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

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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 allows 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.

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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 economic outcomes (UNESCO 2007). However, an important mechanism through which *in utero* environment can affect education and economic outcomes exists. Poor *in utero* health conditions can biologically constrain cognitive development[1]. Using experimental data from Tanzania, and building on pilot data we already collected in 2011, we focus on two key questions (1) How much do biological setbacks - due to pre-natal maternal malnutrition - in brain development *in utero*, influence subsequent child schooling attainment? (2) To what extent do parents allocate resources so as to compensate for or to reinforce inequalities across children in endowments?

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 three of these nutrients, B_6, B_9 and B_{12}, on subsequent cognitive development, school enrollment, attendance rates, educational attainment and parental behavior responses. 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. We plan to address the two research questions above by following up on a randomized individual-level trial of nutritional supplements offered to $HIV-$ pregnant women in Dar in Tanzania conducted between 2000 and 2003. Tracking data collection started in July 2011 and so far effective tracking rate of more than 90% has been maintained.

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). 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[3].

We believe our study improves on previous research in six major ways. First, the original RCT included folic acid and induced variation in a very specific dimension of the child's endowment–cognitive ability–whereas previous studies[4] focus on measure on non-specific proxies which may reflect many dimensions of the child's endowment or many mechanisms through which the effect operates[5]. Second,

[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).

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

[3] 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 in Dar es Salaam revealed a 70% prevalent deficiency of B micronutrients including thiamine, riboavin, and micronutrient B_{12} (Fawzi 1999; Mulokozi 2003).

[4] See Almond (2006), Rosenzweig and Zhang (2009), Rosenzweig and Schultz (1983), Rosenzweig and Wolpin (1988), Pitt et al. (1990).

[5] For example, Maccini and Yang (2009) find long-run effects of early-life variation in rainfall in rural Indonesia. While well-identified, a drawback of the study is that the operative channels are not well understood. The effects are hypothesized to operate via household

previous studies rely on siblings fixed effects models for identification and are likely mis-specified, since even within siblings, unobserved prenatal, neonatal and early-infancy investments in each child likely correlate with whichever endowment measure is used and also with the extent of investment during the remainder of childhood[6]. Third, we expand the set of resource allocation behaviors studied in previous work to include not only neonatal and early-life investments, but also the allocation of resources in later childhood. Fourth, we conduct actual cognitive tests and non-cognitive tests with children. Fifth, we measure parental post-natal investment behavior on various margins including parental care-taking, childhood envionment, psychological care, household and parental expenditures for children. Sixth, and perhaps most importantly, since all the previous work in this area has restricted attention to within-sibling or within-twin estimates, studies have been unable to separately identify own endowment vis-'a-vis sibling endowment effects. Since our empirical strategy does not necessitate the imposition of similar fixed effect models, we are able to identify these two endowment effects separately.

2 Conceptual Framework: Mechanisms by which Health Determines Education Outcomes

Prenatal and early-life health interventions can affect education through four general direct and indirect channels.

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[7] 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).

income (which fluctuates with rainfall in agricultural households), but the authors cannot be sure of this it is possible, for example that a direct health channel might be operative.

[6] For example, see Rosenzweig and Zhang (2009), Rosenzweig and Schultz (1983), Rosenzweig and Wolpin (1988), Pitt et al. (1990).

[7] 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).

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[8]. 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).

2.3 Through Increased Longevity

The third mechanism, an indirect one, works through the nutrients[9] effect on increasing prospective lifespan, which can increase individual 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.

2.4 Through Indirect Parental Responses

Final mechanism relates to parental responses to childrens' biological endowments. Investments in children, as outcomes of the household's allocation problem, are in general jointly determined by parents' preferences for each child; the production function for child quality; and the intra-household distribution of initial endowments. Depending on the parameters of this allocation problem, parents may choose to reinforce or compensate for initial endowments. Second, exogenous shocks to children's health could influence subsequent parental labor supply decisions related to increased time for child care-taking. The sign and magnitude of these behavioral responses indicate whether–and by how much–parental investments magnify or dampen the biological effects of early-life interventions.

3 Simple Model

Schooling choice

We start with a simple model based on the original Ben-Porath's human capital acquisition framework. Abstracting from the household allocation issue for the time being, we start with a set-up in which individuals choose how much education (denoted e) to obtain to maximize discounted lifetime earnings, y, and examine how these schooling investments change as a function of child quality (denoted q). To keep things simple, we posit that child quality comprises both child's health status and cognitive skills. The discounted future income benefits to schooling are $b(e,q)$, and the costs (including both direct tuition costs and the opportunity cost of time spent in school rather than working) are $c(e,q)$. Both the benefits and costs are increasing in education and child quality (b_e, b_q, c_e and c_q are all positive), but the marginal benefit of schooling declines with more education ($b_{ee} < 0$) while costs are convex ($c_{ee} > 0$). Both benefits and costs increase mechanically with child quality (for example, health status if "non-sick" time

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

[9] 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.

increases), thus expanding the effective time budget. An individual's optimal educational investment level $e^\star$ is determined by the first order condition $y_e(e^\star, q) = 0$, and equates marginal benefits to marginal costs, $b_e(e^\star, q) = c_e(e^\star, q)$.

We can show that:

$$\frac{de^\star}{dq} = -\frac{b_{eq} - c_{eq}}{b_{ee} - c_{ee}} \tag{1}$$

By the usual assumptions above, the denominator is negative, but the numerator is more difficult to sign. Both derivatives are likely to be positive, in other words, improved child quality boosts the marginal benefit of both school learning ($b_{eq} > 0$) and the opportunity cost of time (as labor productivity improves, $c_{eq} > 0$), but there is no obvious sign on the difference *ex ante*.

Household Allocation

In addition to initial endowments, household allocation can also influence cognitive development and schooling outcomes. The allocation of resources among children in the household clearly can take place according to at least two alternative types of decision rules. One criterion for such allocation decisions would be equality. There are two potential aspects here, though: equality of inputs (all children receive the same), or equality of outcomes (entailing compensatory distribution, with those who start with less in the way of initial endowments receiving more). An alternative criterion would be to base allocation decisions on efficiency. In this scenario, resources would go to those children who offer the best prospects of gaining the most or providing the greatest return to parents for the "resource investment." This entails reinforcing distribution.

To add the parental response and siblings to the model, we consider a household with two children indexed $i = 1, 2$. Each child is born with an exogenously given endowment of quality q_i. The distribution of endowments determine the within-family distribution of prices of investment in additional quality, h_i, which parents may decide to undertake. We denote the price of quality for child i as $p_i \equiv p(q_i)$, where p is a decreasing function of q, capturing the fact that a dollar of investment in quality will yield larger returns for the child with a relatively higher endowment[10].

Parents value the quality of their children, with preferences $u(h_1, h_2)$[11]. Parents' utility[12] is given by:

$$u(h_1, h_2) = \left(\alpha h_1^{\frac{\gamma-1}{\gamma}} + (1-\alpha) h_2^{\frac{\gamma-1}{\gamma}} \right)^{\frac{\gamma}{\gamma-1}} \tag{2}$$

$\alpha \in (0, 1)$ represents the relative utility weight given to the quality of child 1, and $\gamma \in (0, +\infty)$ is the elasticity of substitution[13].

The household's utility maximization problem is:

$$\underset{h_1, h_2}{max} = \left(\alpha h_1^{\frac{\gamma-1}{\gamma}} + (1-\alpha) h_2^{\frac{\gamma-1}{\gamma}} \right)^{\frac{\gamma}{\gamma-1}} \tag{3}$$

$$s.t. \quad p_1 h_1 + p_2 h_2 \leq W \tag{4}$$

$$p_i = p(q_i), \qquad i = 1, 2 \tag{5}$$

From setting up the first-order conditions and simplifying, we get:

$$h_1 = \left(\frac{\alpha p_2}{(1-\alpha) p_1} \right)^{\gamma} h_2 \tag{6}$$

[10] This assumption is equivalent to assuming that endowments and investments are complements in the production function for quality. Aizer and Cunha (2011) document that the assumption of complementarity is common in the literature.

[11] This is based on Becker's common preference model [Becker (1964, 1974, 1981)]

[12] We adopt a *CES* utility function, which parametrizes the extent to which children's qualities are complements or substitutes.

[13] Child quality is complementary when $\gamma < 1$, and substitutable when $\gamma > 1$.

Denote $\Theta \equiv \left(\frac{\alpha p_2}{(1-\alpha)p_1}\right)^{\gamma}$. The demands for quality as functions of prices, income and the model's primitives are:

$$h_1 = \frac{W\Theta}{\Theta p_1 + p_2} \tag{7}$$

$$h_2 = \frac{W}{\Theta p_1 + p_2} \tag{8}$$

3.1 Comparative Statics

We now examine the effects of a shift in the endowment of child 1 on investments in quality for both children. Because we have modeled endowments as factoring only into prices, equivalently we can examine the effects of a shift in p_1. Differentiating the demand functions (7) and (8) with respect to p_1 and rearranging terms, we obtain:

$$\frac{\partial h_1}{\partial p_1} = \frac{W}{(\Theta p_1 + p_2)^2}\left(p_2 \frac{\partial \Theta}{\partial p_1} - \Theta^2\right) \tag{9}$$

$$\frac{\partial h_2}{\partial p_1} = -W \frac{\Theta + p_1 \frac{\partial \Theta}{\partial p_1}}{(\Theta p_1 + p_2)^2}. \tag{10}$$

The signs of the own- and cross-price elasticities thus depend in part on $\frac{\partial \Theta}{\partial p_1} = -\Theta(\frac{\gamma}{p_1}) < 0$. From here, it is apparent that $\frac{\partial h_1}{\partial p_1} < 0$. The sign of $\frac{\partial h_2}{\partial p_1}$ depends on $\Theta + p_1 \frac{\partial \Theta}{\partial p_1}$. From above, $\frac{\partial h_2}{\partial p_1} < 0$ iff $\Theta + p_1 \frac{\partial \Theta}{\partial p_1} > 0$. We can reduce this condition to $\frac{\partial h_2}{\partial p_1} < 0$ iff $0 < \gamma < 1$.

3.2 Testable Hypotheses

Prediction 1: If $-\frac{b_{eq} - c_{eq}}{b_{ee} - c_{ee}} > 0$, then as q increases $e^{\star}$ increases.

Prediction 2: $\frac{\partial h_2}{\partial p_1} < 0$ iff $\Theta + p_1 \frac{\partial \Theta}{\partial p_1} > 0$. In other words, a rise in a child's own endowment generates increases in parental investments in that child.

Prediction 3: $\frac{\partial h_2}{\partial p_1} < 0$ iff $0 < \gamma < 1$. In other words, a rise in a sibling's endowment increases own investments if child quality is complementary in the household's utility function, and decreases own investments if quality is substitutable.

4 Experimental Design, Survey and Data

4.1 Research Design

Original Randomized Control Trial

We plan on collecting follow-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. 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[14] in Dar es Salaam, Tanzania,

[14] 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.

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 7 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)[15] [16]. 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.

Original RCT Results and Tracking Information

Fawzi finds mean difference of birth weight between treatment and controls groups of 67 g ($P < 0.001$). Treatment had no significant effects on prematurity or fetal death.

As part of the original trial, the research team collected information on socio-economic status of study participants, their family members, bio-marker samples at baseline and after six months following birth. Detailed map cue information was collected in 2001-2003 enabling the original research team to locate study participants in their home and conduct a follow-up interview with them and their households.

Pilot Phase Follow-up

In July 2011, a team, 5 enumerators and tracking personnel, started follow-up tracking effort collecting a number of outcomes outlined in more detail in the next section. All hired enumerators and tracking personnel had had extensive experience with tracking in Tanzania for the The Kagera Health and Development Survey (KHDS), a study into the long-run wealth dynamics of households and individuals within North West Tanzania. The KHDS survey, and the same team we use for this tracking, had maintained a highly successful tracking rate with tracking individuals in Eastern Africa: in 2010 88% of the original 6353 respondents[17] had either been located and interviewed, or, if deceased, sufficient information regarding the circumstances of their death collected.

5 Tanzanian Educational System

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

[15] The active tablets and placebo were similar in shape, size, and color and were packaged in identical coded bottles.

[16] 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.

[17] Baseline for KHDS was collected between 1991-1994; For more information on the KHDS tracking, see http://www.edi-africa.com/research/khds/tracking.htm

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.

6 Identification Strategy

6.1 Empirical Specifications

To test the first of our predictions, we restrict our specification to the following:

$$cognitive\,score_{if} = \alpha + \beta_1(T_{if}) + \beta_2(A_{if}) + \beta_3(H_{if}) + \beta_4(X_{if}) + \mu_f + \delta_{if} \tag{11}$$

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

$$educational\,status_{if} = \alpha + \beta_1(T_{if}) + \beta_2(A_{if}) + \beta_3(H_{if}) + \beta_4(X_{if}) + \mu_f + \delta_{if} \tag{12}$$

where $educational\ status_{if}$ will be proxied both with enrollment and actual school attendance outcomes[18].

In the specifications above, T_{if} is the binary variable that child i of mother f was treated with micronutrient 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 micronutrients 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 makes use of anticipated variation in the impact of micro nutrient supplementation based on individual variation in deficiency for these nutrients.

[18] We outline 6.2.1 and 6.2.2 the specific survey proxies of the outcome variables in (11) and (12).

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 are 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[19] with some information to identify heterogeneous effects.

To test our second and third predictions, we exploit within- and between family sibling variation. Essentially, we 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 randomly select another sibling, born before the trial, from the household. We restrict our specification to the following:

$$parental\,investment_{if} = \alpha + \beta_1(T_{if}) + \beta_2\Delta + \beta_3 * T * \Delta + X_{if} + \delta_{if} \tag{13}$$

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.

Related to the second prediction above, we test for parental responses across treatment/placebo groups when study was unblinded:

$$parental\,investment_{if} = \alpha + \beta_1(T_{if}) + \beta_2(A_{if}) + \beta_3(H_{if}) + \beta_4(X_{if}) + \mu_f + \delta_{if} \tag{14}$$

parental investment in the specification above will focus on post-intervention outcomes that affect infant health after birth other than the miconutrient 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 (Peltzman, 1975). So, we would focus for parental investments feeding into infant health other than micronutrient 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. I outline below under Sub-section *Parental Behavior Responses* empirical proxies that we plan on using to estimate (13) and (14).

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

6.2 Outcomes

6.2.1 Cognitive Development and Non-Cognitive Skills

Because abilities are multiple in nature, we administer a battery of cognitive tests and non-cognitive tests to children for at least 2 children in both treatment and control households. Broadly, we test memory, access to information tasks, speed of processing, verbal acquisition and impatience. All cognitive tests described below were used in the Makwami study, conducted in Bagamoyo, Tanzania and designed by a team of psychologists at the Harvard Graduate School of Education[20].

To test memory, we administer 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 *Corsi block test*, a test designed to test spatial memory. To test verbal ability, we administer the *Peabody Picture Vocabulary Test (PPVT)* with questions equivalent in Swahili[21]. We test psycho-motor skills with a *Pegboard task*, in which the child has to insert a number of awkwardly shaped pegs into a specially made board.

We also administer tests for various non-cognitive character traits such as determination, dependability, persistence, self-esteem, optimism, and time preference based on Sternberg (1985), Goodman (1999) and Goodman and Scott (1999). For example, to test impatience, we conduct a cookie version of the *Marshmallow test* based on Shoda et al. (1990). The cookie test is conducted by presenting a cookie to the child. The child is given an option to eat the cookie now or asked to wait for five minutes by not eating the cookie for a final outcome of two cookies. If the child decides not to wait, he or she could eat the first cookie within the first five minutes. In the case the child eating the cookie within the first five minutes, we record the number of seconds it takes the child until he starts eating the cookie.

6.2.2 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; (2) current and past measures of school attendance. (3) current and past school passing rates, (4) current and part schooling examination results.

6.2.3 Parental Behavior Responses

Parents time allocation and money expenditures on their children's skills, health, learning, motivation, development of 'credentials,' are all important forms of parental investment in the human capital of their children (Becker and Tomes 1986, p. S5) and ultimately could influence both cognitive and non-cognitive development and schooling outcomes [22]. We collect current and recall data on a number of measures: parental perceptions of their children's development throughout various stages in childhood, parental educational expenditures, parental home investment (e.g. hours per week read to own children or played with own children, self-reported warmth, and months breastfed), child vaccinations, parental time for emotional support, post-natal parental behavior and non-cognitive parental assessment based on Goodman (1999) and Goodman and Scott (1999).

[20] See Sternberg et al. (2002).

[21] All measures were piloted and tested for validity and test-retest reliability, assessed through correlation between scores from repeated test sessions one week apart. Tests were administered in one session lasting around 70 minutes in a quiet area in homes of survey participants. All testing was done in the child's language of preference. Children were fed a sandwich before testing to reduce the effects of hunger on performance (Simeon 1998).

[22] Measuring past parental time investment raises telescoping and memory lapse problems, which can lead to severe recall biases (Chen, Mu and Ravallion, 2006).

6.2.4 Other Outcomes Measures

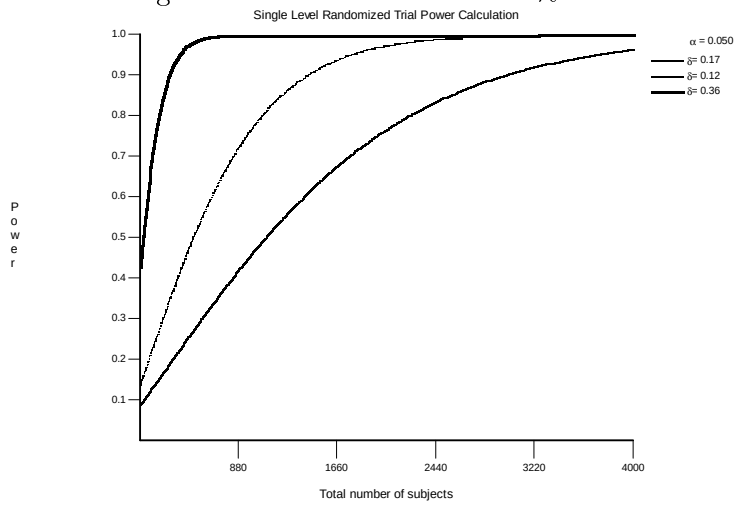
In the survey instrument used in the field, we collect various other variables:

(1) child's past and current physical health based on clinical records and self-reported data, (2) current household and individual wealth, asset and expenditure information, (3) current and past household demographic, fertility and educational history, (5) current and past household income generating activities, (6) current and past household member clinical and self-reported ilness information, (7) location and migration history,

7 Sampling

7.1 Power Calculations

Figure 1: Power for One-sided 5% Test



In choosing the number of participants to follow up with, we conducted power calculations to ensure that we have a sample size that provides us adequate statistical power that a false null hypothesis will be correctly rejected (Cohen 1988). Using the statistical package *Optimal Design*, we computed this probability under the three different scenarios related to the expected effect size (the three scenarios are based on the magnitude of the effect of iodine treatment program on schooling in Tanzania based on Field *et.al.* (2008)). Based on the power test (power level of 90%), the number of subjects we need to track ranged from 400 to 3050 (for the most conservative effect size).

7.2 Two-stage Tracking Exercise

Tracking of children whose mothers were part of the original randomized trial will occur in two phases: regular tracking and intensive tracking. Regular tracking was based on the random sub-sample (approximately 3000) of the original study population (8379 births). After 5 months, we found and successfully interviewed approximately 80% of the children and parents comprising our sub-sample. Of the remaining unfound respondents of this sub-sample, we selected a quarter for more intensive tracking. The intent of the intensive tracking was to observe when regular tracking begins to wind down, to choose 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[23] 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.

8 2011 Pilot Phase Results

8.1 Sample Attrition

Searching for individuals in rural East Africa is an onerous task, and migration of target respondents is could be problematic in the absence of exact address information. Despite the experimental design of the study, causal inference could fail in the presence of high attrition rates and differential attrition across treatment groups. If key explanatory variables, and most importantly micronutrient treatment assignment, were strongly related to attrition, then resulting estimates might suffer from bias. In the pilot phase, we are currently following the sampling strategy described above. Those sampled are being tracked "intensively" (in terms of enumerator time and travel expenses) for the remaining months, while those not sampled are no longer actively tracked. We plan to re-weight those chosen for the "intensive" sample by their added importance to maintain the representativeness of the sample. As a result, we report here "effective" tracking rates (ETR), calculated as a fraction of those found, or not found but searched for during intensive tracking, with weights adjusted properly. The effective tracking rate (ETR) is a function of the regular phase tracking rate (RTR) and intensive phase tracking rate (ITR) as follows:

$$ETR = RTR + (1\text{–}RTR) * ITR \tag{15}$$

This is closely related to the tracking approach employed in the Moving to Opportunity project (Kling et al. 2007, Orr et al. 2003). Based on our pilot effort so far, the RTR is 77.3% and the ITR is 63.4%, which implies an effective tracking rate of 91.69 by the tracking team. Reassuringly, survey tracking rates are nearly identical in the treatment and control groups. We ran a regression of attrition status in the follow-up wave against baseline treatment status and various baseline socio-economic variables and none of the coefficients in this estimation were statistically significant.

8.2 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 plan to 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[24].

8.3 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

[23] 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.

[24] 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=$E[Y_i^T| = 1] - E[Y_i^C| = 0]$. 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, $E[\hat{Y}_i^C| = 0]$ in the sample is not equal to $E[Y_i^C| = 0]$ 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.

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 psychosocial 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[25]. 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.

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 suggests having a control for child gender.

9 Research Contribution and Policy Implications

In utero exposure to four specific micro-nutrients can biologically, irreversibly, and permanently affect subsequent cognitive development, educational attainment and ultimately economic outcomes. Our story is quite simple: the proposed study aims to contribute to the economics literature by calibrating the importance of *in utero* micro-nutrient deficiency for human capital formation, and improves on previous research in several key dimensions. First, the study will be based on a randomized trial, a technique entirely removing any selection bias. Second, in contrast to previous studies - because the initial randomization was at the individual level and because of a much larger sample size (8469 children) - our ability to detect effects is significantly greater[26]. Third, we improve on previous studies' rather narrow focus on academic and cognitive tests not only by administering a wider set of cognitive tests but also by using the Primary School Leaving Examination (PSLE) pass rates by gender, available from the Tanzania Ministry of Education[27]. A fourth area of scientific contribution will cast light on recent field evidence that female fetuses are more sensitive than male ones to *in utero* folic acid exposure[28]. Due to the individual randomization and the follow-up information, we will be able to rule out[29] the possibility that gender differences are driven by sex-specific household responses to improvements in cognition rather than disproportionate increases in female cognitive capacity. Finally, we pin down a specific mechanism through which health can affect income via education by even more narrowly disentangling the individual importance of physical health, cognitive development and increases in longevity.

The proposed research, combined with previous positive findings on the long-term health effects of the prenatal period (Almond 2006), will help explain the gradient between adult health and economic outcomes. That fetal health may be at the fulcrum of this relationship also suffers no shortage of policy implications. First, the large gap in the research literature on *in utero* health may be causing us to miscalculate the benefits of nutrition programs precisely because they do not account for the cognitive development channel, which we focus on in this proposal. Even holding schooling attainment constant, small differences in average IQ at the group level could have large effects on social and economic outcomes.

[25] 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.

[26] For instance, in the well-known INCAP study, Martorell et al. (1995) provided different nutritional supplements to Guatemalan children and later find significant impacts on their cognitive skills during adolescence. However, that study randomly assigned children to the treatment and comparison groups at the village level, and thus has an effective sample size of only four villages.

[27] The current study will use data from the Tanzania Standard 1-4 Examinations.

[28] See Berry et al. (1999).

[29] Field, Robles and Torero (2008) can not distinguish between the biologic mechanism and sex-specific household responses due to natural experiment design at the district level.

Second, our results will provide answer to the practical challenge of identifying the effects of a particular improvement to fetal health and whether public policies that achieve these improvements are cost-effective. Finally, results from this study could help policy makers accurately prioritize nutrition interventions and more clearly understand how to improve education in developing countries. The existence of linkages between fetal health and economic outcomes imply that resources are not being allocated optimally across the life cycle: individual investments and public policies that benefit maternal and fetal health have been under-funded if fetal origins effects have not been accounted for in expenditure decisions, as they presumably have not. Therefore, social welfare can be substantially improved.

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