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Is 1+1 more than 2? Joint evaluation of two public programs in Tanzania *

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Abstract

Human capital accumulation is considered essential for the diffusion of modern economic growth in the developing regions of the world. Due to low levels of personal incomes and missing credit markets, state run policies are of extreme importance in ensuring higher levels of human capital. Traditionally, the cost benefit analyses of such government run development programs have taken a partial equilibrium approach. However, if one (or more) independent programs exhibit complementarity towards an objective, a partial equilibrium approach will understate the net benefits of such programs. In such scenarios, it becomes essential to jointly evaluate the impact of these programs, allowing for possible complementarity between the programs. We evaluate the joint effect of two public programs from Tanzania, the Iodine Supplementation Program and the Primary Education Development Program, and find that these programs exhibit dynamic complementarity. Independent evaluations of these programs understate the benefits.

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1 Introduction

A large body of literature has documented the pivotal role of human capital in development. Romer (1990) incorporates human capital in a model of endogenous growth, and shows that the initial level of literacy can have a big impact on level of investment in and rate of growth of a country's economy. Becker (1993) notes investment in human being paves a way for development, and considers human capital to be the most valuable kind of capital. It is, therefore, crucial to understand the production process for human capital formation. Previous studies have assumed investments made in human capital early and later in life are substitutes in the production of human capital. Based on this assumption, Becker (2009) notes crowding out of parental investment catalyzed by Head Start leads to reduction in effect of Head Start. However, Cunha and Heckman (2007a) provides a different theoretical framework that argues it is more reasonable to consider human capital formation as evolutionary process. According to Cunha and Heckman (2007b), effect of later investment depends on early-life investments in human capital, and that complementarity between investments at different stages exist. Investments made in human capital later in life are likely to be more productive for individuals who receive higher investments in early periods vis--vis those who received lower investments.

The empirical evidence for this model of complementarity is rather scarce. A major empirical challenge in examining whether later investments are complementary to investments at early stage is the endogeneity of such investments. As Almond and Mazumder (2013) points out, an estimation of such a causal model requires "... 'lightning to strike' twice: two identification strategies affecting the same cohort but at adjacent developmental stages. Clearly this is a tall order." Aizer and Cunha (2012) uses parental investment at the very early age calculated by the factor analysis and the Head Start Program of 1996 in the US, and find larger gain in the IQ of children with greater human capital at early age. Bhalotra and Venkataramani (2012) exploits introduction of sulfa drugs and racial segregation in southern states of United States to find bigger impact of the drug in less racially segregated areas. The evidence from developing countries is almost non-existent. There are at least three reasons as to why the question needs to be studied in the developing country context. First, early life investment in human capital in most areas of most developing countries is lower than what the scientific community believes it ought to be. It is, therefore, of immense importance to understand to what extent are later life investments able to compensate for such lack of investments. Second, the complementarity between such inputs might be different in developed versus developing countries if the relationship is non linear. Third, the magnitudes and frequency of negative shocks are higher and the prevention mechanism inadequate and less effective in developing countries. Adhvaryu et al. (2014) uses extreme rainfall shocks in birth years and years of exposure to PROGRESA, a government social assistance (welfare) program, as two exogenous shocks in order to study the complementarity in investments in human capital. They find that the impact of one positive shock is smaller for those who had earlier been exposed to another positive shock. However, the mechanisms through which these rainfall shocks might affect human capital formation is not very straightforward. While lower rainfall may affect the earnings of households, it also might reduce the infectious disease environment and might have a positive impact on human capital formation. Also, it is important to understand if such an observation is specific to the area and shocks being studied or is it more general.

In this paper, we use the Iodine Supplementation Program (ISP) and the Primary Education Development Program (PEDP) in Tanzania as two exogenous shocks that might affect human capital formation of exposed children. ISP started in 1986 and continued till 1997 and provided iodine supplementation to women and children in certain years in areas with high prevalence rates of Iodine deficiency disorder (IDD) Peterson (2000). IDD is closely associated with cognitive development of the foetus in utero. Field et al. (2009) analyze the impact of ISP using the Tanzania Household Budget Survey 2000 (THBS 2000) and find that it had significant positive impact on completed years of schooling of treated children. According to Field et al. (2009), treated children complete 0.345 more years, and effect of ISP was bigger for girls (0.59) than boys (0.19). In contrast, Bengtsson et al. (2013) find no significant impact of the program using various data sets including the one that Field et al. (2009) use. One important difference between the two studies is that Bengtsson et al. (2013) use the data only until 1994. The reason for doing that is according to Peterson (2000), Tanzania took up universal salt iodization in 1994. Our results show that ISP had a negative impact on the completed years of schooling of treated children. However, we do find some suggestive evidence that ISP might have had a positive impact on the cognitive ability of the treated children. ISP treated children tend to make better use of each additional year that they spend at school than others.

Primary Education Development Program (PEDP) was introduced in 2002 in order to increase primary school enrollment rate in Tanzania. PEDP abolished all tuitions and fees for primary school in order to lower barriers preventing children from entering a school. According to Tanzanian census reports, net enrollment rates went up significantly, from 66% in 2001 to 97.3% in 2007, due to PEDP. Consistent with the existing empirical evidence [Riddell (2003),Mbelle (2008)], we find significant positive impact of PEDP on completed years of schooling. We find that the interaction of the two interventions has a negative impact on completed years of schooling but no impact on grade progression. However, those children who are treated by both the programs are the best of the lot in converting extra years of school into completed years of schooling.

We show that ISP treatment status is negatively related to early investment in human capital by parents. Parents tend to compensate for the lack of ISP treatment and this compensation becomes stronger if the cohorts are exposed to PEDP. Therefore, we find evidence of complementarity in early parental investment and later public investment through PEDP. Effect of later investment, PEDP, is stronger for those children who received higher investment at early stages.

Our contribution, through this paper, is multi-fold. First, we evaluate the impact of ISP on both the skill levels and the cognitive ability of the exposed children. Second, we establish that PEDP had positive impact on human capital accumulation by itself, controlling for ISP exposure. Third, we add to the evidence on compensation by parents in response to early negative shocks to children. Our paper illustrates how neglecting parental response behaviour can lead to faulty interpretation of program evaluation results. Fourth, we show that there exists significant complementarity in early and later investment in a developing country. In our knowledge, we are the first to do so.

The remainder of the paper is organised as follows. In section II, we set up the theoretical and empirical framework for testing complementary between inputs in skill formation. In section III, we briefly describe the ISP and PEDP interventions. Section IV describes the data and the empirical strategy. In section V, we report the estimates and the results. Section VI concludes.

2 Theoretical and Empirical framework

2.1 Theoretical framework

Consider a simple model of skill formation where the skill levels of a child in period $t + 1$, S_{t+1}, is function of the skill level of child in period S_t and the investment made in that skill accumulation I_t in period t:

$$S_{t+1} = f(S_t, I_t)$$

with $f_1' > 0$ & $f_1' > 0$. A iterated substitution of skill at each level will lead to a production function of skill that is a function of all the investments made in the child from just before he was conceived, and perhaps even before birth in terms of investment made in the mother's health before pregnancy. However, as Glewwe et al. (2001) point out, it is practically impossible to measure the cumulative inputs in the formation of a skill. We, therefore, use the simple alternative of using the child's skill level in period t as a summary measure for the inputs made in that skill up to period t. The subject of interest is the form of the production function of skill. While the answer to that question might be a rather elusive, we can, however, examine the degree of complementarity or substitutability between the two inputs in production.

Let us suppose that the true functional form is Leontief, one where the two inputs are perfect complements. That is,

$$f(S_t, I_t) = min(aS_t, bI_t)$$

Let us, for exposition, assume that $a = b = 1$. Then a one unit change in I_t will change S_{t+1} by a magnitude equal to 1 if $S_t - I_t \geq 1$, by $S_t - I_t$ if $0 < S_t - I_t \leq 1$ and will not change

S_{t+1} at all if $S_t - I_t \leq 0$. However, suppose the two inputs were perfect substitutes.

$$f(S_t, I_t) = aS_t + bI_t$$

Let us again assume, for simplicity, that $a = b = 1$. Then the impact of the one unit change in I_t will be a full one unit change in S_{t+1}. In other words, the impact of I_t on S_{t+1} will not depend on the value of S_t if the two inputs are substitutes but will depend almost entirely on S_t if the two inputs are complements. It is easy to see that a cobb douglas production function lies between these two extremes. Mathematically, we say that there is complementarity between the two inputs if

$$\frac{\partial^2 f(S_t, I_t))}{\partial S_t \partial I_t} > 0$$

A bigger positive value of this expression implies a greater degree of complementarity between the two inputs. The closer the value of this expression is to zero, greater is the degree of substitution.[1]

2.2 Empirical framework

The empirical analogue of the above model is the following:

$$S_{t+1} = \alpha + \beta_1 S_t + \beta_2 I_t + \beta_3 S_t * I_t + \epsilon_t$$

where β_3 is the coefficient of interest. It tells us about the change in the impact of I_t on S_{t+1} with change in S_t. The empirical challenge in estimating such an empirical model is that both S_t and I_t are endogenous. Therefore, we need to find exogenous variation in both these inputs. We need, as mentioned before, a 'lightning strikes twice' situation, two shocks affecting the same cohort but at adjacent developmental stages.

Given two such exogenous shocks, the identification of the interaction effect, that is whether there is any complementarity between the two inputs or not, is akin to a difference in difference estimation. We want to emphasize here that this framework does not estimate the production function for skill S_{t+1} but merely examines the complementarity between two inputs in the production process.

Consider the situation described in **??**, where we are interested in the interactive effect of treatment 1 and 2. Sub-population A does not receive any treatment. Sub-population B receives treatment 1 and Sub-population C receives treatment 2. Sub-population D receives both treatment 1 and treatment 2. Under difference in difference assumptions, treatment effect

[1] Cunha and Heckman (2007a), & Cunha et al. (2010) call this dynamic complementarity, which they argue, unlike static complementarity, implies that investments in one period increase the marginal productivity of investments in a later period.

Table 1: Empirical methodology

	Pre treatment mean	Post treatment mean
Sub-pop. A: Control 1, Control 2	p	w
Sub-pop. B: Control 1, Treatment 2	q	x
Sub-pop. C: Treatment 1, Control 2	r	y
Sub-pop. D: Treatment 1, Treatment 2	s	z

of treatment 1, $\beta_1 = E\{y - r - w + p, z - s - x + q\}$ and treatment effect of treatment 2, $\beta_2 = E\{x - q - w + p, z - s - y + r\}$. Treatment effect of Shock 1 & 2 combined, $\beta_1 + \beta_2 + \beta_3 = E\{z - s - w + p\}$. Therefore, Interaction effects of the two shocks $\beta_3 = \beta_1 + \beta_2 + \beta_3 - \beta_1 - \beta_2$. In our study, we use the Iodine Supplementation Program in Tanzania as a shock to S_t and the Primary Education Development Program of Tanzania as an exogenous change to I_t. The details of the two program are discussed in the following section.

3 Background

3.1 Iodine Supplementation Program in Tanzania

Lack of proper nutrient, especially in utero, have been proven to be detrimental to the physical and cognitive development of the individual (Cao et al. (1994), Zimmermann et al. (2005), Barker (1995), Barker (1990), Barker et al. (2002)). It impairs the body's ability to develop and work at its full capacity and also weakens the immune responses and capability to the body to protect it against infectious diseases. These impact an individual's capacity to accumulate skills, which, in turn, might have long term impacts on the labour market outcomes of the individuals (Almond and Currie (2011), Almond (2006)). Both the short term and long term impacts of lack of macro and micro nutrients have been studied extensively (Thomas et al. (2006), Basta et al. (1979), Scholz et al. (1997), Sommer et al. (1981), Sommer et al. (1986), West Jr et al. (1989), Glasziou and Mackerras (1993), Beaton et al. (1994), Maluccio et al. (2009)).

Iodine is one such micro nutrient the availability of which in sufficient quantity in a pregnant mother's body in the first trimester of the pregnancy is considered extremely crucial for the cognitive development of the in-utero child. Iodine is required for the synthesis of thyroid hormones, the adequate supply of which are important for physical and mental development of the foetus. According to Lamberg (1991), lack of requisite amounts of thyroid hormones impairs the body's capacity to develop adequate number of interconnections among brain cells and greatly impairs an individual's cognitive ability. Brain development is sensitive to minor adjustments in thyroid hormone and mild maternal iodine deficiency can impair the full cognitive development of individual (**?**, **?**, Dugbartey (1998)).

Iodine exists naturally in the soil, but its natural prevalence varies across regions.People from Tanzania, like many countries on the African continent, traditionally suffered from high rates of Iodine Deficiency Disorder (IDD). Van der Haar et al. (1988) report that according to a nationwide survey of iodine levels in the early 1970s, about 40% of the Tanzanian population lived in iodine-deficient areas and 25% of the population was estimated to have iodine deficiency disorders. The prevalence amongst pregnant and lactating women was as high as 52%.

With the increased awareness of the benefits of IDD prevention, the government of Tanzania undertook a large scale iodized oil capsule (IOC) supplementation program (henceforth, ISP) in 1984. The intention of the ISP was to target the regions with the highest level of iodine deficiency prevalence until the universal salt iodization program began in the early 1990s. A total of 27 districts from across the country were to be covered by the programs. These districts were selected on the basis of prevalence of goiter. While the program was scheduled to begin in 1988 and planned to distribute iodized oil capsules containing 400 mg of iodine amongst males and females aged 2 to 45 years and a dose of 200 mg for children aged 12 to 23 months Peterson et al. (1999). However, The time line was not strictly followed and three districts had received the supplementation by 1988 while two districts did not receive it until 1992. The coverage rate was never perfect in any district and the average coverage rate across all districts across all years in which they received the supplementation was 64% (See Table A2).

Despite the distributional problems, the program reached a substantial number of individuals, pregnant mothers is particular. A conservative estimate by Peterson et al. (1999) claims that the program provided 12 million person-years of protection from Iodine deficiency (ID). By early 1994, the Universal Salt Iodization (USI) program had begun. The focus of the IOC program then shifted from districts with high IDD prevalence to districts not yet reached by the USI program. Thus, during this period, the absence of an IOC program does not necessarily indicate that the population is unprotected from ID, even in districts with previously very high levels of IDD.

3.2 Primary Education Development Program in Tanzania

In 2001, Tanzanian government launched the Primary Education Development Plan (PEDP) wherein tuition fees and other mandatory cash contributions to schools were abolished. The target was to ensure the enrollment of all 7-13 year-olds by 2006. A gross enrollment ratio of 98% in 1980 had declined to 46.7% in the year preceding the launch of PEDP. The program began by targeting those who were seven to eight year old in 2001. These were cohorts born in 1993 and 1994. While the coverage of the program was extended to 12 and 13 years old in 2004 (nine and ten years old in 2001), the effort and impact for these children was substantially lower and delayed. As a result of PEDP, the gross enrollment ratio increased to 100.4% and the net enrollment ratio to 80.7%.

The program worked towards bringing down the cost of free primary education by abolishing all tuition fees. A $10 capitation grant was also introduced and controlled by school committees. This was intended to cover some of the additional school-based costs. However, substantial indirect costs, such as for instructional materials, remained, the provision of which has not been sufficient to date.Measures such as not requiring uniforms were not implemented in practice (by parents) because of the social ostracism this would involve.

We use the ISP and PEDP interventions as the two exogenous shocks for analysis. In the nest section we describe the data set and sample that we use and how we construct dependent and independent variables of our interest.

4 Data and Empirical Strategy

4.1 Data

For our analysis, we study a sample representative of the population of Kagera region from Tanzania. Kagera, located in the northwestern corner of Tanzania, is one of Tanzania's 30 administrative regions. Kagera is Tanzania's fifteenth largest region and accounts for more than three percent of the country's area (CIA (2010)). During 1980s, Kagera suffered from high rates of IDD and four of its seven districts were targeted by the Iodine Supplementation Program. We use the data from Kagera Health and Development Survey (KHDS). KHDS was designed to provide data to understand the long-run wealth dynamics of households and individuals within North West Tanzania. The households were originally interviewed in 4 rounds from 1991 to 1994. Resurveys were then organised in 2004 and 2010. KHDS is one of the longest-running African panel data set of this nature and boast of an impressive tracking rate of around 90%. (Beegle et al. (2006), De Weerdt et al. (2012)). We use the 1991-94 and 2004 rounds for information on individuals years of education, primary school starting age, early investments by the parents in the child and a variety of covariates.

For iodine supplementation, we use the district-year coverage rates from Field et al. (2009). We match this coverage rate for each year for each district with the corresponding observation from KHDS using the year and name of the district information contained in KHDS. We follow Field et al. (2009) in calculating the probability that the mother of a child received iodine supplementation while pregnant with this child conditional on whether and when the iodine supplementation program was implemented in a district. In that, we make the assumption that mothers lived in district where they delivered the child through out their pregnancy. The details about the method followed are provided in the next section. We restrict our sample to cohort born in between 1991 and 1994. We do this because nation-wide iodine supplement began in early 1994. Since PEDP covered both 7 year-old and 8 year the first two years, cohorts born after 1993 are defined as treated by PEDP.

We check the robustness of our results using Demographic and Health Survey (DHS) 2010 & 2011-12 and Tanzania Household and Budget Survey (THBS) 2007. One main reason why we choose not to use DHS or THBS even though they are representative of the entire country is because the relevant rounds from both these surveys do not have information on the birth districts for individuals being studied here. As documented in Kudo (2015) , there is a lot of internal migration in Tanzania (See Table A3). Migration of women is much more common as opposed to migration of men. Whenever we use data from these surveys for our robustness analysis, we restrict our sample to males and unmarried females. The major reason for migration of men is business opportunities. We assume that migrations due to this reason have not been realized by 2004 when the exposed children were relatively young to participate in economic activity. In Kagera, the region for which we have migration information from KHDS 2004, migration outside the region is relatively smaller. However, we restrict our attention to the sample who report not having moved in the last ten years and report being a part of the household in preceding years. While children born in 1991 and 1992 were born twelve and eleven years before the 2004 round, respectively, the probability that they moved in the first two years of their birth is relatively small.

4.2 Outcome variables

Variables related to educational attainment are the main outcomes of interest. In particular, we use completed years of schooling and grade progression. We define grade progression as the ratio of completed years of schooling to the years spent in school. While years of schooling will reflect the composite effect of cognitive ability, which was related to ISP, and the subsequent investment, the effect of differences in cognitive ability is more likely to show up in grade progression. As mentioned before, Tanzanian government launched a universal salt iodization program in 1994 Peterson (2000). The universal free primary program started in 2002. Therefore, the cohorts which were exposed to both ISP and PEDP but not to the universal salt iodization program were those born in 1993 and 1994.

When we examine the plausible mechanism that might be operating behind the results we observe, we use primary school starting age as our dependent variable. Following Glewwe et al. (2001), we treat primary school starting age as an investment variable. Glewwe and Jacoby (1995) argue that delays in enrollment can be related to the malnutrition status of the child. To test for this, we look at the differences in height of the the ISP treated and non-treated children in 1991-94 and 2004.

4.3 Independent variables

The three independent variables of our interest are the two exogenous shocks, ISP and PEDP, and their interaction. As mentioned before, the probability of exposure varied across district for cohorts born in a particular year and across cohorts in particular district. The variation in iodine exposure is, therefore, at the district-year of birth level.

As described before, sufficient levels of iodine are most crucial in the first trimester. Therefore, the child of an iodine deficient mother who received an iodised oil capsule in the first month of any year would not be protected unless the child was born in month eight of that year or later. Following Peterson et al. (1999) and Field et al. (2009), we assume that the timing of the distribution was uniform over the months of an year that the district received the supplementation in. We also maintain the assumption made in Field et al. (2009) that given the starting month, it took three months for the distribution of these capsules.[2]. Therefore, for a district that received the supplementation program in year t, children born in the first seven months in that district were not protected by the supplementation program. Research shows that the body stock of iodine depletes at a certain rate after every such iodine supplementation. To account for this depletion we use the method used in Field et al. (2009). For those born in the eighth month or later, protection, therefore, depended on whether the program started early enough to have reached their mothers in time (first trimester or earlier) and whether their mothers had retained adequate amounts of iodine throughout their first trimester after accounting for the depletion of body iodine stocks with time. The detailed table of probability of protection calculation is reproduced from Field et al. (2009) in the appendix. We present here the calculation for those born in the eighth, the ninth and the tenth month of year t. For those born in month 8, probability of protection is equal to the probability that the program started in January that year (equal to 1/12 using uniform timing assumption) and their mothers were reached in that very month (equal to 1/3 using three month diffusion time assumption.) Therefore, it is equal to 1/36. For those born in the ninth month, the program reached their mother in the first trimester if it started in January (1/12) and reached them by February (2/3) or if it started in February(1/12) and reached them that very month(1/3), therefore, 1/12. For those born in October, the program reached their mothers in time if it started in January(1/12) and reached the mother by March (1) or started in February (1/12) and reached them by March (2/3) or started in March (1/12) and reached them in March itself (1/3), therefore, 1/6. Given the assumption on the rate of depletion made in Field et al. (2009), one we maintain here, stocks of iodine retained in the body are above the required levels for 24 months after the administration of the pill. Therefore, one does not need to adjust for depletion for these months.

This calculated probability then needs to be multiplied by the coverage in a particular district in a particular year to get the treatment probability. To check the robustness of our treat-

[2]Field et al. (2009) make this assumption based on Zafari reports stored in the archives of Tanzania Food and Nutrition Centre (p. 149)

ment probability variable, we use four alternative ISP treatment definitions for our analysis, some of the definitions use the iodine depletion formula used in Field et al. (2009) while some others do not. Definition 1 is as described, a product of probability of protection and district-year specific coverage. In definition 2, we use probability of protection only. The depletion, however, could depend on the nutritional status of the mother. In definition 3, therefore, we use coverage as our ISP treatment variable. In definition 4, we use a dummy variable that takes value 1 the product of probability of protection and coverage is more than zero. The results, presented in Table ..... show that the definitions yield very similar results.

The Primary Education Development Program, which was a universal free primary education program, is our source of exogenous variation in investment across cohorts. In the sample that we use, cohorts who are 10 and 11 year old are exposed to this treatment while the ones that are older are not. This variation, therefore, is at the year of birth level. We include a host of other covariates - a dummy each for whether the mother and father are have some level of education or not, a dummy for sex of the child, a quadratic in age, tribe, religion and primary enumeration area fixed effect. Wherever, the variation is not solely based on year of birth, we replace the quadratic in age by a year of birth fixed effects.

4.4 Empirical Strategy

We begin by examining the direct impact of the iodine exposure on completed years of schooling in 2004 to establish that iodine exposure, indeed, predicts complete years of schooling. Since iodine exposure varies at the district X year level, we use the following specification:

$$Y^1_{idb} = \alpha^1 + \beta^1_1 * ID_{idb} + \gamma^1 * X_{idb} + \tau^1_b + \omega^1_d + \epsilon^1_{idb} \quad (1)$$

where Y_{icdb} is the years of schooling completed by an individual i born in district d in year b by 2004. It depend on the ISP exposure status ID_{idb} of the individual's mother in the first trimester of the pregnancy, which is constructed as explained in the previous section. X_{idb} is a vector of covariates that include a dummy each for whether the mother and father have some education or not, a dummy for gender of the individual and total land ownership of the household to which individual i belongs. We also include birth year fixed effect, τ, and district fixed effect, ω. Since the identification of treatment status of the PEDP program is based the cohort of birth, we cannot use a birth fixed effects in specifications that follow. Therefore, we also present the impact of iodine with birth year fixed effect replaced by a quadratic in age in (1) and argue that a specification with quadratic in age does a good job at approximating the year fixed effect specification. Standard errors are clustered at the district X year level in order to allow for arbitrary correlation in the error terms of observations within the districts for births in the same year.[3] We also add religion and tribe fixed effects to test for robustness of

[3]Clustering at the district level does not change our results. However, since the number of districts is just seven, we

the results.

We, then, look at the combined impact ISP and PEDP on completed years of schooling using the specification:

$$Y_{idb}^2 = \alpha^2 + \beta_1^2 * ID_{idb} + \beta_2^2 * P_{idb} + \beta_3^2 * ID_{idb} * Pidb + \gamma^2 * X_{idb} + \tau^2 * age + \delta^2 * age2 + \omega_d^2 + \epsilon_{idb}^2 \quad (2)$$

For individual i, living in district d and born in year b, ID_{idb} represents the probability that individual is mother received iodine supplementation during the first trimester of her pregnancy. P_{idb} indicates individual i's exposure to PEDP and takes value '1' if individual was born in or after 1993, '0' otherwise. Our specification includes district fixed effect (ω_d), a quadratic in age, a dummy for gender, a dummy each for whether the father and the mother have some education and a continuous variable for total land ownership. Again, standard errors are clustered at the district X year of birth level. β_1 and β_2 represent the independent impacts of ISP and PEDP on the schooling attainments, respectively. The coefficient of interest, β_3, is the measure of complementarity between the two shocks.

To provide suggestive evidence for the hypothesis we propose, we begin by showing that ISP and PEDP treatment status and their interaction predict the primary school start age in a manner that is consistent with impact of these three variables on years of schooling. The specification is similar to (2), except now the outcome variable is the age at which they start primary school. To compare other parental investment in children, we compare the means of the height of ISP treated and non treated children in 2004 and also in 1991-94. The specification used for this mean comparison is the following:

$$Y_{idb}^3 = \alpha^3 + \beta_1^3 * ID_{idb} + \gamma^3 * X_{idb} + \tau_b^3 + \omega_d^3 + \epsilon_{idb}^3 \quad (3)$$

where now Y_{idb} is now the height of the child in 2004 or in 1991-94 survey. Note that not all household are surveyed in each of the four initial rounds or the fifth round of the survey. So, we make comparisons for each of the rounds separately. We also do this analysis for only those individuals for which we have panel information and the results (not reported here) do not change much. ID_{idb}, τ_b and ω_d are as before. We also use the vaccination information from the 1991-94 rounds. We create five investment variables using the 1991-94 rounds.

1. Does the household keep a vaccination progress report card for the child?
2. Has the child ever been administered a vaccination against poliomyelitis?
3. Has the child ever been administered a vaccination against tetanus?
4. Has the child ever been administered a vaccination against tuberculosis?
5. Has the child ever been administered a vaccination against measles?

prefer the district X year clustering. In the appendix, we present result where we use cgm wildboot cluster method to correct for standard errors.**cite Cameron et al.**

We, then, compare the probability of an affirmative answer for all these five questions for treatment and control children using a specification similar to (3) except now the model becomes a linear probability model with the dependent variable taking value equal to one for an affirmative answer in any of the four baseline rounds of the 1991-94 survey and equal to zero if the response is a no. We do not use the observations for which the respondent does not know the answer to this question. For three out of the five questions, the proportion of the sample that does not know the answer to the question is less than one percent. For the remaining two questions, it is 20%. We also add the tribe and religion dummy in each of these regressions as different religions and tribes living in the same enumeration area might have different attitudes towards vaccination. Ideally, one would want to create a vaccination variable that incorporates information on timing and completion of each of the vaccination. Unfortunately, KHDS does not contain detailed information about vaccinations. The vaccination results, therefore, are suggestive at best.

5 Results and Discussion

Table 3 presents the impact of ISP on completed years of schooling. ISP exposure, indeed, predicts completed years of schooling.The magnitudes of the coefficients have to be interpreted with caution. Since the independent variable is in percentage terms, a one percentage point increase in probability of iodine exposure leads to 0.0054 extra years of schooling. However, note that the mean probability of exposure in treated districts across the treatment years was 62%. Therefore, a movement from non treated district to a treated district in a treatment year decreases completed years of schooling by 0.32 years.[4] Second, the sign of the coefficient looks somewhat counter-intuitive at this stage. We present a hypothesis and supporting evidence as to why that might be later in this section. The results are quite robust across different specification. Since we cannot use year of birth fixed effects in some specifications we use ahead, we present the results for both year of birth fixed effects and a quadratic in age here. It is clear that a quadratic term in age seems to do very well at approximating the impact of year of birth fixed effects.

Having established the impact of ISP on years of schooling, we present the results for the combined effect of ISP and PEDP on completed years of schooling in table 4. ISP exposure continues to have a negative impact on completed years of schooling, PEDP has the expected positive impact. PEDP increases the average years of schooling by a 0.37 years. The unit change in the interaction of the two program tends to decrease completed years of schooling by a little over 0.8 years on average. Therefore, the two programs together tend to decrease completed years of schooling by 0.62 years, on average [5]. Again, the magnitudes have to be

[4] It is important to remember that all our estimates are intent-to-treat extimates (ITT)

[5] As for column (1) in table 3, the joint effect of two programs can be calculated in the way; -(0.0086+0.0074)*62

interpreted with caution. In any case, the signs of the coefficient are of greater relevance for the present purposes.

We now put forth a hypothesis to explain the negative impact of iodine exposure completed on years of schooling. Exposure to ISP treatment may affect parental expectations about the cognitive ability of a child, regardless of whether there is, in fact, an effect or not. In such scenarios, parents whose children were not exposed to ISP treatment might make extra investments in their children in an attempt to compensate for the negative effects of lack of sufficient iodine availability in-utero. Even within households, if parental preferences are those of inequality aversion in children quality, parental investment response might be compensatory in nature (Behrman et al. (1982)). Empirical evidence in support of such inequality averse preference and compensatory behavior, even in developing countries, (Li et al. (2010), Pitt et al. (1990)) exist side by side empirical evidence in support of reinforcement (Rosenzweig and Schultz (1982), Adhvaryu and Nyshadham (2014)). The question, therefore, is of an empirical nature where reinforcement or compensation are equally likely. We begin by looking at the primary school starting age for our sample. As mentioned before, according to Glewwe et al. (2001), one way in which the parents might invest differentially in their children is by sending them to school earlier. The age at which a child starts school is an investment variable. The results for our sample are presented in Table 5. Higher probability of ISP treatment status seems to delay the age at which a child starts primary school. PEDP, as expected, might slacken the budget constraint and the PEDP treated might go to school earlier than the non treated, on average. The interaction of the two treatments tends to delay the school start age even further. It is possible that the slackening of the budget constraint provides greater scope for compensation and, hence, the negative impact of the interaction. The results suggest that parents might be compensating for the perceived negative effect of lack of iodine in utero. The impact of iodine on completed years of schooling that we observe, therefore, is the combined effect of ISP treatment and subsequent differences in investment made by the parents in their children.

If similar compensatory behavior in other investments made in children is observed, the hypothesis that the presented effect of ISP treatment status reflects the combined effect gains further credence. As pointed out by Glewwe et al. (2001), height of the child adjusted for age can be used a summary measure of the investments that have been made in the child in past. As Glewwe et al. (2001) acknowledge, a criticism of this approach is that height reflects more than just a childs cumulative nutritional history, because it also captures the effects of illness and other environmental and genetic influences. However, in the present scenario using height as a measure might be desirable because a lower incidence of illness for certain groups might also indirectly reflect, on an average, higher investments made by the parents in their children to protect them against diseases. Table 6 compares the average heights of children treated by

+0.3781 = 0.62

ISP with the average height of those not treated by ISP. We find that, on average, the height of the ISP treated children in lower than those not treated by ISP. Even though the difference is not significant across all specification, it is significant in the most robust specification amongst those presented. Also, the sign and the magnitude is consistent across specification. Table 7 does the same comparison for the data from the four waves of the 1991-94 round and finds similar results. While the differences are not significant for data from wave 1 and 2, the signs are in the expected direction and magnitude consistent with the results from wave 3 and 4 of the 1991-94 survey.

We also look investments in terms of likelihood of having a vaccination card and of being vaccinated against four major diseases for the ISP treated and non treated children. The results are presented in tables 8 - 12. While the differences are insignificant for four of the five questions talked about in the preceding section, ISP treated children are significantly less likely to have received a vaccination against poliomyelitis. Therefore, there is some evidence, albeit weak, that investments made in non treated children was higher, which is consistent with the compensation hypothesis we proposed.

Can we say something about the impact of ISP on the cognitive ability of the children exposed? It is crucial at this juncture to make a distinction in prior and posterior parental perception. As we mentioned before, the sheer fact that some children were treated by ISP and others were not might make parents believe that the ISP treated kids have higher cognitive ability than non-treated kids. This is what we call prior parental perception, where prior signifies that this judgement is made before parents can access cognitive ability of a child through her action. Once the child is born, parents revise their perceptions as they can now better assess the ability of the child through the child's actions. As pointed out by Adhvaryu and Nyshadham (2014), the evidence from the medical literature suggests that mothers are able to recognize, assess and react to signals of cognition in their infant children from very early ages (Bullowa (1979), Meltzoff and Moore (1983), Brazelton and Nugent (1995), Meltzoff and Moore (1997)). This revised perception is what we call posterior parental perception. While posterior parental perception still happens to be a perception, it might be more accurate than their prior perception. Given that the investments differences continue after birth for a significant number of years might be indication of the fact that the posterior parental perception did point to possible lower cognitive ability of non-treated children, on average.

However, given that there could be significant measurement errors in posterior parental perceptions as a measure of cognitive ability of the child, we present two separate ways to, very roughly, evaluate if the ISP treatment had any impacts on the cognitive ability of the treated children that has persisted even after the compensatory investments made by the parents subsequently. First, we look at the combined effect of the two interventions of grade progression. We define grade progression as the ratio of completed years of schooling to numbers of years spent at school. Cognitive ability is expected to play a bigger role here that in completed years of schooling as the latter is invested more by parental investment than the former. We expect to find a positive impact of ISP treatment on this variable. PEDP, on the other hand, should not affect this variable. It is, however, possible that the quality of education infrastructure, for example, teacher to pupil ratio, suffers, on the margin, due to such a large scale program. In that case, there might be some negative effects on grade progression. The sign of the interaction could go either way depending on whether education infrastructure is more or less important for children with higher cognitive ability children. The results are presented in Table 13. While none of the coefficients are significant, the signs are in the expected direction. If we were to judge on the basis of the sign of the ISP treatment coefficient, it seems that ISP treatment has a positive ability on the cognitive ability of the children. However, as Cunha et al. (2010) point out, investments made early in life can lead to changes in cognitive ability of the individuals. It is, therefore, possible that the early investment made in the children in our sample by their parents could mitigate the differences in cognitive ability and, hence, we find no significant effects.

The second method we use is that we use the following relationship:

$$\frac{\partial(years of schooling)}{\partial(school starting age)} = \frac{\partial(years of schooling)}{\partial(treatment)} * \frac{\partial(treatment)}{\partial(school starting age)} \tag{4}$$

Assuming linear but heterogeneous relationship between primary school start age and completed years of schooling, we calculate the slope of this relationship for ISP treated, PEDP treated and both ISP and PEDP treated children. If cognitive ability differences persist, we expect that the slope will be lowest for PEDP treated children and higher for ISP and both ISP and PEDP treated children. That is, those children who received ISP treatment should be better able to convert one additional year into completed years of schooling if the cognitive differences still persist. The results are presented in Table 14. The results seem to suggest that the cognitive ability difference might have persisted even after the compensatory parental investment response. Those who received PEDP treatment but no ISP treatment seem to convert very extra year at spent at school into 0.6 additional years of completed schooling. ISP treated children did much better, converting every extra year at school into 0.94 additional years of completed schooling. While the slope of the ISP-PEDP group is not directly interpretable, the sign suggests that there seems to be a further higher slope for children who received both the treatment.

Therefore, combining the results from the two methods, there seems to be some evidence suggesting that there might be some differences in cognitive abilities that persist even after compensatory parental investments. If that is so, as time goes by ISP treated children might be able to catch up to non-treated children in terms of completed years of schooling even though the former started school later. If the difference in cognitive ability persists and helps children with higher cognitive ability make better use of each additional year then the negative impact of iodine should vanish over the years. We run robustness using THBS 2007 and DHS 2010 and 2011-12 rounds. Since we do have the birth district information in these datasets and migration is a major concern, we present the results for married and unmarried and, within unmarried, females and males samples separately. For THBS 2007, we find that even though the signs are exactly as they were for KHDS, none of the coefficients are significant [Table A4]. Also, while the PEDP coefficient is comparable to the ones in our main results, the coefficient for ISP and the interaction are much smaller now. This is exactly what we expect to find if differences in cognitive ability due to ISP treatment persist. The higher cognitive ability of the ISP treated children should help them catch up to the non treated children who started school first. For the DHS analysis we pool the 2010 and 2011-12 rounds of the survey. The results are presented in Table A5. While ISP treatment tends to have negative impact as before, those who get both ISP and PEDP seem to be able to mitigate the impact of the delay in start by making better use of the extra years due to PEDP. Since these results are from around 6 to 8 years later compared to KHDS and 3 to 5 years later compared to THBS, the period for which the catching up might have continued is larger. It is, therefore, possible that the ISP treated children caught up and started doing better than non treated children due to their better cognitive ability.

In light of the results above, it seems that ISP treatment is an indicator of lower investment by parents in the treated children vis--vis non-treated children more than a positive shock to cognitive ability. However, ISP treatment still predicts completed years of education. If we reinterpret the absence of ISP treatment as the positive investment shock due to compensatory response by the parents, the absence of ISP treatment and the PEDP treatment can be seen as having a positive impact on the completed years of schooling. That is there seems to be dynamic complementarity between parental investments and PEDP treatment. The magnitude of the coefficient suggests that the complementarity is rather strong.

6 Conclusion

There is now a broad consensus amongst demographer, sociologist and economists alike that the diffusion of modern economic growth to the developing regions requires human capital accumulation by the population in these regions (Counts (1931), Inkeles (1969), North (1973), Davis et al. (1971), Rosenberg et al. (1986), Easterlin (1981), Easterlin (2009)). A higher level of human capital is desirable in its own right (Pigou (1952), Adelman (1975), Grant (1978), Grant (1978), Streeten et al. (1981), Sen (1984)). However, low levels of incomes, coupled with imperfect credit markets, in these regions limit the possibilities of private investment in human capital accumulation. State run policies, therefore, are of extreme importance in ensuring higher levels of human capital (Easterlin (1981)). Given the limited state budget, the decision of whether or not to roll out a particular program depends a lot on the cost benefit analysis of the program. Traditionally, the cost benefit analyses of such development programs have taken a partial equilibrium approach and have rightly done so as a general equilibrium model poses tractability challenges. However, if two independent programs exhibit complementarity towards the objective, a partial equilibrium might greatly understate the net benefits of such programs. In such scenarios, it becomes essential to jointly evaluate the impact of the two (or more) programs, allowing for possible complementary between the programs.

Keeping this in mind, we evaluate the Iodine Supplementation Program and Primary Education Development Program in Tanzania. We find that ISP might have small positive effects on cognition and this might show up in achievement like completed years of schooling even in under subsequent adverse investment environments. Additional years at school made affordable by PEDP might have allowed for better manifestation of the impact of ISP on outcomes. We also find that that absence of ISP treatment makes parents make compensatory investment in the non treated children. Besides having direct positive impact on completed years of schooling, these investments are complementary to PEDP, i.e., the investment are higher for non ISP but PEDP treated children and result in higher attainments. In light of the our results, the gains from ISP and PEDP estimated till now seem to be an under report of the true benefits of these programs. The consistency of the results across data sets seems suggestive of the robustness of these results.

Another important policy implication of the present study is that the government policies have to be careful about what parental response might a program that treats children randomly but nit universally might generate. The implications on intra household resource allocation may or may not be desirable. The government might need to design a placebo along with the treatment if such adjustments in intra household resource allocation are undesired.Studies in future should aim to replicate the exercise presented here in other parts of the world to see if complementarity between such intervention programs are generally true. One might also, with the availability of required data, attempt to recalculate the cost and benefits of such programs so as to better inform policy.

Figure 1: Iodine Supplementation Program in Tanzania (from Field et al. (2009)

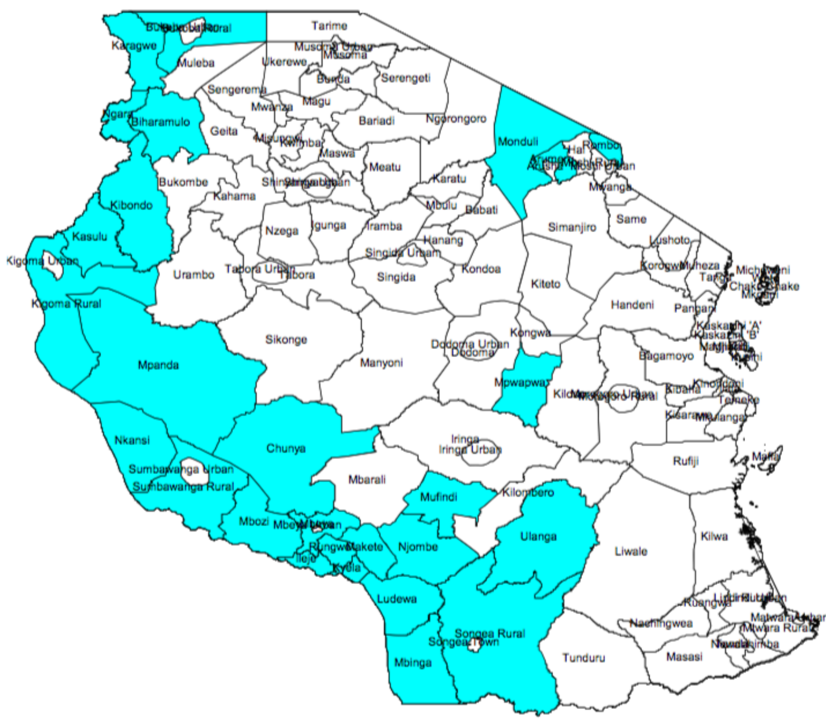

Table 2: Summary Statistics

Outcomes	Control		Treatment	
	Ages 10-11			
	Mean	***SD***	***Mean***	***SD***
Years of Schooling	1.87	1.00	1.44	0.96
Primary school start age	7.98	1.02	8.22	1.15
School Progression	0.82	0.30	0.79	0.40
HAZ in 2004	130.50	8.99	127.77	7.61
Proportion with				
Vaccination card	0.92		0.85	
Tb vaccination	1.00		0.97	
Measles vaccination	1.00		0.92	
Tetanus vaccination	0.46		0.39	
Polio vaccination	0.52		0.55	
	Ages 12-13			
	Mean	***SD***	***Mean***	***SD***
Years of Schooling	3.09	1.38	2.78	1.42
Primary school start age	8.57	1.45	9.01	1.46
School Progression	0.79	0.25	0.81	0.24
HAZ in 2004	141.21	8.47	139.64	8.44
Proportion with				
Vaccination card	0.95		0.99	
Tb vaccination	0.99		0.99	
Measles vaccination	0.94		0.97	
Tetanus vaccination	0.80		0.88	
Polio vaccination	0.84		0.86	

Independent variables	Control		Treatment	
	Ages 10-11			
	Mean	***SD***	***Mean***	***SD***
Protection due to ISP	0	0	14.26	17.17
Age	10.34	0.47	10.13	0.34
Mother has any education	0.95	0.21	0.92	0.27
Father has any education	0.92	0.27	0.92	0.27
Household land per capita	0.48	0.53	0.55	0.47
Proportion				
Sex = Male	0.54		0.48	
Tribe = Mhaya	0.93		0.37	
Religion = Catholic	0.65		0.53	
N	133		185	
	Ages 12-13			
	Mean	***SD***	***Mean***	***SD***
Protection due to ISP	0	0	70.05	27.72
Age	12.57	0.50	12.55	0.50
Mother has any education	0.97	0.17	0.89	0.32
Father has any education	0.95	0.21	0.88	0.32
Household land per capita	0.56	0.56	0.80	0.66
Proportion				
Sex = Male	0.47		0.48	
Tribe = Mhaya	0.94		0.01	
Religion = Catholic	0.65		0.50	
N	161		87	

Table 3: Impact of Iodine Supplementation Program on completed years of schooling

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
VARIABLES	yos	yos	yos	yos	yos	yos	yos	yos
ISP	-0.0079***	-0.0081***	-0.0080***	-0.0083***	-0.0078***	-0.0079***	-0.0079***	-0.0081***
	(0.0024)	(0.0025)	(0.0025)	(0.0026)	(0.0023)	(0.0025)	(0.0025)	(0.0026)
Age fixed effect	YES	NO	YES	NO	YES	NO	YES	NO
Quadratic in age	NO	YES	NO	YES	NO	YES	NO	YES
Religion dummy	NO	NO	YES	YES	NO	NO	YES	YES
Tribe dummy	NO	NO	NO	NO	YES	YES	YES	YES
Constant	3.7598***	-10.8691*	3.7065***	-10.4015	3.5351***	-10.9240*	3.4337***	-10.4588
	(0.3048)	(6.2352)	(0.3185)	(6.3954)	(0.3294)	(6.0501)	(0.3344)	(6.2223)
Observations	466	466	466	466	466	466	466	466
R-squared	0.4632	0.4625	0.4674	0.4666	0.4656	0.4650	0.4710	0.4704

Notes: *** p<0.01, ** p<0.05, * p<0.1. Robust standard errors in parentheses. The standard errors are clustered at geoage level where geoage are district X year of birth groups. Other controls include total land ownership of the household in which the child was born in the 1991-1994 survey, a dummy each indicating whether the mother and the father of the child had some education, gender and primary enumeration area fixed effects.

Table 4: Impact of ISP and PEDP on completed years of schooling

VARIABLES	(1) yos	(2) yos	(3) yos	(4) yos
ISP	-0.0086***	-0.0088***	-0.0084***	-0.0085***
	(0.0022)	(0.0024)	(0.0022)	(0.0023)
PEDP	0.3781**	0.3893**	0.3381**	0.3417**
	(0.1546)	(0.1546)	(0.1507)	(0.1536)
ISP * PEDP	-0.0074**	-0.0074***	-0.0067**	-0.0065**
	(0.0027)	(0.0026)	(0.0029)	(0.0029)
Religion dummy	NO	YES	NO	YES
Tribe dummy	NO	NO	YES	YES
Constant	-15.9174**	-15.5661**	-15.4486**	-14.9910**
	(6.0053)	(6.0664)	(5.9983)	(6.0763)
Observations	466	466	466	466
R-squared	0.4651	0.4694	0.4672	0.4725

Notes: *** $p<0.01$, ** $p<0.05$, * $p<0.1$. Robust standard errors in parentheses. The standard errors are clustered at geoage level where geoage are district X year of birth groups. Other controls include a quadratic in age, total land ownership of the household in which the child was born in the 1991-1994 survey, a dummy each indicating whether the mother and the father of the child had some education, gender and primary enumeration area fixed effects.

Table 5: Impact of ISP and PEDP on primary school starting age

VARIABLES	(1) startage	(2) startage	(3) startage	(4) startage	(5) startage	(6) startage	(7) startage	(8) startage
ISP	0.0076***	0.0074***	0.0077***	0.0074***	0.0075***	0.0073***	0.0076***	0.0073***
	(0.0020)	(0.0018)	(0.0020)	(0.0018)	(0.0021)	(0.0018)	(0.0021)	(0.0019)
PEDP		-0.5910***		-0.6024***		-0.5779**		-0.5827**
		(0.1980)		(0.2050)		(0.2101)		(0.2201)
ISP * PEDP		0.0030		0.0028		0.0027		0.0025
		(0.0027)		(0.0029)		(0.0028)		(0.0030)
Religion dummy	NO	NO	YES	YES	NO	NO	YES	YES
Tribe dummy	NO	NO	NO	NO	YES	YES	YES	YES
Constant	7.1757	12.9913**	6.6917	12.5469**	7.2145	12.8517**	6.7408	12.3305**
	(5.9487)	(5.4035)	(6.2991)	(5.5206)	(5.9717)	(5.4539)	(6.3734)	(5.5854)
Observations	437	437	437	437	437	437	437	437
R-squared	0.3319	0.3375	0.3380	0.3438	0.3324	0.3378	0.3391	0.3446

Notes: *** $p<0.01$, ** $p<0.05$, * $p<0.1$. Robust standard errors in parentheses. The standard errors are clustered at geoage level where geoage are district X year of birth groups. Other controls include a quadratic in age, total land ownership of the household in which the child was born in the 1991-1994 survey, a dummy each indicating whether the mother and the father of the child had some education, gender and primary enumeration area fixed effects.

Table 6: Impact of ISP on height of the child (Height-for-age) in 2004

VARIABLES	(1) Height	(2) Height	(3) Height	(4) Height
ISP	-0.0059**	-0.0066**	-0.0059**	-0.0065**
	(0.0028)	(0.0027)	(0.0028)	(0.0027)
Religion dummy	NO	YES	NO	YES
Tribe dummy	NO	NO	YES	YES
Constant	-1.4482***	-1.2730***	-1.4264***	-1.2918***
	(0.3370)	(0.3604)	(0.3741)	(0.4099)
Observations	491	491	491	491
R-squared	0.1468	0.1604	0.1468	0.1604

Notes: *** $p<0.01$, ** $p<0.05$, * $p<0.1$. Robust standard errors in parentheses. The standard errors are clustered at geoage level where geoage are district X year of birth groups. Other controls include a quadratic in age, total land ownership of the household in which the child was born in the 1991-1994 survey, a dummy each indicating whether the mother and the father of the child had some education, gender and primary enumeration area fixed effects. Observations with body mass index of more than 100 were not used for the analysis. We used the WHO Child Growth Charts and WHO Reference 2007 Charts for our height for age analysis.

Table 7: Impact of ISP on height of the child (Height-for-age) in 1991-1994

VARIABLES	(1) Height_91	(2) Height_92	(3) Height_93	(4) Height_94
ISP	0.0483	0.0398	-0.0166	-0.0299***
	(0.0286)	(0.0291)	(0.0156)	(0.0085)
Religion dummy	YES	YES	YES	YES
Tribe dummy	YES	YES	YES	YES
Constant	-1.4653	-2.6897***	-1.1018	-0.3107
	(2.1488)	(0.7375)	(0.8646)	(0.7161)
Observations	146	175	218	231
R-squared	0.4954	0.3040	0.3248	0.2526

Notes: *** p<0.01, ** p<0.05, * p<0.1. Robust standard errors in parentheses. The standard errors are clustered at geoage level where geoage are district X year of birth groups. The four columns correspond to the data collected in the four waves of the first round of the KHDS survey. Other controls include a quadratic in age, total land ownership of the household in which the child was born in that wave of the 1991-1994 survey, a dummy each indicating whether the mother and the father of the child had some education, gender and primary enumeration area fixed effects. Observations with body mass index of more than 100 were not used for the analysis.

Table 8: Impact of ISP on probability of a vaccination card ownership

VARIABLES	(1) Vaccination Card	(2) Vaccination Card	(3) Vaccination Card	(4) Vaccination Card
ISP	0.0011	0.0013	0.0011	0.0013
	(0.0022)	(0.0023)	(0.0022)	(0.0023)
Religion dummy	NO	YES	NO	YES
Tribe dummy	NO	NO	YES	YES
Constant	-5.0816	-5.5715	-4.9677	-5.4633
	(4.1952)	(4.3543)	(4.1851)	(4.3500)
Observations	321	321	321	321
R-squared	0.1772	0.1847	0.1780	0.1854

Notes: *** p<0.01, ** p<0.05, * p<0.1. Robust standard errors in parentheses. The standard errors are clustered at geoage level where geoage are district X year of birth groups. Other controls include a quadratic in age, total land ownership of the household in which the child was born in 1991-1994, a dummy each indicating whether the mother and the father of the child had some education, gender and primary enumeration area fixed effects.

Table 9: Impact of ISP on probability of vaccination against measles.

VARIABLES	(1) Measles vaccination	(2) Measles vaccination	(3) Measles vaccination	(4) Measles vaccination
ISP	0.0005	0.0002	0.0004	-0.0000
	(0.0018)	(0.0019)	(0.0018)	(0.0019)
Religion dummy	NO	YES	NO	YES
Tribe dummy	NO	NO	YES	YES
Constant	3.0093	2.4771	3.3236	2.8191
	(2.6662)	(2.7156)	(2.6298)	(2.7340)
Observations	246	246	246	246
R-squared	0.2528	0.2623	0.2557	0.2651

Notes: *** p<0.01, ** p<0.05, * p<0.1. Robust standard errors in parentheses. The standard errors are clustered at geoage level where geoage are district X year of birth groups. Other controls include a quadratic in age, total land ownership of the household in which the child was born in 1991-1994, a dummy each indicating whether the mother and the father of the child had some education, gender and primary enumeration area fixed effects.

Table 10: Impact of ISP on probability of vaccination against polio.

VARIABLES	(1) Measles vaccination	(2) Measles vaccination	(3) Measles vaccination	(4) Measles vaccination
ISP	-0.0072*	-0.0076*	-0.0075*	-0.0077*
	(0.0042)	(0.0043)	(0.0041)	(0.0043)
Religion dummy	NO	YES	NO	YES
Tribe dummy	NO	NO	YES	YES
Constant	4.3664	6.4827	4.1239	6.2964
	(7.2862)	(7.4066)	(7.2520)	(7.4470)
Observations	216	216	216	216
R-squared	0.3665	0.4167	0.3698	0.4179

Notes: *** p<0.01, ** p<0.05, * p<0.1. Robust standard errors in parentheses. The standard errors are clustered at geoage level where geoage are district X year of birth groups. Other controls include a quadratic in age, total land ownership of the household in which the child was born in 1991-1994, a dummy each indicating whether the mother and the father of the child had some education, gender and primary enumeration area fixed effects.

Table 11: Impact of ISP on probability of vaccination against tuberculosis.

VARIABLES	(1) Tb vaccination	(2) Tb vaccination	(3) Tb vaccination	(4) Tb vaccination
ISP	0.0007	0.0010	0.0007	0.0010
	(0.0006)	(0.0008)	(0.0006)	(0.0008)
Religion dummy	NO	YES	NO	YES
Tribe dummy	NO	NO	YES	YES
Constant	0.7148*	0.4989	0.7110*	0.4855
	(0.3902)	(0.4788)	(0.3879)	(0.4786)
Observations	306	306	306	306
R-squared	0.1650	0.2779	0.1650	0.2780

Notes: *** p<0.01, ** p<0.05, * p<0.1. Robust standard errors in parentheses. The standard errors are clustered at geoage level where geoage are district X year of birth groups. Other controls include a quadratic in age, total land ownership of the household in which the child was born in 1991-1994, a dummy each indicating whether the mother and the father of the child had some education, gender and primary enumeration area fixed effects.

Table 12: Impact of ISP on probability of vaccination against tetanus.

VARIABLES	(1) Tetanus vaccination	(2) Tetanus vaccination	(3) Tetanus vaccination	(4) Tetanus vaccination
ISP	-0.0052	-0.0061	-0.0056	-0.0063
	(0.0044)	(0.0047)	(0.0045)	(0.0047)
Religion dummy	NO	YES	NO	YES
Tribe dummy	NO	NO	YES	YES
Constant	8.5183	8.8449	8.1786	8.5086
	(6.0348)	(6.0963)	(6.1083)	(6.1590)
Observations	211	211	211	211
R-squared	0.4176	0.4341	0.4196	0.4352

Notes: *** p<0.01, ** p<0.05, * p<0.1. Robust standard errors in parentheses. The standard errors are clustered at geoage level where geoage are district X year of birth groups. Other controls include a quadratic in age, total land ownership of the household in which the child was born in 1991-1994, a dummy each indicating whether the mother and the father of the child had some education, gender and primary enumeration area fixed effects.

Table 13: Impact of ISP and PEDP on grade progression

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
VARIABLES	Grade progress	Grade progress	Grade progress	Grade progress	Grade progress	Grade progress	Grade progress	Grade progress
ISP	0.0003	0.0005	0.0003	0.0005	0.0003	0.0005	0.0003	0.0005
	(0.0008)	(0.0008)	(0.0008)	(0.0008)	(0.0007)	(0.0007)	(0.0008)	(0.0008)
PEDP		-0.0218		-0.0191		-0.0221		-0.0197
		(0.0358)		(0.0378)		(0.0384)		(0.0396)
ISP * PEDP		0.0017*		0.0017*		0.0017*		0.0017
		(0.0008)		(0.0010)		(0.0009)		(0.0010)
Religion dummy	NO	NO	YES	YES	NO	NO	YES	YES
Tribe dummy	NO	NO	NO	NO	YES	YES	YES	YES
Constant	0.0971	0.7084	0.1806	0.7714	0.0987	0.7108	0.1811	0.7772
	(1.2624)	(1.3342)	(1.2645)	(1.3683)	(1.2704)	(1.3270)	(1.2708)	(1.3590)
Observations	443	443	443	443	443	443	443	443
R-squared	0.1828	0.1847	0.1859	0.1878	0.1828	0.1847	0.1859	0.1878

Notes: *** p<0.01, ** p<0.05, * p<0.1. Robust standard errors in parentheses. The standard errors are clustered at geoage level where geoage are district X year of birth groups. Other controls include a quadratic in age, total land ownership of the household in which the child was born in the 1991-1994 survey, a dummy each indicating whether the mother and the father of the child had some education, gender and primary enumeration area fixed effects.

Table 14: Conversion of an additional year into additional years of schooling

Treatment	**School entering age**	**Years of schooling**	$\frac{\partial(years of schooling)}{\partial(school starting age)}$
Free Primary only	-0.4729	0.2928	-0.6191
Iodine only	0.00064	-0.0060	-0.9375
Iodine * Free Primary	0.7961	-0.8118	-1.0197

7 Appendix

This section draws heavily from Field et al. (2009). Information has been reproduced for clarity in understanding of how the iodine treatment variables were defined.

Calculation of probability of protection: The treated mothers received and iodine dosage of 380 mg via the IOC [Peterson (2000), Peterson et al. (1999)]. However, cited in Field et al. (2009), Wolff (2001), Jun and Jianqun (1985) and Untoro et al. (1998) provide a review of literature that finds that majority of iodine stored in the fatty tissue is depleted rapidly within the first week and an hyperbolic rate thereafter. Following Field et al. (2009), we assume that 85 percent (323) of the 380 mg dose was extracted away immediately within the first month and the depletion followed the simple hyperbolic discounting formula $V = A/(1 + kt)$ after that, where k^{-1} is the half life of iodine in months. Using the observation from Cao et al. (1994) and Eltom et al. (1985), which use similar dosages of IOC provides full protection for 24 months and that 6.5 mg is the minimum iodine requirement for one full month of protection, Field et al. (2009) calculate the half life to be 3 months. This implied half life is consistent with other studies of the approximate half lives of urine iodine excretion after oral iodine administration to human populations with iodine deficiency (See Wolff (2001)). The probability of protection in a month of the first trimester, therefore, is the probability that the program had started and reached the mother of the child by that month and the stocks of iodine had not depleted to levels insufficient for protection (< 4.2 mg as per Field et al. (2009)) in that month. [6] A child is protected in the first trimester if she is protected throughout weeks 1 to 12 (roughly three months)

Based on the information and assumption above, probability of protection from in utero IDD if the child district of birth received the ISP in year t (by month of birth):

Table A1: Probability of protection

Year	Jan	Feb	March	April	May	June	July	Aug	Sep	Oct	Nov	Dec	Birth year average
t	0	0	0	0	0	0	0	0.028	0.083	0.167	0.250	0.333	0.072
t+1	0.417	0.5	0.583	0.667	0.75	0.833	0.917	1	1	1	1	1	0.806
t+2	1	1	1	1	1	1	1	1	1	0.998	0.991	0.977	0.997
t+3	0.955	0.927	0.891	0.849	0.802	0.749	0.69	0.627	0.559	0.488	0.419	0.353	0.668
t+4	0.292	0.237	0.189	0.148	0.112	0.082	0.057	0.037	0.022	0.011	0.004	0.001	0.099

Note that the above calculation assumes that every mother in the district received the pill in an ISP year. However, as reported before, the coverage was not 100 percent. Therefore, we multiply the protection probability with the coverage rate to get to our final ISP treatment variable definition.

[6] The 6.5 mg and 4.2 mg figures are calculated based in the recommended daily allowance (RDA) for pregnant women

Table A2: ISP treatment variation (from Field et al. (2009))

Region	District	Year 1	Coverage 1	Year 2	Coverage 2	Year 3	Coverage 3	Year 4	Coverage 4	Year 5	Coverage 5
Dodoma	Mpwapwa	1990	0.65	1992	0.58						
Arusha	Monduli	1992	0.71								
Arusha	Arumeru	1991	0.89								
Kilimanjaro	Rombo	1990	0.68								
Morogoro	Ulanga	1988	0.73	1991	0.61	1992	0.34				
Ruvuma	Songea Rural	1987	0.91	1991	0.74	1995	0.85				
Ruvuma	Mbinga	1995	0.92								
Iringa	Mufindi	1986	0.41	1991	0.63	1995	0.54				
Iringa	Makete	1986	0.2	1991	0.62	1993	0.62	1996	0.49		
Iringa	Njombe	1989	0.76	1992	0.68	1995	0.64				
Iringa	Ludewa	1989	0.59	1992	0.62	1995	0.47				
Mbeya	Chunya	1990	0.49								
Mbeya	Mbeya Rural	1986	0.44	1989	0.84	1990	0.9	1993	0.53	1997	0.53
Mbeya	Kyela	1989	0.91	1993	0.57						
Mbeya	Rungwe	1986	0.35	1990	0.73	1993	0.49				
Mbeya	Ileje	1989	0.94	1992	0.71						
Mbeya	Mbozi	1989	0.67	1991	0.63						
Rukwa	Mpanda	1987	0.79	1991	0.6	1993	0.72				
Rukwa	Sumbawanga	1987	0.76	1990	0.89	1993	0.72	1996	0.51		
Rukwa	Nkansi	1987	0.89	1991	0.49						
Kigoma	Kibondo	1989	0.73	1992	0.75	1996					
Kigoma	Kasulu	1987	0.5	1990	0.66	1996	0.49				
Kigoma	Kigoma Rural	1991	0.91								
Kagera	Karagwe	1990	0.96	1994	0.85						
Kagera	Bukoba Rural	1994	0.78								
Kagera	Biharamulo	1990	0.96	1994	0.38						
Kagera	Ngara	1989	0.29	1994	0.51						

Table A3: Migration in Tanzania (from Kudo (2015))

	Move out from wave 1 to 5		Move in by wave 1	
	Male	Female	Male	Female
(1) Economic reason				
No job/ wanted better job	0.07	0.02	0.08	0.01
Business opportunities	0.21	0.04	0.02	0.00
Land not available	0.11	0.03	0.18	0.05
(2) Schooling	0.09	0.06	0.03	0.02
(3) Family-related reason				
Marriage	0.03	0.47	0.00	0.44
Divorce	0.00	0.03	0.00	0.06
Widowhood	0.05	0.02	0.00	0.02
Death of parents	0.00	0.00	0.12	0.06
Illness of household members	0.00	0.01	0.01	0.01
Other family problems	0.05	0.02	0.15	0.11
(4) Political reason				
Posted to new area	0.02	0.00	0.07	0.03
Political/ economic problems	0.07	0.06	0.02	0.00
(5) Other	0.24	0.17	0.26	0.14
No. of migrants	626	955	995	1521

Notes: (1) Migrants in the first two columns are those who migrated out their original village at some point between 1991 and 2004, whereas migrants in the latter two columns are those interviewed in wave 1 who migrated into their surveyed village prior to wave 1. (2) The number is the proportion relative to the total number of migrants in each category.

Table A4: Impact of ISP and PEDP on completed years of schooling (THBS 2007)

	All	Unmarried	Unmarried females	Unmarried males
	(1)	(2)	(3)	(4)
VARIABLES	yos	yos	yos	yos
ISP	-0.0009	-0.0011	-0.0018	-0.0004
	(0.0053)	(0.0056)	(0.0071)	(0.0051)
PEDP	0.2917	0.3778	0.5048	0.1518
	(0.3734)	(0.3386)	(0.3744)	(0.4698)
ISP * PEDP	-0.0029	-0.0030	-0.0049	-0.0007
	(0.0053)	(0.0057)	(0.0077)	(0.0052)
Constant	1.9105	6.0709	16.0070	-8.6869
	(13.2078)	(12.4373)	(12.1626)	(20.1989)
Observations	4,364	4,319	2,218	2,101
R-squared	0.2885	0.2950	0.3023	0.3305

Notes: *** $p<0.01$, ** $p<0.05$, * $p<0.1$. Robust standard errors in parentheses. The standard errors are clustered at geoage level where geoage are district X year of birth groups. Other controls include a quadratic in age, a dummy each indicating whether the mother and the father of the child had some education, a dummy indicating if the residence is in an urban area, gender and primary enumeration area fixed effects.

Table A5: Impact of ISP and PEDP on completed years of schooling (DHS 2010 & DHS2011-12)

	All	Unmarried	Unmarried females	Unmarried males	All
	(1)	(2)	(3)	(4)	(5)
VARIABLES	yos	yos	yos	yos	yos
ISP	-0.0392***	-0.0397***	-0.0438***	-0.0252**	-0.0343***
	(0.0048)	(0.0047)	(0.0058)	(0.0095)	(0.0076)
PEDP	0.0855	0.1959	0.2273	-0.0612	0.0092
	(0.2849)	(0.3748)	(0.4466)	(1.4006)	(0.4953)
ISP * PEDP	0.0521***	0.0478***	0.0513***	0.0301**	0.0435***
	(0.0026)	(0.0032)	(0.0038)	(0.0131)	(0.0033)
Constant	5.2434	1.7128	2.5900	8.6869	-15.2251
	(8.3975)	(9.3925)	(10.4767)	(56.1428)	(16.7798)
Observations	1,804	1,304	1,143	161	542
R-squared	0.5816	0.5782	0.5736	0.8091	0.6840

Notes: *** p<0.01, ** p<0.05, * p<0.1. Robust standard errors in parentheses. The standard errors are clustered at geoage level where geoage are district X year of birth groups. Other controls include a quadratic in age, number of members in the houshold, a dummy indicating if the residence is in an urban area, gender, survey year and primary enumeration area fixed effects. Column (5) included land ownership control.

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