The Cognitive Link between in Utero Nutrition and Development: Micronutrient Deficiency, Schooling Attainment and Economic Outcomes in Tanzania

Plamen Nikolov*

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Abstract

Because of the high returns of schooling in developing countries, policy-makers have placed considerable attention to increasing school access. However, an important mechanism through which brain development in utero can affect demand for education exists. Cognitive development in utero, due to maternal deficiency in folic acid, can biologically constrain children's demand for education. Using a more scientifically credible research designed to detect causal effects than has been used in previous research, we examine how reductions in micronutrient deficiency in utero impact subsequent child schooling attainment in Tanzania. We also examine to what extent parents allocate resources so as to compensate for or to reinforce inequalities across children in cognitive endowments. Capturing the behavioral response to the biological intervention will allow us to disentangle the biological effect from the household response to the original randomized intervention. To execute this strategy, we follow up on a randomized control trial with micronutrient supplements offered to HIV-negative pregnant women in Dar es Salaam, Tanzania between 2001-2003.

1 Introduction

Over the past several decades, there has been considerable attention placed on increasing schooling in developing countries. Psacharopoulos (1994) estimates microeconomic returns to education as high as 42% per annum in Botswana (for primary education) and 47.6% per annum in Zimbabwe (for secondary education). These large returns have stimulated a concerted effort in investing in education to stimulate growth (UNESCO 2007). However, an important mechanism through which *in utero* environment can affect education exists. Poor *in utero* health conditions can biologically constrain cognitive development[1]. By not accounting for this important channel, estimates of returns to education may be inaccurate and possibly biased upwards. This paper seeks to challenge the magnitude of previous microeconomic estimates of returns to education by accounting for the effect of health investments *in utero* on subsequent educational attainment. How much do biological setbacks - due to pre-natal maternal malnutrition - in brain development *in utero*, influence subsequent child schooling attainment? To what extent do parents allocate resources so as to compensate for or to reinforce inequalities across children in endowments? Does health environment *in utero* play an important causal role in constraining lifetime social mobility and how big exactly is that role? These questions are of importance to economists in a variety of fields

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[1]Health's direct impact on labor productivity and household earnings has been extensively documented (Thomas et al., 2003; Basta et al., 1979; Sommer et al., 1986, 1981; Glasizou et al., 1993; Beaton et al., 1992).

(development economics, macroeconomics, population economics, health economics, etc.) and have both positive and normative implications. Results from this project will provide answers to the three questions presented and will inform education and nutrition policy.

While most micronutrient deficiencies are likely to be resolved with improvements in economic outcomes by way of rising caloric intake, deficiency *in utero* for four nutrients in particular (B_6, B_9, B_{12}, and iodine) has been biologically linked to irreversible and continuous damage to cognitive development throughout an individual's lifetime (Bottiglieri et al., 1995; Bryan et al., 2002; Guilarte 1993; Fioravanti et al., 1997; Hankey 1999; Alpert and Fava, 1997; Schneede 1994; Cao et al., 1994; Hetzel and Mano, 1989; Pharoah and Connolly, 1987). Because of this persistent effect on learning over the lifespan, the proposed research project will examine the effect of these nutrients on subsequent cognitive development, enrollment and attendance rates and educational attainment. We will accomplish this by following up on a randomized individual-level trial of nutritional supplements offered to non-HIV pregnant women in Dar in Tanzania conducted between 2000 and 2003 Specifically, we plan on estimating several reduced-form specifications to test how (1) micronutrient supplementation affects children's cognitive development, (2) micronutrient supplementation affects children's school attendance and school enrolment, (3) how parents invest in children with various cognitive and health endowments.

In Tanzania, as in other Sub-Saharan African countries, dietary intake of nutrients in pregnant women is marginal or lower than the recommended intakes and therefore these women are at high risk for deficiencies (Mulokozi 2003; FAO, 1995; FAO, 1996a; FAO, 1996b; WHO, 1994; WHO/UNICEF, 1995). However, unlike general nutrition shortages in utero that can lead to lower educational attainment, increased rates of physical disability, lower income and lower socioeconomic status (Almond 2006), fetal deficiency in B_6, B_{12} and B_9 (folic acid) permanently limits intellectual ability. Thus, the impact of this particular deficiency is likely to be particularly acute and persistent across individuals' lifespans. B_9 (folic acid) and B_{12} deficiencies occur mainly due to the increased requirements during pregnancy which are not balanced by adequate dietary intake, and the high prevalence of malaria (Kavishe, 1987; Kavishe, 1991). The prevalence of deficiency in B_9 ($<$5ng/mL[2]) and deficiency in B_{12} ($<$200pg/mL) is respectively as high as 80% (Fleming, 1989; Baker, 1981; Massawe et al., 1996) and 60% (DeMayer, 1985; Van den Broek, 2000) for women in some Sub-Saharan African countries.

We focus on Tanzania due to the high general micronutrient deficiency and the particular deficiency in the micronutrients on which we intend to focus (Kavishe, 1987; Kavishe, 1991). Recent studies with blood analyses among pregnant women and non-pregnant adults[3] in Dar es Salaam revealed a 70% prevalent deficiency of B micronutrients including thiamine, riboavin, and micronutrient B_{12} (Fawzi 1999; Mulokozi 2003). If true, the resulting loss in cognitive capacity could have important consequences for aggregate human capital accumulation in afflicted settings, leading to a lower fraction of children enrolling in or attending school, slower rates of grade attainment for age, and fewer students progressing to institutions of higher education.

The remainder of this paper is structured as follows. Section 2 introduces the mechanisms through which micronutrient deficiency can affect the demand for education. Section 3 sets up the field experimental design. Section 4 provides more details about the data collection methodology and the project schedule during 2010-12. Section 5 outlines the specific empirical identification strategy.

2 Conceptual Framework: Mechanisms by which Health Determines Education Outcomes

Schooling can enhance the production of health (Adams et al., 2003; Auster et al., 1969; Grossman, 1976; Grossman and Joyce, 1987; Taubman and Rosen, 1982; Berger and Leigh, 1989; Behrman and Wolfe,

[2]Deficiency levels are based on Hillman and Ault's 'Hematology in clinical practice' (1994).

[3]Not only that deficiency levels for B6, B12 and folic acid differ between pregnant and non-pregnant women but deficiency for these nutrients can cause physical health problems even in non-pregnant adults.

1989; Kenkel, 1991, 1995) but unobserved variables such as one's preferences, time discounting (Fuchs, 1982; Farrell and Fuchs, 1982) and upbringing can affect both health and education in the same direction (Rosenzweig and Schultz, 1983). We bypass the limitations of causal interpretations based on associations between education and health outcomes by exploiting an exogenous health shock with the use of a medical randomized trial. Prenatal and early-life health interventions can affect education through three direct and indirect mechanisms.

2.1 Through Permanent Limitation of Intellectual Ability

The first mechanism is a biological one. Growing evidence that the health and nutrition of young children has a long-term effect on their cognitive development exists (Liebenstein 1957; Walker 2000; Chang 2002; Grantham-McGregor 1994; Pollitt 1995; Berkman et al. 2002). Unlike nutritional shortages during childhood or adulthood, fetal malnutrition for the nutrients chosen in this study (B_6 and B_{12} and folic acid) is believed to permanently limit intellectual ability, so its impact is likely to be particularly acute and persistent. Micronutrient B_6, comprising three chemically distinct compounds, pyridoxal, pyridoxamine, and pyridoxine, is involved in the regulation of mental function and mood. B_6 is also an essential homocysteine re-methylation cofactor, and deficiency is associated with an increase in blood homocysteine levels. Homocysteine is a risk factor for cerebrovascular disease and may also have directly toxic effects on neurons of the central nervous system. Recent work in animal models suggests that micronutrient B_6 deficiency during gestation and lactation alters the function of N-methyl-D-aspartate receptors, a subtype of receptors of the glutamatergic neurotransmitter system thought to play an important role in learning and memory (Guilarte 1993). Maternal blood levels of folic acid (B_9) and B_{12} have also been causally linked to neural tube defects (Botto et al., 1999; Christensen et al., 1999ab; Czeizel and Dudás, 1992; Laurence et al. 1981; MRC, 1991). There have been at least two observational studies among children[4] with B_{12} deficiency. In the first study, infants of macrobiotic mothers in The Netherlands had delayed motor and language development compared to infants of omnivores (Schneede 1994). At age 12, the children had higher methylmalonic acid levels and scored lower than the omnivores on standardized assessments, including the Raven's progressive matrices, Digit Span and Block Design, even though their current diet contained almost their recommended daily intake of micronutrient B_{12} (Louwman 2000). In the second study, Guatemalan children with micronutrient B_{12} deficiency had slower reaction time on neuropsychological tests of perception, memory and reasoning (Allen 1999; Penland 2000).

2.2 Through Physical Health and Immune Deficiency Leading to Improved School Attendance

The second mechanism, also a biological one, works through the immune system and improved general physical health, both of which can lead to improved school attendance. Ample evidence that better physical health improves education levels exists for the US (Edwards and Grossman, 1979; Shakotko et al., 1981; Shakotko and Grossman, 1982; Perri, 1984; Wolfe, 1985; Berger and Leigh, 1989). Miguel and Kremer (2004) find that deworming of children in Kenya increases school attendance. An underactive immune system leads to an increased risk of disease[5]. Studies have associated B micronutrients with immune function (Bendich and Cohen, 1988). B_9 has hematological benefits during pregnancy (Fleming et al. 1986). Lack of pyridoxine (B6) appears consistently to inhibit cell-mediated immune function as well as humoral responsiveness to a variety of test antigens. Through randomized trials, B_6 and B_{12} deficiencies have been causally linked with fetal development (Ramakrishnan et al. 1999).

[4]Research linking micronutrient B12 deficiency to cognitive functioning has also been conducted in the elderly, where it has been associated with dementia and neurobehavioral decits (Rosenberg 1992).

[5]Neuropsychiatric disorders including seizures, migraine, chronic pain and depression have been linked to micronutrient B6 deficiency (Malouf and Grimley 2008).

2.3 Through Increased Lifespan

The third mechanism, an indirect one, works through the nutrients[6] effect on increasing prospective lifespan, which can increase incentives to invest in human capital. This effect occurs for the individual for whom the benefits of education are now greater (Kalemli-Ozcan, Ryder and Weil, 2000). In addition, lower infant mortality may encourage parents to invest more resources in fewer children, leading to low fertility but high levels of human capital investment in each child (Kalemli-Ozcan, 2002). Evidence for this effect is limited, though Bils and Klenow (2000) do find an effect of life expectancy on investments in education at the national level.

3 Experimental Design, Survey and Data

3.1 Research Design

The paper uses follows-up data on a recent randomized medical trial of pregnant women (Fawzi 2007). The double-blind trial, assigning 8468 pregnant women to receive a daily oral dose of either a micro-nutrient supplement or placebo, examined the effect of nutrient supplementation on low birth weight (<2500 g), prematurity, and fetal death. We will collect follow-up survey information on parental post-natal child investments, educational attainment and cognitive development of the children born to the women who participated in the original medical trial.

In 2001-2004, Fawzi (2003) conducted a randomized trial with pregnant women in Dar es Salaam, Tanzania to account for differences in physical health outcomes at birth for children. Pregnant women who attended antenatal clinics[7] in Dar es Salaam, Tanzania, between August 2001 and July 2004 were invited to participate in the trial. Simple random sampling was used. Requirements for eligibility included a negative test for HIV infection, a plan to stay in the city until delivery and for 1 year thereafter, and an estimated gestational age between 12 and 27 weeks according to the date of the last menstrual period. A list was prepared according to a randomization sequence in blocks of 20; at enrollment, each eligible woman was assigned to the next numbered bottle. The treatment tablet included 20 mg of micronutrient B_2 , 25 mg of micronutrient B_6 , 50 mg of micronutrient B_{12}, and 0.8 mg of B_9 (folic acid). The active tablets and placebo were similar in shape, size, and color and were packaged in identical coded bottles[8].

3.2 Outcomes

We follow up with a sub-sample of the original population of 8468 children to collect additional outcomes.

3.2.1 Enrollment and attendance status

We focus on an array of current and past year's schooling status of the children in the interviewed households. In particular, we collect information for both the treatment and control subjects for several key variables: (1) enrollment rate status (E); (2) various current and past measures of school attendance.

[6]While it is likely that the studied micro-nutrients exert a direct effect on lowering mortality and therefore increased life expectancy, they likely have a much stronger effect on life expectancy through proximate determinants, such as improved cognitive development and improved morbidity, both of which ultimately can decrease mortality on the population level.

[7]According to a DHS 1996 Bureau of Statistics Tanzania Report (Bureau of Statistics, 1996), 97% of pregnant women attend antenatal care (ANC), and 70% do so at least four times.

[8]At every monthly visit, a new bottle was given to each woman, and the pills remaining in the used bottles were counted. All women completed a baseline questionnaire that included their socio-demographic characteristics and obstetrical history. Laboratory investigations at baseline included tests for syphilis, gonorrhea, and trichomoniasis; routine urine and stool tests; and evaluation of blood lms for malaria.

3.2.2 Cognitive Development

In addition to recording enrollment rate and school attendance differences, a battery of cognitive tests will be administered to children. Four tests will measure cognition, memory and general intelligence. The first, and primary, of these is a version of the Peabody Picture Vocabulary Test (PPVT), a test of receptive vocabulary that has been widely used in developed and developing countries[9]. Children are shown a series of slides with four pictures each (for example, the first slide has a picture of an ashlight, a boat, a basket, and a hot-air balloon), and are asked to point at a given object stated by the enumerator (for example, boat). Test items gradually become more difficult. The enumerator records the number of correct and incorrect responses, and the test stops when a child is making as many errors as she would be expected to make if she were randomly guessing[10]. We will also use a digit span test, assessing short-term memory for strings of orally presented digits (in order of presentation and then, in a separate test, in reverse order), a categorical fluency test, assessing the number of animals and food types children can name in two one-minute sessions, and a visual search test, assessing the speed at which children identify target pictures from amongst distracters. There will be two tests corresponding to the two constituent factors of general intelligence, fluid intelligence (inductive reasoning) and crystallized intelligence (knowledge) (Cattell 1943). Raven's Coloured Progressive Matrices Test will assess children's reasoning ability, and a Tanzanian adaptation of the Mill Hill Vocabulary Test (Raven 1998) will measure knowledge. For tests of verbal ability (the vocabulary and proverbs tests), only questions that will be equivalent in the local languages will be used[11].

3.2.3 Parental Investment

Parents expenditures on their children's skills, health, learning, motivation, 'credentials,' and their home investment are all forms of parental investment in the human capital of their children (Becker and Tomes 1986, p. S5). Measuring past parental time investment raises telescoping and memory lapse problems, which can lead to severe recall biases (Chen, Mu and Ravallion, 2006). Therefore, we will attempt to collect some recall data on parental home investment (e.g. hours per week participated with children, self-reported warmth, and months breastfed) but primarily, we will rely on school expenditures and early childhood health data collected from the trial on vaccinations and visits with the child to doctors. To the extent possible, we intend to capture parental investment information for both parents.

3.2.4 Other Outcomes Measures

We capture various other outcome measures:

(1) child's physical health, (2) wealth index for each individual and household of subject who was part of the initial randomization, (3) child's other socio-economic characteristics (wealth index for each individual and household of subject which was part of the initial randomization), (4) parents' socioeconomic characteristics, (5) parental investment indicators, (6) children's physical health (P), (7) location and migration history, (8), fertility outcomes after the randomized trial, (9) parent expectations of future child mortality collected retrospectively, (10) parent beliefs about child health endowment at birth, (11) parental human capital investment on siblings (PI).

[9] See, for example, Paxson and Schady (2007, 2008), Umbel et al. (1992), Baydar and Brooks-Gunn (1991), Blau and Grossberg (1992), Rosenzweig and Wolpin (1994), and Fernald, Gertler, and Neufeld (2008).

[10] Before the test starts, the enumerator explains the test with the help of a few example slides. She proceeds to the actual test slides only once the child has demonstrated understanding of the test.

[11] All measures will piloted and tested for validity and testretest reliability, assessed through correlation between scores from repeated test sessions one week apart. Tests will be administered in one session lasting around 70 minutes in a quiet area in homes of survey participants. All testing will be done in the child's language of preference. Children will be fed a sandwich before testing to reduce the effects of hunger on performance (Simeon 1998). The additional information will enable us to test the hypotheses outlined below.

4 Causal Identification and Hypothesis Testing

4.1 Hypotheses

The objective of this research paper is ultimately to test the following null hypotheses related to the outcomes (let T and C denote treatment and control group outcomes respectively) as defined in Section 3.2:

Table 1: Hypotheses To Be Tested

Hypothesis 1	H_0: E_T=E_C	Does a statistically significant difference in cognitive development
	H_a: E_T>E_C	between the treatment and control groups exist?
Hypothesis 2	H_0: PI_{T1}=PI_{T2}	Does a statistically significant difference in parental investment between
	H_a: PI_{T1}>PI_{T2}	the treatment group siblings and born-prior-to-study siblings exist?
Hypothesis 3	H_0: PI_T=PI_C	Does a statistically significant difference in parental investment affecting
	H_a: PI_T≠PI_C	infant health between the treatment and control groups exist post study?

4.2 Methods and Identification Strategy

We estimate the effect of micronutrient supplementation on child schooling in a specification in which the primary outcome of interest is a school attendance/enrollment outcome and cognitive development measured by one of the tests as outlined earlier.

The Tanzanian formal education system involves seven years of primary education, four years of junior secondary (ordinary level), and two years of senior secondary (advanced level). Although primary enrollment rates have been high since the late 1990s, very few children transition to secondary school. In 2001, gross enrollment in primary school was 85% but only 7% in secondary school, largely due to an insufficient supply of secondary schools. In 2001, one quarter of rural households reported being over 20 kilometers from a secondary school (THBS, 2001), while only 8% reported the nearest primary school to be more than 6 kilometers away.

In Tanzania, as in many African countries, there is high variance in the rate at which children progress through primary school. Meanwhile, since few children drop out in the age range to which our analysis is restricted (primary school), progression is presumably a considerably more sensitive indicator of final schooling attainment than enrollment. Throughout the country, primary schooling is characterized by high variation in age of entry, high rates of grade retention and intermittent enrollment. Particularly in rural areas, gross enrollment ratios are substantially higher than net ratios because many over-age children are present in primary schools due to beginning schooling late and progressing slowly. Although teachers have some room to retain students for attendance and behavioral problems, the main reason for repetition in primary school is exam failure, and repetition rates are highest in grades at which students take national standardized tests and in grade 1. Despite the fact that there is no national examination in the first year of school, Standard 1 has the highest repetition rate of all grades (12:3% in 2000). The large fraction of children that fails to pass school-specific Standard 1 assessments is attributed to repetition-related overcrowding in grade 1, and high variation in preparedness at school entrance on account of differences in age of entrance and access to preschool education (World Education Forum, 2001). Retention is lower in grades 2 and 3, but then jumps again in grade 4 when students take the Standard 4 exam.

To test the first hypothesis, we restrict our specification[12] to the following:

$$cognitive\,score_{if} = \alpha + \beta_1(T_{if}) + \beta_2(A_{if}) + \beta_3(H_{if}) + \beta_4(X_{if}) + \mu_f + \delta_{if} \quad (1)$$

[12] cognitive score$_{if}$ will be replaced with the respective outcome variable (e.g. enrollment status/physical health/test score) which will allow me to test one of the four hypotheses outlined earlier.

We plan on estimating several specifications with current educational enrollment and various continuous measures of current or past year's educational attendance outcomes:

$$educational\ status_{if} = \alpha + \beta_1(T_{if}) + \beta_2(A_{if}) + \beta_3(H_{if}) + \beta_4(X_{if}) + \mu_f + \delta_{if} \quad (2)$$

, where $educational\ status_{if}$ will be captured both with enrollment and actual school attendance outcomes.

In the specifications above, T_{if} is the binary variable that child i of mother f was treated with vitamin supplement in the original medical trial, A is a vector of birth-month dummies, H is a vector of physical health variables, and X includes binary controls for gender, sex-specific birth order and other socio-economic variables. We will also include interaction effects. β_1 in the equation above is the key parameter of interest and it captures the educational attainment effect of mother's vitamin endowment during pregnancy. To examine whether the fetal effects of vitamins are stronger for females, we will also run the above regression separately by gender. The characteristics known prior to randomization (X) are also analytically useful. For example, age, race, marital status, family structure, education, employment, mobility history, social contact can all be derived from information collected by the initial study at the time of random assignment. Due the randomization technique, the distribution of X's should be the same within the treatment and control groups because they are statistically independent of group assignment. Unless these characteristics known prior to randomization happen to differ between groups due to the variability in a small sample including in a regression like the one above will not change the coefficient. As in all fixed effect estimates, identification of the causal effect of T requires that the error term be uncorrelated with treatment, conditional on the observables contained in X and sibling average grade attainment (μ_f). The difference in outcomes between the treatment and control group, known as "Intent-to-Treat" (ITT) effect, is captured by the ordinary least squares (OLS) estimate of the coefficient β_1. We can use treatment assignment as an instrumental variable to estimate the parameter commonly known as "the effect of Treatment-on-Treated" (TOT). The TOT parameter measures the average effect of the treatment on those in the treatment group who actually receive the treatment. One estimate of TOT is ITT divided by the proportion receiving the treatment.

To refine our estimate of the treatment impact, our analysis will make use of anticipated variation in the impact of micro nutrient supplementation based on individual variation in deficiency for these nutrients. Given that the level of micro-nutrients provided to the treatment group was uniform across individuals and treatment coverage was not based on level of need, the relationship between baseline deficiency rates and treatment impact is likely to be non-linear. In other words, we anticipate a threshold level of micro-nutrient deficiency below which rates are too low to observe a significant treatment effect, and a second threshold (e.g. 400 mcg of folic acid) above which the treatment will be insufficient to protect against maternal micro-nutrient deficiency due to factors which raise daily requirements for micro-nutrient intake. The treatment impact among individuals in the lower tercile of the treated population is likely to be larger than it is for individuals with the highest baseline rates of micro-nutrient deficiency. These predictions will be tested by studying variation in program effect by level of consumption of foods rich in naturally occurring folate (or naturally occurring B_6 and B_{12}). Both participants' blood sample information at baseline but also the food consumption for foods rich in naturally occurring folate (lentils; legumes; dried beans and peas; broccoli, spinach, collard or turnip greens, okra, and asparagusthat) will provide us[13] with some information to identify heterogeneous effects.

To test the second hypothesis, we will exploit within- and between family sibling variation. Essentially, we will use a combination of the randomization and a difference-in-difference technique to use the previous siblings as a control group ($\Delta = S_{ift} - S_{ift-1}$). In addition to collecting information for the outcome variable below on the index child which was part of the original trial, we will randomly select another sibling, born before the trial, from the household. We restrict our specification to the following:

[13]The district rate of food consumption for food items 8-9 years prior can be used to proxy for variation in dietary intake.

$$parental\,investment_{if} = \alpha + \beta_1(T_{if}) + \beta_2\Delta + \beta_3 * T * \Delta + X_{if} + \delta_{if} \quad (3)$$

Here T_{if} is the binary variable that child i of mother f was treated with vitamin supplement in the original medical trial, $\triangle$ is a difference of parental investment between siblings within the same family, and X includes binary controls for gender, sex-specific birth order and other socio-economic variables. β_3 in the equation above is the key parameter of interest for the difference-in-difference estimation and it captures the parental investment response within the family in response to the vitamin endowment increase during pregnancy.

To test the third hypothesis, we restrict our specification to the following:

$$parental\,investment_{if} = \alpha + \beta_1(T_{if}) + \beta_2(A_{if}) + \beta_3(H_{if}) + \beta_4(X_{if}) + \mu_f + \delta_{if} \quad (4)$$

parental investment in the specification above will focus on post-intervention outcomes that affect infant health after birth other than the vitamin supplements. The idea is that the treatment group might in theory become more complacent than the control group about engaging in health investments into infant health once the trial's purpose is "unblinded". Simple microeconomic framework will predict that as parents realized that their infant's health has been positively affected by their participation in the trial, the parents could become complacent about the incentives for further private health investments into infant health and could reduce further investing in their infant health post the intervention. So, we would focus for parental investments feeding into infant health other than vitamin supplementation. Here T_{if} is the binary variable that child i of mother f was treated with vitamin supplement in the original medical trial, A is a vector of birth-month dummies, H is a vector of physical health variables, and X includes binary controls for gender, sex-specific birth order and other socio-economic variables. β_1 in the equation above is the key parameter of interest and it captures the difference between treatment and control groups in post-intervention health investments affecting infant health.

4.3 Potential Threats to the Identification Strategy

Several factors complicate an estimation of treatment effects on children. These factors include: (i) the possibility of health externalities; (ii) sample attrition, and (iii) intent to treat effect on mediating factors.

4.3.1 Estimating Treatment Externalities

The estimation of treatment effects in this paper is complicated by the possibility of health externalities. Health externalities arise when the benefits of treatment (such as a medicine) are felt beyond the actual recipient of the treatment. In this case, it might be reasonable to assume that within-household externalities are present. We will follow the approach of recent papers, such as Duflo and Saez (2002), Miguel and Kremer (2002), Katz, Kling, and Liebman (2001), Kremer and Levy (2001), and Sacerdote (2001) that use individual-level randomization of treatment to estimate peer effects. To see how spillovers can lead to biased estimates of treatment effects, consider the simple situation in which a treatment is randomly allocated across a population of individuals and compliance is perfect. Using the potential outcome framework, the intention-to-treat estimate is ITT=$E[Y_{\mathrm{i}}^T| = 1] - E[Y_{\mathrm{i}}^C| = 0]$. In order to interpret this difference as the effect of the treatment, the standard unit treatment value assumption (SUTVA) must hold. SUTVA says that the potential outcomes for each individual are independent of his treatment status, as well as the treatment group status of any other individual (Angrist, Imbens, and Rubin 1996). If this is violated, $E[\hat{Y}_{\mathrm{i}}^C| = 0]$ in the sample is not equal to $E[Y_{\mathrm{i}}^C| = 0]$ in the population, since the sample contains both treated and untreated individuals. The potential outcome for each individual (and therefore the ITT) now depends on the entire vector of allocations to treatment and comparison groups. If the spillover effects on untreated individuals are generally positive, then the intention-to-treat estimate ITT will generally be smaller than it would have been without spillovers. Similar to Miguel and Kremer

(2002) and Katz, Kling, and Liebman (2001), we attempt to estimate regressions of the form that captures the outcome of interest for each individual and include a measure for the number of treated individuals within distance d of a treated individual for treated subjects and all subjects.

4.3.2 Sample Attrition

Finally, despite the experimental design of the study, causal inference could fail in the presence of high attrition rates and differential attrition across treatment groups. Recent work on panel surveys in developing countries has demonstrated that focus respondents who move away from the location in which they were originally surveyed differ appreciably from those who stay put and thus have substantial informational value (Thomas 2001). In order to combat the bias arising from such sample attrition, all reasonable efforts will be made to track and interview the full sub-sample selected from the original sample, as described earlier. First, if attrition is due primarily to refusal to participate in the follow-up, subjects who receive vitamin supplementation benefit and subjects who received the placebo do not benefit, it is possible that controls will be more likely to attrit than treatments. On the other hand, if the treatment results in side-effects, they may be more likely to attrit[14]. If attrition is due to geographic mobility, and if the treatment results in an increase in children's physical health, then treated subjects may more likely to move in search of new economic opportunities. Treatments would be more likely to attrit. At the very least, we will look for evidence that attrition is not related to treatment-control status. We will compare birth and children's health and mothers health characteristics at baseline and compare whether it is related to attrition. We will also report a regression that includes an interaction between treatment status and each of the co-variates (health characteristics of child and mother's SES collected at baseline and age in years), for all observations and by gender. We will also conduct an F-test to test whether the interactions are jointly significant for males and females. If, however, we do find evidence of attrition being nonrandom with respect to treatment, we will use matching methods described by Hirano, Imbens and Ridder (2000)

4.3.3 Intent to Treat Effects on Mediating Factors

The supplementation treatment may also create differential change in a nexus of several mediating factors that ultimately could influence the outcome of interest. For example, it may be that the mechanism through which the treatment affects educational attainment works though a child's physical health in early childhood rather than the child's cognitive development. Therefore, by collecting information on psycho-social health characteristics, we can examine the impact of the treatment on general health status of the children. A more complicated mechanism can even work through parental labor force participation. For example, children with poor health require more parental time, which in turn decreases the amount of time their parents spend in the labor force and decreases their earnings potential; this, in turn, will affect the investment in their children's human capital. Another important factor to account for is parental education (Lin, Liu and Chou 2007). Parental education could serve as a proxy for the parent's ability to respond quickly and efficiently to early childhood health shocks due to poorer child's health. A final issue of concern relates to whether interviewer surveys are blinded to the randomization status. To address this, we plan to provide no *ex ante* knowledge of the original treatment status of each household they interview[15]. To the extent that such differences differ across the treatment and control groups but become more pronounced only in response to treatment, we would need to account for them.

[14] Of course, we are not so concerned that one group attrits more than the other but particularly interested in the possibility of selective attrition.

[15] Unfortunately, we have no way of controlling if household members casually mention their treatment status to interviewers in the course of the survey instrument. We will, however, emphasize in the enumerator training sessions we intend to organize for the interviewers/surveyors to strictly adhere to question protocol and not ask any other questions outside of the survey instrument.

Another interesting issue is parental attitudes towards differences in gender. Becker (1981) suggested that parents may discriminate against daughters if the returns from investing in sons are higher. This will suggest having a control for child gender.

5 Sampling

5.1 Tracking Exercise

Of the 8379 women with known birth outcomes in the original medical randomized trial study (Fawzi 2007), 8137 gave birth to live babies and were eligible for the analyses of birth weight and prematurity outcomes. This subsection briefly outlines the tracking procedure that we execute.

Table 2: Schedule of Project Activities

Activity/Year	**1 (2004)**	**2 (2010)**	**3 (2011)**	**4 (2012)**
Data Collection (Round 1: Fawzi 2007)	X			
Data Collection (Round 2: Follow-up)		X	X	X
Data entry and cleaning			X	X
Analysis				X
Dataset publicly available				

5.1.1 Stratified Sampling With Optimal Allocation

Due to health and socio-economic differences in different ethnic groups of the original population, a stratified sampling wherein the population is divided into ethnic strata is most appropriate. We will divide the total population of the random sample into N members of H mutually exclusive and collectively exhaustive groups[16]. Given the fixed budget C, we would like to select a sample of size n among different strata to minimize the variance of the sample estimate, allocation known as *optimal allocation of sample* (Snedecor and Cochran, 1980). The optimal proportion of the sample that should be given to the jth stratum is $n_j = \frac{N_j s_j}{\sum N_i s_j}.n$, where $s_j$is the sample standard deviation for the jth stratum. The total sample n will be given by $n = \frac{1/N\,(\sum N_j s_j)2}{N\sigma^2_{\bar{x}_{st}} + 1/N \sum N_j s_j^2}$, where $\sigma^2_{\bar{x}_{st}}$ is the desired variance of the sample mean. With varying costs per strata c_h, the allocation rule for sample size within strata is $n_h = \frac{N_h \sigma_h I \sqrt{c_h}}{\sum_{i=1}^{H} N_i \sigma_i I \sqrt{c_i}}$,where $N_h$is stratum population size, $\sigma_h$is stratum standard deviation, and $\frac{1}{\sqrt{c_h}}$ is inverse square root of stratum data collection costs.

5.1.2 Two-stage Tracking

Tracking of children whose mothers were part of the original randomized trial will occur in two phases: regular tracking and intensive tracking. Regular tracking will be based on the random sub-sample (approximately 3000) of the original study population (8379 births). After 3 months, we anticipate that we would find and successfully interview approximately 60% of the children and parents comprising our sub-sample based on a very similar endeavor in Kenya (Baird, Hamory and Miguel, 2007). Of the remaining unfound respondents of this sub-sample, we would select a quarter for more intensive tracking. The intent of the intensive tracking will be to observe when regular tracking begins to wind down, to choose

[16]Because the strata are mutually exclusive, we know that $N_1 + N_2 + ... + N_n = N$, where $N_i$is the number of members from each strata.

one quarter of the remaining unfound focus respondents to be sought intensively for another 6 months, and then to multiply the sampling weights of these individuals by four to account for the unfound focus respondents who were not tracked during the final months of field interview enumeration. We could then maintain a representative sample by merely re-weighting individuals[17] of this intensive sub-sample in the final analysis, so that each respondent in the intensive sample represents the other unfound individuals who were not intensively tracked.

6 Results

Data from the field experiment is being finalized and will be available in November 2011. I plan to present preliminary results, on the four hypotheses outlined in Section 4.2, at the conference.

[17] The probability weights applied to individuals in this intensive tracking sample will be adjusted in the final data set. Sampling will be performed in STATA 11 and, in general, precede as follows: within each stratum, children will be assigned a uniform (0,1] random variable, children will be sorted by this variable, and the first n children in that stratum will be assigned to the sample. Creation of the intensive tracking sample will be performed in STATA 11 using the "sample" command.

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