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Enhancing Cognitive Functioning: Medium-Term Effects of a Health and Family Planning Program in Matlab†

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It is believed that early life circumstances are crucial to success later in life. Yet causal evidence that the impacts of early childhood health interventions continue into late childhood and adolescence is sparse. This paper exploits a quasi-random placement of the Matlab Maternal and Child Health and Family Planning Program in Bangladesh to determine whether children eligible for child health interventions in early childhood had better cognitive functioning at ages 8–14. I find a program effect of 0.39 standard deviations on cognitive functioning and similar effects for height and educational attainment (*JEL* I15, I18, J13, J18, O15).

Cognitive development is a key contributor to future educational attainment, labor market outcomes, and overall wellbeing; but over 200 million children under five years of age are failing to reach their cognitive potential owing to deprivation early in life (Grantham-McGregor et al. 2007). Many programs in developed and developing countries, such as Head Start in the United States and conditional cash transfer programs, aim to improve the early life circumstances of children through improved health, nutrition, and other services. 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 child's health or nutrition early in life, such as from flu pandemics or famines, lead to worse outcomes later in life.[1] However, there is little causal evidence on the long-run effects of *interventions* designed to improve the health and nutrition of young children, both in general and on cognitive functioning in particular. This is because there are few well-designed programs that took place ten or more years ago and that have data on cognitive functioning.[2] The few

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[1] See Glewwe and Miguel (2008), Strauss and Thomas (2008), and Currie (2009) for recent reviews of the literature.

[2] Evidence on short- and long-run effects of nutrition and infectious diseases on cognitive development is reviewed in Grantham-McGregor, Fernald, and Sethuraman (1999a,b), Currie (2009), and Walker et al. (2007).

studies that do examine the effects of such programs on longer-term cognitive development show mixed results.[3]

This paper exploits quasi-random variation in eligibility for the Matlab Maternal and Child Health and Family Planning (MCH-FP) program in Bangladesh to estimate the causal effect of improvements in early-life health and nutrition and in reduced maternal fertility on cognitive functioning of children at older ages. Program interventions were phased-in over time in the treatment area starting with family planning in 1977, shortly followed by maternal health interventions. Intensive child health interventions began with measles vaccination in 1982 and the program was expanded in 1986 to include other child health interventions, such as vaccinations against DPT,[4] 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, which in turn improves cognitive development. The geographic separation of the treatment and comparison groups minimizes the possibility that the estimates will be biased by 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 preprogram data for the study site shows that the treatment and comparison groups were similar on a variety of preprogram characteristics.

I use the phasing-in of the program over time and geographic variation to estimate the intent-to-treat double difference effects on cognitive functioning on three cohorts: those born prior to the program and who may have experienced sibling competition (20–24-year-olds); those born after the program started but before the intensive child health interventions were introduced (15–19-year-olds), and those born after the intensive child health interventions were introduced but before child health interventions were available in the comparison area (8–14-year-olds). Cohorts born before the program and unlikely to have been affected by the program are used to measure baseline differences between the treatment and comparison areas. Surprisingly, to my knowledge, this is the first paper to take advantage of the program's phase-in to examine its longer-term effects. The findings demonstrate that there were important program effects on the cognitive functioning for 8–14-year-olds who were born after the child health interventions were introduced. In particular, cognitive functioning was 0.39 standard deviations higher in the treatment than the comparison area. The program also led to a small negative effect, that was only marginally significant in some of the models, for the 15–19-year-olds, and had no effect on the 20–24-year-olds. Instrumental variables estimates show that the effects on children who received the majority of the child health interventions are almost twice as large as the intent-to-treat effects for the 8–14-year-olds. The pattern of program effects is similar for height, a indirect measure of nutrition in childhood

[3] For example, a nutritional supplementation study in Jamaica found a significant positive impact on children's development two years after the program, but no statistically significant effects when the children were ages 7–8 (Grantham-McGregor et al. 2007). In contrast, Maluccio et al. (2009) found that childhood nutritional supplementation in the well-known INCAP study in Guatemala led to better adult nonverbal cognitive functioning.

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

and a mechanism through which the program may affect cognitive functioning, and educational attainment.

Two related papers examine the effect of the MCH-FP program on various measures of human capital including height-for-age (Chaudhuri 2005, Joshi and Schultz 2007) and educational attainment (Joshi and Schultz 2007). This paper differs in that it exploits the phasing-in of program interventions over time in the treatment area and pays attention to the introduction of child health interventions in the comparison area to determine the age groups of interest. As a result, this paper finds significant effects on height and educational attainment of 8–14-year-olds in contrast to Joshi and Schultz (2007), but similarly to Chaudhuri (2005). Furthermore, this paper also examines a new measure of human capital—cognitive functioning. While educational attainment is often thought of as a proxy for cognitive functioning, it is a function of many other factors, such as cost of enrollment, school quality and access, policies on automatic promotion, and labor market opportunities. As a result, educational outcomes are unlikely to accurately reflect one's cognitive abilities.

While the paper provides some insight into the effect of the child health interventions alone, it is difficult to separate out these effects from the other program interventions. Even so, given that the early childhood vaccination and family planning programs are arguably two of the most important and widespread health programs in developing countries in the latter part of the twentieth century, the combined effect is important.

The rest of the paper proceeds as follows. Section I describes the MCH-FP program and the mechanisms through which the program may affect cognitive functioning. Section II describes the data. Section III lays out the identification and estimation strategy. The findings and robustness analysis are discussed in Section IV, and Section V concludes.

I. The Matlab MCH-FP Program

The MCH-FP project was initiated in October 1977 by the International Centre for Diarrheal Disease Research, Bangladesh (ICDDR,B) in a rural area of Bangladesh called Matlab. It was designed as an integrated family planning and maternal and child health (MCH) services project, but the initial focus was on family planning, with limited MCH services (Bhatia et al. 1980; Phillips et al. 1984). Further MCH services, including the childhood vaccinations, were phased in over time in a planned manner. Government health services were provided in the comparison area, but most of the program 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 the interventions. The MCH-FP program is still ongoing.

The 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). At least quarterly demographic surveillance of vital events (births, deaths, migration, and marriage) and provision of diarrheal health services in both the treatment and comparison area started in the mid-1960s (Chen et al. 1983).

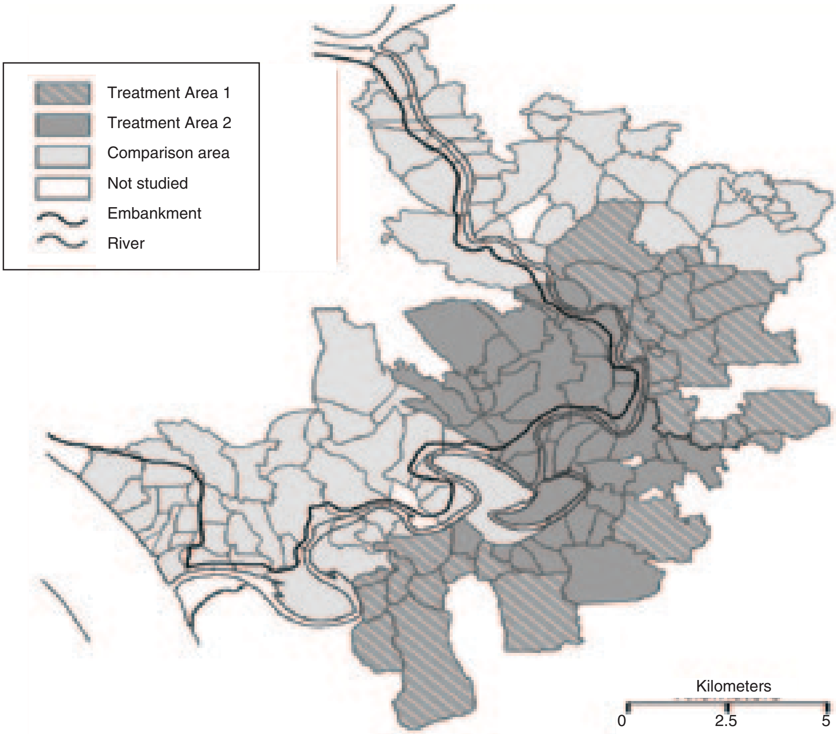

Figure 1. Map of Matlab Study Area

A. *The Intervention*

Between 1977 and 1981, the program interventions focused on family planning through the provision of modern contraception (starting October 1977), but also included tetanus toxoid vaccinations for pregnant women (starting June 1978) and iron and folic acid tablets for women in the last trimester of pregnancy (Bhatia et al. 1980). Tentanus toxoid was expanded to all women of reproductive age in 1982. The program is unique in that the interventions were provided free and administered in the house of the beneficiary during monthly visits made by local female health workers hired and trained by the program (Bhatia et al. 1980). During these visits, the female health workers also provided counseling about contraceptives, nutrition, hygiene, and breastfeeding, and instructions on how to prepare oral rehydration solution. These services were supported by well-developed follow-up and referral systems to ensure management of side effects and continued use of contraceptives (Phillips et al. 1984).

Starting in 1982, child health interventions were intensified in half the treatment area, referred to as Treatment Area 1, while interventions were delayed in the other half, Treatment Area 2 (Figure 1). In particular, the measles vaccine was introduced in March 1982 and was available to children between the ages of 9 and 59 months in Treatment Area 1. It was expanded to Treatment Area 2 in November 1985 (Koenig et al. 1990). Additional child health interventions were phased in between 1986

Table 1—MCH-FP Program Eligibility by Birth Year

Birth cohorts	Birth cohort label[a]	Program eligibility[b]
October 1947–September 1972	25–49	<u>*Pre-intervention group*</u>
October 1972–September 1977	20–24	<u>*No interventions, potential sibling competition*</u> i. Not eligible for child health interventions and unlikely to use family planning and maternal health interventions. ii. Potentially affected by the program through sibling competition.
October 1977–February 1982	15–19	<u>*Intensive family planning and maternal health interventions*</u> i. Mother eligible for family planning, tetanus toxoid vaccine, folic acid, and iron in last trimester of pregnancy. ii. Children under age five eligible for mainly late measles vaccination in Treatment Area 1. iii. Potentially affected by sibling competition from younger groups.
March 1982–December 1988	8–14	<u>*Child health interventions added*</u>
March 1982–October1985	12–14	*Child health interventions added in Treatment Area 1* i. Mother eligible for family planning, tetanus toxoid vaccine, folic acid, and iron in last trimester of pregnancy. ii. Children under age five eligible for on-time measles vaccination in Treatment Area 1, but for late DPT, polio, and tuberculosis vaccination in entire treatment area.
November 1985–December 1988	8–11	*Child health intervention extended to entire treatment area* i. Mother eligible for family, tetanus toxoid vaccine, folic acid and iron in last trimester of pregnancy. ii. Children under age five eligible for on-time vaccination (measles, DPT, polio, tuberculosis) and vitamin A supplementation. iii. Nutrition rehabilitation for children at risk.

[a] The label is based on age in years rounded to approximate age in years as of December 1996. The exact year and month cutoffs are used to create groups for the analysis.
[b] See Section IA for a detailed description of the interventions and the exact dates when they were introduced.

and 1988. In January 1986, DPT, polio, and tuberculosis immunizations were introduced, and later in that year, vitamin A supplementation for children under age five and nutrition rehabilitation for those who were nutritionally at risk were added.[5] These interventions were also administered in the home of the recipient.

In sum, these interventions fall into three main time periods. In the first, October 1977–February 1982, the program's focus was on family planning and maternal health. In the second, March 1982–October 1985, the program introduced child health interventions in half the treatment area. In the third, November 1985–December 1988, child health interventions were intensified and expanded to the entire treatment area. Table 1 summarizes program eligibility by birth cohorts for these three groups as well as for those born before the program, and highlights the variation in program eligibility used to identify the program effects.

[5] Other child health interventions, such as control for acute respiratory infections and dysenteric diarrhea, became available in 1988 or later, after the experimental period for this study.

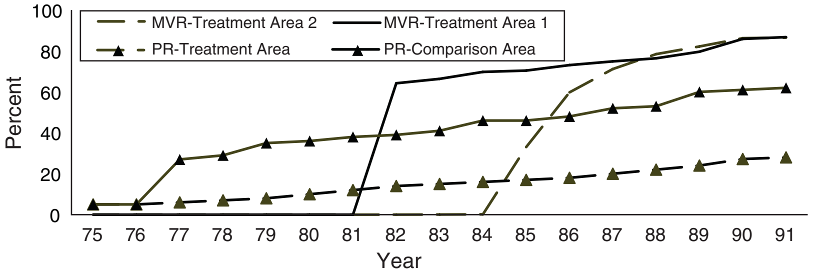

Figure 2. Trends in Measles Vaccination Rates (MVR) for Children 12–59 Months and Contraceptive Prevalence Rate (CPR) by Calendar Year

B. *Program Take-up*

Figure 2 demonstrates that implementation followed the planned timeline and that uptake was rapid for two main program interventions (the measles vaccine and contraception). The measles take-up data for children 12–59 months are presented separately for Treatment Areas 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 before 1989 are believed to have been near zero, since government clinics in the comparison area did not provide vaccines for children until around 1989 (Koenig, Fauveau, and Wojtyniak 1991). Nationally, measles vaccination was less than 2 percent in 1986 (Kahn and Yoder 1998), and it remained below 40 percent for children under age five in the comparison area in 1990 (Fauveau 1994).

Figure 2 shows that the contraceptive prevalence rate (CPR) for married women 15–49 years old 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), but the comparison area did not benefit from home visits. As a result, the CPR in the comparison area was much lower, with rates below 20 percent in 1988.

C. *Mechanisms Linking the MCH-FP Program and Cognitive Functioning*

Potentially, the MCH-FP interventions could directly or indirectly affect the cognitive development of young children through a variety of channels. This section highlights many of the better-known mechanisms. In particular, direct effects on cognitive functioning may result from reducing the incidence of measles and pertussis, since encephalitis, a complication of both these diseases, leads to long-term brain damage (Greenberg, von König, and Heininger 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 may be exacerbated in children who are vitamin A deficient because vitamin A reduces the severity of diarrheal diseases and measles, as well as vision impairments (Martorell 1999). These effects are likely to be much larger in developing than developed countries, partly because lower levels of nutrition before infection may weaken the immune system. Infections reduce appetite and food intake, impair absorption of nutrients, and increase metabolic demands from the body via fever and immune response (Reddy 1987; Grantham-McGregor, Fernald, Sethuraman 1999a, b). Measles, in particular, is known to severely impair the child's nutritional status through secondary complications such as pneumonia and diarrhea, and through 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 can do so for months (Greenberg, von König, and Heininger 2005). Both nonrandomized and randomized studies show that undernutrition, especially before the age of three, affects the cognitive development of young children (Grantham-McGregor, Fernald, Sethuraman 1999a, 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. In turn, lack of stimulation and learning opportunities 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 who did not in order to compensate that child for not having received the interventions. Given that the first few years of life are generally believed to be the most important for cognitive development (Grantham-McGregor et al. 2007), the effects of sibling competition on cognitive function may be greatest for siblings who are five or fewer years apart in age.

The nonchild 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, such as improved nutrition, or could allow the parents to spend more quality time with their child, both of which could improve cognitive functioning. Furthermore, children in the treatment area may benefit more from cognitive stimulation from their mothers because the program has reduced maternal mortality and improved maternal health. In addition, greater birth spacing resulting from the family planning interventions and some of the maternal health inputs (e.g., iron and folate supplementation, nutritional counseling, referral for pregnancy complications) may also directly affect the cognitive development of a child through the improved nutrition and health of the mother while the child is in utero (Walker et al. 2007; Almond and Mazumder 2011).[6]

Maternal immunization for tetanus may reduce neonatal mortality from tetanus but is less likely to affect the cognitive functioning of children. It is true that those few infants who survive the disease may suffer long-term cognitive impairment, but without quality hospital care, the likelihood of death from neonatal tetanus is extremely high (Roper, Vandelaer, and Gasse 2007).

II. Data

A. *Data Sources*

This paper draws on the unusually rich data available for the Matlab area, and benefits from the ability to link the various data sources by person and/or household identification number. Four main data sources are used to construct the dataset. The first one, the 1996 Matlab Health and Socioeconomic Survey (MHSS), is the main data source used to create the dataset for the analysis (ICDDR, B 1996).[7] The MHSS is a multi-purpose survey, but, unlike many household surveys taken before 2000, the MHSS includes a measure of cognitive functioning, the Mini Mental State Exam (MMSE).

The MMSE should have been collected on all household members age six and older selected for in-depth interviews. Because of a fieldwork error, the MMSE was administered to children aged 6–14 only in the last quarter of households that were surveyed. To partly correct this error, a 10 percent random sample of all survey households was drawn (referred to as the *random kid sample* in the user guide). Surveyors returned to these households and administered the MMSE to children aged 6–14 who were missed in the initial field work. To ensure sufficient sample size, I do not restrict the sample to the random kid sample, but use all available observations (referred to as the *full sample*). It is possible that the data on 6–14-year-olds for the full sample might suffer from selection bias. Luckily, this does not appear to be the case. As Section IIIA shows, baseline characteristics are similar between treatment and comparison areas for this age group for the full sample; and in Section IVB, the regression results are the same regardless of whether the full sample or the random kid sample is used, though statistical significance improves with the full sample. Preliminary tests performed by the survey administrators also showed no difference between the random kid sample and those initially given the cognitive tests prior to discovering the field error (Rahman et al. 1999).

The MHSS 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. Within each bari, one household was selected at random, and within households, members were selected

[6] Joshi and Schultz (2007) and Schultz (2009) show that the MCH-FP led to longer birth spacing and improved women's health (as measured by the body mass index).

[7] Data including the individual identification number to link to other data sources must be requested from ICCDR,B.

for detailed adult and child interviews and testing.[8] Survey weights are included to adjust for the sampling scheme, but have an unusually large spread since sampling clusters are not similarly sized.[9] This combination of large spread and small sample sizes for the under 15-year-olds[10] will likely make the weighted least squares estimates not only inefficient but also biased (Deaton 1997). It is important to note that the relatively small sample size for cognitive functioning for the under 15-year-olds is a result of the data collection error, and the weights are not likely to pose a problem for analysis on other outcomes. Indeed, when examining height, an indirect measure of long-term nutrition, the weighted and unweighted results are the same, but when the sample is restricted to having nonmissing MMSE scores, the weighted results are much lower (results not reported). So, the sample weights are not used in the analyses in this paper. Results are internally valid, but should not be interpreted as representative of the population of Matlab. Weighted least squares results for the main analyses, where the weight is top coded at the ninetieth percentile, and outliers with high weights removed are on the whole similar to those reported in this paper and available in the online Appendix.[11]

The second source, periodic censuses (e.g., 1974, 1982) collected by ICCDR,B on the entire study population (treatment and comparison area), provides preprogram information on household location, composition, assets, employment, and education. The 1974 census (ICDDR, B 1974) offers the opportunity to test for preprogram similarity between the treatment and comparison areas, and the 1982 census (ICDDR, B 1982) is used in tracing 1996 MHSS respondents back to the 1974 preprogram census.

The third source, demographic surveillance site data on vital events (e.g., births, deaths, migration) collected by ICCDR, B on the entire study population of Matlab, is used to help create the intent-to-treat variable and examine attrition from mortality or outmigration in the study area. The demographic surveillance data have been collected at least quarterly since 1966 on the whole study population and are known to be of high quality.

The last source, the record keeping system (RKS) collected by ICDDR,B, contains data on receipt of program interventions for each individual in the treatment area. These data include information on the date and type of each childhood and tetanus toxoid vaccine received, date of vitamin A supplementation, and types of family planning methods used.

[8] Separate criteria were used to select adults (age 15 plus) and children (age 0–14). For the adult sample, all household heads, spouses of household heads, household members over 50, and their wives were given detailed interviews. Of the remaining 15–49-year-olds in the household roster, one individual was identified at random, and both this person and his/her spouse were selected for in-depth interviews. For the child sample, up to two 0–14-year-olds were selected at random. These selection criteria resulted in a high probability of household members being chosen. In particular, 50 percent of individuals were chosen for detailed interview with a probability of 1. 95 percent of children 15 and under year were selected with a probability of approximately 0.5.

[9] Because the number of households in a bari varies, the range of the weight is wide (2.54 to 465) and the standard deviation of the weight is large (26) and more than twice the median (10.3). Even top coding the weight at the ninetieth percentile (42) leads the highest weights to be four times the median.

[10] Because of the data collection error for the under 15 age group and the inclusion of birth year fixed-effects in the analysis, the sample sizes used to identify program effects on cognitive functioning for those who are less than 15 years of age in the MHSS are small (e.g., there are 37 observations in the treatment area for those born in 1984).

[11] Controlling for bari size in 1974 and the sampling weight in the unweighted regressions did not change the results.

The final sample includes 4,672 observations on individuals born between 1947 and 1988 for whom cognitive functioning test scores were collected.[12] Individuals age 50 or older in the household roster are dropped in order to exclude those who may be suffering from dementia and have reduced MMSE scores. Individuals who migrated into the study area, or were born to a family who migrated into the study area, after the 1977 census are also not included in the sample, since they arrived after the program was already under way.[13]

B. *Defining the Intent-to-treat Indicators*

Village of residence is used to determine MCH-FP program eligibility. A variable indicating program eligibility based on a person's 1996 MHSS village 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, individuals are linked to the 1974 census, and village of residence from the 1974 census is used to determine treatment status. Many individuals cannot be linked directly to the 1974 census since they were born after 1974 or moved into the study area between 1975 and 1977. To trace these individuals back to a household in 1974, I create a dataset that indicates each time a person entered or exited the study area and identifies the household head at that time, using census and demographic surveillance data. For those who moved in after 1974, their village of residence the first time they moved into the survey area determines treatment status. For those born after the 1974 census, the head of the household at the time they were born is traced back to the 1974 census and the village of residence of this household head is used to determine treatment status. If this household head cannot be traced back, I determine when the person of interest first entered the study site and who was the head of his or her household at that time, and trace that person back to the 1974 census. The intent-to-treat variable, *Treatment Area*, takes on the value 1 if the individual (or the household head who is traced back to the 1974 census data) resided in a treatment area in 1974, or if the individual (or household head who is traced back) migrated to a treatment village in the study area between 1974 and 1977. The 1974 baseline information is linked to individuals in the MHSS using the same method of tracing back individuals to 1974. Baseline information is missing for 48 people who migrated into or were born to a family who migrated into the study area after the 1974 census.[14]

The eligibility of a person's mother for the MCH-FP program is measured by a binary variable that takes on the value 1 if a person's mother was eligible for the MCH-FP program during her fertile years (age 11–45). In particular, she is eligible if she was 45 years old or younger in 1977. In the MHSS, mothers' information is missing for many adults since her information is available only if she lives in the same household as her child. Individuals were linked to their birth records and the 1974 and 1982 censuses in order to capture some of the missing mother

[12] Twenty-nine observations were excluded because the respondent had not been selected for a detailed interview.
[13] Information on date of entry into the study area is from the demographic surveillance site data.
[14] The baseline information for these individuals is filled in with treatment or comparison area baseline means.

information. Since mother information is still missing for many older adults, a person's mother was coded as ineligible for the program if the person themselves was age 54 or older in the MHSS survey.[15]

C. *The Mini Mental State Exam*

The MMSE measures five areas of cognitive functioning: orientation, attention-concentration, registration, recall, and language (Folstein, Folstein, and McHugh 1975). The test has been widely used to assess higher mental functioning and detect cognitive impairment among adults. Modest to high correlations have been found between the MMSE total score and other tests of intelligence, memory, attention, and executive functioning, such as the Wechsler Adult Intelligence Scale (Rush, First, and Blacker 2000). Adaptations of the MMSE are effective at evaluating the cognitive development of children as young as three years old (Ouvrier et al. 1993; Jain and Passi 2005; Rubial-Álvarez et al. 2007), and it has been shown to correlate fairly well with the Kaufman Brief Intelligence Test for children (Rubial-Álvarez et al. 2007).

The MMSE used in this study is based on the Bangla Adaptation of the Mini Mental State Examination (BAMSE) created by Kabir and Herliz (2000). The BAMSE was designed for an illiterate population and for cultural relevance to Bangladesh, and tests show that the changes made to adapt the instrument do not change the ranking of scores (Kabir and Herliz 2000).

The MMSE asks 33 questions and gives one point for each correct response, for a maximum score of 33.[16] 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. The story makes six main points (e.g., there are three children in the household, the house is on fire) and the respondent is given a point for each main point he or she repeats. 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 increases with age for children (Ouvrier et al. 1993), is on average constant for adults, and then decreases after age 55 or 60 (Strauss, Sherman, and Spreen 2006). The decline in adults is known to persist even when education is controlled. This issue is particularly salient for this paper because of the wide age range being examined. Table 2, panel A, demonstrates that the MMSE score does vary by age in the sample. The MMSE score for adults in this sample is fairly constant until the age of 50, when it starts to decline. Birth year fixed effects are included in the regression analysis in order to control for this association between age and the MMSE score.

[15] The oldest child a mother could have if she was 45 in 1977 is 34 years old in 1977 (assuming fertility starts at age 11). This 34-year-old in 1977 could be 54 by the end of 1996.

[16] The MMSE question on counting backward by seven is not included in the total score because this question is biased with respect to race and ethnicity (Strauss, Sherman, and Spreen 2006) and was determined to be culturally inappropriate in Bangladesh (Kabir and Herliz 2000).

Table 2—1996 MHSS Characteristics

	Treatment area			Comparison area			Difference in means		
	Mean	SD	Obs.	Mean	SD	Obs.	Mean	T-stat	Mean/SD
MMSE score 25–49	25.9	(8.3)	1,405	26.1	(7.3)	1,595	−0.18	−0.67	−0.02
MMSE score 20–24	27.3	(5.6)	250	27.3	(4.7)	294	0.05	0.12	0.01
MMSE score 15–19	26.7	(6.3)	281	27.6	(6.2)	355	−0.93	−1.91	−0.14
MMSE score 8–14	23.7	(9.4)	188	21.8	(11.0)	304	1.84	2.00	0.16
Height (cm) 25–49	154.5	(11.4)	1,369	155.0	(8.1)	1,558	−0.5	−1.3	−0.10
Height (cm) 20–24	155.6	(11.5)	245	155.6	(9.5)	284	0.0	0.0	0.00
Height (cm) 15–19	149.8	(8.6)	323	149.6	(12.0)	396	0.2	0.3	0.00
Height (cm) 8–14	126.3	(12.5)	675	124.8	(15.5)	876	1.6	2.3	0.10
Years of education 25–49	2.9	(6.4)	1,518	2.6	(5.6)	1,659	0.3	1.8	0.00
Years of education 20–24	5.9	(4.4)	274	5.1	(4.7)	311	0.8	2.2	0.20
Years of education 15–19	4.8	(2.9)	365	4.8	(3.3)	449	0.0	−0.2	0.00
Years of education 8–14	2.2	(2.3)	794	2.0	(2.2)	972	0.2	2.3	0.10
Age	28.9	(17.3)	2,124	28.3	(15.5)	2,548	0.61	1.25	0.04
Female (=1)	0.57	(0.41)	2,124	0.55	(0.41)	2,548	0.02	1.68	0.06
Hindu (=1)	0.16	(1.44)	2,124	0.05	(0.56)	2,548	0.11	3.42	0.10

Notes: Standard deviations (SD) are clustered at the village level. The standard deviation of the whole sample is used to create the ratio of the difference in means to standard deviation. Age, female, and Hindu is presented for the whole sample (8–49).

III. Estimation Strategy

A. *Quasi-random Program Design*

A comparison group was built into the design of the MCH-FP program. Randomization was not used to determine which households or villages belonged to the treatment and comparison areas. Instead, the treatment and comparison areas are contiguous geographic areas (Figure 1), which were socially and economically similar and geographically insulated from outside influences at the time (Phillips et al. 1982). This scheme was used to mitigate potential contamination of the comparison area from the family planning interventions (Huber and Khan 1979), and was also likely important for reducing spillovers 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 and Phillips 1990; Joshi and Schultz 2007). This is important since it means the program was 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 pre-intervention household and household head characteristics from the 1974 census. Table 3, panel A provides the means and standard deviations (SD) of the characteristics for the treatment and comparison areas for the entire sample. The differences in means are statistically insignificant at the 5 percent level for all variables except drinking water sources, number of cows, and age of household head and household head spouse. Table 2 also shows that religion differed between treatment and comparison area in 1996. [17] I also examine the normalized differences (difference in the means divided

[17] Interaction effects show that the program impacts do not differ by religion (results not reported).

Table 3—1974 Baseline Characteristics

	Treatment Area			Comparison Area			Difference in Means		
	Mean	SD	Obs.	Mean	SD	Obs.	Mean	T-stat	Mean/SD
Panel A. Full Sample									
Family size	7.01	(5.15)	2,124	6.82	(4.20)	2,548	0.18	1.34	0.04
Owns a lamp (=1)	0.65	(1.18)	2,124	0.61	(0.92)	2,548	0.04	1.37	0.04
Owns a watch (=1)	0.16	(0.94)	2,124	0.16	(0.62)	2,548	0.00	0.05	0.00
Owns a radio (=1)	0.08	(0.63)	2,124	0.08	(0.47)	2,548	0.00	0.15	0.00
Wall tin or tinmix (=1)	0.32	(1.08)	2,124	0.31	(0.78)	2,548	0.01	0.27	0.01
Tin roof (=1)	0.83	(0.66)	2,124	0.84	(0.69)	2,548	0.00	−0.10	0.00
Latrine (=1)	0.83	(0.78)	2,124	0.85	(0.86)	2,548	−0.03	−1.22	−0.03
Number of rooms per capita	0.21	(0.15)	2,124	0.21	(0.18)	2,548	0.00	0.39	0.01
Number of cows	1.55	(3.30)	2,124	1.37	(3.05)	2,548	0.19	2.02	0.06
Number of boats	0.68	(1.50)	2,124	0.68	(1.42)	2,548	−0.01	−0.20	−0.01
Drinking water, tubewell (=1)	0.31	(1.41)	2,124	0.16	(0.93)	2,548	0.15	4.35	0.11
Drinking water, tank (=1)	0.38	(1.72)	2,124	0.33	(1.68)	2,548	0.05	1.01	0.03
Drinking water, other (=1)	0.31	(2.21)	2,124	0.51	(1.84)	2,548	−0.20	−3.39	−0.09
HH age	47.8	(22)	2,124	46.5	(22)	2,548	1.28	1.97	0.06
HH years of education (edu.)	2.52	(6.72)	2,124	2.34	(5.25)	2,548	0.17	1.25	0.02
HH works in agriculture (=1)	0.61	(0.89)	2,124	0.59	(0.99)	2,548	0.02	0.79	0.02
HH works in fishing (=1)	0.05	(0.53)	2,124	0.06	(0.49)	2,548	−0.01	−0.57	−0.02
HH spouse's age	37.0	(17)	2,124	36.2	(16)	2,548	0.86	1.72	0.05
HH spouse's years of edu.	1.14	(3.58)	2,124	1.21	(2.56)	2,548	−0.07	−0.95	−0.02
Panel B. Age 8–14									
Family size	6.59	(3.09)	188	6.87	(3.20)	304	−0.27	−0.97	−0.08
Owns a lamp (=1)	0.63	(0.52)	188	0.60	(0.69)	304	0.03	0.63	0.05
Owns a watch (=1)	0.16	(0.71)	188	0.17	(0.43)	304	−0.01	−0.13	−0.01
Owns a radio (=1)	0.09	(0.51)	188	0.09	(0.29)	304	0.01	0.14	0.01
Wall tin or tinmix (=1)	0.29	(0.72)	188	0.32	(0.43)	304	−0.03	−0.49	−0.05
Tin roof (=1)	0.81	(0.45)	188	0.88	(0.52)	304	−0.07	−1.57	−0.14
Latrine (=1)	0.85	(0.39)	188	0.85	(0.52)	304	−0.01	−0.13	−0.01
Number of rooms per capita	0.21	(0.08)	188	0.21	(0.18)	304	0.00	0.09	0.01
Number of cows	1.52	(2.51)	188	1.38	(2.22)	304	0.14	0.60	0.06
Number of boats	0.67	(0.98)	188	0.67	(0.92)	304	−0.01	−0.07	−0.01
Drinking water, tubewell (=1)	0.27	(0.71)	188	0.13	(0.58)	304	0.14	2.28	0.21
Drinking water, tank (=1)	0.37	(0.84)	188	0.31	(0.99)	304	0.06	0.75	0.07
Drinking water, other (=1)	0.36	(1.12)	188	0.56	(0.97)	304	−0.21	−2.09	−0.18
HH age	48.9	(17)	188	46.3	(18)	304	2.58	1.61	0.15
HH years of education	1.92	(3.84)	188	2.10	(3.15)	304	−0.18	−0.61	−0.05
HH works in agriculture (=1)	0.67	(0.45)	188	0.55	(0.67)	304	0.12	2.29	0.19
HH works in fishing (=1)	0.07	(0.35)	188	0.07	(0.38)	304	0.00	0.11	0.01
HH spouse's age	37.6	(15)	188	35.8	(14)	304	1.82	1.37	0.13
HH spouse's years of edu.	1.00	(1.39)	188	1.20	(1.58)	304	−0.20	−1.58	−0.12

Note: Standard deviations (SD) are clustered at the village level.

by the SD of the difference) to get a sense of the size of the differences. Imbens and Wooldridge (2009) argue normalized differences greater than 0.25 are substantial. The mean of the differences that do exist are relatively small and less than 0.10 for all the differences except access to tubewell water, which is 0.11. These findings, together with previous results on fertility and mortality, strongly suggest that the two areas had very similar observable characteristics. Baseline characteristics for the 8–14 year old group, presented in Table 3, panel B, confirm that the treatment and comparison groups still look similar despite the loss of observations in this age group due to the fieldwork error discussed in Section IIA.

Before the program, 15 percent greater proportion of households used tubewell water for drinking, which is concerning since tubewell water is often thought to be cleaner than other sources of water. Because a larger percent of treatment area households had access to this water, the program effect might be biased upwards. Unfortunately, there is widespread groundwater arsenic contamination in the tubewells in Bangladesh

(Chowdhury et al. 2000; Alam et al. 2002). Arsenic is a serious health concern and has been shown to reduce IQ among 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.

B. *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 variation in 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 five cohorts of interest referred to by the age label: aged 8–11, 12–14, 15–19, 20–24, and 25–49.[18]

I would like to be able to show that the level of cognitive functioning 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 predating the intervention, it is not possible to examine the before-after program difference in cognitive functioning for any one individual or age cohort. Instead, cognitive functioning for 25–49-year-olds is used to measure the pre-intervention difference between the treatment and comparison areas. It is doubtful that the cognitive functioning of the 25–49 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 years. They were also unlikely to be affected indirectly through sibling competition, as they had reached age five by the time of the program.[19] While the MMSE score does vary by age, it is known to be constant among adults in this age group (Strauss, Sherman, and Spreen 2006). As I expected, the mean MMSE scores in 1996 for this group are the same between the treatment and comparison areas, at approximately 26 out of 33 (Table 2).

The 8–14-year-olds are the only group to have benefited directly from the intensive child health interventions, and are the main group of interest in seeking to determine the effect of these interventions on cognitive functioning. All children 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 age. The mothers of these children were eligible for family planning and maternal health interventions; so to the extent that these interventions indirectly

[18] Exact year and month of birth shown in Table 1 is used to create age groupings for the analyses.

[19] Results do not differ if the 25–29-year-olds, who were between the ages of 5 and 9 at the program start, are excluded.

affect the 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 provided only family planning and maternal health interventions in the treatment area. Given the rapid increase of contraceptive use, this age cohort could provide an early estimate of the effect of the family planning and maternal health interventions on cognitive functioning, and could be used to help partial out the effects of the family planning and maternal health interventions from those of the whole program for the 8–14-year-olds.[20] However, this group may be affected by sibling competition from younger siblings who became eligible for the child heath interventions, though it is unclear whether this would bias the effects up or down.

It is also possible that the family planning program affected the cognitive function of the 20–24 year old group through sibling competition. These children would have been between the ages of one and five 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.

C. *Empirical Specification*

The ITT effect is estimated using a double difference model. The 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 seems likely to hold given that the MMSE score was similar between the treatment and comparison areas for the 25–49 year old pre-intervention age cohort. The double difference model for person i from village v is estimated using the following linear regression:

$$(1) \qquad C_{im} = \beta_0 + \beta_1 T_v + \beta_2 AG_i^{8-14} + \beta_3 AG_i^{15-19} + \beta_4 AG_i^{20-24} + \beta_5 (T_v \times AG_i^{8-14}) + \beta_6 (T_v \times AG_i^{15-19}) + \beta_7 (T_v \times AG_i^{20-24}) + \alpha_{by} + X'Z + \varepsilon_{iv},$$

where C is the measure of cognitive functioning, MMSE z-score. 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, resided in a treatment village before the MCH-FP program started, and 0 if from the comparison area (see Section IIB for more details). β_1 represents the difference in mean cognitive functioning between the treatment and comparison areas for the 25–49 age group (the preintervention cohort). AG^Y is a binary variable used to indicate whether person i is in age group Y, so β_2–β_4 controls for differences

[20] Some children born during this time were eligible for measles vaccination past the recommended age of nine months since the measles vaccination was available to all children under the age of five 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 already have had measles before they became eligible for the measles vaccination. To the extent that some children did benefit from the measles vaccine, estimates of the effect of the family planning and maternal health interventions on cognitive functioning will be biased upward.

in cognitive functioning between the age groups. β_5–β_7 are the double difference ITT effects, and represent 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 preprogram differences in the two areas (using the 25–49 year-old cohort). α_{by} are birth year fixed-effects to control for differences in the MMSE score due to age as well as other events that may be correlated with birth year. **X** is a vector of individual (gender and religion) and baseline household and household head characteristics.[21] Standard errors are clustered at the village level to account for the likely intracluster correlation in the error term. Village fixed effects are included as a robustness check to determine whether these unobservable characteristics could bias the results.

IV. Program impacts

A. *Intent-to-Treat Program Effects*

ITT effects controlling for birth year fixed-effects and individual characteristics are presented in column 1 of Table 4.[22] Importantly, the point estimate is almost zero for the variable *Treatment Area*, which gives the difference in means between the treatment and comparison areas for the 25–49 year-old age group.

There is a statistically significant 0.37 SD difference between the treatment and comparison area 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 (0.39 SD) with the inclusion of pre-program characteristics in Table 4, column 2. This provides some confidence that the few differences in baseline characteristics are not biasing the results. The effect size is similar to that found in studies of the beneficial effects of nutrition, in particular iron, on cognitive abilities (Walker et al. 2007).

For the 15–19 year-old group the point estimates are negative and small but not significant when preintervention characteristics are included. Any positive effect the family planning and maternal health interventions may have had on cognitive development of these children may have been swamped by sibling competition from younger siblings who received more intensive child health interventions.

For the 20–24 year-old group, the point estimates are close to zero and insignificant with and without controls for pre-intervention characteristics. The lack of a significant positive or negative effect highlights that this age group does not appear to be affected by sibling competition.

B. *Robustness Checks*

It is possible that these results are affected by nontime varying unobservable village characteristics. Village fixed effects are included in column 3 of Table 4. Because of the inclusion of the village fixed effects, the coefficient on *Treatment Area* cannot

[21] Mother's and father's age and education are not included since the program may have affected them.
[22] Results without the individual controls (sex and religion) are similar.

Table 4—Intent-to-Treat Program Effects for the MMSE Z-Score by Age Group

	Double Difference OLS						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Treatment Area (=1)	−0.02	−0.04	−0.09	−0.03	−0.05	−0.05	
	(0.05)	(0.05)	(0.10)	(0.05)	(0.05)	(0.04)	
Treatment Area*(Age 8–14)	0.37**	0.39**	0.36**	0.44*	0.38**	0.40***	0.39**
	(0.16)	(0.15)	(0.15)	(0.23)	(0.15)	(0.15)	(0.16)
Treatment Area*(Age 15–19)	−0.13*	−0.12	−0.14*	0.19	−0.12	−0.12	−0.11
	(0.08)	(0.08)	(0.07)	(0.20)	(0.08)	(0.09)	(0.07)
Treatment Area*(Age 20–24)	0.02	0.00	−0.02	−0.01	0.01	−0.00	−0.03
	(0.07)	(0.07)	(0.06)	(0.22)	(0.07)	(0.08)	(0.06)
Treatment Area*(Age 8–14) * Tubewell drinking water in 1974					−0.20		
					(0.29)		
Mother ever eligible for FP (=1)						0.01	
						(0.04)	
Individual characteristics	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Preintervention characteristics	No	Yes	Yes	Yes	Yes	Yes	Yes
Village fixed-effects	No	No	Yes	No	No	No	No
Random child sample	No	No	No	Yes	No	No	No
Education fixed-effects	No	No	No	No	No	No	Yes
Observations	4,672	4,672	4,672	654	4,672	4,095	4,624
Adjusted R^2	0.13	0.19	0.22	0.21	0.19	0.19	0.26

Notes: Standard errors are clustered at the village level. Individual characteristics include year of birth fixed effects and controls for gender and religion. Pre-intervention characteristics include all variables in Table 3. OLS = Ordinary least square, FP = family planning.
***Significant at the 1 percent level.
**Significant at the 5 percent level.
*Significant at the 10 percent level.

be identified. The age group left out is still the 25–49 year-old cohort, and the interpretation of the coefficients does not change. The results show that the ITT effects do not change.

To further investigate if the results are biased for the 8–14-year-olds due to the field work error when collecting the MMSE data for the under 15-year-olds, the sample is restricted to the smaller random kid sample (Section IIA) in column 4. The point estimate is similar but slightly higher, 0.44 SD, indicating that the estimates are not likely biased due to the data collection error.

As Section IIIA explains, it is possible that the findings may be biased by differences in access to tubewell water before the program began. 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 interaction between age group and tubewell water was also included but is not reported. 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 prior to the program, this imbalance is likely to bias the estimates of the program effect downward, consistent with the presence of arsenic in some tubewell water in Bangladesh.

A control for the family planning program, a variable that indicates whether 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 of the effect of the family planning program on cognitive functioning. The point estimate is small, indicating that there is little direct effect from this variable. The double difference estimates also remain unchanged. The percent of reproductive years the mother was eligible for the family planning program may be a better estimate of her exposure to the family planning interventions. Unfortunately, this variable is highly correlated with the mother's age, which is endogenous, so is not included.

Spatially Correlated Errors.—Since the treatment and comparison areas are contiguous, 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 likelier to affect the cognitive development of younger children than of older ones, so the double difference model will not control for such shocks. 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 and 15–19 age groups I use the following test. First, I predict the errors from the base model in Table 4, column 2, and average the errors at the village level for the two age groups separately. To test whether these village-level error terms are correlated; I use Moran's I test using the Euclidean distance between village centroids as a weight. I examine whether village-level error terms are correlated for the whole sample and for each age group in the treatment and comparison areas separately. In all cases, I find no spatial correlation.

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. Mortality selection is likely to bias the results downward since frailer individuals (or those with lower health endowments) are more likely to survive in the treatment area due to the intervention, leaving a higher probability of observing someone with a lower level of cognitive functioning in the treatment than in the comparison area in the follow-up period. Indeed, the negative effect for the 15–19 year-old group in Table 4 may result from mortality selection from late measles vaccination in Treatment Area 1. To examine this possibility, I drop from the analysis children who were most likely to benefit from late vaccination in the 15–19 year-old group—that is, children who were age 2 or younger in March 1982, when the measles vaccination was introduced. The results remain the same (results not reported).

The direction of the bias due to differential migration selection between treatment and comparison area is uncertain. Out-migration probabilities in Matlab are highest among educated males, families with few resources (such as the landless), and women of marriage age (Kuhn 2005). If those with better cognitive functioning are more likely to leave for employment or higher education, the positive human capital effects of the program may result in higher migration rates of those with better cognitive functioning in the treatment than the comparison area, biasing results downward. The program may have also encouraged worse off families who might have migrated to stay in the treatment area, as the program subsidizes the cost of raising children. This incentive to remain may lead to a higher proportion of children who have lower human capital (since they come from worse off families) in treated areas, again biasing the results downward. Conversely, if those with higher cognitive functioning are more likely to delay marriage or employment in order, for example, to obtain more schooling, the results could be upward biased.

To investigate the migration bias, I use data from the demographic surveillance system to determine migration rates for boys and girls separately for the 8–14 and 15–19 year-old groups. The migration bias is likely to be negligible for the 8–14 year-old groups given that migration rates for this age group as a whole, as well as for 14 year-olds themselves, prior to the survey in 1995, are less than 2 percent, and are similar in the treatment and comparison area. This is not surprising given that in the MHSS marriage rates are less than 1 percent for girls by age 14, and school enrollment rates are over 90 percent for 12–14-year-old boys in 1996. Migration rates are higher for the 15–19 year-old group, with the 1995 rates being highest for those 19-year-olds, who were 18 in 1995, at approximately 4.5 percent for males and 5.9 percent for female. The difference in migration rates between the treatment and comparison area even for the 18-year-olds in 1995 for this age group are not statistically different.[23] In addition, people from this age group who migrated from the treatment and comparison area in 1995 are similar based on marital status, and baseline household and household head characteristics.[24] So, there doesn't appear to be large compositional change in types of migrants at least based on observable characteristics. Given the similarity in migration rates between treatment and comparison area and in the composition of migrants, it seems unlikely that selection bias due to differential migration are driving the program effects.

Spillover Effects.—The ITT effects may also be biased by the program's indirect effects on nonparticipants: informational spillovers, and the positive externalities of some of the interventions, such as vaccinations. In both of these cases, spillovers are more likely to occur in areas that border or are relatively close to the treated villages, since knowledge about the programs is likely to be spread by word-of-mouth, and the positive externalities of vaccination are largely local.

I explore this possibility using the following linear regression:

$$(2) \qquad C_{im} = \beta_o + \beta_1 C_v + \beta_2 AG_i^{8-14} + \beta_3 AG_i^{15-19} + \beta_4 AG_i^{20-24} + \beta_5 (C_v \times AG_i^{8-14}) + \beta_6 (C_v \times AG_i^{15-19}) + \beta_7 (C_v \times AG_i^{20-24}) + \beta_8 B_v + \beta_9 (B_v \times AG_i^{8-14}) + \beta_{10} (B_v \times AG_i^{15-19}) + \beta_{11} (B_v \times AG_i^{20-24}) + \alpha_{by} + X'Z + \varepsilon_{iv},$$

[23] Double difference estimates of the impact of the MCH-FP program on out-migration from the study area using the demographic surveillance data for 18-year-olds also show no statistically significant impact for either sex (results not reported).

[24] Differences in baseline characteristics of migrants aged 15–18 in 1995 between those who came from the treatment and comparison are similar to those reported in Table 3, panel A (results not reported).

Table 5—Spillover Effects on MMSE Z-Score

	(1)	(2)
Comparison area	0.03	0.04
	(0.05)	(0.05)
Comparison area*(Age 8–14)	−0.41**	−0.40***
	(0.16)	(0.16)
Comparison area*(Age 15–19)	0.11	0.12
	(0.08)	(0.08)
Comparison area*(Age 20–24)	0.00	0.01
	(0.07)	(0.07)
Comparison area*Border treatment village	0.01	
	(0.07)	
Comparison area*(Age 8–14)*Border treatment village	0.11	
	(0.20)	
Comparison area*(Age15–19)*Border treatment village	0.03	
	(0.09)	
Comparison area*(Age 20–24)*Border treatment village	−0.03	
	(0.08)	
Comparison area*Border treatment village–closest quartile		−0.04
		(0.07)
Comparison area*(Age8–14)*Border treatment village–closest quartile		0.18
		(0.32)
Comparison area*(Age15–19)*Border treatment village–closest quartile		−0.05
		(0.15)
Comparison area*(Age20–24)*Border treatment village–closest quartile		−0.13
		(0.11)
Observations	4,672	4,672
Adjusted R^2	0.19	0.19

Note: All regressions include year of birth fixed effects, controls for gender and religion, and pre-intervention characteristics from Table 3.

***Significant at the 1 percent level.
**Significant at the 5 percent level.
*Significant at the 10 percent level.

where C_v is $1 - T_v$, and where T_v is defined as in equation 1. B_v takes on the value 1 if person i, or person i's household, lived (in 1974) in a comparison village that borders a treatment village, and 0 otherwise. All other variables are defined as in equation 1. Equation 2 examines the spillover effect by splitting the comparison area into two groups: those who lived (in 1974) in a village that borders a treatment village and those who did not. $\beta_5 - \beta_7$ 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 it is in the treatment area. $\beta_9 - \beta_{11}$ are the differences in effect for the various age groups between those who lived (in 1974) in a comparison area village that borders a tretment village and one that does not border a treatment village.

The point estimates in Table 5, column 1 indicate a small, positive spillover effect of 0.11 in control areas that border treatment areas for the 8–14 age group, but the effect is not statistically significant. However, 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 therefore 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). With this specification, the spillover effect for the 8–14-year-olds is 0.18 SD higher but still insignificant. These findings weakly indicate that there may have been a spillover effect in the comparison area, so that the intent-to-treat effects may be an underestimate.

Program Impacts Using the Phasing-in of Interventions in 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 that better controls for the family planning and maternal health interventions. It is used as a robustness check due to concern about small sample sizes. As explained in Section IA, children under the age of five in half the treatment area (Treatment Area 1) were eligible to receive the vaccine starting in March 1982, and children in the other half (Treatment Area 2) in were eleigible in November 1985. As a result, children aged 12–14 in Treatment Area 1 had been eligible to receive the measles vaccination at the recommended age of 9 months, while those in Treatment Area 2 had been eligible only past the recommended age. In both areas, 12–14-year-olds had been eligible for the other child health interventions at the same time, and all of their mothers had been eligible for the family planning and maternal health interventions. Eight to 11-year-olds in both areas had been eligible for the child health interventions, including measles vaccination, at the recommended age, and, again, all their mothers had been eligible for the family planning and maternal health interventions. Therefore, the 12–14-year-olds provide an opportunity to examine whether the program effect differs for children who were eligible to receive the measles vaccination at the recommended age of nine months versus those were eligible later. As a result, the program effects are likely to be downward bias since some children in Treatment Area 2 may have benefited from late vaccination.

This phasing-in of the measles vaccine in the treatment area has been used to study the effect of the vaccine on mortality (Clemens et al. 1988; Koenig et al. 1990) and examine the morbidity and mortality risk by age of measles in vaccinated and unvaccinated populations (Fauveau et al. 1991). These studies show there was a 36–46 percent decline in all cause mortality among 9–60 month olds, and that children under the age of 12 months were at greatest risk from the disease.

I first examine 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 6, panel A demonstrates that the intent-to-treat effects are large, 0.54 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, 0.31 SD, and marginally significant at the 10 percent level for those who were eligible for the measles vaccine past the recommended age (12–14-year-olds in Treatment Area 2).

Table 6—Intent-to-Treat Effects on MMSE Z-Score Disaggregated in the Treatment Area

Panel A. Full sample	
Treatment area	−0.04
	(0.05)
Treatment area*(Age 8–11)	0.35*
	(0.21)
Treatment Area 1*(Age 12–14)	0.54***
	(0.17)
Treatment Area 2*(Age 12–14)	0.31*
	(0.18)
Treatment area*(Age 15–19)	−0.12
	(0.08)
Treatment area*(Age 20–24)	0.00
	(0.07)
Observations	4,672
Adjusted R^2	0.19

Panel B. Treatment area only sample	
Treatment Area 1	−0.11*
	(0.06)
Treatment Area 1*(Age 8–11)	0.47**
	(0.23)
Treatment Area 1*(Age 12–14)	0.33*
	(0.19)
Treatment Area 1*(Age 15–19)	−0.13
	(0.11)
Treatment Area 1*(Age 20–24)	−0.11
	(0.10)
Observations	2,124
Adjusted R^2	0.18

Note: All regressions include year of birth fixed effects, controls for gender and religion, and pre-intervention characteristics from Table 3.

***Significant at the 1 percent level.
**Significant at the 5 percent level.
*Significant at the 10 percent level.

To estimate this difference more rigorously, the analysis in Table 6, panel B is restricted to observations from the treatment area only. The intent-to-treat double difference results are estimated using Treatment Area 1 as the treated group and Treatment Area 2 as a comparison group. The sample sizes are small. There are 44 observations on children aged 12–14 in Treatment Area 1 and 53 in Treatment Area 2. The coefficient on *Treatment Area* 1 in panel B of Table 6 is small, negative, and marginally significant, demonstrating that the MMSE z-score was slightly lower for the 25–49-year-olds in the treated area.

For the 12–14-year-olds, being in the group that was eligible to receive the measles on time resulted in cognitive functioning being 0.32 SD higher in Treatment Area 1 than Treatment Area 2, and the results are marginally significant at the 10 percent level. These estimates used a different source of variation but are similar to the main finding.

Surprisingly, there continues to be a larger program effect in Treatment Area 1 for the children aged 8–11, 0.46 SD, even though for this age group the two areas received the same interventions. It is possible that part of this effect is a result of positive spillovers to children less than nine months old, who were not eligible to receive the measles vaccine in Treatment Area 1, from their healthier older siblings who already received the measles vaccination. Measles is highly contagious, and children who are too young to receive the measles vaccine are at high risk of contracting the disease and may have severe symptoms due to their younger age. In addition, delivery of the child health vaccinations, which requires careful attention to the cold chain to keep the vaccines cool, may have been better in Treatment Area 1 due greater experience.

Table 7—Effects by Subcomponent of MMSE (Z-Scores)

	Orientation (1)	Attention-Concentration (2)	Recall (3)	Registration (4)	Language (5)
Treatment Area	−0.03	−0.01	−0.04	−0.10	0.09
	(0.04)	(0.03)	(0.05)	(0.07)	(0.07)
Treatment Area*(Age 8–14)	0.28**	0.29**	0.15	0.44***	0.21
	(0.14)	(0.13)	(0.13)	(0.16)	(0.16)
Treatment Area*(Age 15–19)	−0.06	−0.08	−0.10	−0.13	−0.07
	(0.08)	(0.07)	(0.08)	(0.09)	(0.08)
Treatment Area*(Age 20–24)	0.03	−0.06	0.05	0.03	−0.07
	(0.07)	(0.07)	(0.08)	(0.07)	(0.08)
Observations	4,672	4,672	4,672	4,672	4,672
Adjusted R^2	0.22	0.21	0.06	0.08	0.05

Note: All regressions include year of birth fixed effects, controls for gender and religion, and pre-intervention characteristics from Table 3.

***Significant at the 1 percent level.
**Significant at the 5 percent level.
*Significant at the 10 percent level.

C. *Results by Subcomponent of MMSE*

Table 7 presents the results by the main subcomponents of the MMSE (orientation, attention-concentration, recall, registration, and language) for the 8–14-year-olds. The effects of the program by subcomponent are significant at the 5 percent level for orientation (0.28), attention-concentration (0.29), and registration (0.44). There are no significant impacts for language or recall, though the point estimates are positive. The reason for the lack of effect on language is likely that 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 6 out of 7, and the standard deviation is 1.

D. *Treatment on the Treated Effects*

It is important to determine the impact on those who participated in the program, as well as those who were eligible. Since program take-up is likely to suffer from selection bias, I use an instrumental variables approach in which program take-up (or receipt) is instrumented by program eligibility. Using data on receipt of the preventative child health interventions (vaccines for tuberculosis, measles, and third dose of DPT, and vitamin A) from the RKS, I create two indicator variables of receipt of child health interventions measuring different intensities of treatment: a binary variable indicating whether the child received two or more child health interventions or less (*received two plus child health interventions*), and another binary variable indicating if the child received all four of the interventions or less (*received four child health interventions*). Receipt of child health interventions is instrumented by a variable indicating whether the child was born during the period when the child health interventions were introduced (*Treatment Area * Age* 8–14), and the two measures of receipt intensity are investigated in different regressions since there is only one instrument. The main family planning interventions for mothers' of these children

Table 8—Treatment-on-the-Treated Effects

	First-stage equations			Second-stage equations	
	Received 2 plus child health intervention	Received 4 child health intervention	Mother ever used modern contraception	MMSE z-score	
	(1)	(2)	(3)	(4)	(5)
Endogenous variables					
Received 2 or more preventative child health interventions (=1)				0.46***	
				(0.17)	
Received 4 preventative child health interventions (=1)					0.76**
					(0.30)
Mother ever used modern contraception (=1)				−0.22	−0.19
				(0.17)	(0.17)
Instruments					
Eligible*(Age 8–14) (=1)	0.82***	0.49***	0.05		
	(0.04)	(0.04)	(0.05)		
Mother Eligible for MCH-FP (=1)	0.02	0.00	0.25***		
	(0.00)	(0.00)	(0.02)		
F-statistic on excluded instruments	270	74	95		
Observations	3,758	3,758	3,758	3,758	3,758

Note: All regressions include year of birth fixed effects, controls for gender and religion, and pre-intervention characteristics from Table 3.
***Significant at the 1 percent level.
**Significant at the 5 percent level.
*Significant at the 10 percent level.

was modern contraception. Program data on receipt of modern contraception from the RKS and MHSS is used to determine if a child's mother ever used modern contraception.[25] A mother's use of contraception 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 mother information is missing for many of the adults in the sample.

Two-stage least square (2SLS) is used to estimate the model and results are presented in Table 8. The 2SLS model assumes no spillovers in the treatment area, so 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 regressions in column 1–3 of Table 8 show that the instruments are highly correlated with a child receiving preventative health interventions and their mother ever using contraception (*F*-statistics are 74 or greater). The effect of the program on those who received 2 or more child health interventions is, 0.46 SD. Only 16 percent of the 8–14-year-olds in the treatment area received less than two interventions, so it is not surprising the effects are so close to the intent-to-treat effects. The effects for those children who received the 4 child health interventions is 0.76 SD, almost double the intent-to-treat effects, and demonstrates that together these interventions had a large effect on cognitive functioning. The effect

[25] Unfortunately, data is not available to measure duration of use of family planning in both intervention areas.

Table 9—Intent-to-Treat Effects on Height and Educational Attainment Z-Scores

	Double-difference OLS Height z-score		Single-difference OLS Education attainment z-score	
	(1)	(2)	(3)	(4)
Treatment Area (=1)	−0.04	−0.04		
	(0.05)	(0.05)		
Treatment Area*(Age 8–14)	0.22**	0.21**	0.17***	0.25**
	(0.09)	(0.10)	(0.05)	(0.11)
Treatment Area*(Age 15–19)	0.06	0.00	−0.05	0.00
	(0.10)	(0.10)	(0.07)	(0.08)
Treatment Area*(Age 20–24)	−0.03	0.04	0.11	0.12
	(0.12)	(0.10)	(0.07)	(0.08)
Treatment Area*(Age 25–49)			0.06	0.06
			(0.04)	(0.04)
MMSE Sample	No	Yes	No	Yes
Observations	5,726	4,468	6,342	4,562
Adjusted R^2	0.02	0.02	0.23	0.26

Note: All regressions include year of birth fixed effects, controls for gender and religion, and pre-intervention characteristics from Table 3.
***Significant at the 1 percent level.
**Significant at the 5 percent level.
*Significant at the 10 percent level.

of contraception use by the child's mother on cognitive functioning is small and statistically insignificant.

E. *Intent-to-Treat Effects for Height and Education*

One important mechanism through which the MCH-FP program may affect a child's cognitive functioning is improved nutrition in childhood. To investigate if the program indeed led to improved childhood nutrition, intent-to-treat double difference estimates on height (a common indicator of early childhood nutrition) are presented in Table 9. To account for differences in height by age and gender, height is normalized by subtracting the comparison group mean and dividing by the comparison group standard deviation for people of the same age and gender. Results are presented for both the full sample and a sample restricted to observations with nonmissing MMSE scores. The pattern of results resembles the MMSE results and are the same regardless of which sample is used. There is no program impact on the 25–49-year-olds, a statistically significant 0.22 SD increase for the 8–14-year-olds, and small and statistically insignificant effects for the 15–19 year-old and 20–24 year-old groups.

Improvements in childhood nutrition and cognitive functioning may have led to improved educational attainment and is examined in Table 9. Similarly to Joshi and Schultz (2007), a normalized z-score of the highest grade attended adjusting for age and gender, in the same manner as height, is used. The normalized z-score accounts for the fact that schooling completed differs by gender and varies more as cohorts age. Educational attainment is based on self-reports, and single difference intent-to-treat estimates are presented since the family planning interventions could have led to changes in educational attainment for some in the 25–49 year-old group through delayed child bearing. The pattern of results is similar to height z-scores. There is a statistically significant 0.17 SD increase in the education attainment z-score for the 8–14-year-olds, and no statistically significant effects for the other age groups. Interestingly, there are insignificant, but small positive effects for both the 20–24 and 25–49 year-old groups highlighting that the MCH-FP program may have affected the educational attainment of men and women who were eligible for the family planning program.

It is possible the cognitive functioning did not improve as a result of improved health or nutrition but rather due to greater education. To examine whether increased education is a mechanism through which the program led to improved cognitive functioning, education-level fixed effects are included to equation 1 (Table 4 column 7). Education is obviously endogenous, but inclusion of education fixed effects can give insight into this particular mechanism. The point estimates are stable, providing evidence that the MCH-FP program effect on cognitive functioning for the 8–14-year-olds is not a result of increased levels of education.

V. Conclusions

This paper examines the medium-term effects of a maternal and early childhood health and family planning program in Bangladesh on cognitive functioning. The findings show that cognitive functioning score of children between the ages of 8–14 in the treatment area was 0.39 SD higher than in the comparison area. These children were eligible for the early child health interventions at birth, and their mother was eligible for family planning and maternal health interventions. This effect size is similar to effect sizes in studies of the benefit of nutrition programs. The effect among children who actually received the majority of preventive child health interventions was almost twice as large. Important program effects were also found on height and educational attainment for the 8–14-year-olds. These large program effects provide needed evidence that typical maternal and child health and family planning programs improve human capital of children between the ages of 8–14, even in a high-disease environment with competing health risks. However, more research is needed to determine if these effects will continue or fade-out when children become adults.

There was a small negative effect on cognitive functioning for the 15–19 year-old group. This may be a result of sibling competition with younger siblings who were eligible for the child health interventions. However, the effects for this group were only marginally significant for some of the analyses indicating this finding may not be robust.

One limitation of this study is that the interventions were not randomly introduced in a cross-over design, making it difficult to determine the separate effects of the interventions. An important contribution of this paper has been to provide some evidence of the separate effect of the child health interventions. First, estimation by age group finds a large and significant effect of the child health program on children born after the child health interventions were introduced, but a small, negative, and statistically insignificant effect of the program for children who were born before the child health interventions began, but whose mothers were eligible for the family planning and maternal interventions. While this analysis is by itself insufficient, since family resources differ for earlier- and later-born children, adding a control for the family planning program in the ITT and TOT models still yields no evidence that the family planning program improves children's cognitive function. Lastly, using the phasing-in of the measles vaccine within the treatment area to hold constant the effect of the family planning program and other interventions, the ITT effect was similar at approximately 0.33 standard deviations. While any one of these analyses is insufficient, taken together they suggest the child health interventions likely played a large role in improving cognitive functioning.

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