

Copyright Permission

Note: In accordance with 'publisher's copyright policies and self-archiving' the International Food Policy Research Institute (IFPRI) has posted the post-print after peer reviewed of this article to author's websites and/or institutional repositories.

The correct citation for this article is Field, E.M., O. Robles, and M. Torero. 2009. Iodine deficiency and schooling attainment in Tanzania. *American Economic Journal: Applied Economics* 1(4):140-169. <http://dx.doi.org/10.1257/app.1.4.140>

For additional information, contact IFPRI-library@cgiar.org.

Iodine Deficiency and Schooling Attainment in Tanzania

By ERICA FIELD, OMAR ROBLES, AND MAXIMO TORERO*

Cognitive damage from iodine deficiency disorders (IDD) has important implications for economic growth through its effect on human capital. To gauge the magnitude of this influence, we evaluate the impact on schooling of reductions in IDD from intensive iodine supplementation in Tanzania. Our findings suggest a large effect of in utero iodine on cognition and human capital: treated children attain an estimated 0.35 – 0.56 years of additional schooling relative to siblings and older and younger peers. Furthermore, the effect appears to be substantially larger for girls, consistent with laboratory evidence indicating greater cognitive sensitivity of female fetuses to maternal thyroid deprivation. (JEL I10, I21, I38, O1, O12, O15)

A number of recent cross-country studies provide evidence that ecological conditions related to health environment, such as malaria transmission rates, have a direct effect on economic growth (Sachs, 2001; Malaney and Sachs, 2002). One critical aspect of health environment that has received little attention in the literature is the concentration of trace elements in soil and rock, which differs widely across settings as a result of geographic variation in the age of surface rock (Marett, 1936). Among minerals found in soil, iodine is potentially one of the most important for human growth and development since it is the only micronutrient known to have significant, irreversible effects on brain development (Cao et al., 1994; Hetzel and Mano, 1989; Pharoah and Connolly, 1987).

* Field and Robles: Department of Economics, Harvard University, Cambridge, MA (email: efield@latte.harvard.edu, orobles@fas.harvard.edu). Torero: International Food Policy Research Institute, Washington, DC (email: m.torero@cgiar.org). We thank Lisa Vura-Weis and Sonali Murarka for excellent research assistance. We are also grateful for feedback and discussion from three anonymous referees and numerous seminar participants.

If dietary iodine is indeed a key determinant of cognitive capacity in humans, its deficiency could have important consequences for human capital accumulation and labor productivity in afflicted settings. Given that an estimated one billion people globally are at risk of brain damage from iodine deficiency disorders (IDD) worldwide, this influence may account for a significant fraction of unexplained variation in cross-country growth rates. Iodine deficiency may also constitute an important missing link in explaining abysmal rates of growth in Africa: Geological “shields”, which cover a large and populous ring of Central Africa, are associated with particularly low concentrations of iodine in soil and ground water due to their geological age.¹ Although dietary patterns vary geographically with respect to a variety of micronutrients important for human development, iodine availability is likely to exert a stronger independent influence on economic outcomes than dietary prevalence of other micronutrients and many climatic conditions due to the fact that it has little correlation with local food availability. Hence, while other micronutrient deficiencies are likely to be resolved with economic development by way of rising caloric intake, iodine deficiency is more likely to exert a persistent influence on economic outcomes.

This research looks for evidence of the influence of iodine deficiency on rates of learning disability by examining the effect on child schooling of an intensive and repeated distribution of iodine supplements in several districts of Tanzania between 1986 and 1997. Since iodine is thought to matter most at the time of fetal brain development, we look for evidence of improvements in cognitive ability attributable to the intervention by assessing whether children who benefited from supplements in utero exhibit higher rates of grade progression ten to fourteen years later. Since supplements offer protection for two years but distribution rounds occurred less frequently, we exploit gaps in coverage specific to each district using fixed effects models that compare children likely to benefit from the program in utero based on year of birth to slightly

¹ Shields are large areas of exposed Precambrian rocks. These areas have been relatively unaffected by tectonic processes, which tend to occur near plate boundaries, so the age of surface rock is at least 570 million years.

older and younger cohorts within the district who were in utero during program gaps and delays.

In addition to providing evidence of a direct link between geography and development our analysis contributes to the growing body of micro-level studies on the effects of malnutrition on schooling and labor outcomes.² Assessing the importance of physiological determinants of schooling informs a fundamental debate in the development literature surrounding the importance of supply-driven explanations for low levels of human capital investment relative to differences across settings in returns to education. Schooling responses to reductions in fetal brain damage provide evidence that patterns of human capital investment also reflect biological differences in the cognitive cost of schooling. Since fetal IDD permanently limits intellectual functioning, its impact is likely to be particularly acute and persistent.

Of particular interest is the possible role of iodine deficiency in explaining gender differences in schooling outcomes in light of recent scientific evidence of biological gender differences in iodine sensitivity in utero. If girls are more susceptible to IDD in utero, geography may contribute directly to gender disparities in schooling outcomes by way of sex differences in rates of learning disability. This is a particularly compelling explanation for gender differences in schooling in Tanzania, where lower female attainment is almost entirely accounted for by the extremely low rate at which girls pass the national secondary school qualifying exam.

The long-run effect of fetal iodine intake is also of interest in light of recent worldwide progress in reducing IDD through universal salt iodization legislation passed in many countries during the 1990s. Between 1980 and 2000, at least 28 countries reduced goiter, a common indicator of IDD, by more than 20% through national salt iodization, and several others that lack data are believed to have made similarly important gains. Because children born after the

² The relationship between macro-nutrients (energy and protein intake) and education has been examined through subsidized school meal programs in Kenya and nutrition supplements in Guatemala, which were found to increase school participation and test scores (Vermeersch, 2003; Behrman et al, 2003). While little attention has been paid to the effect of specific micronutrients on schooling, many studies have examined the benefits of micronutrient supplements on health and labor productivity (Thomas et al, 2003; Basta et al, 1979; Husani and Gunadi, 1981; Sommer et al., 1986, 1981; West et al., 1989; Glasizou et al, 1993; Beaton et al, 1992).

majority of these changes are only now reaching school age, there has been little opportunity to evaluate the impact of these reforms on health and well-being or to determine whether resulting reductions in IDD will alter the global pattern of schooling attainment in the near future.

Our findings suggest that reducing fetal IDD has significant benefits for children's cognitive capacity as evidenced by its effect on schooling attainment: Children likely to be protected from iodine deficiency during their first trimester in utero attain an average of 0.35 years of education above older and younger children in their district who were not. When district-level coverage rates are incorporated into the estimates, the implied effect of supplementation rises to 0.56 years. This result supports the common claim that the first three months of fetal growth are a critical period for cognitive development.

Furthermore, the estimated effects are substantially larger and more robust for girls, indicating a potentially important role of micronutrient deficiencies in explaining gender differences in schooling attainment in many parts of the developing world. The finding is consistent with new evidence from laboratory studies in animals which find greater sensitivity of the female fetus to maternal thyroid deprivation on cognitive development. The pattern of results is similar in household and district fixed effects models and consistent across datasets and points in time. Furthermore, the size of the program effect rises steeply with the baseline level of IDD in the district. Since there is no evidence of program effects on health status or school days missed due to illness, the schooling results appear to be driven by cognitive rather than physical health benefits of maternal iodine availability.

I. Background

A. Iodine Deficiency

Iodine is produced in the ocean and deposited in the soil, where it is stored in underground rock layers. Hence, dietary iodine availability is determined primarily by soil

composition and amount of seafood consumed. Because soil is depleted of iodine gradually over time, older soil surfaces are more iodine deficient, so rates of IDD increase with distance to coast and altitude and decrease with level of recent tectonic activity. Since iodine deposits are concentrated in deep soil layers, well water is also an important source in places where bedrock is rich in iodine (Hetzel, 1989).

Humans require iodine for the biosynthesis of thyroid hormone. Although thyroid hormone plays a role in daily metabolism and nervous system activity, the human body is most sensitive to thyroid hormone availability during fetal development: In utero development of the central nervous system required for intellectual functioning depends critically on an adequate supply of thyroid hormone, which influences the density of neural networks established in the developing brain (Lamberg, 1991).³ In contrast, morbidity from child and adult thyroid fluctuations is relatively low.

IDD has also been associated with physical impairments in the fetus other than brain damage such as congenital anomalies, perinatal mortality and deaf mutism, as well as retarded physical development, although the evidence is mixed.⁴ In general, evidence from human studies suggests that damage from fetal IDD is overwhelmingly cognitive and that physical birth defects (including fetal death) occur only under extreme deprivation (Zimmerman, 2005; Hetzel, 1983). Furthermore, animal studies indicate that cognition is sensitive to iodine deficiency exclusively during early fetal life (prior to mid-gestation) whereas growth and psychomotor development are believed to be most affected by deficiency in infancy (Cao et al., 1994a; Zaleha et al., 2000).

Although iodine deficiency has been associated with goiter for centuries, the effect of iodine deficiency on mental development is no longer believed to be limited to rare cases of severe mental retardation from extreme deprivation. Recent evidence from laboratory studies

³ Cretinism, a relatively rare form of mental retardation that occurs under extreme deprivation, is the most severe manifestation of cognitive damage from insufficient maternal thyroid hormone. Though cretinism is rare, severely affected populations may have rates as high as 15%, imposing a major social and economic burden on the community (Boyages et al., 1988; Halpern et al., 1991; Pandav and Kochupillai, 1982)

⁴ See Allen and Gillespie (2001) for a review of the evidence.

indicates a continuous process by which fetal brain development is sensitive to minor adjustments in thyroid hormone (Lavado-Autric et al., 2003; Sundqvist et al, 1998; Dugbartey, 1998; Pop et al, 1999). As a result, even mild maternal iodine deficiency is now hypothesized to reduce intelligence quotients by a noticeable margin.⁵

While there have been no experimental or large-scale observational studies of the cognitive effects of moderate iodine deficiency in humans, there is suggestive but mixed evidence from community-based assessments of iodine intervention trials that supplementation can improve performance on cognitive tests (Bleichrodt et al., 1994; Bautista et al., 1982). One oft-cited study in Ecuador found a 10-15 point difference in IQ between 50 offspring of women given iodine prophylaxis in early pregnancy compared with children in untreated communities (Shrethsa, 1994). To our knowledge, the long-term impact of in utero iodine on children's human capital attainment has not been measured in any setting.

B. Gender Differences in Iodine Deficiency

Evidence from multiple sources indicates gender differences in the importance of iodine for brain development. Notably, in the only two of the above studies that analyzed results by gender, cognitive improvements were only found among girls, although in both cases the findings were merely suggestive given limited sample sizes (Bautista et al., 1982; Shrethsa, 1994). Consistent with this pattern, practitioners have noted that adolescent IDD, including rates of goiter and average severity among sufferers, is systematically higher among females (Allen and Gillespie, 2001; Simon et al., 1990).

More conclusive evidence of biologically-driven gender differences in iodine sensitivity comes from recent laboratory experiments of maternal thyroid deficiency in animals. Scientific investigation of gender differences in in utero iodine sensitivity has only recently been

⁵ The World Health Organization labeled IDD “the most common cause of preventable mental retardation (WHO, 1992).”

undertaken, consistent with the general absence of research into the role of maternal biochemicals on sex differences in fetal neurodevelopment. However, two studies lend strong support to the hypothesis of sex-specific sensitivity to iodine deficiency in utero. First, Friedhoff et al. (2000) found that the effect of artificially restricting maternal thyroid hormone in utero on fetal neurodevelopment and behavioral outcomes was significantly larger in female relative to male rat progeny. Although the mechanism underlying sex-selective effects of maternal nutrient deprivation on brain development could not be directly addressed by their experiment, a recent study of gene expression in nutrient deprived fetal guinea pigs by Chan et al (2005) provides insight into the cellular pathways.⁶

II. Setting

Our study examines the long-run impact of an iodized oil supplementation program in Tanzania. Although a number of countries undertook iodine supplementation programs during the 1990s, there are two important advantages to studying the case of Tanzania. First, Tanzania was one of the largest and most intensive programs, ultimately reaching approximately 25% of the population. As a result, an estimated 1.9 million babies born during and immediately after the program were protected from fetal IDD.⁷ The breadth of the program and well-defined target population are critical for retrospective evaluation because they enable follow-up studies of these cohorts based solely on year and district of birth. Second, Tanzania was one of the earliest countries to distribute iodine supplements. Hence, evaluation of the initial effect on children born during the program provides a first glimpse of long-run patterns that can be expected to emerge over the coming decade in a number of other settings.

⁶ In this experiment, in utero nutrient deprivation led to a significant *increase* in the male fetal brain and *decrease* in the female fetal brain of mRNA expression of nuclear thyroid hormone receptors (TRs), which mediate thyroid hormone action. Increased TRs in key regions of the fetal brain help regulate thyroid hormone during development and thereby have the potential to compensate for lower maternal thyroid transfers. Although the biological pathway underlying the gender difference is not fully understood, the finding was hypothesized to be related to elevated male androgen levels at the height of neural TR expression, a gender difference also found in humans.

⁷ Estimate based on 1988-1994 population and crude birth rate.

A. Iodine deficiency in Tanzania

Tanzania, like many countries in Africa, traditionally suffered high rates of IDD. According to a nationwide survey of iodine levels in the 1970s, about 40% of the Tanzanian population lived in iodine-deficient areas and 25% was estimated to suffer from IDD, including 3% with severe and 22% with moderate symptoms. In endemic regions, 13% of children under five and 52% of pregnant and lactating women showed manifestations of iodine deficiency prior to the intervention (van der Haar et al., 1988). According to the WHO global database on micronutrients, in 1980, goiter prevalence was 57% among girls 13-18 living in endemic areas relative to 43% among boys of the same age.

II.B Schooling in Tanzania

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.⁸

Throughout the country, primary schooling is characterized by high variation in age of entry, high rates of grade retention and intermittent enrollment. 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. The National Examinations Council of Tanzania (NECTA) conducts two primary school examinations used for promotion at the end of Standard 4 and Standard 7 (MOEC, 1995). In examinations at both levels, failure rates are high (United

⁸ 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.

Republic of Tanzania, MoEC, 2003). In 2002/2003 over 20% of students repeated the Standard 4 exam (United Republic of Tanzania, MoEC, 2003b).

Although gender parity in primary enrollment was more or less achieved by 1998, female students represented only 36% of the secondary level student population in 2000. Gender differences in secondary enrollment are almost entirely accounted for by differences in the Standard 7 Primary School Leaving Exam (PSLE), which is required for admittance to any lower secondary school in the country. Alarming, although in 2001 roughly the same numbers of boys and girls completed primary school and sat for the PSLE, boys were 69% more likely to pass. As there is cost and no benefit other than admission to taking the test, this pattern alone suggests that parental preferences for male over female schooling are not fully responsible for gender differences in education.

II.C Iodized Oil Capsule (IOC) Distribution in Tanzania

Tanzania was targeted for iodine supplementation early compared to similarly afflicted countries. In 1986, a massive supplementation intervention was scheduled to begin in the most affected districts of the country as a short-term measure until nationwide production of iodized salt could be phased in in the mid-1990s. The objective of the program was to cover all iodine deficient sub-populations for ten years with IOC. Iodized oil, taken either orally or through injection, is considered one of the most effective short-term measures for combating IDD on account of the immediacy of health improvements and duration of coverage, which lasts from one to four years depending on the dosage (Delange, 1998). Program districts were chosen based on 1984 field measurements of visible goiter rate (VGR) among school children. The minimum VGR for inclusion was 10%, which resulted in 25 treatment districts encompassing 25% of the country's population (Peterson, 2000).⁹

⁹ Two districts, Bukoba Rural, Kagera, and Mbinga, Ruvuma, were added late so do not contribute to the sample of treated children in our study.

In program districts, all women of child-bearing age were targeted to receive 380 mg capsules once every two years, the expected duration of protection from this dose.¹⁰ From 1986–1994, approximately five million women and children received at least one supplement through the program. Program roll-out and coverage rates across districts, collected from the archives of the Tanzania Health and Nutrition Office annual reports of program activity, are detailed in Table 1. Although all districts were scheduled to begin IOC by 1988, in practice there were significant delays in program implementation in many of them. Only ten of the districts had begun by 1988, and three did not start until 1992. Finally, districts were reached less frequently than once every two years due to administrative problems and caution over administering supplements frequently (Peterson, 2000). Penetration rates were lower than planned but still relatively high, ranging from 60 – 90% of the target population with average coverage across all districts and all years of 64%. The average coverage rate among districts and years included in our analysis sample was 68%, although the coverage rate among the target population of women of child-bearing age is unknown, and likely to be higher.

Although the long-term impact of the program has not been evaluated, the program was deemed a success early on due to the number of IOC distributed, overall cost-effectiveness (the average cost per dose was \$0.51–\$0.56), and a handful of initial studies indicating that visible and total goiter rates (VGR and TGR) had decreased. A 1991 evaluation in three districts found that VGR had decreased by over 50% and TGR by over 25% among children who received supplements directly (Peterson, 2000). In light of the importance of adequate thyroid hormone during brain development and increased need for iodine during pregnancy, the implied program impact on children of women protected from IDD during pregnancy is substantially higher.

¹⁰ The target groups for supplementation were, in order of importance: 1) women of childbearing age; 2) children 1-5 years; 3) older children; and 4) adult men 15-45 years of age. In people older than 45, iodized oil was not encouraged due to increased risks of hyperthyroidism (Peterson, 2000).

III. Empirical Analysis

III.A Data

We examine the program effect on children born to mothers targeted for IOC during pregnancy using micro-level data from the 2000 Tanzanian Household Budget Survey (THBS), to which we append the district-level information from Table 1 on timing and coverage rates of IOC distribution rounds in intervention districts. The THBS is a nationally representative survey of 22,178 households conducted by the National Statistics Office of Tanzania, 25.2% of which live in districts targeted for IOC. The survey collects individual information on school enrollment and grade attainment of all household members in addition to a variety of community and family background characteristics.

We restrict the analysis samples to all children between ages 10 and 13 who are residing in the household and who could be linked to mothers in the household. In total, 20.8% of children were dropped because they could not be matched to mothers based on age and relationship to household head.¹¹ Excluding non-resident children is necessary due to the fact that schooling outcomes are only available for household members. Excluding observations that cannot be linked to mothers was chosen to minimize the number of children born outside the district given relatively high incidence of orphanhood in rural Tanzania. As a robustness check we verify that the regression results are robust to using the full sample.

The upper age limit reflects the fact that oldest children in intervention districts affected by the program are 13 in 2000. The lower bound on age was based on the modal age of school enrollment, which peaks at 10 for both boys and girls. Restricting the sample to ages by which

¹¹ We matched mothers to children with the following algorithm: A woman was considered the mother of the child of the head or spouse if she herself was the head or spouse and fell within the right age range (12 to 45 at birth of that child). Out of 3397 children 8 to 14, 725 could not be linked to mothers; of these, 342 are not the child of the head, 191 live in households in which there are no eligible women, and 192 live in households in which there is more than one eligible mother due to polygamy. By this method, some fraction of mother-child pairs is likely to be matched incorrectly, reducing the precision of the estimates without introducing any obvious bias since the match rate is balanced across treated and untreated kids. Furthermore, incorrectly matching sibling pairs in the household fixed effects estimates is unlikely to matter for either the efficiency or consistency of the estimates since the predictions regarding fetal iodine deficiency are the same for children born in the same district.

most children have entered school maximizes the predictive power of our treatment variable: across the entire rural sample, only half of nine-year-olds were enrolled.¹² We also verify that the findings are statistically robust to including children as young as 6 and present evidence that both retention and age of entry matter by examining the effect of treatment on school enrollment and grade-by-grade attainment among the larger sample of children 6-13.

The analysis sample contains 1508 children living in the 25 intervention districts that began IOC by 1992, 89% of whom are enrolled in primary school.¹³ Within-household estimates reduce the sample to 880 children in households with more than one child aged 10-13. We supplement the analysis with an analogous sample from the 2004 Tanzanian Demographic and Health Survey (TDHS), which contains 683 children ages 10-13 residing in intervention areas.

III.B Definition of Program Participation

To analyze the impact of IOC distribution, we defined an indicator of treatment based on the likelihood that the mother of a child was protected from IDD at some point during her first trimester of pregnancy given an IOC dosage of 380 mg.¹⁴ First trimester was chosen based on numerous laboratory studies indicating that maternal hypothyroxinemia increases the risk of neuro-developmental deficits of the fetus only prior to mid-gestation, a period during which the mother is the only source of thyroid hormone (Cao et al., 1994a; Hetzel & Mano, 1989; Pharoah & Connolly, 1987; Zoeller & Rovet, 2004).¹⁵ Furthermore, since brain development of the fetus takes place during the first month of pregnancy, it is believed that most of the consequences

¹² The signal-to-noise ratio of grade attainment as an indicator of cognitive capacity will automatically improve with age over this pre-drop-out range (6-10) even if grade differences are driven entirely by differences in age of entry. However, if retention rates are also an important mechanism by which cognition influences school progression, the relationship between grade attainment and cognition will be particularly weak at young ages.

¹³ The remaining 11% are not studying. Three 13-year-olds report enrollment in secondary school.

¹⁴ According to program rules, women under age 23 were instructed to receive half the dosage of older women (200mg). However, according to one program report, this rule was rarely followed on account of the distribution scheme designed to administer as many pills as possible in a short amount of time (Peterson et al., 1998).

¹⁵ In addition, one experimental study in humans on the timing of iodine supplements for preventing cretinism found that iodine treatment during the first trimester protects the fetal brain from the effects of iodine deficiency, while treatment later in pregnancy or after delivery does not improve neurologic status (Cao et al, 1994).

become permanent by the second trimester. This view is consistent with a wider body of scientific thought regarding the importance of micronutrients during the “critical period” of the first three months of pregnancy (Barker, 1990, 1995; Painter et al., 2005).

The likelihood that the mother of a child born t months after a program year p was protected from IDD at any point during the first trimester of pregnancy is equal to the probability that the mother received IOC on or before $t-7$ (in time to protect the child prior to end of the first trimester given 9-month gestation) multiplied by the probability that sufficient stores of maternal iodine (≥ 6.5 mg) were remaining at $t-9$ to protect the child for at least one month of this critical period. Without data on month of IOC distribution, the first probability calculation requires an assumption regarding the length of distribution periods, which we assume to be three months based on project reports, and the timing of distribution periods over the year, which we assume to be uniform (Peterson et al, 1998). This implies that mothers of children born t months after the start of a program year were treated in time to protect that child with probability equal to: $\frac{1}{36}$ if $t = 8$; $\frac{1}{18}$ if $t = 9$; and $\min(1, \frac{1}{36} + \frac{1}{18} + \frac{t-9}{12})$ if $t > 9$.

The second probability calculation, which pertains to children born 2-4 years after the program, requires an assumption regarding the depletion pattern of iodine from 380 mg supplements, which is stored in the adipose tissue and excreted gradually from the body. To account for depletion in the treatment indicator, we make the following assumptions based on existing evidence: First, we assume that 85% of iodine is extracted in urine immediately, implying an initial loss of 323 mg of iodine in the first month, after which point it is depleted hyperbolically.¹⁶ Second, based on results from three separate human studies in settings with comparable levels of IOC, we assume that iodine stores adequate to fully protect against fetal

¹⁶ Several studies have established that iodine stored in fatty tissue is depleted hyperbolically with the majority of urinary extraction occurring in the first week and then tapering off gradually. See Wolff (2001), Untoro et al. (1998) and Wei and Li (1985) for a review of the literature. To calculate iodine stores, we use the following simple hyperbolic discounting formula: $V = \frac{A}{1+kt}$, where k^{-1} is the half-life of iodine.

IDD remain in the body for 24 months (Eltom et al., 1985; Cao et al., 1994; Furnee, 1997).¹⁷ Given that baseline iodine deficiency varies across treated individuals, the fraction of treated who are adequately covered will presumably decline after the point of full protection (24 months) at a gradual and decreasing rate, which can be calculated based on our assumed depletion formula.¹⁸ In particular, the above two assumptions together imply a half-life (at 1) of 3 months, which means that iodine levels will continue to fall for an additional 14 months after $t=24$ until they reach ineffective levels to protect anyone in the population ($>4.2\text{mg}$) at $t=38$. Exact probabilities for each birth month are described in on-line Appendix A. Importantly, the implied half-life is consistent with four studies of the approximate half-lives of urinary iodine excretion after oral iodine administration to iodine-deficient human populations (Wolff, 2001).

We calculate the birth-year-specific likelihoods of receiving adequate coverage by averaging the monthly probabilities weighted by district-specific seasonality in births observed between 1996 and 2004 in the 2004 TDHS (after the intervention). The unadjusted and seasonality adjusted likelihoods for children born x years after IOC are the following:

<i>Birth year - program year (x):</i>	-1	0	1	2	3	4
Likelihood of IDD protection in trimester 1, immediate depletion of 223mg followed by simple hyperbolic depletion with half-life of 3 at $t=1$ (380mg):	0	0.072	0.806	0.997	0.668	0.099
Seasonality-adjusted likelihood, averaged across districts:	0	0.070	0.802	0.997	0.696	0.101

While there is arguably insufficient information on which to base the assumed depletion pattern, it is important to note that our estimation strategy does not depend on any of the above assumptions.¹⁹ First, we introduce flexibility into the regression equation to account for

¹⁷ Recommended daily iodine intake is 200 μg for pregnant and lactating women (WHO, 1996). Epidemiological criteria for sufficient iodine intake is 100 μg or above.

¹⁸ Based on the recommended daily allowance for pregnant women (of which 90% is depleted per day, unlike iodine stores), 6.5 mg is considered the minimum level of iodine stores in adipose tissue sufficient to ensure at least 1 full month of coverage in the population (~ 0.2 mg RDA for 31 days).

¹⁹ Large variance across populations and individuals in the speed of iodine depletion has been documented and few scientific studies follow subjects for more than a year. Patterns of iodine extraction have been found to be specific to the amount, method of delivery, and population characteristics. One study in Malawi found that type of iodized oil, goitre, parasites, sex, adipose tissue, cassava consumption and seasonality influence duration of IOC (Furnee, 1997).

misspecification by including a correction factor for potential measurement error in the estimated treatment probability due to incorrect assumed rate of depletion. This variable is equal to the number of years after the point of full protection a child was born, relevant only for children born 3-4 years after a distribution round. We also include a correction factor that accounts for the possibility that women under age 23 received half the amount of IOC, an initial program guideline that was reportedly followed very rarely. This is simply the previous correction factor interacted with an indicator that the child's mother was under 23 at the time of IOC.

In addition to the above measure of protection based on annual likelihood, we also construct a binary indicator that treats only those individuals born one to three years after IOC distribution as protected from IDD in utero. Since the binary measure encompasses a wide range of alternative depletion assumptions, the robustness of our results to this treatment indicator confirms that the estimates are insensitive to the exact choice of depletion function. Lastly, since not all women in a district can be assumed to have been covered by the program, we construct a third measure of exposure by interacting the initial exposure measure with the district program coverage rate reported in Table 1.²⁰ This alternative exposure variable more accurately captures the program effect on women who took the pill as opposed to the district average treatment effect.

III.C Estimation Strategy

We estimate the effect of IOC on education in a regression analysis in which the primary outcome of interest is years of completed schooling. Table 2 presents summary statistics from the full THBS sample divided according to the timing of IOC. Comparisons across intervention and non-intervention districts show clearly that the program favored needier areas, underscoring

²⁰ Since program personnel were instructed to administer and not just hand out pills (when they reached their turn in line, a patient was handed a capsule with a cup of water and instructed to swallow), reported coverage rates are likely to reflect the true rate of treatment exposure

the importance of relying on within-districts comparisons for estimating a treatment effect. Comparisons among participating districts according to program timing are less clear. School enrollment and access to safe drinking water fall monotonically with program start date, while illness rates and average distance to school are significantly higher for late districts. In contrast, annual durables consumption is significantly higher in late districts, while number of meals and fish consumed are relatively constant across program start dates. Nonetheless, the general patterns suggest that districts in which IOC began early were better off, such that program effect estimates based on comparisons across participating districts are also likely to be biased upward.

For this reason, we restrict our regression estimates to within-district comparisons with the following fixed-effects regression:

$$grade_{id} = \alpha + \beta_1 (T_{id}) + \beta_2 (A_{id}) + \beta_3 (X_{id}) + \mu_f + \varepsilon_{id} \quad (1)$$

Here T_i is the continuous or binary likelihood child i in district d was protected from IDD during the first trimester described in the last section, A is a vector of birth year dummies, and X is a vector of household- and child-level control variables, including gender, birth-order and the following measures of household socio-economic status: urbanicity, whether mother was under 23 at birth, household total children in sample and total boys in sample, whether household reported experiencing food shortages in past month, whether roof made of grass, home ownership, distance to nearest secondary school, distance to nearest health clinic, month of survey, and education of the head and spouse. Standard errors are clustered at the district-age level. To examine whether the fetal effects of IDD are stronger for females, we also run the above regression separately by gender.

Since within districts treatment is determined entirely by age, in the above equation β_1 reflects the program effect averaged across treated cohorts. Identification of the causal effect of T requires that the error term be uncorrelated with treatment conditional on the observables contained in X and district or sibling average grade attainment (μ_f). If cohort differences in

treatment are correlated with other trends that positively affect grade attainment, the estimates will overstate the true effect of iodine on schooling.

The district fixed effects model minimizes the potential confounding role of unobservable cohort effects that might vary systematically with treatment and district. As a robustness check, we also estimate the above equation using household rather than district fixed effects, which potentially increases the precision of our estimates by holding family background constant. As a final robustness check, we estimate the same regression using 2004 DHS in place of 2000 TDHS data. The only differences in specifications across the 2000 and 2004 analyses are the use of birth month data in the 2004 estimates, which is used to refine the definition of treatment and added to the set of controls to account for the independent effect of small differences in age on school entrance, and differences in the exact household- and child-level control variables available in each survey (covariates available in 2004 are reported in the notes to Table 3).²¹

IV. Results

IV.A Grade Attainment

Regressions of grade attainment on program participation yield large and significant estimates of the impact of IOC on progression through school, presented in Table 3. In district fixed-effects regressions from 2000, adequate maternal iodine in utero is associated with 0.347 years of additional schooling relative to peers who are unprotected (column 1). When the regressions are run separately by gender, the estimated effect is even larger – 0.594 years – and statistically significant among girls but not boys. When the binary measure of program participation is used in place of the continuous likelihood measure (columns 4-6), the results are similar in magnitude and retain significance.²²

The measured effects in columns 1-6 underestimate the cognitive impact of IDD to the

²¹ For this reason in the TDHS sample we exclude children who are missing birth month data. Date of birth is missing for 20.1% of children of the head between ages 9 and 17 because their mother did not participate in the birth history survey. Attrition rates are not significantly different for treatment and control households.

²² The magnitudes are almost identical when the coefficient estimate on the binary treatment variable is scaled by the likelihood of treatment among those who are considered protected with probability one in the binary measure.

extent that not all pregnant women in a district were reached by the program. When schooling is instead regressed on program exposure multiplied by the district rate of program coverage (columns 7-9), as expected the point estimate rises by roughly the inverse of the average coverage rate, implying that actual protection from in utero IDD is associated with 0.559 additional years of schooling.

Reassuringly, when coverage information is incorporated into the exposure variable the estimated program effect also becomes more precise, suggesting that reported coverage constitutes meaningful variation in iodine exposure. In gender-specific estimates, the program effect on boys is an estimated 0.384 years, and remains roughly half that of girls, who gain an estimated 0.824 years of schooling. While these are considerably larger, it is important to bear in mind that the estimates that incorporate average coverage rates are likely to overstate the true program effect since coverage is likely to be higher than average among women of child-bearing age due to the fact that they are more likely to respond to public health initiatives and were reportedly first on the priority list for receiving IOC (Peterson, 2000).

The pattern of results is robust to replacing district with household fixed effects (columns 10-12). In household fixed effects regressions by gender, the estimated treatment effect is once again only significant for girls but somewhat larger in magnitude than the district fixed effect result. The higher point estimate is likely driven by the fact that household fixed effects estimates can only be estimated on the sample of households with many children close in age, which are likely to be more vulnerable to IDD on account of the observed correlation between family size and poverty. This interpretation is supported by the fact that the difference between household and district fixed effects estimates disappears when the latter is restricted to households with more than one child (unreported).

Finally, the 2004 estimates of schooling attainment (columns 13-15) are strikingly similar to the 2000 estimates despite the fact that sample members were born in different years and

enumeration areas from the THBS sample. Since treatment status pertaining to children of a given age and district is not constant across samples, the similarity of 2000 and 2004 estimates alone indicates that the findings are not driven by time-invariant patterns of grade attainment by age that are spuriously correlated with treatment.

In all specifications, the coefficient estimates on the correction factor for rate of iodine depletion 3-4 years after the program are insignificant and close to zero, indicating that coverage falls at the assumed rate. While the coefficient estimates on the correction factor for mothers who received iodine below age 23 are generally insignificant, the point estimates are large and approach significance in a number of cases, suggesting that a number of young women may have actually received 200 mg supplements as intended and therefore experienced little program effect after two years. More general robustness checks verify that the program effect is robust to the inclusion of the ~20% of children that could not be precisely matched to mothers, suggesting that orphaned or fostered children are likely to live in districts where they were born (Table 5, columns 1-3), and to including children as young as 6 (on-line Appendix B, columns 1-6). The basic estimates are also robust to excluding control variables other than age, gender and birth order (unreported).

Since there is virtually no drop-out over this interval, the results presumably reflect the influence of fetal IDD on children's rate of progression through school, a function of age of entrance, attendance, and grade retention.²³ As described in Section II.B, in rural Tanzania, all such mechanisms are likely to matter. Without information on age of entrance or attendance, we cannot disentangle the precise mechanism by which educational attainment increases with treatment, however using the larger sample of children 6-13 we look for evidence of the first mechanism by regressing the binary indicator of whether a child has started primary and pre-school on the treatment variable (on-line Appendix B, columns 7-10). These results provide

²³ Furthermore, our coefficient estimates also encompass any externalities of the program on schooling outcomes, which could either be positive (e.g. learning spillovers) or negative (e.g. school crowding).

weak evidence of treatment influence on age of entry into primary but not pre-school, which appears to be larger for girls.

Regressions on grade-by-grade attainment (on-line Appendix C) provide strong evidence of a treatment effect on retention rates. In particular, there is a large estimated effect of treatment on the likelihood of passing at least one primary school grade (column 1), no observed treatment effect on completing early primary school (column 2), and a large estimated treatment effect on the likelihood of starting late primary school (column 3). As described in Section II.B, children take their first national standardized test at the completion of Standard 4, which determines entry to Standard 5 (traditionally middle school). The pattern of regression results in Appendix C indicates that, if age of entry is indeed lower for protected children, unprotected children catch up in terms of grade attainment by the end of early primary school and then fall behind again at Standard 4 exams. This pattern of results implies that test-taking ability conditional on grade 4 attainment is sensitive to in utero iodine availability, which is inconsistent with a story in which the effect of IOC on schooling operates exclusively through earlier entrance among the treated.

One of the most striking patterns in both sets of results is the consistently higher estimated program effect on girls. In all estimates, girls appear to benefit more than twice as much as boys from IOC in utero. While observed gender differences in the impact of IOC may reflect physiological differences in the importance of iodine for fetal brain development similar to those observed in animal studies, there are two other possible interpretations for the gender findings. First, gender differences may reflect the fact that girls in Tanzania systematically enter school at an earlier age than boys. If the importance of cognitive ability on school pass rates increases with grade, girls between 10 and 13 will benefit more from the intervention simply because they are more likely to be on the margin of influence. Here, it is important to note that baseline gender differences in age of entry are quite small. To account for the full gender difference in IOC, a 0.2 year difference in age of entry would have to correspond to twice the

effect of IOC on attainment, which could only happen if the influence of ability on pass rates were highly non-linear with age.

A more compelling reason that we might observe differences across girls and boys in the impact of IOC in utero is that parents' decision to invest in girls' schooling may be more sensitive to differences in cognitive capacity. This could be the case if, for instance, the cost of enrollment is higher for girls than for boys due to girls' higher productivity at home or greater opportunities for marrying young. If this is true, boys and girls might experience the same cognitive benefits of IOC at a biological level, but these benefits translate into greater schooling improvements for girls. Unfortunately, without data on cognitive capacity, there is no clear way to distinguish the last story from a disproportionate improvement in female cognitive capacity. However, the fact that gender differences in treatment effects are observed in examination years only (Appendix C) provides important evidence that the gender difference is not due to parents' choices over schooling.

Importantly, the above estimates of grade attainment are biased measures of the program effect on final schooling attainment since education outcomes are unobserved, although the direction of bias is ambiguous. In general, differences in grade attainment widen over time as a disproportionate number of slow achievers drop out of school. On the other hand, if there is sufficient catch-up at the point of primary school transition, the program effect on final schooling attainment could be significantly lower. Unfortunately, treated children in all available datasets are too young to enable examination of secondary school outcomes, and censored data models are unlikely to be appropriate for estimating the total effect of the program on schooling attainment given the substantial barriers to secondary school enrollment which are likely to generate sharp discontinuities in grade attainment around age 14.

In Table 4 we also explore heterogeneity in program effect according to two proxies for baseline rate of iodine deficiency. Columns 1-3 examine variation in the program effects with

baseline total goiter rate (TGR), measured between 1980 and 1988. TGR data are available at the district level from the World Health Organization for 21 of the 25 districts in our sample. As expected, in these estimates we observe a strong negative interaction between 1980 TGR and iodine supplementation, implying that the importance of iodine supplementation rises with the population rate of iodine deficiency. In columns 4-6 we examine within-district variation in the importance of iodine supplementation by studying the difference in program effect in relatively poor households, as measured by reported food insecurity in the 2000 survey. As expected, the coefficient estimate on the interaction term indicates that girls in relatively poor households benefit more from iodine supplementation, as would be expected if poor households have less nutrient-rich diets. These patterns provide consistency checks on our empirical model.

IV.B Robustness Checks

Importantly, our estimation strategy leaves little room for omitted factors to bias the results, and simple robustness checks rule out obvious competing stories. First, the consistency of the treatment effect across sample years provides strong evidence that the estimated program effects are not driven by time invariant district-specific patterns of schooling attainment by age. We also confirm that the treatment effect is absent in a placebo regression run on children too old to benefit from IOC in utero by regressing grade attainment among children in sample districts who were 10 to 13 in 1988 on a pseudo-indicator of IOC that pertains to children of the same age and district in 2000 (on-line Appendix D).

Second, we confirm that the treatment effect can be separately identified off of both program gaps and program delays, which rules out the possibility that our results simply reflect time trends that vary systematically with program start dates. For instance, the age gap between 10 and 12 year-olds may be lower in districts in which younger but not older siblings were treated simply because education is increasing faster in districts that received IOC later.

Importantly, because IOC was rarely distributed according to the intended two-year schedule, there are multiple instances of an older but not a younger sibling receiving IOC. In total, among all sibling pairs in the 2000 sample in which only one individual was treated, the older sibling was treated in 23% of cases. This variation in treatment patterns allows us to check whether the program both *reduces* the grade attainment gap when a younger sibling is treated and *increases* the gap when an older sibling is treated.

Figure 1 shows the average difference in grade attainment for three categories of siblings classified according to the binary indicator of treatment: (1) those in which both or neither benefited from IOC; (2) those in which the older but not the younger sibling benefited from IOC; and (3) those in which the younger but not the older sibling benefited from IOC. Comparison across these groups reveals that the program effect is symmetric across the latter two cases: When an older but not younger sibling is protected from IDD in utero, the difference in schooling attainment widens, and when the younger but not older sibling is protected, the difference narrows. Regression estimates of grade differences on sibling age gaps reveal statistically significant (at the 10% level) program effects of the expected sign in both comparisons (on-line Appendix E).

Third, we restrict the control group to children born during a program year in order to confirm that the results are robust to comparisons between treated children and siblings or peers in utero or recently born at the time of the program (Table 5, columns 4-6). The fact that the treatment effect remains constant in both size and significance among this subsample significantly reduces the set of confounding influences that could bias our results. To account for all findings, omitted variables of concern would have to be factors that impact offspring during early but not late fetal development or neonatal stages.

Fourth, we confirm that the estimated treatment effect is at least as strong two years after IOC, as our biological model implies, by verifying that the estimates are robust in magnitude to

excluding from the *treatment* group children born one year after the program (Table 5, columns 7-9). Given that the estimated treatment effect does not fall within the two-year window, any omitted factor that accounts for the observed patterns would have to have an equally lasting effect on maternal health.

This collection of evidence reduces the set of potential confounders to district-specific changes in early fetal health environment *other than iodine* that: (1) coincided with multiple rounds of IOC distribution in timing and duration (i.e. match Table 1), (2) were not accompanied by equally large improvements in neonatal health, and (3) had a sustained effect on maternal health for 24 to 36 months.

IV.C Threats to Validity

Within this set, there are two categories of possible confounding influences. First, treatment may have influenced early fetal outcomes through channels other than iodine availability, either through interaction with health care workers or the offer of alternative health inputs at the time of IOC. For two reasons, other aspects of treatment are unlikely to explain our results. First, given the nature of distribution, in which villagers were assembled on an appointed day to receive supplements en masse, and the government's emphasis on quick distribution rounds, it is unlikely that other health services would have been offered at the same moment (Peterson, 2000).²⁴ Second, it is unlikely that any information or alternative health inputs that could have been provided in conjunction with IOC would have had a sustained effect on mother's physical health or health behaviors for two years after treatment, and those that could have had a lasting effect would generally also improve outcomes of children born immediately after iodine depletion, contradicting the evidence in Table 5. For instance, Vitamin E supplements or nutritional supplements given to women at the same time as IOC would only

²⁴Nor is there any discussion of this occurring in lengthy program implementation reports (and no incentive to hide).

benefit children born very soon after the program and not those born two to three years after. Similarly, changes in mothers' behavior as a result of the treatment are unlikely to affect children born 3 but not 4 years after the program and also not affect children born during the program.

A second possible concern is that the timing of distribution rounds was driven by intermittent declines in the quality of district prenatal (but not neonatal) health services. In this case, children in utero during program gaps may have experienced other deficiencies in fetal health inputs relative to those born immediately before or after, which could lead to permanently poorer health – and possibly schooling – among children who did not benefit from IOC that is independent of reductions in IDD. Similarly, if program timing was driven by district-level income shocks, children in utero during the program may have received better nutrition at critical stages of development.

Once again, the duration of IOC coverage makes such stories difficult to construct. Essentially, since most treated children are born two to three years after IOC and a significant fraction of *control* children are born the year of and the year before IOC, treatment is not well correlated with program activity. The robustness of our results to restricting the control group to children born during a program year (Table 5) confirms the limited potential for a treatment effect driven by unobservable district trends that are correlated with IOC. To illustrate, suppose there are district-level trends in income strong enough to generate improvements in health care in year t (a program year) followed by a worsening of health care in year $t+3$ (a program gap year). In this case, unobservable income should be *higher* for children born in t (the control group) when services are upgraded relative to children born in $t+2$ or $t+3$ (the treatment group) when the district fails to replenish services, biasing our estimates downwards if at all.

Information on sources of coverage delays and gaps also provides qualitative evidence that variation in treatment was independent of other shocks to fetal health environment. A post-intervention study by Peterson (2000) provides a detailed account of sources of delay gleaned

from IOC program reports and administrative records, interviews with past and present program managers, and supervision visits to selected districts. The key piece of information from this study is that, in all cases of delay, lags in program start date were due to administrative delays resulting from the logistical challenges of district-wide IOC distribution and start date was ultimately determined by an external rather than an internal force. Delays of one to three years most likely resulted from delayed receipt of IOC from the government, which sent capsules to district health centers as late as 1989. Meanwhile, the eight districts delayed beyond 1989 started late because they were slow to organize a distribution system, which was eventually resolved externally through the establishment by the central government of national district teams.²⁵ Given the central role of external resource provision in determining distribution timing there is little reason to suspect that variation in program timing was related to income shocks or changes in the quality of health care services within districts.

Finally, since all possible sources of bias relate to unobservable influences on health that in turn reduce schooling, in the following section we further assess the degree to which either unobservable impacts of treatment or omitted trends in district income or services pose a threat to our estimation strategy by testing whether variation in IOC is related to observable health status of children and reported school days missed due to illness. The latter is a particularly strong test of our identifying assumption since alternative explanations would almost by definition operate through increased schooling absence due to sickness. Hence, the absence of observable differences in current health status between treated and untreated children provides strong evidence that the program effect is driven by IOC. These data also allow us to explore

²⁵ Distribution involved organizing mass campaigns on one particular day in each village through one of two strategies: In addition to IOC, some districts received central funding for fuel and health worker per-diems and set up a “district team” which toured the area using government vehicles. Other districts initially received only IOC and were told to integrate distribution into primary health care facilities. Eight of nine districts attempting the latter did not accomplish this before the capsules were close to expiring. To ensure rapid distribution before expiration, in four of the eight districts, the central government established “national district teams” in which staff from the national program initiated and supported distribution with cars, money for fuel and per-diem pay. This discussion and the empirical analysis ignore two districts that were added late and began 1994 to 1995.

whether IOC operates through reducing childhood illness rather than improving cognition.

IV.D Health Effects of IOC

The patterns of results observed in Tables 3-4 are consistent with a change in the cognitive cost of schooling resulting from lower incidence of fetal IDD. However, since IQ is unobservable and IDD has also been associated with infant and child health outcomes in some but not all experimental studies in humans, it is possible that program participation influenced schooling attainment of children in utero by improving their long-run physical health which in turn increased the ability to progress through school.²⁶ Although iodine deficiency has been demonstrated in laboratory studies to influence fetal brain development much more readily than physical development, it is possible that its influence is distinct in this particular setting such that the impact on physical health is more acute than the impact on cognition. Furthermore, even if cognitive damage from IDD exceeds physical health damage, schooling attainment may be more sensitive to physical health status than to cognitive ability at this level.

To examine this possibility, we make use of data on the health status in 2000 of children in utero during the program, which include indicators of six different health problems over the past year in addition to number of school days missed due to sickness. The latter outcome is a particularly useful test of whether the program effect operates through illness since, in an environment of near universal primary enrollment, school attendance is the mechanism through which in utero health damage would most likely account for the observed program effect on schooling. The following health outcomes are available from the THBS: whether the child experienced fever/malaria, diarrhea, an ear/nose/throat condition, a skin condition, an eye condition, an accidental injury or any other health episode during the last four weeks, frequency of illness over the past month, and total days of work or school missed during the last four weeks

²⁶ The evidence on the health effects of in utero IDD is mixed. See Allen and Gillespie (2001) for an overview.

due to any sickness or injury.

Regression estimates in Table 6 indicate that children in utero during the program are no more likely to experience illness at ages 10-13 conditional on surviving to that age, suggesting no significant program effect on the average health status of survivors. The estimates indicate no relationship between IOC and school days missed due to illness (column 10), nor is there any evidence that children covered by the program report fewer episodes of ill health (column 1). Furthermore, the estimates in columns 2-9 indicate no program effect on a wide range of observable measures of child health at ages 10-13, all of which have the potential to be influenced by an overall weakening of the immune system resulting from fetal IDD. Only one symptom out of six - diarrhea – is weakly associated with the program, but there is no consistent pattern in the signs of point estimates across the measures of illness. This set of findings suggests that the measured program effect of IOC operates through cognitive rather than physical health improvements.

Similarly, the Table 6 results provide evidence that the estimated program effects are not driven by shocks to fetal health environment *other* than IOC that coincided with the program. As discussed in Section IV.B, our principal identifying assumption is that variation in program activity (including program gaps and delays) is uncorrelated with within-district changes in other fetal health inputs that affected schooling through persistent differences in health status. Given that there is no evidence of changes in average health status of children treated with iodine supplements in utero, it is unlikely that the pattern could be driven by changes in health inputs such as nutrition. Furthermore, since the effects of fetal iodine deficiency are thought to be overwhelmingly cognitive whereas deficiencies in other health inputs are more likely to show up in physical outcomes, the Table 6 results favor the interpretation that the estimated program effect operates through IOC. Although it is impossible to rule out in utero *cognitive* damage resulting from the absence of health inputs other than IOC, there is no obvious candidate

influence on fetal cognition other than IOC.

IV.E International Implications

The Universal Salt Iodization (USI) movement was based on the notion that IDD is easily and inexpensively preventable through iodized salt (Mannar, 1996). Approximately 40 countries passed USI legislation between 1970 and 2000, the majority during the 1990s, resulting in an increase of iodized salt intake from 20% of the world population to over 70%. On account of USI legislation and local distribution efforts, approximately two-thirds of the previously IDD-affected population of Africa now consumes adequately iodized salt (Unicef, 2005). The magnitude of the estimated effect of IOC on schooling in Tanzania implies that comparable reductions in iodine deficiency worldwide that have resulted from USI over the past two decades should be visible in improvements in aggregate schooling between 1980 and 2000. Indeed, cross-country regression estimates suggest that reductions in IDD between 1980 and 2000 have had an positive effect on both male and female primary school participation, evidenced by the fact that both measures are increasing in the fraction of households consuming iodized salt.²⁷

Based on the estimated impact of IOC in Tanzania, we calculate the expected gains in education that should be observed by 2015 among the 42 countries that experienced unambiguous reductions in IDD through USI legislation passed in the 1980s and 1990s. In each country, we estimate the number of children that were newly protected from fetal IDD over the past decade by multiplying the number of children at risk pre-legislation by the fraction of households using adequately iodized salt in 2000.²⁸

According to our estimates, approximately 41.1 million children between the ages of 5

²⁷ Cross-country regression estimates are available in an earlier version of the paper (Field, 2006) posted on the author's website.

²⁸ The number of children previously at risk is the population of children aged 5-9 in 2002 times the rate of in utero IDD. The pre-legislation rate of in utero IDD is conservatively assumed to be twice the baseline TGR among school-age children based on the fact that TGR is approximately three times more prevalent and the ratio of recommended iodine intake twice as high in pregnant women compared to school-age children.

and 9 in 2002 have benefited from increases in iodine intake over the past decade, with the largest populations of newly protected children found in Algeria, Indonesia and Nigeria (on-line Appendix F). Based on our previous estimates, the expected increase in grade attainment for a child protected from fetal IDD is a minimum of 0.73 years.²⁹ Multiplying the expected increase in schooling per treated child by the estimated number of children who are newly protected, we calculate an anticipated overall impact of USI for each country ranging from 0.5% to 40%, with the largest gains in Africa. Based on our estimates, 13 countries should experience more than a 10% improvement in schooling attainment by the year 2015. For Central and Southern Africa, the predicted improvement in average schooling due to USI across all countries is 7.5%.

VI. Conclusions

Our findings provide micro-level evidence of the influence of iodine availability on cognition. Variation in iodine availability is likely to play an important role in growth patterns to the extent that human capital investment falls with rates of learning disability. Even holding schooling attainment constant, small differences in average IQ at the group level could have large effects on social and economic outcomes. Given how inexpensive iodization campaigns are to implement, as New York Times columnist Nicholas Kristof pointed out in a recent op-ed, “[T]here may be no investment that gets more bang for the buck.” (Kristof, 2008)

Our results also support laboratory evidence that the female fetus is more sensitive to in utero iodine exposure, such that endemic iodine deficiency may give rise to gender differences in cognitive ability. The possibility that physiological gender differences exert a significant influence on schooling has important implications for how we interpret gender differences in schooling attainment across the globe and over time. An important caveat is that we cannot fully rule out the possibility that gender differences are driven by sex-specific household responses to

²⁹ This effect is calculated from the baseline effect (0.39 years) observed in Tanzania adjusted for an average IOC take-up rate assumed to be 75% and the average rate of maternal IDD (60%) in the target population.

improvements in cognition rather than disproportionate increases in female cognitive capacity. However, the corresponding evidence of gender differences in fetal sensitivity to maternal iodine levels from controlled laboratory studies and gender differences in rates of iodine deficiency should not be discounted.

Based on these estimates, reduced levels of IDD due to wide-scale salt iodization in the 1990s are likely to have a visible impact on schooling attainment in previously afflicted areas over the next two decades, and these changes are likely to disproportionately benefit girls. In much of Africa, universal salt iodization could go far towards achieving gender parity in schooling attainment. Reduced fetal IDD among the birth cohorts of 1990-2000 will be important to bear in mind when interpreting changes in schooling in much of the developing world over the coming decade.

References

- Allen L and Gillespie S (2001). What works? A review of efficacy and effectiveness of nutrition interventions. *UN (ACC/SN)*.
- Barker DJP (1990). Fetal and infant origins of adult disease. *British Medical Journal* 301: 1111.
- Barker DJP (1995): Fetal origins of coronary heart disease. *British Medical Journal* 311:171-174.
- Basta SS, Soekirman Karyadi D, and Scrimsha NS. (1979). Iron deficiency anemia and the productivity of adult males in Indonesia. *American Journal of Clinical Nutrition* 32: 916-25.
- Bautista A, Barker PA, Dunn JT, Sanchez M, and Kaiser DL (1982). The effects of oral iodized oil on intelligence, thyroid status, and somatic growth in school-age children from an area of endemic goiter. *The American Journal of Clinical Nutrition* 35:127-134.
- Beaton GH, Martorell R, L'Abbe KA, Edmonston B, McCabe G, Ross AC and Harvey B (1992). Effectiveness of Vitamin A supplementation in the control of young child morbidity and mortality in developing countries. SCN State-of-the-Art Nutrition Policy Discussion Paper No. 13.
- Behrman JR, Hodinott J, Maluccio J, Martorell R, Quisumbing A, and Stein A (2003). The Impact of experimental nutritional intervention in childhood on education among guatemalan adults. International Food Policy Research Institute: Brief Discussion Paper 207.
- Bilabina I, Brazier M, and Bour H (1994). Evaluation of iodide deficiency in Togo using an optimized potentiometer method for iodide estimation in urine; *Annales de Biologie Clinique* 52(4):261-4.
- Benoist B, Andersson M, Egli I, Takkouche B, and Allen H (2004). Iodine Status Worldwide: WHO Global Database on Iodine Deficiency. World Health Organization. ISBN 92 4 159200 1.
- Bleichrodt N, and Born MA (1994). A meta-analysis of research on iodine and its relationship to cognitive development. *Report of the Franklin Symposium: Iodine deficiency and brain damage*. Cognizant Communications Corporation.
- Boyages SC, Halpern JP, Maberly GF, Eastman CJ, Morris J, Collins J, Jupp JJ, Jin CE, Wang ZH, and You CY. (1988). A comparative study of neurological and myxedematous endemic cretinism in Western China. *Journal of Clinical. Endocrinology & Metabolism*. v67: 1262-66.
- Cao XY, Jiang XM, and Dou ZH (1994). Timing of vulnerability of the brain to iodine deficiency in endemic cretinism. *New England Journal of Medicine* 331: 1739-44.
- Chan SY, Andrews MH, Lingas R, McCabe CJ, Franklyn JA, Kilby MD, and Matthews SG (2005). Maternal nutrient deprivation induces sex-specific changes in thyroid hormone receptor and deiodinase expression in the fetal guinea pig brain. *Journal of Physiology* 566(Pt 2): 467-480.
- Delange F (2000). The role of iodine in brain development, *Proceedings of Nutrition Society* 59(1):75-80.
- Delange F (1998). Risks and benefits of iodine supplementation. *Lancet* 351(9107): 923-924.
- Dugbartey, A (1998). Neurocognitive aspects of hypothyroidism, *Archives of Internal Medicine*. 158(13):1413-18.
- Eltom M, Karlsson FA, Kamal AM, Bostrom H, and Dahlberg PA (1985). The effectiveness of oral iodized oil in the treatment and prophylaxis of endemic goiter. *Journal of Clinical Endocrinology and Metabolism* 61(6):1112-7.
- Field E, Robles O and Torero M (2006). The Cognitive link between geography and development: Iodine deficiency and schooling attainment in Tanzania. Mimeo, Harvard University. Available at: http://www.economics.harvard.edu/faculty/field/papers_field.

- Friedhoff AJ, Miller JC, Armour M, Schweitzer JW, and Mohan S (2000). Role of maternal biochemistry in fetal brain development: effect of maternal thyroidectomy on behaviour and biogenic amine metabolism in rat progeny. *International Journal of Neuropsychopharmacology* 3:89–97.
- Furnee CA (1997). Prevention and control of iodine deficiency: a review of a study on the effectiveness of oral iodized oil in Malawi. *European Journal of Clinical Nutrition* 51(Suppl 4):S9-10.
- Gaitan E and Dunn J (1990). Goitrogens in food and water. *Annual Review of Nutrition* 10:21-39.
- Glasizou PP and Mackerras DEM (1993). Vitamin A supplementation in infectious diseases: a meta-analysis. *British Medical Journal* 306:366-70.
- Haddow, J, Palomaki G, Allan W, Williams J, Knight G, Gagnon J, O’Heir C, Mitchell M, Hermos Rosalie, Waisbren S, Faix J, and Klein R. (1999). Maternal thyroid deficiency during pregnancy and subsequent neuropsychological development of the child, *New England Journal of Medicine*. August; 341(8):549-555.
- Halpern JP, Boyages SC, Maberly GF, Collins JK, Eastman CJ, Morris JG. (1991). The neurology of endemic cretinism: A study of two endemias. *Brain* 114:825-41.
- Hetzel, BS (1983). IDD and Their Eradication. *Lancet* ii:1126-1129.
- Hetzel BS (1989). The story of iodine deficiency. Oxford University Press, Oxford, UK.
- Hetzel BS (2000). Iodine and neuropsychological development. *Journal of Nutrition* 130(2S Suppl): 493S-495S.
- Hetzel BS and Mano M (1989). A review of experimental studies of iodine deficiency during fetal development, *Journal of Nutrition* 119:145-151.
- Husani, KD and Gunadi H (1981). Evaluation of nutritional anemia intervention among anemic female workers on a tea-plantation. *Iron Deficiency and Work Performance* 73. Washington DC: Nutrition Foundation, Washington DC, USA. IDPAS# 127.
- Kristof, Nicholas “Raising the World’s IQ.” *New York Times*, p. A43, December 4, 2008.
- Lamberg BA (1991). Endemic Goiter-Iodine Deficiency Disorders. *Annals of Medicine* 23: 367-372.
- Lavado-Autric R, Auso E, Garcia-Velasco JV, Arufe Mdel C, Escobar del Rey F, Berbel P, and Morreale de Escobar G. (2003). Early maternal hypothyroxinemia alters histogenesis and cerebral cortex cytoarchitecture of the progeny. *Journal of Clinical Investigation* 111:1073–82.
- Malaney, Pia and Jeffrey Sachs (2002) The Economic and social burden of malaria. *Nature*, Vol. 415, no. 6872, Feb. 7, 2002.
- Mannar M (1996). The iodization of salt for the elimination of iodine deficiency disorders. *SOS for a Billion*. Hetzel & C. Pandav. Dehli: Oxford University Press.
- Marett JRH (1936). Race, sex and environment: A Study of mineral deficiency in human evolution. London: Hutchinson’s Scientific and Technical Publications, 342 pgs.
- Merke F (1984). The History and Ichnography of Endemic Goitre and Cretinism. Lancaster: MTP Press.
- Painter RC, TJ Roseboom, and OP Bleker (2005). Prenatal Exposure to the Dutch Famine and disease in later life: an overview. *Reproductive Toxicology* 0(3):345-352
- Pandav CS and Kochupillai N. (1982). Endemic goitre in India: Prevalence, etiology, attendant disabilities and control measures. *Indian Journal of Pediatrics* 50:259
- Peterson S (2000). Controlling iodine deficiency disorders: Studies for program management in Sub-Saharan Africa. *Uppsala Dissertations from the Faculty of Medicine* 943.

- Peterson S, Assey V, Forsberg B, Greiner T, Kavishe FP, Mduma B, Rosling H, Sanga B, and Gebre-Medhin M (1998). Coverage and cost of iodized oil capsule distribution in Tanzania. *Health Policy and Planning* 14(4):390-399.
- Pharoah PO and Connolly KJ (1987). A controlled trial of iodinated oil for the prevention of endemic cretinism: a long-term follow-up. *International Journal of Epidemiology* 16:68-73.
- Rao PS and Lakshmy R (1995). Role of goitrogens in iodine deficiency disorders & brain development. *Indian Journal of Medical Research* 102:223-6.
- Research and Analysis Working Group (R&AWG) (2005). Tanzania poverty and human development report 2005. Ministry of Planning, Economy and Empowerment, Poverty Eradication Division. Dar es Salaam, Tanzania. Available at: www.povertymonitoring.go.tz
- Sachs, Jeffrey (2001). Tropical underdevelopment. NBER Working Paper 8119. February 2001.
- Simon PA, Jamison DT and Manning MA (1990). Gender differences in Goiter prevalence: A review. Los Angeles, CA: University of California Press.
- Sommer A, Tarwotjo I, Hussaini G, Susanto D, and Soegiharto T (1981). Incidence, prevalence, and scale of blinding malnutrition. *Lancet* 1:1407-8
- Sommer A, Tarwotjo I, Djunaedi E, West K, Loeden A, Tilden R, and Mele L (1986). Impact of vitamin A supplementation on childhood mortality: A randomized controlled community trial. *Lancet* 1:1169-73.
- Sundqvist J, Wijetunga M, Assey V, Gebre-Medhin M, and Peterson S (1998). Salt iodation and risk of neonatal brain damage. *Lancet* 352(9121):34-35.
- Tanzanian Census (1988). Dar Es Salaam: Bureau of Statistics, Ministry of Finance, Economic Affairs and Planning.
- Thomas D, Frankenberg E, Friedman J, Habicht J-P, Hakimi M, Jaswadi, Jones N, McKelvey C, Pelto G, Sikoki B, Seeman T, Smith H, Sumantri C, Suriastini W, and Wilopo S. (2003). Iron deficiency and the wellbeing of older adults: Early results from a randomized nutrition intervention. Mimeo, UCLA
- UNICEF (2004). A pinch of salt can go further In West Africa. Press center: http://www.unicef.org/media/media_23686.html
- United Republic of Tanzania, Ministry of Education and Culture (MoEC) (2003). *Poverty and Human Development Report*, Mkuki na Nyota Publishers.
- United Republic of Tanzania, Ministry of Education and Culture (MoEC) (2003b). *Basic Statistics in Education 1999-2003*. Dar es Salaam.
- Untoro, J, Schultink W, Gross R, West CE, Hautvast JG (1998). Efficacy of different types of iodised oil. *Lancet* 351(9104): 752-753.
- Van der Haar F, Kavishe P, Medhin MG (1988). The public health importance of IDD in Tanzania. *Central African Journal of Medicine* 34(3):60-5.
- Vermeersch, C (2003). School meals, educational achievement and school competition: Evidence from a randomized evaluation. Mimeo, University of Oxford.
- West KP, Howard JR, Sommer A (1989). Vitamin A and Infection: Public health implications. *Annual Review of Nutrition* 9:63-86.
- Wei J and Li J (1985). Metabolism of iodized oil after oral administration in guinea pigs. *Nutrition Reports International* 31:1085-1087.
- Wolff, J (2001). Physiology and Pharmacology of Iodized Oil in Goiter Prophylaxis. *Medicine* 80:20-36.

- WHO (1991). National strategies for overcoming micronutrient malnutrition, EB 89/27.
- WHO (1992). National strategies for overcoming micronutrient malnutrition, A45/17.
- Zaleha MD, Isa PhD, Iskandar Zulkarnain Alias MSc, Khalid Abdul Kadir PhD, Osman Ali PhD (2000). Effect of iodized oil supplementation on thyroid hormone levels and mental performance among *Orang Asli* schoolchildren and pregnant mothers in an endemic goitre area in Peninsular Malaysia. *Asia Pacific Journal of Clinical Nutrition* 9(4):274–281.
- Zoeller, RT and Rovet J (2004). Timing of thyroid hormone action in the developing brain: Clinical observations and experimental findings. *Journal of Neuroendocrinology* 16:809–818.

Appendix A:

Probability of protection from in utero IDD relative to program year t by month of birth, 380mg IOC ^{1,2,3}

	Jan	Feb	March	April	May	June	July	Aug	Sept	Oct	Nov	Dec	Birth year average	Seasonality adjusted birth year average
<i>Program year t</i>	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.028	0.083	0.167	0.250	0.333	0.072	0.070
<i>t + 1</i>	0.417	0.500	0.583	0.667	0.750	0.833	0.917	1.000	1.000	1.000	1.000	1.000	0.806	0.802
<i>t + 2</i>	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	0.998	0.991	0.977	0.997	0.997
<i>t + 3</i>	0.955	0.927	0.891	0.849	0.802	0.749	0.690	0.627	0.559	0.488	0.419	0.353	0.668	0.696
<i>t + 4</i>	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	0.101

Notes:

¹ Calculations make the following assumptions about IOC distribution over the year: Three months are required for the program to reach all individuals in a district, and the distribution of program start dates over the year is uniform. This implies that children born t months after the start of the program year were treated in time with probability equal to: $\frac{1}{36}$ if $t = 8$; $\frac{1}{18}$ if $t = 9$; and $\min(1, \frac{1}{36} + \frac{1}{18} + \frac{t-9}{12})$ if $t > 9$.

² Iodine contained in IOC is assumed to be stored in the body after an immediate extraction of 90% during month 0, and depleted during months 1-38 following a simple hyperbolic discounting function ($V = \frac{A}{1+kt}$) with a half-life at month 1 of 3 months ($\rightarrow k = 0.33\bar{3}$).

³ Minimum iodine requirement for one full month of protection from IOC was calculated to be 6.5mg based on recommended daily requirement for pregnant women of 1.4mg – 2.1mg (multiplied by 30 days), assuming daily depletion of dietary iodine of 90%. Based on this range of required iodine across the population, iodine stores below 4.2mg were assumed to offer inadequate protection from fetal IDD.

⁴ Seasonality adjustment based on district-level number of births per month between 1996 and 2004 in the 2004 TDHS.

Appendix B: 2000 Grade Attainment and IOC Supplementation in Utero, Ages 6-13

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	Highest Grade Attained						Started primary school		Started pre-school	
	All	Girls	Boys	All	Girls	Boys	All	All	All	All
Pr(IOC in utero)	0.172 [0.063]**	0.135 [0.110]	0.130 [0.101]	0.119 [0.059]*	0.200 [0.086]*	0.059 [0.081]	0.026 [0.020]	0.030 [0.017]+	0.006 [0.019]	0.020 [0.018]
Age 7	-0.001 [0.105]	-0.093 [0.190]	0.128 [0.176]	0.022 [0.086]	0.158 [0.124]	-0.128 [0.119]	0.199 [0.033]**	0.225 [0.025]**	0.166 [0.032]**	0.192 [0.026]**
Age 8	0.196 [0.102]+	0.011 [0.174]	0.272 [0.182]	0.207 [0.090]*	0.339 [0.126]**	0.053 [0.129]	0.387 [0.032]**	0.424 [0.026]**	0.263 [0.031]**	0.307 [0.027]**
Age 9	0.515 [0.099]**	0.253 [0.188]	0.377 [0.178]*	0.57 [0.088]**	0.686 [0.129]**	0.402 [0.122]**	0.609 [0.031]**	0.603 [0.026]**	0.472 [0.031]**	0.48 [0.026]**
Age 10	1.137 [0.098]**	0.741 [0.200]**	0.991 [0.180]**	1.183 [0.083]**	1.312 [0.123]**	1.012 [0.116]**	0.663 [0.031]**	0.713 [0.025]**	0.512 [0.030]**	0.549 [0.025]**
Age 11	1.595 [0.112]**	1.19 [0.229]**	1.352 [0.222]**	1.531 [0.095]**	1.617 [0.142]**	1.322 [0.134]**	0.769 [0.035]**	0.741 [0.028]**	0.56 [0.035]**	0.563 [0.029]**
Age 12	2.526 [0.109]**	2.181 [0.234]**	2.162 [0.231]**	2.42 [0.089]**	2.512 [0.131]**	2.206 [0.130]**	0.743 [0.034]**	0.775 [0.026]**	0.552 [0.034]**	0.586 [0.027]**
Age 13	3.064 [0.119]**	2.805 [0.254]**	2.635 [0.255]**	3.08 [0.097]**	3.363 [0.145]**	2.678 [0.140]**	0.809 [0.037]**	0.789 [0.029]**	0.599 [0.037]**	0.589 [0.029]**
<i>Fixed effects</i>	<i>Household</i>	<i>Household</i>	<i>Household</i>	<i>District</i>	<i>District</i>	<i>District</i>	<i>Household</i>	<i>District</i>	<i>Household</i>	<i>District</i>
<i>Observations</i>	2805	888	926	3590	1765	1825	2805	3590	2805	3590

Notes: Data from the 2000 Tanzanian Household Budget Survey, sample restricted to children ages 6-13 in 25 districts targeted for iodized oil capsule (IOC) distribution between 1986 and 1992. All estimates exclude children that cannot be matched to mothers in the household. Outcome in columns 1-6 is highest grade completed; outcome in columns 7-8 is whether child ever enrolled in primary school; outcome in columns 9-10 is whether child ever enrolled in either primary or pre-school. *Pr(IOC in utero)* is the probability that IOC was distributed in the district before or during the first trimester of pregnancy times the likelihood that sufficient iodine stores remain in the mother's body to protect the fetus during month 1 of pregnancy. Precise values are given in Appendix A. All regressions also control for binary indicators of sex-specific birth order. + significant at 10%; * significant at 5%; ** significant at 1%

Appendix C: 2000 Grade Attainment and IOC Supplementation in Utero, Ages 6-13

	(1)	(2)	(3)	(4)	(5)	(6)
	At least one year early primary (≥ Standard I)		Completed early primary (≥ Standard IV)		At least one year late primary (≥ Standard V)	
Pr(IOC in utero)	0.044 [0.018]*	0.053 [0.021]**	0.007 [0.015]	0.011 [0.019]	0.018 [0.009]+	0.032 [0.012]**
Age 7	0.052 [0.026]*	0.048 [0.034]	0.002 [0.022]	-0.010 [0.031]	-0.008 [0.014]	0.000 [0.020]
Age 8	0.232 [0.027]**	0.202 [0.033]**	0.003 [0.023]	0.011 [0.030]	-0.006 [0.014]	-0.004 [0.020]
Age 9	0.417 [0.026]**	0.411 [0.033]**	0.043 [0.022]+	0.035 [0.029]	-0.002 [0.014]	-0.004 [0.019]
Age 10	0.623 [0.025]**	0.62 [0.032]**	0.168 [0.021]**	0.152 [0.029]**	0.005 [0.013]	-0.003 [0.019]
Age 11	0.685 [0.028]**	0.7 [0.037]**	0.288 [0.024]**	0.303 [0.033]**	0.023 [0.015]	0.023 [0.022]
Age 12	0.809 [0.027]**	0.852 [0.036]**	0.528 [0.023]**	0.546 [0.032]**	0.111 [0.014]**	0.115 [0.021]**
Age 13	0.857 [0.029]**	0.874 [0.039]**	0.675 [0.025]**	0.691 [0.035]**	0.255 [0.016]**	0.236 [0.023]**
Female	0.015 [0.013]	0.01 [0.017]	0.039 [0.011]**	0.046 [0.015]**	0.012 [0.007]+	0.013 [0.010]
<i>Fixed effects</i>	<i>District</i>	<i>Household</i>	<i>District</i>	<i>Household</i>	<i>District</i>	<i>Household</i>
<i>Observations</i>	2805	3590	2805	3590	2805	3590

Notes: Data from the 2000 Tanzanian Household Budget Survey, sample restricted to children ages 6-13 in 25 districts targeted for iodized oil capsule (IOC) distribution between 1986 and 1992. All estimates exclude children that cannot be matched to mothers in the household. Outcomes are binary indicators of whether child has passed a certain grade in school. *Pr(IOC in utero)* is the probability that IOC was distributed in the district before or during the first trimester of pregnancy times the likelihood that sufficient iodine stores remain in the mother's body to protect the fetus during month 1 of pregnancy. Precise values are given in Appendix A. All regressions also control for binary indicators of sex-specific birth order. + significant at 10%; * significant at 5%; ** significant at 1%

Appendix D: Control Experiment, IOC Distribution and Grade Attainment of Older Cohort

	(1)	(2)	(3)	(4)	(5)	(6)
	Boys and girls	Boys	Girls	Boys and girls	Boys	Girls
Pr(IOC in utero)	-0.023	0.069	-0.028	-0.042	-0.035	-0.050
	[0.025]	[0.047]	[0.045]	[0.019]*	[0.027]	[0.026]+
Age 11	0.699	0.692	0.596	0.716	0.784	0.646
	[0.022]**	[0.043]**	[0.041]**	[0.014]**	[0.020]**	[0.020]**
Age 12	1.622	1.558	1.423	1.394	1.445	1.341
	[0.019]**	[0.047]**	[0.047]**	[0.013]**	[0.019]**	[0.019]**
Age 13	2.547	2.446	2.319	2.239	2.301	2.176
	[0.023]**	[0.062]**	[0.061]**	[0.015]**	[0.022]**	[0.021]**
Female	0.268			0.324		
	[0.015]**			[0.010]**		
<i>Fixed effects</i>	<i>Household</i>	<i>Household</i>	<i>Household</i>	<i>District</i>	<i>District</i>	<i>District</i>
<i>Observations</i>	113932	57613	56319	113932	57613	56319

Notes: All data from the 1988 Census of Population and Housing, sample restricted to children ages 10-13 in 1988 in 25 districts targeted for iodized oil capsule (IOC) distribution between 1986 and 1995. In all regressions, Pr(IOC in utero) is equal to the value of the variable described in the notes to Table 3 for children born 12 years later in the same district, such that kids born 11 years before a distribution round receive the value pertaining to kids in the same district born 1 year after the distribution round, etc. Regressions also control for sex-specific birth order and household or district fixed effects. + significant at 10%; * significant at 5%; ** significant at 1%

Appendix E: Difference in Grade Attainment and IOC Supplementation by Birth Order

IOC in utero, eldest only	0.383 [0.201] ⁺	0.383 [0.212] ⁺
IOC in utero, youngest only	-0.225 [0.129] ⁺	-0.225 [0.134] ⁺
IOC in utero, both		-0.001 [0.127]
Age difference = 1 year	0.616 [0.176]**	0.616 [0.176]**
Age difference = 2 years	0.990 [0.160]**	0.990 [0.159]**
Age difference = 3 years	1.333 [0.197]**	1.333 [0.197]**
Age oldest	0.157 [0.057]*	0.157 [0.088] ⁺
Both female	-0.041 [0.123]	-0.041 [0.124]
Both male	-0.115 [0.117]	-0.115 [0.117]
Birth order	-0.008 [0.030]	-0.008 [0.030]
<i>Observations</i>	667	667

Notes: Data from the 2000 Tanzanian Household Budget Survey, sample restricted to children ages 10-13 in 25 districts targeted for iodized oil capsule (IOC) distribution between 1986 and 1992. Observations are sibling pairs from 667 different households in sample in which more than one child between 10 and 13. To balance across treatment orders, in households with more than one sibling pair, pair in which older sibling treated and younger not was selected first, pair in which younger sibling treated and older not was treated second, otherwise two siblings chosen at random. *IOC in utero* is the binary indicator of treatment based on probability that IOC was distributed in the district before or during the first trimester of pregnancy, defined in Notes to Table 3. + significant at 10%; * significant at 5%; ** significant at 1%

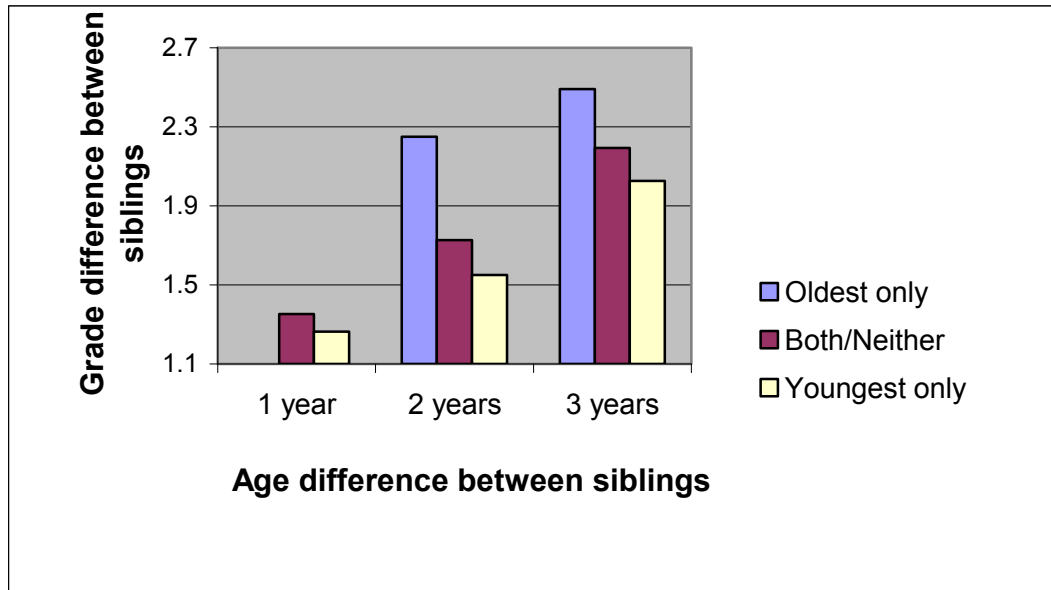
Appendix F: Projected impact on school participation worldwide

Country	% of households using adequately iodized salt	Year Salt Iodization Measured	Total Goiter Rate ¹	Year TGR Measured	Population 5-9 yr 2002 ²	Expected Treated Population ³	Average Years of Schooling ⁴	Percentage increase in grade attainment ⁵
Algeria	92.0	1995	48	1995	3,628	3,204	5.37	11.9%
Argentina	92.0	1996	19.0	1995	3,373	1,179	8.83	2.9%
Bangladesh	44.0	1995	10.5	1982	13,782	1,273	2.58	2.6%
Bhutan	82.0	1996	21.0	1988	276	95	9	2.8%
Croatia	70.0	1997	20.0	1995	267	75	6.28	3.2%
Indonesia	62.1	1997	25.0	1988	23,114	7,177	4.99	4.5%
Jordan	95.0	1997	37.7	1993	677	485	6.91	7.5%
Kazakhstan	52.9	1995	52.1	1993	1,379	760	8.87	4.5%
Kyrgyz Republic	27.0	1997	49.1	1993	530	141	8	2.4%
Malaysia	85.0	1998	36.9	1993	2,618	1,642	6.8	6.7%
Maldives	55.0	1999	23.6	1995	49	13	7	2.7%
Mongolia	46.0	1999	22.0	1993	256	52	8	1.8%
Myanmar	64.8	1997	33.1	1994	4,019	1,724	2.77	11.3%
Nicaragua	86.1	1998	35.8	1994	653	403	4.58	9.8%
Niger	7.4	1996	20.0	1993	1,661	49	1.02	2.1%
Oman	35.0	1996	10.0	1994	376	26	9	0.6%
Pakistan	19.0	1995	13.2	1990	19,761	991	3.88	0.9%
Panama	91.6	1996	13.2	1990	302	73	8.55	2.1%
Paraguay	64.0	1995	48.7	1988	762	475	6.18	7.3%
Philippines	14.6	1996	29.5	1991	10,180	877	8.21	0.8%
Russian Federation	30.0	2000	50	1990	7,069	2,121	10.03	2.2%
Syrian Arab Republic	40.0	2000	42	1994	2,152	723	5.77	4.2%
Thailand	60.2	1999	32	1992	5,264	2,028	6.5	4.3%
Tunisia	63.0	1996	30.5	1988	926	356	5.02	5.6%
Turkey	18.2	1995	23.0	1994	6,274	525	5.29	1.1%
Uzbekistan	16.7	1996	17.2	1981	2,906	167	8	0.5%
Venezuela, RB	90.0	1998	39.7	1986	2,601	1,859	6.64	7.8%
Vietnam	49.4	1996	22.0	1993	8,312	1,807	3.84	4.1%
Central/Southern Africa:								
Angola	35.0	2001	35.3	1965	1,493	369	4	4.5%
Botswana	60.2	1994	16.5	1994	214	43	6.28	2.3%
Burundi	80.0	1993	30	1990	932	447	1.38	25.3%
Cameroon	82.5	1998	26.5	1993	2,142	937	3.54	9.0%
Central African Republ	86.0	2002	80	1991	520	716	2.53	39.5%
Congo	75.0	2000	69	1987	379	392	5.14	14.6%
Congo, Dem. Rep.	12.3	1995	20.0	1995	8,806	433	6	0.6%
Cote d'Ivoire	31.0	2000	43	1992	2,490	664	4	4.8%
Gabon	15.0	2000	34.4	1989	179	18	6	1.2%
Guinea	36.8	1996	26.4	1992	1,277	248	0.84	16.8%
Kenya	100.0	1995	16.3	1984	4,420	1,441	4.2	5.6%
Lesotho	73.0	1996	42.9	1993	234	147	4.23	10.8%
Madagascar	7.0	1995	45.2	1992	2,426	154	6	0.8%
Malawi	58.1	1995	51.2	1993	1,734	1,032	3.2	13.5%
Mozambique	62.0	1995	34.5	1991	2,409	1,031	1.11	28.0%
Namibia	59.0	1996	34.5	1990	270	110	10	3.0%
Nigeria	83.2	1995	10.0	1993	18,766	3,123	5	2.4%
Rwanda	90.0	2000	50.0	1993	982	884	2.56	25.5%
Tanzania	73.8	1995	15.3	1991	5,196	1,173	2.71	6.1%
Uganda	69.0	1995	75.0	1991	4,241	4,389	3.51	21.4%
Zambia	78.1	1996	65.0	1990	1,570	1,594	5.46	13.5%
Zimbabwe	93.0	1999	42.7	1989	1,617	1,284	5.35	10.8%

Total Projected Increase Among Beneficiary Countries Worldwide: **4.83%**

Total Projected Increase Among Beneficiary Countries in Central/Southern Africa: **7.50%**

Figure 1: Sibling differences in schooling by age difference and IOC



Notes: Data from the 2000 Tanzania Household Budget Survey. 576 observations comprise all sibling pairs in 25 pre-1994 project districts in which both children are between the ages of 10 and 13 and are children or grandchildren of the household head or spouse. Mother-child linkages are not perfectly recorded, so children may not be true siblings. Because month of birth is unobservable, there is no variation in likelihood of IOC in utero for siblings of the same age. Hence, siblings of same age are excluded from the analysis. Y-axis is sibling difference in completed years of schooling. IOC categories refer to whether iodized oil capsules distributed in district 1 or 2 years prior to the birth year of each child. Since IOC prevent iodine deficiency for 24 months, this corresponds to higher likelihood that sufficient maternal iodine levels in utero during first two trimesters of pregnancy, what is considered to be the critical intervention period for fetal brain development.

Table 1: Summary of Timing and Coverage of Intervention Across Districts

	Region	District	Year of Intervention (Coverage - %)*					Average Frequency (yr)
			1	2	3	4	5	
1	Dodoma	Mpwapwa	1990 (65)	1992 (58)				2.00
2	Arusha	Monduli	1992 (71)					n/a
3	Arusha	Arumeru	1991 (89)					n/a
4	Kilimanjaro	Rombo	1990 (68)					n/a
5	Morogoro	Ulanga	1988 (73)	1991 (61)	1992 (34)			1.33
6	Ruvuma	Songea Rural	1987 (91)	1991 (74)	1995 (85)			2.67
7*	Ruvuma	Mbinga	1995 (92)					n/a
8	Iringa	Mufindi	1986 (41)	1991 (63)	1995 (54)			3.00
9	Iringa	Makete	1986 (20)	1991 (62)	1993 (62)	1996 (49)		2.50
10	Iringa	Njombe	1989 (76)	1992 (68)	1995 (64)			2.00
11	Iringa	Ludewa	1989 (59)	1992 (62)	1995 (47)			2.00
12	Mbeya	Chunya	1990 (49)					n/a
13	Mbeya	Mbeya Rural	1986 (44)	1989 (84)	1990 (90)	1993 (53)	1997 (53)	1.75
14	Mbeya	Kyela	1989 (91)	1993 (57)				4.00
15	Mbeya	Rungwe	1986 (35)	1990 (73)	1993 (49)			2.33
16	Mbeya	Ileje	1989 (94)	1992 (71)				3.00
17	Mbeya	Mbozi	1989 (67)	1991 (63)				2.00
18	Rukwa	Mpanda	1987 (79)	1991 (60)	1993 (72)			2.00
19	Rukwa	Sumbawanga	1987 (76)	1990 (89)	1993 (72)	1996 (51)		2.25
20	Rukwa	Nkansi	1987 (89)	1991 (49)				4.00
21	Kigoma	Kibondo	1989 (73)	1992 (75)	1996 n/a			2.33
22	Kigoma	Kasulu	1987 (50)	1990 (66)	1996 (49)			3.00
23	Kigoma	Kigoma Rural	1991 (91)					n/a
24	Kagera	Karagwe	1990 (96)	1994 (85)				4.00
25*	Kagera	Bukoba Rural	1994 (78)					n/a
26	Kagera	Biharamulo	1990 (96)	1994 (38)				4.00
27	Kagera	Ngara	1989 (29)	1994 (51)				5.00
Total			27	20	12	3	1	2.76

Notes: Dates and coverage rates collected from various Tanzanian Food and Nutrition Centre (TFNC) Zafari Reports stored in the archives of TFNC library. Coverage was calculated using 1988 Tanzanian Census data and adjusted for proportion of population in target age group. * indicates district that was added to the intervention area post-1990; these two districts were excluded from the analysis since children who benefited from IOC in these areas were too young in 2000 and 2004 to exhibit improvements in schooling.

Table 2: Summary Statistics by Timing of Intervention Across Districts

	(1)	(2)	(3)	(4)	t _A
	IOC Program Timing				
	No Program	1986-1987	1988-1989	1990-1995	2 - 4
Total members per household	4.86 (3.13)	5.02 (2.90)	4.58 (2.50)	5.08 (3.11)	-0.52
Enrollment (ages 5-15)					
Boys	65.4%	68.7%	63.2%	61.6%	4.00
Girls	67.0%	66.8%	67.4%	61.2%	3.09
Total	66.2%	67.7%	65.4%	61.4%	5.01
Urban	69.9%	52.2%	49.6%	56.8%	-2.83
Purchases of durables, services (Tsh - 12 mo)	32,362.47	21,341.32	25,126.56	25,626.35	-4.19
Head of household farmer	40.6%	56.8%	62.3%	55.8%	0.62
Main source of cash income					
Harvest crops	31.9%	53.1%	56.3%	43.8%	5.71
Business income	23.8%	17.8%	16.6%	15.8%	1.60
Wage income	21.5%	14.8%	12.1%	14.4%	0.35
Safe Water	73.15%	79.58%	73.63%	67.62%	8.52
Drinking water source					
Private Indoor	12.7%	7.4%	4.9%	4.5%	3.78
Private Outdoor	13.4%	11.3%	6.1%	9.4%	2.01
Community/Neighbor	31.7%	38.1%	33.6%	41.5%	-2.07
Private/Public Well	27.4%	30.3%	34.8%	25.5%	3.32
Hunger (self-reported)					
Never	33.3%	44.5%	43.9%	34.6%	6.26
Seldom	42.8%	39.1%	36.1%	42.9%	-2.37
Sometimes	7.2%	5.5%	7.8%	4.8%	0.99
Often	15.7%	10.3%	11.5%	16.7%	-5.87
Meals per day	2.74 (0.48)	2.51 (0.53)	2.49 (0.52)	2.48 (0.55)	1.45
Fish per week	2.27 (1.80)	1.87 (1.59)	1.59 (1.45)	1.79 (1.84)	1.48
Toilette facilities					
Flush toilette	9.1%	4.4%	2.8%	2.0%	3.97
Pit Latrine	84.2%	92.1%	90.8%	74.4%	2.03
Illness in previous month					
Fever/Malaria	66.2%	60.4%	63.4%	67.6%	-5.42
Diarrhea	10.0%	11.0%	12.0%	12.2%	-1.39
Ear/Nose/Throat	7.1%	9.0%	8.7%	8.2%	1.08
Dirt floor	53.3%	65.5%	67.6%	68.0%	-1.81
Mud or grass roof	36.7%	53.4%	46.2%	40.8%	8.87
Metal roof	60.3%	45.9%	53.6%	58.6%	-8.94
Distance to nearest Health Center (km)	2.29	2.78	2.33	2.59	1.39
Distance to nearest Hospital (km)	10.19	20.18	12.94	22.88	-2.73
Distance to nearest Primary School (km)	1.07	1.06	1.30	1.48	-5.76
Distance to nearest Secondary School (km)	1.63	2.59	3.41	3.88	-2.79
<i>Observations</i>	<i>17067</i>	<i>2152</i>	<i>819</i>	<i>1711</i>	

Source: 2000 Tanzanian Household Budget Survey (THBS). IOC Program refers to government-sponsored iodized oil capsule distribution that was initiated between 1985 and 1995 in 27 districts of the country.

Table 3: Grade Attainment and IOC Supplementation in Utero

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)
				<i>Binary treatment indicator</i>											
	All	Girls	Boys	All	Girls	Boys	All	Girls	Boys	All	Girls	Boys	All	Girls	Boys
Pr(IOC in utero)	0.347	0.594	0.190	0.246	0.429	0.134									
	[0.148]*	[0.170]**	[0.160]	[0.114]*	[0.135]**	[0.136]									
Pr(IOC in utero)							0.559	0.824	0.384	0.632	1.611	1.045	0.554	1.200	0.008
* District coverage rate							[0.197]**	[0.262]**	[0.240]	[0.283]*	[0.461]**	[0.548]+	[0.327]+	[0.472]*	[0.484]
Pr(IOC in utero) _{3st<5}	0.033	0.208	-0.095	0.106	0.223	-0.017	0.009	0.196	-0.117	-0.149	0.795	-0.262	0.017	0.017	0.020
	[0.159]	[0.296]	[0.210]	[0.122]	[0.199]	[0.147]	[0.151]	[0.296]	[0.208]	[0.238]	[0.540]	[0.360]	[0.011]	[0.015]	[0.018]
Pr(IOC in utero) _{3st<5}	-0.055	-0.283	0.080	-0.056	-0.313	0.121	-0.075	-0.339	0.075	0.286	-0.790	0.168	-0.031	-0.018	-0.051
* Young mom	[0.161]	[0.354]	[0.200]	[0.081]	[0.180]+	[0.112]	[0.163]	[0.364]	[0.201]	[0.308]	[0.549]	[0.492]	[0.013]*	[0.015]	[0.022]*
Age 11	0.377	0.310	0.360	0.437	0.362	0.412	0.381	0.326	0.356	0.546	1.056	0.240	0.721	0.768	0.661
	[0.115]**	[0.137]*	[0.132]**	[0.126]**	[0.154]*	[0.147]**	[0.111]**	[0.134]*	[0.130]**	[0.192]**	[0.320]**	[0.485]	[0.117]**	[0.163]**	[0.187]**
Age 12	1.129	1.113	1.115	1.187	1.146	1.170	1.141	1.130	1.121	1.306	1.707	0.974	0.964	1.147	0.741
	[0.125]**	[0.162]**	[0.137]**	[0.130]**	[0.176]**	[0.154]**	[0.121]**	[0.157]**	[0.136]**	[0.252]**	[0.347]**	[0.439]*	[0.153]**	[0.198]**	[0.252]**
Age 13	1.914	2.062	1.735	1.958	2.079	1.778	1.936	2.070	1.761	1.959	2.975	1.604	1.566	1.830	1.340
	[0.143]**	[0.172]**	[0.160]**	[0.148]**	[0.193]**	[0.191]**	[0.132]**	[0.165]**	[0.158]**	[0.245]**	[0.367]**	[0.552]**	[0.218]**	[0.294]**	[0.339]**
<i>Fixed effects</i>	<i>District</i>	<i>District</i>	<i>District</i>	<i>District</i>	<i>District</i>	<i>District</i>	<i>District</i>	<i>District</i>	<i>District</i>	<i>Household</i>	<i>Household</i>	<i>Household</i>	<i>District</i>	<i>District</i>	<i>District</i>
<i>Data source</i>	<i>2000 THBS</i>	<i>2000 THBS</i>	<i>2000 THBS</i>	<i>2000 THBS</i>	<i>2000 THBS</i>	<i>2000 THBS</i>	<i>2000 THBS</i>	<i>2000 THBS</i>	<i>2000 THBS</i>	<i>2000 THBS</i>	<i>2000 THBS</i>	<i>2000 THBS</i>	<i>2004 DHS</i>	<i>2004 DHS</i>	<i>2004 DHS</i>
<i>Observations</i>	<i>1395</i>	<i>678</i>	<i>717</i>	<i>1395</i>	<i>678</i>	<i>717</i>	<i>1395</i>	<i>678</i>	<i>717</i>	<i>690</i>	<i>192</i>	<i>208</i>	<i>683</i>	<i>356</i>	<i>327</i>

Notes: Data in columns 1-12 from the 2000 Tanzanian Household Budget Survey, data in columns 13-15 from the 2004 Tanzanian Household Budget Survey. Sample restricted to children ages 10-13 in 25 districts targeted for iodized oil capsule (IOC) distribution between 1986 and 1992. All estimates exclude children that cannot be matched to mothers in the household. Outcome is highest grade completed. *Pr(IOC in utero)* is the probability that IOC was distributed in the district before or during the first trimester of pregnancy times the likelihood that sufficient iodine stores remain in the mother's body to protect the fetus during month 1 of pregnancy. Precise values are given in Appendix A. *Pr(IOC in utero)_{3st<5}* is the same probability for children born 3-4 years after IOC was distributed in the district, during which time iodine is being depleted from the body at an unobservable rate, and equal to 0 otherwise. *Young mom* is an indicator of whether mother was under 23 years of age at the time of IOC distribution, in which case she might have received 200mg rather than 380mg of iodine and therefore experienced faster depletion 3-4 years after the program.

All regressions control for binary indicators of sex-specific birth order, gender, and whether mother under 23 at birth. All district FE regressions control for urbanicity, household number of children in sample and number of boys in sample, whether household reported ever experiencing food shortages, and whether roof made of grass. Column 1-9 regressions also control for home ownership, distance to the nearest secondary school, distance to the nearest health clinic, month of survey, and education of the head and spouse. Column 13-15 regressions also control for distance to the nearest market, whether electric connection, whether piped water, whether bank account, and month of birth. + significant at 10%; * significant at 5%; ** significant at 1%

Table 4: Variation in Program Effect by Underlying Need

	(1)	(2)	(3)	(4)	(5)	(6)
	All	Girls	Boys	All	Girls	Boys
Pr(IOC in utero)	-0.115	0.113	-0.081	0.311	0.497	0.199
	[0.303]	[0.331]	[0.363]	[0.151]*	[0.179]**	[0.175]
Pr(IOC in utero)	0.009	0.010	0.004			
* TGR 1980	[0.005]*	[0.005]*	[0.006]			
Pr(IOC in utero)				0.217	0.540	-0.057
* Food insecure				[0.203]	[0.296]+	[0.264]
Age 11	0.314	0.225	0.353	0.375	0.293	0.375
	[0.101]**	[0.138]	[0.128]**	[0.103]**	[0.121]*	[0.124]**
Age 12	1.224	1.113	1.270	1.143	1.095	1.134
	[0.108]**	[0.147]**	[0.119]**	[0.100]**	[0.128]**	[0.118]**
Age 13	1.973	2.068	1.799	1.920	2.045	1.753
	[0.121]**	[0.163]**	[0.150]**	[0.121]**	[0.145]**	[0.142]**
<i>Fixed effects</i>	<i>District</i>	<i>District</i>	<i>District</i>	<i>District</i>	<i>District</i>	<i>District</i>
	<i>2000</i>	<i>2000</i>	<i>2000</i>	<i>2000</i>	<i>2000</i>	<i>2000</i>
<i>Data source</i>	<i>THBS</i>	<i>THBS</i>	<i>THBS</i>	<i>THBS</i>	<i>THBS</i>	<i>THBS</i>
<i>Observations</i>	<i>1193</i>	<i>574</i>	<i>619</i>	<i>1395</i>	<i>678</i>	<i>717</i>

Notes: Data from the 2004 Tanzanian Household Budget Survey, sample restricted to children ages 10-13 that reside in the household and have non-missing data. "TGR 1980" is district average total goitre rate measured in the 1980s, available from the WHO for 21 out of 25 districts in the sample. "Food insecure" is a dummy variable equal to one if the household reports having trouble satisfying food needs "sometimes", "often" or "always". For a description of other variables and list of control variables, see notes to Table 3. + significant at 10%; * significant at 5%; ** significant at 1%

Table 5: Robustness Checks of Grade Attainment and IOC Supplementation in Utero

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	Full sample, including children with unmatched mothers			Restricted control group			Restricted treatment group		
	All	Girls	Boys	All	Girls	Boys	All	Girls	Boys
Pr(IOC in utero)	0.398 [0.132]**	0.657 [0.188]**	0.177 [0.139]	0.338 [0.133]*	0.612 [0.194]**	0.240 [0.150]	0.574 [0.145]**	0.767 [0.198]**	0.408 [0.179]*
Pr(IOC in utero) _{3≤t<5}				-0.266 [0.172]	-0.197 [0.302]	-0.351 [0.214]	-0.205 [0.203]	0.193 [0.336]	-0.380 [0.293]
Pr(IOC in utero) _{3≤t<5} * Young mom				-0.073 [0.150]	-0.226 [0.325]	0.054 [0.210]	0.013 [0.236]	-0.342 [0.400]	0.140 [0.357]
Age 11	0.419 [0.088]**	0.355 [0.118]**	0.421 [0.103]**	0.217 [0.125]+	0.040 [0.148]	0.205 [0.156]	0.278 [0.119]*	0.274 [0.147]+	0.239 [0.132]+
Age 12	1.169 [0.088]**	1.083 [0.112]**	1.253 [0.099]**	0.818 [0.160]**	0.786 [0.196]**	0.714 [0.193]**	1.125 [0.147]**	1.176 [0.177]**	1.116 [0.164]**
Age 13	1.995 [0.097]**	2.082 [0.136]**	1.862 [0.101]**	1.427 [0.182]**	1.595 [0.271]**	1.274 [0.164]**	1.948 [0.156]**	2.155 [0.184]**	1.730 [0.191]**
Fixed effects	District	District	District	District	District	District	District	District	District
Observations	1663	812	851	921	436	485	1160	555	605

Notes: Data from the 2000 Tanzanian Household Budget Survey, sample restricted to children ages 10-13 in 25 districts targeted for iodized oil capsule (IOC) distribution between 1986 and 1992. Full sample (columns 1-4) includes all children in the analysis sample, including those that could not be matched to mothers in the household; restricted control group sample (columns 5-8) excludes children born more than one year before a treatment year who are not also born 1-3 years after a treatment year; restricted treatment group sample (columns 9-12) excludes children born the year after an intervention year. All estimates exclude children that cannot be matched to mothers in the household. Outcome is highest grade completed. *Pr(IOC in utero)* is the probability that IOC was distributed in the district before or during the first trimester of pregnancy times the likelihood that sufficient iodine stores remain in the mother's body to protect the fetus during month 1 of pregnancy. Precise values are given in Appendix A. See notes to Table 3 for a full list of the control variables. Columns 1-3 estimates exclude control variable for young mother. All r

Table 6: Effect of IOC on Reported Health Status at Ages 10-13

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	<i>Whether any sickness last 4 weeks</i>	<i>Whether fever</i>	<i>Whether diarrhea</i>	<i>Whether ear/nose/throat condition</i>	<i>Whether eye condition</i>	<i>Whether skin condition</i>	<i>Whether dental condition</i>	<i>Whether accident-related condition</i>	<i>Whether other health problem</i>	<i>Days school/work missed due to illness</i>
Pr(IOC in utero)	-0.025 [0.044]	0.009 [0.035]	-0.014 [0.007]+	0.012 [0.016]	-0.007 [0.006]	-0.012 [0.009]	0.002 [0.009]	0.002 [0.006]	-0.025 [0.019]	-0.045 [0.036]
Age 11	-0.045 [0.025]+	0.002 [0.026]	0.006 [0.009]	-0.005 [0.011]	-0.007 [0.005]	-0.014 [0.007]*	-0.003 [0.003]	-0.002 [0.004]	-0.016 [0.009]+	-0.009 [0.029]
Age 12	-0.012 [0.027]	-0.026 [0.025]	-0.001 [0.007]	-0.016 [0.012]	0.001 [0.005]	-0.004 [0.008]	0.004 [0.006]	0.008 [0.006]	0.003 [0.012]	0.057 [0.033]+
Age 13	-0.010 [0.026]	-0.004 [0.026]	0.004 [0.009]	-0.009 [0.016]	-0.006 [0.005]	-0.013 [0.008]+	0.010 [0.005]+	0.003 [0.006]	0.006 [0.011]	0.008 [0.032]
<i>Fixed effects</i>	<i>District</i>	<i>District</i>	<i>District</i>	<i>District</i>	<i>District</i>	<i>District</i>	<i>District</i>	<i>District</i>	<i>District</i>	<i>District</i>
<i>Observations</i>	1395	1395	1395	1395	1395	1395	1395	1395	1395	1395

Notes: Outcome is whether child reported by respondent to have experienced any of above health conditions during last four weeks; last column is amount of absence from school or work due to illness during past four weeks, a four category variable indicating: none, 0-1 week, 1-2 weeks, and 2-4 weeks. All data from the 2000 Tanzanian Household Budget Survey, sample restricted to children ages 10-13 in 1988 in 25 districts targeted for iodized oil capsule (IOC) distribution between 1986 and 1992. In all regressions, *Pr(IOC in utero)* is the probability that IOC was distributed in the district before or during the first trimester of pregnancy times the likelihood that sufficient iodine stores remain in the mother's body to protect the fetus during month 1 of pregnancy. Precise values are given in Appendix A. For a description of other variables and list of control variables, see notes to Table 3.+ significant at 10%; * significant at 5%; ** significant at 1%