal health interventions and by the mother's age and her child's birth.

[6] 1974 individual or household information was not available for 592 observations. For these observations 1982 location information was used. The results do not change if these 592 observations are excluded.

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 groups are contiguous geographic areas (Figure 2). It is possible that village randomization was not used in order to mitigate the possible spill-over effects to the comparison area from the positive externalities created by the vaccines. Prior research shows that the MCH-FP and comparison areas are indeed similar with respect to a number of pre-intervention variables including prior rates of mortality and fertility (Koenig et al., 1990; Menken & Phillips 1990; Joshi & Schultz, 2007). This is important as it shows that the MCH-FP program was probably not placed first in areas that had poor child health or high fertility –a potential targeting criteria for such programs. For this reason prior papers using these data argue that the treatment and comparison areas mimic a randomized evaluation or was quasi-randomly placed. However, it is important to note that even if the observables characteristics look similar, without randomization is it not clear that the unobservable characteristics are similar.

We would like to be able to show that the level of cognition was similar between treatment and comparison areas in the pre-intervention period. Since there is no pre-intervention data on cognition, we instead examine differences in the MMSE score for those age 25 and older since their cognition was unlikely to have been affected by the MCH-FP program (those age 20 and older were born before the program began) and any potential sibling competition (those who are at least 5 years older than an eligible younger sibling. Panel C of Table 1 shows that the mean MMSE score for this group was almost exactly the same at approximately 29 out of 30.

Panel C in Table 1 also highlights that mother's in the treatment and comparison area have a similar level of education. While this data is not available for the pre-intervention period, it is likely that almost all these mothers would have finished their education prior to the program. This is important since mother's education is usually correlated with child health which affects cognition. In addition, more education mothers may be a signal of better cognition which may be pass onto the child through their genetic make-up.

We further test the areas are similar using a wider array of household and household head characteristics from the 1974 census. Table 1 displays the means and standard deviations of each characteristic for the treatment and comparison area, the difference in the means between the two areas, as well as the difference in means divided by the standard deviation of the whole sample.[7] We do not report the t-statistic for the difference because it partly reflects a larger sample size. Instead, Imbens and Wooldrige (2007) suggest using the normalize difference (where the mean is normalized by dividing by the standard deviation) since it unambiguously indicates when the means are different. A difference of 0.25 standard deviations between means of the two groups is considered to be substantial (Imbens and Wooldridge 2007).[8] None of the standardized differences are more than 0.25 standard deviations. Using this method, most of the 1974 characteristics are similar expect except for drinking water source and household head's religion. In the treatment area there is a larger Hindu population, and households were more likely to use tube well water for drinking. There is a fairly large difference in the use of tube well water for drinking of 12 percent between treatment and comparison areas. Indeed, shallow tubewells were introduced by donors and the government starting in the 1970s and served 95 percent of the

[7] The t-statistics are clustered at the village level. All analysis will be clustered at the treatment group age level (i.e. at a higher level).

[8] The results are the same if comparison were made using the t-statistics.

Bangladeshi population by the mid-1990s. Usually tube well water is cleaner than other sources of water resulting in a healthier population which could potentially bias the estimates of the effect of the program on cognitions upwards. However, there is widespread groundwater arsenic contamination in the tube wells in Matlab and Bangladesh (Smith et al. 2000) which is a serious health concern (Chowdhury et al. 2000; MM Rahman et al. 2001; Alam et al. 2002) including detriments to the IQ of school-aged Bangladeshi children (Wasserman et al. 2006), and may actually lead the results to be downward bias as a result of greater access to tube well water.

4.2. Intent-to-Treat Single and Double Differences Models.

We first ordinary least square to estimate single difference model. The assumption of this model is that the mean MMSE score would have been the same between the treatment and comparison group in the absence of the MCH-FP program. This is not a testable assumption, however it seems likely given that the mean MMSE for those who likely did not benefit from the program, the 25 plus age group, were similar. The estimation equation is:

$$(1)\quad \begin{aligned} C_{imv} &= \beta_1(E_v * AG_{imv}^{6-7}) + \beta_2(E_v * AG_{imv}^{8-14}) + \beta_3(E_v * AG_{imv}^{15-19}) + \beta_4(E_v * AG_{imv}^{20\text{-}24}) \\ &+ \beta_5(E_v * AG_{imv}^{25+}) + \delta_1 FP_{imv} + \alpha_a + \lambda_s + X'Z + v_{imv}, \end{aligned}$$

where C is a measure of cognition of person i, of mother m in village v. E is a binary variable which takes on the value 1 if person i or i*'s* household was from a treatment village in the pre-interventions period, and 0 if in a control village. AG^{Y} is a binary variable used to indicate if the person i is or is not in age group Y. Thus, the β's are the single difference ITT effect for the various age groups. The ITT effect for the group of children who received the most intensive health interventions during the time period when the interventions were likely not available in the comparison area, age 8 to 14, is given by β_2. B_3 is the ITT estimate for the age group who received the less intensive child health interventions. FP is the percent of reproductive years up to 1996 person i's mother, m, was eligible for the family planning program. α_a represents age fixed effects to control for differences in cognition due to age as well as other events which may be correlated with age and common to the study population. λ_s are school level fixed-effects to control for differences in cognition that may be due to differences in years of education completed. Due to the potential endogeneity of schooling attainment (i.e. there may be reverse causality between cognition and education level) results are presented with and without school level fixed-effects. $\boldsymbol{X}$ is a vector of individual, mother controls including age, sex, education and religion as well as baseline household and household head characteristic presented in Table 2. Including these variables controls for any observable pre-intervention differences resulting from the quasi-random design, and may increase statistical precision. It also allows us to check if the few differences between treatment and comparison area found at baseline are biasing the results.

The error term $v_{imv} = \varepsilon_i + \varepsilon_m + \varepsilon_v + \varepsilon_{imv}$ is a composite of three terms: ε_i, which represents an individual effects such as genetics; ε_m, which captures time invariant mother characteristics and household environment such as if the child had stimulating parents that might have enhanced the child's cognition; ε_v, any village or treatment area variable that may be correlated cognitive development when the child was young for example, exposure to arsenic in the tube-well; and ε_{imv}, an idiosyncratic error term. This model assumes that various individual, mother, household, and village or treatment area unobservables are not correlated with E_v due to the quasi-random placement of the program. Standard errors are clustered at the treatment group-year level to account for the likely intra-cluster correlation in the error term.

Equation 1 does not take into account potential indirect effects of the MCH-FP due to sibling competition. Future versions of this paper will try to determine if those sibling competition is indeed an issue.

Even when interventions are successfully randomized, there is a chance that the particular outcome of interest will vary slightly during the pre-intervention period between MCH-FP and comparison areas. In this case a *double-difference estimator* may be more appropriate because it takes into account these pre-program differences. In addition, this estimator will control for non-time varying unobservables that may differ between the treatment and comparison areas, such as level of arsenic in the wells, and could be correlated with the eligibility variable, E_v. Given the long time span between the pre- and post-intervention time period and that the cognition data is only available post-intervention in the MHSS, it is not possible to examine the difference in cognition on any one individual pre- and post-intervention. Instead, the analysis will use the 25 year and older group to determine pre-intervention difference between treatment and control areas. The double difference estimator can be determined using the following linear regression:

$$(2)\quad \begin{aligned} C_{imv} &= \beta_1 E_v + \beta_2 (E_v * AG_{imv}^{6-7}) + \beta_3 (E_v * AG_{imv}^{8-14}) + \beta_4 (E_v * AG_{imv}^{15-19}) + \beta_5 (E_v * AG_{imv}^{20\text{-}24}) \\ &+ \delta_1 FP_{imv} + \alpha_a + \tau_s + X'Z + v_{imv}, \end{aligned}$$

Where the variables are defined as in Equation (1) but the interpretation differs because the 25+ age group is not interacted with *T*. Therefore, β_1 provides the difference in means between the treatment and comparison area for the 25+ years olds, or the differences in the treatment and comparison groups in the pre-intervention period.

4.3. Maternal Fixed-Effect Model

The single and double difference models will be bias if unobservable mother or household characteristics are not similar between the treatment and comparison. As discussed in section 3.1, most of the observable baseline characteristics as well as mother's education are similar. However, we are still concerned that there may be unobservables that are biasing the results. We include mother fixed-effects to the double difference model to partial out the ε_m component of the error term in Equation (1). This model identifies the effect of the MCH-FP program by comparing siblings who were and were not eligible for interventions but born to the same mother. The sample size is substantially smaller for this models since the mother identification code is missing for some individuals because their mother is dead (3,849 observations)[9], and because some individuals do not have any siblings in the dataset (3,715 observations). The large number of individuals missing a sibling in the data is partly due to the MMSE scores not being collected from all 6 to 14 years olds (see section 3.1).

4.4 Single and Double Difference Models with Propensity Score Weighting

Finally, if the treatment effect is not homogenous our estimates may suffer for two additional sources of bias. The first arises because there may be no comparison observations with comparable characteristics to the treated observations, or the two groups do not have a common support of all values of the characteristics.[10] The second bias may arise if the distribution of the

[9] Efforts were made to obtain identification codes for mothers who had died prior to 1996 by using the census data. Approximately 50 percent of the observations with missing mother information are for those under age 43.

[10] If there is a lack of overlap in the distributions the assumption of unconfoundness may not hold.

vector of covariates differs between the treatment and control group within the common support.[11]

To address the first potential bias we estimate a propensity score (Rosenbaum and Rubin 1983) and following Crump et al. 2008 only keep those observations for which the propensity score is greater than .1 or less than .9. Pre-intervention census data presented in Table 2, which included many variables that may be correlated with cognitive development such as measures of household wealth, occupation (as a proxy for income), and sources of drinking water (to proxy for cleanliness of drinking water), as well as mother's education and the age, sex, and religion of the person (in the 1996 data), are used to estimate the propensity score using logistic function form. Higher powers of these variables and interaction terms were included until the propensity score was balanced between treatment and controls areas in 14 blocks created using the propensity score.[12] Figure 4 presents the kdensity graph for the distribution of propensity score for the treated and comparison areas separately. It shows that there is substantial overlap of the propensity scores between the treatment and comparison area prior to determining the common support.

There are a number of different methods that are used in the literature to control for the second bias.[13] Imbens and Wooldridge (2008) suggest using a combination of regression adjustment with matching, weighting, or blocking techniques because these methods achieve some robustness to misspecification of the parametric models. In order to present result in a similar format to the other results in this paper we use a combination of propensity score weighting and regression adjustment. Robins and Rotnitzky (1995) demonstrate that when these two techniques are combined the estimator is consistent if either the parametric model for the propensity score or the regression functions are misspecified. The weights are the inverse probability of treatment or non-treatment, where the probability of treatment for those in the treatment group is the estimated propensity score and for the comparison group is one minus the estimated propensity score. As suggested by Imbens and Wooldridge (2008) we bootstrap the standard errors.

It is important to remember that these models cannot ensure that the unobservable characteristics are similar. Also given the program was quasi-randomly placed, and that there are few pre-intervention differences in the observable characteristics, we use propensity score weighting with our single and double difference models as a robustness check. If the program is really quasi-randomly placed, the estimates of the impact should be similar to those from Equations 1 and 2.

4.5 Birth Order and Gender Effects

Patterns of mortality risk, which in part may reflect differential investments in children, vary by family composition in Bangladesh. In particular, first born children and boys tend to have lower mortality risk (Muhuri and Menken 1997), and the presence of older siblings increases the chance of wasting (Trapp et al. 2004). Given the family planning program reduced fertility in the treatment area (Joshi and Schultz 2007), there are approximately 6 percent more first born children in the treatment than in the control area for those aged 6 to 19. The difference between these two groups for those aged 20 to 25 is less than 1 percent. It is possible then, that the

[11] Heckman et al (1998) and suggests that, in practice, the first of these two sources of bias is likely to be the most severe.

[12] There were no significant differences in the propensity score between the treatment and comparison area at the 5 percent significance level in any of the 14 blocks.

[13] See Imbens and Wooldridge 2008 for a review of these methods.

positive program impact on cognitive development is a result of there being a higher percent of first born children in the treatment area rather than an effect of the child health interventions. To explore this hypothesis, we interact the eligibility age group indicators with an indicator variable for if a child is first born. An binary variable indicating whether or not a child is first born is not included in the analysis since this variable is missing for more than 50 percent of the sample for those 20 years and older.

4.6 Spillover Effects

Spillover effects occur when the program indirectly affects non-program participants and will bias the ITT effects. Spillover effects could affect the untreated in MCH/FP or comparison villages due to the positive externalities of some of the interventions such as vaccinations (i.e. if most children in an area are vaccinated against polio then the non-vaccinated children in the area will be less likely to contract polio) or informational spillovers. In the comparison area, spillovers are likely to occur in those areas that border the MCH-FP villages since knowledge about the programs would be spread by word-of-mouth or occur as a result of marriage.

We explore the possibility of spillovers to comparison area villages which boarder a treatment village using the following linear regression:

(3)

$$
\begin{aligned}
C_{imv} = {} & \beta_1 NE_v + \beta_2 (NE_v * AG_{imv}^{6-7}) + \beta_3 (NE_v * AG_{imv}^{8-14}) + \beta_4 (NE_v * AG_{imv}^{15-19}) + \beta_5 (NE_v * AG_{imv}^{20\text{-}24}) \\
& + \beta_6 \mathrm{B}_v + \beta_7 (NE_v * AG_{imv}^{6-7} * B_v) + \beta_8 (NE_v * AG_{imv}^{8-14} * B_v) + \beta_9 (NE_v * AG_{imv}^{15-19} * B_v) + \delta_1 FP_{imv} + \alpha_a + \tau_s + X'Z + v_{imv},
\end{aligned}
$$

NE_v is defined as 1-E_v, where E_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 boarders a treatment village in 1982 and 0 otherwise.[14] All other variables are defined as above. Equation 3 examines the spillover effect by splitting the comparison area into two groups, those who live in a village that borders a treatment village and those that do not. Using a double difference estimator, $\beta_2 - \beta_5$, will show how much lower the outcome variable is in the comparison area where which does not boarder the treatment village, and $\beta_7 - \beta_9$ gives the difference in the effect between those who live in a comparison village that boarders and does not boarder a treatment village. If there are positive spill-over effects as a result of the program we would expect that $\beta_7 - \beta_9$ to be positive. If spillovers only resulted from the health interventions then β_9 would be zero. Lastly, β_6 may be smaller or zero since some child health interventions were available in the comparison when the 6 to 7 year olds were born.

In future versions of this paper we will include a variable which indicates the proportion of children in a village that received vaccinations to control for the possible within treatment area spillovers.

4.7 Other Econometric Issues

Two potentially important econometric issues in this context are attrition bias, and spatially correlated errors. Two prominent causes of attrition are mortality and migration. Even if the MCH-FP were truly randomized, the program itself is likely to cause mortality and migration to differ between MCH-FP and comparison areas over time, potentially biasing the results. For

[14] The results do not change if the 1974 location is used. We use 1982 to better reflect the possibility of spillover effects when the child health component commenced.

example, if frailer individuals (or those with lower health endowments) are more likely to survive in the MCH-FP area, then there will likely be a higher probability of observing someone with a lower level of cognition in the MCH-FP than in the comparison area in the follow-up period, biasing the results downwards. Differential mortality is likely to cause the treatment effect to also be biased downwards. Since migration from rural areas in Matlab tends to occur among families with few resources (Kuhn 2003, 2006b), 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 will leave a higher proportion of children who likely have lower human capital (since they come from worse off families) in eligible versus ineligible areas biasing the results downwards. In both of these cases the individual and mother fixed-effect components of the error term in Equation (1) are likely to be correlated with the treatment variable E_v. Since we do not have access to data on those who were benefited from the program but died or migrated before the 1996 MHSS, we control for non-time varying household variables that may be correlated with the attrition by using a maternal fixed-effect model.

Since the MCH-FP and comparison area are contiguous geographic areas, it is possible that errors are spatially correlated, biasing the treatment effects. This could arise for example if there was a health shock, such as a famine or a disease outbreak, in a given year in the comparison but not the MCH-FP area. Since these outbreaks are likely to affect younger children's human capital development more than those who are older, even the double difference models will be biased as they only control for time invariant unobservables between treatment and comparison area. Future versions of this paper will check if the errors are spatially correlated and will control for some village level variables such as access to health clinics and education facilities and any other known major outbreaks.

5. Results

5.1. Summary Statistics

Figure 3 displays the mean MMSE score by age groups of interest. The figure highlights how the level of cognition is higher in the treatment area than in the comparison area for children who were eligible for various interventions in the MCH-FP program (i.e. those under the age of 19). Surprisingly, the level of cognition is lower in the MCH-FP program area than in the comparison area for the 20 to 24 year old group. We will test if these differences are statistically significant in the regression analysis. It is possible that this difference are a result of sibling competition as these individuals are at most 5 years beyond the eligibility age. However, it could also reflect some negative shock that took place in the comparison area that affected the development of cognition of the 20-24 year old age group when they were children. For the rest of the age groups the mean MMSE score are essentially the same between the treatment and comparison groups.

5.2. Main Intent-to-Treat Regression Results

The regression results for the various models are presented in Table 3. Columns one through five presents the results for the single difference model where the standard errors are clustered at the treatment group-age level. Results in column 1 shows that there is a small, negative effect, -0.1 standard deviations (SD) of the program on the age 15 to 24 year olds, which is significantly at the 5 percent level for the 15-19 year olds. The MCH-FP program also lead to a statistically significant increase in the MMSE score of 0.3 SD for age 8-14 year olds and of 0.2 SD for the age 6-7 year olds. An effect size of 0.3 SD is similar to effects size in studies of the benefit of nutrition, and in particular iron, on cognitive-language abilities (Walker et al. 2007). It is also equivalent to the effects size for completing 3 years of primary school education on the MMSE in the sample.

The point estimates remain almost unchanged for all age groups with the inclusion of the variables to control for the family planning program (columns 2), pre-intervention characteristics (columns 3), mother's level of education (column 4), and for education level fixed-effects (column 5). However, the effects on the 6 to 7 and 15-19 years olds are no longer statistically significant. This provides some confidence that the differences at baseline in some of the household characteristics (such as access to tube well water) are not biasing the results, and that the effect of the MCH-FP program on cognition functioning for the 8-14 years olds is not a result of increased levels of education. Lastly, for most of the models there was a very small negative and insignificant effect of the family planning controls on the MMSE score, and of the program on the group of children who were primarily exposed to the family planning program but not the child health interventions (i.e. the 15-19 year olds). These results provides some evidence that it is likely the child health interventions and not the family planning interventions of the MCH-FP program which are resulting in improved cognitive functioning.

Double difference estimates without and with the mother fixed-effects are presented in columns 9 to 12. The results without the fixed effects are exactly the same as the single difference estimates except the coefficients for the 15-24 years olds is now marginally significance at the 10 percent level. The number of observations used for identification in the mother fixed-effects model is much smaller, 1844 (see section 4.3 for an explanation). Column 10 presents the double difference estimates without the maternal fixed-effects but for the same sample that is used to identify the fixed effects results in order to separate out the changes in the coefficient that are due to the smaller sample versus inclusion of mother fixed-effects. With the exception of the effect on the 8-14 year olds, the results for the smaller sample are very similar. The point estimate for the 8-14 year olds is double that of the full sample, 0.6 as compared to 0.3, and is statistically significant at the 1 percent level. Adding the maternal fixed-effect does not change the findings. In particular, the point estimate for the 8-14 years olds increased by 0.1 SD to 0.7. If anything these results indicate that not including maternal fix-effects may bias the results for this age group downwards.

Lastly, restricting the support and reweighing observations by the propensity score to account for differences in the distribution of the pre-intervention characteristics also leaves the findings unchanged (columns 7, 8 and 14). The program results in 0.3 SD increase in cognitive functioning for the 8 to 14 year olds which is statistically significant at the 1 percent level, and there are no other statistically significant effects for other age group. It is not surprising the results are unchanged because the pre-intervention characteristics are very similar between the treatment and comparison group (Table. 2).

5.2 *Birth Order and Gender Effects*

Panel A of Table 4 presents the double-difference OLS models where interactions for birth born and gender are included. The second column in Table 4 shows that the effect of the program is not statistically different for first born children. In fact the negative and small point estimates indicate that the first born may have experienced slightly less of a program effect for the 6 to 7 and 8 to 14 year old age groups. Indeed if a first born child's health status was better prior to the health intervention, then there would be less scope for the program to affect their health status and hence cognitive development.

Program effects by gender are in columns 3 and 4 of Table 4. These results demonstrate that the program effect is a statistical significant 0.2 SD higher for females than males for the 8-14 year olds, resulting in a total program effect for females of 0.4 SD, as compared to 0.2 SD for males. The difference between males and females are smaller and insignificant when education level fixed-effects are included indicating that one of the mechanisms through which the program may have increased girl's cognitive development is through higher levels of educational attainment.

5.3 Spillover Effects

Panel B of Table 4 displays the spillover results. The MMSE score is a statistically significant 0.3 SD higher for those who live in comparison villages which border a treatment village than in those that do not (as shown by the triple interaction) for the 8 to 14 year olds. The MMSE score is not different between the two groups for the age 15-19 year olds, suggesting that the family planning program did not have spillover effects. Finally, the point estimates on the difference is small (0.1 SD) and insignificant for the 6-7 year olds. The lack of spillover effects for the 6 to 7 year olds is likely a result of vaccinations being available in the comparison area when these children were in their first year of life.

5.4 Results by Sub-Component of MMSE

Table 5 presents the results by the main sub-components of the MMSE (orientation, attention-concentration, registration, and language) for the double difference models with and without propensity score weighting. The effects of the program by sub-component are similar to the aggregate effect for the 8 to 14 year olds of 0.3 SD with the exception of language, for which there is no statistically significant impact. The average score on the language section for 8 to 14 year olds is six out of seven and the standard deviation is one. The high average and lack of variation for the language components shows that it is not a good measure of cognitive functioning for children of this age group.

6. Conclusion

This paper examines the effect of a family planning and child health program that took place in the late 70s and 80s in Matlab Bangladesh on the cognitive functioning of those same children when they are between the ages of 8 and 19. The program concentrated on family planning interventions for the first five year and then introduced important child health interventions such as vaccination against measles, DPT, and polio. The analysis takes advantage of the quasi-random placement of this program and demonstrates that the treatment and comparison areas look similar with respect to most pre-intervention characteristics. The paper uses single and double difference models and shows that the program led to an approximately 0.3 SD increase in Mini Mental State Exam score for the 8 to 14 year olds who were born when both the family planning and child health interventions were in operation, but had no effect on the 15 to 19 year olds who were born when the program only had family planning component. The results also remain the unchanged when variables to control for the family planning program (percent of a woman's fertile years she was eligible for the program at mother's age at birth) are included. So the effects are likely to be mainly from the child health interventions. It is possible that results under-estimated due to positive spill-over effects in comparison villages that border a treatment village, and due to the mortality selection that these programs create.

An effect size of 0.3 SD is similar to effect sizes in studies of the benefit of nutrition, and in particular iron, on cognitive-language abilities (Walker et al. 2007), and is equivalent to the effects size for completing 3 years of primary school education on the MMSE in the sample.

The findings remain the same when pre-intervention controls and mother fixed-effects are included, and propensity score weighting is used. The results also remain the same when education level fixed-effects are included highlighting that the improvements in cognitive functioning are not due to an increase in education levels.

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Figures

Figure 1: Proportion of children aged 12-59 months who received the measles vaccination by year and treatment block

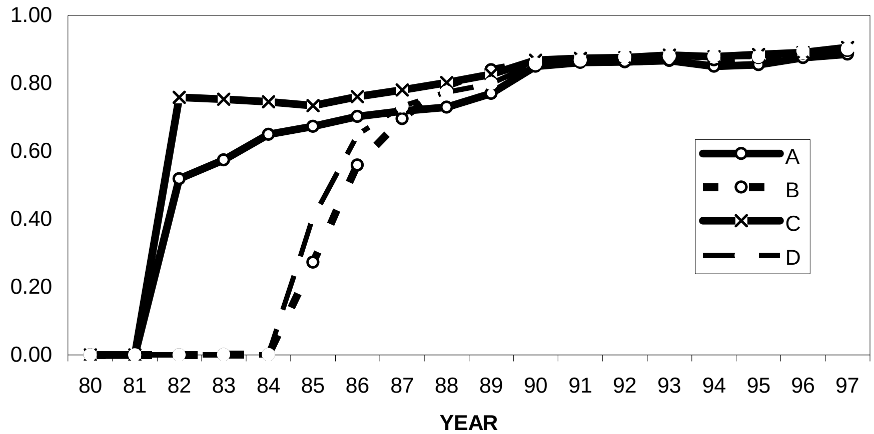


Figure 2: Map of Matlab study area

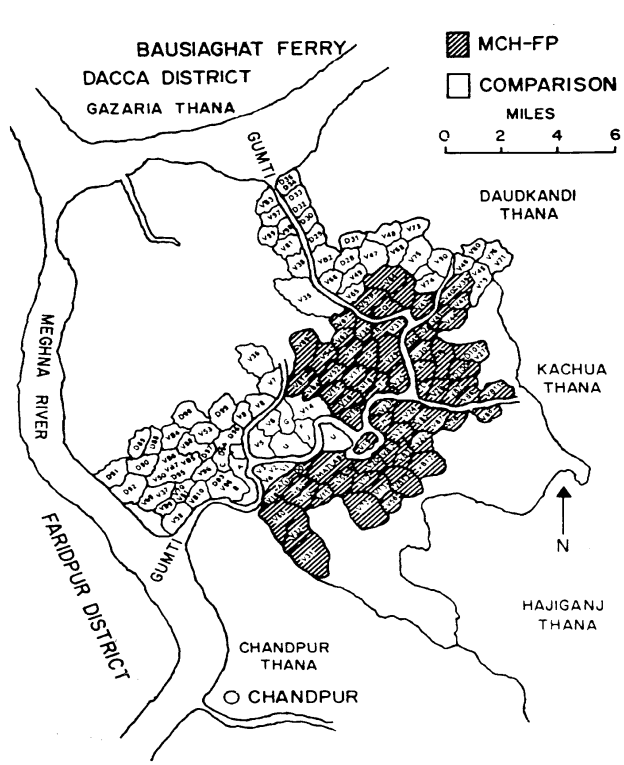
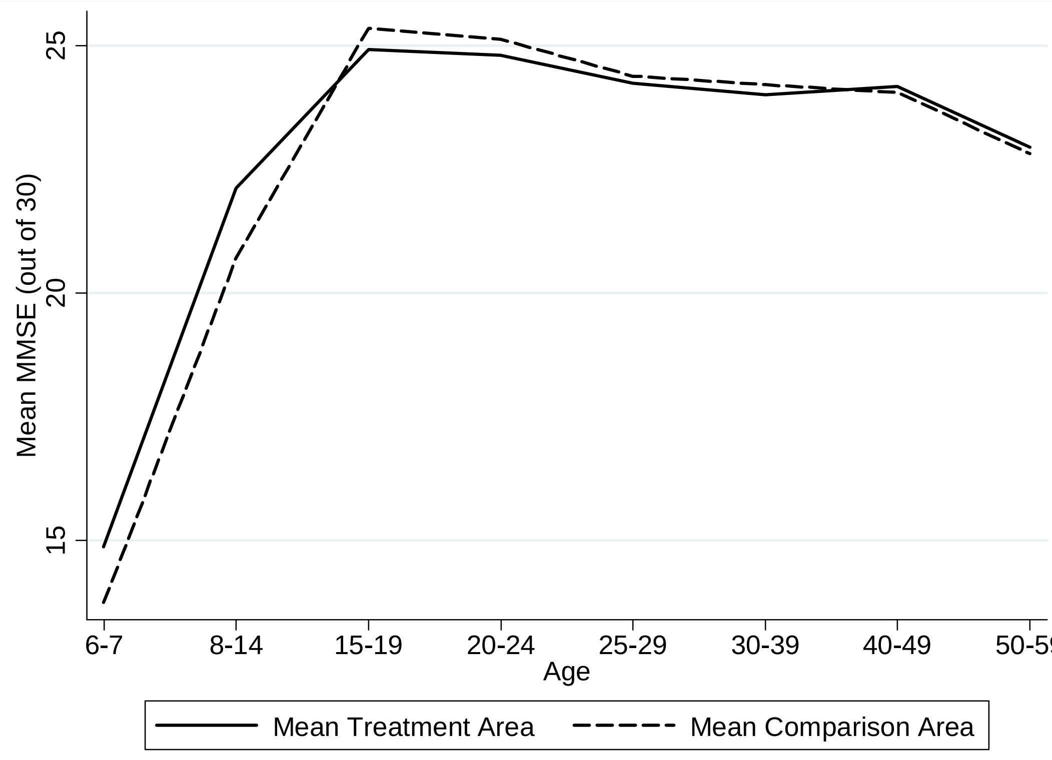
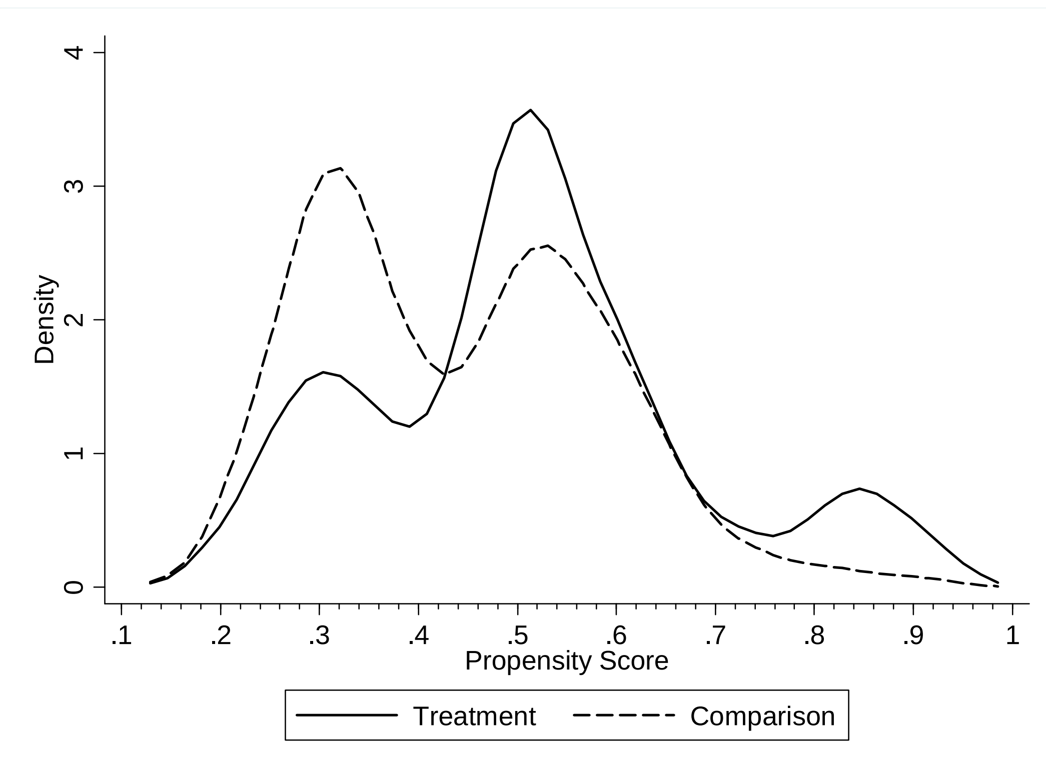


Figure 3: Mean MMSE Score by Age Group and Eligibility Status





Tables

Table 1: MCH-FP Eligibility by Age

Eligibility	**Year Born**	**Age in MHSS**
Not eligible for MCH-FP.	Pre 1938	59
Reproductive age during MCH-FP program, eligible for FP and MH.	1938-1973	23-58
Born during MCH-FP program, eligible for CH.	1977-1989	7-19
Non-intensive treatment: FP, ORT, mainly late measles vaccination.	1977-1981	15-19
Intensive treatment: FP, ORT, on-time or late measles/DPT/polio/BCG, vitamin A.	1982-1988	8-14
Some interventions now available in comparison area.	1989-2010	0-7

Notes: FP=family planning; MH=maternal health; CH=child health; ORT=Oral Rehydration Therapy; DPT=Diphtheria-Pertussis-Tetanus; BCG=Bacille Calmette-Guerin (vaccine against tuberculosis).

Table 2: Differences in Means between Treatment and Comparison Area

	Treatment Group			Comparison Group			Difference		
	Mean	SD	Obs	Mean	SD	Ob	Mean	Mean/SD	T-stat
A. Household Characteristics from 1974 Census									
Family size	7.0	3.11	4108	6.9	3.04	4494	0.2	0.06	1.34
Owns a lamp (=1)	0.66	0.47	4108	0.61	0.49	4494	0.05	0.1	1.44
Owns a watch (=1)	0.18	0.38	4108	0.16	0.37	4494	0.01	0.04	0.77
Owns a radio (=1)	0.08	0.27	4108	0.08	0.28	4494	0.00	-0.01	-0.27
Wall made of tinmix (=1)	0.23	0.42	4038	0.25	0.43	4424	-0.02	-0.05	-1.07
Roof made of tin (=1)	0.82	0.39	4095	0.83	0.37	4459	-0.02	-0.05	-0.86
Latrine in household compound (=1)	0.83	0.38	4108	0.85	0.36	4494	-0.02	-0.06	-0.95
Number of rooms per capita	0.2	0.1	4108	0.2	0.11	4494	0.0	0.01	0.13
Number of cows	1.5	1.75	4108	1.4	1.79	4494	0.1	0.07	1.29
Number of boats	0.7	0.65	4106	0.7	0.61	4494	0.0	0	0.06
Drinking water from tube well (=1)	0.30	0.46	4108	0.17	0.38	4491	0.12	0.3	3.48
Drinking water from tank (=1)	0.39	0.49	4108	0.33	0.47	4491	0.06	0.13	1.26
Drinking water from river, canal, or ditch (=1)	0.31	0.46	4108	0.42	0.5	4491	-0.22	-0.38	-3.17
B. Household Head Characteristics 1974 Census									
Age	47.1	14.37	4099	46.5	13.92	4489	0.6	0.05	0.97
Hindu (=1)	0.17	0.37	4053	0.05	0.22	4462	0.12	0.38	3.34
Years of education	2.6	3.12	4108	2.4	2.81	4494	0.2	0.06	1.29
Primary occupation is agriculture (=1)	0.61	0.49	4108	0.61	0.49	4494	0.00	0	0.00
Primary occupation fishing or boatman (=1)	0.05	0.22	4108	0.06	0.24	4494	-0.01	-0.05	-0.95
C. Characteristics from 1996 MHSS									
MMSE for 25+ age group	23.8	3.89	2996	23.9	3.9	3223	0.0	-0.01	-0.14
Mother's years of education	1.1	2.18	4217	0.9	2.02	4718	0.2	0.07	1.58
Hindu (=1)	0.16	0.37	4333	0.05	0.22	4831	0.11	0.3	3.41
Islamic (=1)	0.84	0.37	4333	0.95	0.22	4831	-0.11	-0.3	-3.41
Age	33.1	13.83	4333	32.3	14.24	4831	0.8	0.05	1.40
Female (=1)	0.57	0.5	4333	0.55	0.5	4831	0.02	0.04	2.40
Years of education	3.5	3.68	4310	3.1	3.46	4807	0.4	0.12	2.48

Notes: SD = standard deviation, Obs = observation; T-stat = T- statistic; The standard deviation of the whole sample is used to create the ratio of the difference in means to standard deviation; Standard errors clustered at the village level.

Table 3: Main Regression Results

	Single Difference							Double Difference						
	OLS					PS Weighting		OLS			Maternal FE		PS Weighting	
	(1)	(2)	(3)	(4)	(5)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)
Eligible (=1)								0	0	-0.0	0.1	0.5	-0.0	-0.0
								(0.0)	(0.0)	(0.1)	(0.6)	(0.6)	(0.0)	(0.0)
Eligible*(Age 25+)	0.0	0.0	-0.0	0.0	-0.0	-0.0	-0.0							
	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)							
Eligible*(Age 20-24)	-0.0	-0.1	-0.1	-0.1	-0.1	-0.1	-0.1	-0.1	-0.1+	-0.2	-0.4**	-0.2	-0.1	-0.1
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.0)	(0.1)	(0.1)	(0.2)	(0.1)	(0.1)
Eligible*(Age 15-19)	-0.1*	-0.1	-0.1	-0.1	-0.1+	-0.1	-0.1	-0.1+	-0.1*	0.0	0.3	0.3	-0.1	-0.1+
	(0.0)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.0)	(0.0)	(0.1)	(0.2)	(0.2)	(0.1)	(0.1)
Eligible*(Age 8-14)	0.3**	0.3**	0.3**	0.3**	0.3**	0.3**	0.3**	0.3**	0.3**	0.6**	0.7**	0.7**	0.3**	0.3**
	(0.0)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.0)	(0.0)	(0.1)	(0.2)	(0.2)	(0.1)	(0.1)
Eligible*(Age 6-7)	0.2*	0.2	0.2	0.2	0.1	0.1	0.1	0.2	0.1	0.2	-0.1	-0.1	0.1	0.1
	(0.1)	(0.2)	(0.2)	(0.2)	(0.1)	(0.3)	(0.3)	(0.1)	(0.1)	(0.2)	(0.3)	(0.3)	(0.3)	(0.3)
% of fertile years mother eligible		-0.0	-0.0	-0.1	-0.1	-0.0	-0.1	-0.0	-0.1	-0.3	-0.2	-0.6	-0.0	-0.1
		(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.3)	(0.6)	(0.7)	(0.1)	(0.1)
Mother's age at child's birth		-0.0	-0.0	-0.0	-0.0	-0.0	-0.0	-0.0	-0.0	-0.0**	-0.0	-0.0	-0.0	-0.0
		(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)
Mother's years of education				0.1**										
				(0.0)										
Age fixed-effects	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Individual characteristics	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Baseline characteristics	N	N	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Education level fixed-effects	N	N	N	N	Y	N	Y	N	Y	N	N	Y	N	Y
Mother fixed-effect sample	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	N
Mother fixed-effects	N	N	N	N	N	N	N	N	N	N	Y	Y	N	N
Observations	9164	8566	8566	8447	8522	7712	7671	8566	8522	1844	1844	1836	7712	7671
Adjusted R-Squared	0.21	0.21	0.26	0.28	0.33	0.28	0.34	0.26	0.33	0.37	0.58	0.60	0.28	0.34

Notes: Standard errors are clustered at the treatment-age level. An asterisks indicates that the difference in the coefficient from zero is statistically significant at the 95 percent confidence level, and + at the 90 percent confidence level. Individual characteristics include a dummy for female and being Islamic. Baseline characteristics include all household and household head characteristics from 1974 presented in Table 2.

Table 4: Birth Order, Gender, and Spillover Effects (Double-Difference OLS Models)

Panel A	(1)	(2)	(3)	(4)	Panel B	(5)
Eligible	-0.0	-0.0	-0.0	-0.0	Not Eligible (=1)	-0.0
	(0.0)	(0.0)	(0.0)	(0.0)		(0.0)
Eligible*(Age 20-24)	-0.1	-0.1	-0.1	-0.1	Not Eligible*(Age 20-24)	0.1*
	(0.1)	(0.1)	(0.1)	(0.0)		(0.1)
Eligible*(Age 15-19)	-0.1+	-0.1**	-0.2**	-0.1	Not Eligible*(Age 15-19)	0.1+
	(0.0)	(0.0)	(0.1)	(0.1)		(0.0)
Eligible*(Age 8-14)	0.3**	0.3**	0.2**	0.3**	Not Eligible*(Age 8-14)	-0.4**
	(0.0)	(0.1)	(0.1)	(0.1)		(0.1)
Eligible*(Age 6-7)	0.2	0.1	0.1	0.2	Not Eligible*(Age 6-7)	-0.2+
	(0.1)	(0.2)	(0.2)	(0.2)		(0.1)
Eligible*(Age 15-19)*First Born		0.1			Boarder Treatment (=1)	0.1*
		(0.1)				(0.0)
Eligible*(Age 8-14)*First Born		-0.1			Not Eligible*(Age15-19)*Border Treatment	-0.0
		(0.1)				(0.0)
Eligible*(Age 6-7)*First Born		-0.1			Not Eligible*(Age8-14)*Border Treatment	0.3*
		(0.0)				(0.1)
Female			-0.4**	-0.3**	Not Eligible*(Age6-7)*Border Treatment	0.1
			(0.0)	(0.0)		(0.1)
Eligible*(Age 15-19)*Female			0.2**	0.1		
			(0.1)	(0.1)		
Eligible*(Age 8-14)*Female			0.2*	0.1		
			(0.1)	(0.1)		
Eligible*(Age 6-7)*Female			0.2	-0.1		
			(0.1)	(0.2)		
Education level fixed-effects	N	N	N	Y		N
Observations	8566	8229	8566	8522		8566
Adjusted R-Squared	0.26	0.26	0.26	0.34		0.26

Notes: Standard errors are clustered at the treatment-age level. Regressions include age fixed-effects, and individual, pre-intervention and family planning controls. An asterisks indicates that the difference in the coefficient from zero is statistically significant at the 95 percent confidence level, and + at the 90 percent confidence level. Individual characteristics include a dummy for female and being Islamic. The variable border treatment indicates if a control village borders a treatment village.

Table 5: Effects by Subcomponent of the MMSE

	Double Difference							
	Orientation		Attention-Concentration		Registration		Language	
	OLS	**PSW**	**OLS**	**PSW**	**OLS**	**PSW**	**OLS**	**PSW**
Eligible (=1)	0.0	0.0	-0.0	-0.0	-0.1*	-0.1*	0.1	0.0
	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)
Eligible*(Age 20-24)	-0.0	-0.0	-0.1	-0.1	-0.1	-0.1	-0.2**	-0.1
	(0.1)	(0.1)	(0.0)	(0.1)	(0.1)	(0.1)	(0.0)	(0.1)
Eligible*(Age 15-19)	-0.0	-0.0	-0.1+	-0.1	-0.0	-0.1	-0.1*	-0.1
	(0.1)	(0.1)	(0.0)	(0.1)	(0.0)	(0.1)	(0.0)	(0.1)
Eligible*(Age 8-14)	0.3**	0.3**	0.2**	0.3*	0.3**	0.3**	0.1	0.1
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)
Eligible*(Age 6-7)	0.2+	0.2	-0.0	-0.1	0.2*	0.2	0.1	0.1
	(0.1)	(0.2)	(0.1)	(0.3)	(0.1)	(0.2)	(0.2)	(0.3)
Family planning controls	Y	Y	Y	Y	Y	Y	Y	Y
Age fixed-effects	Y	Y	Y	Y	Y	Y	Y	Y
Individual characteristics	Y	Y	Y	Y	Y	Y	Y	Y
Pre-intervention characteristics	Y	Y	Y	Y	Y	Y	Y	Y
Observations	8566		8566		8566		8566	
Adjusted R-Squared	0.28		0.28		0.1		0.07	

Notes: PSW = propensity score weighting. Standard errors are clustered at the treatment-age level and are bootstrapped for the PSW models. Regressions include age fixed-effects, and individual, pre-intervention and family planning controls. An asterisks indicates that the difference in the coefficient from zero is statistically significant at the 95 percent confidence level, and + at the 90 percent confidence level. Individual characteristics include a dummy for female and being Islamic. The variable border treatment indicates if a control village borders a treatment village.