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The Cognitive Link Between Geography and Development: Iodine Deficiency and Schooling Attainment in Tanzania

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ABSTRACT

An estimated 20 million children born each year are at risk of brain damage from in utero iodine deficiency, the only micronutrient deficiency known to have significant, non-reversible effects on cognitive development. Cognitive damage from iodine deficiency disorders (IDD) has potentially important implications for economic growth through its effect on human capital attainment. To gauge the magnitude of this influence, we evaluate the impact of reductions in fetal IDD on child schooling attainment that resulted from an intensive distribution of iodized oil capsules (IOC) in Tanzania. We look for evidence of improvements in cognitive ability attributable to the intervention by assessing whether children who benefited from IOC in utero exhibit higher rates of grade progression at ages 10 to 14 relative to siblings and older and younger children in the district who did not. Our findings suggest that reducing fetal IDD has significant benefits for child cognition: Protection from IDD in utero is associated with 0.36 years of additional schooling. Furthermore, the effect appears to be substantially larger for girls, consistent with new evidence from laboratory studies indicating greater cognitive sensitivity of the female fetus to maternal thyroid deprivation. There is no indication that IOC improved rates of illness or school absence due to illness, suggesting that IOC improves schooling through its effect on cognition rather than its effect on health. However, there is weak evidence that the program also reduced child but not fetal or infant mortality, which may bias downward the estimated effect on education. Cross-country regression estimates corroborate the results from Tanzania, indicating a strong negative influence of total goiter rate and strong positive influence of salt iodization on female school participation. Together, these findings provide micro-level evidence of the direct influence of ecological conditions on economic development and suggest a potentially important role of variation in rates of learning disability in explaining cross-country growth patterns and gender differences in schooling attainment.

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1 Introduction

There is an unresolved debate in the economic growth and development literature regarding the role of geographic variation in health environment on long-run economic outcomes. 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, 1997; Sachs and Gallup, 1998, 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).

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 household and district fixed effects models that compare children likely to benefit from the

[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.

program in utero based on month and year of birth to slightly 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 literature on barriers to educational attainment in developing countries 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 differences between males and females 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 (USI) 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 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.

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

[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; Husain et al, 1981; Sommer et al., 1986, 1981; West et al., 1995; Glasizou et al, 1993; Beaton et al, 1992).

from fetal IDD.[3] 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.

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 0.36 – 0.51 years of education above siblings and older and younger children in their district who were not. 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. In addition, the observed variation in estimated effects matches predictions regarding the relative vulnerability of specific subpopulations to fetal IDD based on local diet. Finally, our results indicate that the program reduced child but not fetal or infant mortality. Since there is no evidence of gender differences in the program effect on survival or of program effects on the average health status of survivors, this influence is unlikely to account for the schooling results and could bias downward the estimated effect on education.

To examine the implications of our findings at a macro level, we estimate cross-country regressions of school participation on baseline IDD and fraction of population consuming adequately iodized salt, two measures that have not been considered previously in the empirical growth literature. The results reveal a negative correlation between baseline iodine deficiency and female secondary schooling, and indicate that early salt iodization has already had a positive effect on female primary schooling attainment. Based on our micro-level estimates from Tanzania, we calculate that the average increase in schooling attainment in Central and Southern Africa attributable to USI could ultimately be as large as 7.2% of baseline average schooling levels.

A remaining policy question is the value of devoting further resources necessary to fully eradicate IDD. Although USI is arguably the most successful micronutrient intervention in world history, legal regulations on salt production are rarely sufficient to guarantee dietary change among rural populations that consume mainly subsistence food products. In several African countries less than 30% of households

[3] Estimate based on 1988-1994 population and crude birth rate.

presently consume iodized salt despite universal legislation, and even iodized salt may be insufficient to reduce IDD in populations whose diets contain sufficient amounts of iodine-depleting foods (UNICEF, 2005). With respect to the estimated 10% of the population that remains at risk in spite of salt iodization legislation and the more than 40 countries that have yet to undertake any control program, the magnitudes of our estimated returns to eliminating IDD well justify more costly approaches.[4]

2 Background

2.1 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. For this reason, Precambrian rock shields are associated with particularly low concentrations of iodine. 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).[5] In contrast, morbidity from child and adult thyroid fluctuations is relatively low. 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.[6] 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.[7] In general, evidence from human studies suggests that damage from fetal IDD is overwhelmingly cognitive and that physical birth defects 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

[4] Hetzel (2000) estimates that less than half the population in 83 developing countries consumed adequately iodized salt in the mid-1990s.
[5] Recommended daily iodine intake is 50 μg for infants under 12 months, 150 μg for adults and 200 μg for pregnant and lactating women (WHO, 1996). Epidemiological criteria for sufficient iodine intake is 100 μg or above.
[6] 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 et al., 1982)
[7] See Allen and Gillespie (2001) for a review of the evidence.

most affected by iodine deficiency in infancy (Cao et al., 1994a; Zaleha et al., 2000).

Although iodine deficiency has been associated with goiter and cretinism for centuries, only during the past decade have IDD been widely recognized as a leading cause of intellectual impairment (Merke, 1984; Delange, 2000; Haddow, 1999).[8] Furthermore, 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 indicates a continuous process by which fetal brain development is sensitive to minor adjustments in thyroid hormone (Lavado-Autric R, 2003; Sundqvist et al, 1998; Dugbarty, 1998; Pop et al, 1999). As a result, even mild maternal iodine deficiency is now hypothesized to reduce intelligence quotients by a noticeable margin.

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.

2.2 Gender Differences in Iodine Deficiency

Evidence from multiple sources indicates gender differences in the importance of iodine for brain development. Notably, in both 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 numbers of subjects and lack of statistical significance (Bautista et al., 1982; Shrethsa, 1994). Similarly, practitioners have noted that adolescent IDD, including rates of goiter and average severity among sufferers, is systematically higher among females (Allen et al, 2001; Simon, 1990). A central limitation of observational studies is their inability to attribute gender differences in IDD to physiological sex differences in iodine sensitivity as opposed to sex-specific dietary patterns.

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 undertaken, consistent with the general absence of research into the role of maternal biochemicals on sex differences in fetal neurodevelopment (Friedhoff et al., 2000). However, two studies lend strong support to the hypothesis of sex-specific sensitivity to iodine deficiency in utero. First, Friedhoff et al. found that the effect of

[8] The World Health Organization labeled IDD "the most common cause of preventable mental retardation (WHO, 1992)."

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. 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.

3 Setting

3.1 Iodine deficiency in Tanzania

Our study examines the long-run impact of an iodized oil supplementation program in Tanzania. Tanzania, like many countries on the African continent, traditionally suffered high rates of IDD. According to a nationwide survey of iodine levels in the early 1970s, about 40% of the Tanzanian population, or 10 million people, lived in iodine-deficient areas and 25% of the population 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., 1998).

3.2 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. 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.

Throughout the country, primary schooling is characterized by high variation in age of entry, high rates of grade retention and intermittent enrollment. Particularly in rural areas, gross enrollment ratios are

substantially higher than net ratios because many over-age children are present in primary schools due to beginning schooling late and progressing slowly. Although teachers have some room to retain students for attendance and behavioral problems, the main reason for repetition in primary school is exam failure, and repetition rates are highest in grades at which students take national standardized tests and in grade 1. 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 and girls perform significantly worse than boys (United Republic of Tanzania, MoEC, 2003). In 2002/2003 over 20% of students repeated the Standard 4 exam (United Republic of Tanzania, MoEC, 2003b). Pass rates for the Standard 7 Primary School Leaving Exam (PSLE), which is required for admittance to any lower secondary school in the country, are even worse: Despite the government's ongoing goal of increasing the proportion of children passing to 50%, in 1997 only 20.1% of pupils who sat for the PSLE passed the examination, which has since increased to 22.0% in 2000 and 28.6% in 2001.

Despite the fact that there is no national examination in the first year of school, Standard 1 has the highest repetition rate of all grades (12.3% in 2000). The large fraction of children that fails to pass school-specific Standard 1 assessments is attributed to repetition-related overcrowding in grade 1, and high variation in preparedness at school entrance on account of differences in age of entrance and access to preschool education (World Education Forum, 2000). Retention is lower in grades 2 and 3, but then jumps again in grade 4 when students take the Standard 4 exam.

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. In the standard primary age group of 7 to 13, boys have a slightly lower participation rate than girls because they start school slightly later, while the reverse is true for older children since girls drop out earlier. Not all of this is due to faster progression. Girls are less likely than boys to be in school beginning at 13, and the difference increases steadily thereafter as a disproportionate fraction of boys proceed to secondary school.

Gender differences in secondary enrollment are almost entirely accounted for by differences in PSLE pass rates. Alarmingly, 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. In total, only 21.4% of female and 36.2% of male test-takers passed the exam. By 2004, the pass rate had improved considerably, most likely due to a reported (though poorly documented) change in the grading policy: 43% of female and 57% of male test-takers passed the exam in 2004, reducing the gender difference considerably in terms of pass rates while maintaining roughly the same percentage point gender gap. 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.

3.3 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 iodized oil capsules (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] As shown in Figure 1, intervention districts were spread across ten regions of the country but concentrated geographically in the lake district of the western border, opposite the coast, which corresponds to a major geological shield and region of endemic IDD.

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%.[11]

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 among children who received supplements directly. A 1991 evaluation in three districts found that VGR had decreased by over 50% and TGR by over 25% (Peterson, 2000). Among

[9] Two districts, Bukoba Rural, Kagera, and Mbinga, Ruvuma, were added late and are excluded from the study.
[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 (Peterson, 2000). In people older than 45, iodized oil was not encouraged due to increased risks of hyperthyroidism (Dunn 1987b).
[11] 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 in unknown, and likely to be higher.

school children aged 7-18 in the district of Mahenge, TGR was 74.9% before IOC and 51.9% three years after (Kavishe, 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.

4 Empirical Analysis

4.1 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) and the 2004 Tanzanian Demographic and Health Survey (TDHS), to which we append the district-level information from Table 1 on timing 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 2004 TDHS covers a total of 4,987 households, 1,034 of which reside in intervention areas. Both surveys collect individual information on school enrollment and grade attainment of all household members in addition to a variety of community and family background characteristics. The THBS has particularly rich information on household consumption and production and childhood health status, while the TDHS focuses heavily on reproductive health histories, including fertility and infant health.

Although the TDHS sample is considerably smaller than the THBS, it has two principal advantages. First, information on month of birth allows us to construct a more precise indicator of IOC treatment, described in the proceeding section. Second, the data capture schooling outcomes for children born during a wider set of program years, allowing us to make use of greater variation in program activity within and across districts. In particular, as a result of the delayed start of the program in ten districts, only 17 districts contain program activity that affects children in the 2000 sample, while treated kids are found in all 25 districts in 2004.

4.2 Analysis Sample

For the empirical analysis, we restrict the analysis samples to all children between ages 10 and 13 in 2000 or between 10 and 14 in 2004 who are residing in the household and who could be linked to mothers in the household. In the THBS, 20.8% of children were dropped because they could not be matched to mothers based on age and relationship to household head, and in the TDHS, 20.1% of children are missing month of birth data.[12,13] Excluding non-resident children is necessary due to the fact that in

[12] Birth month is missing for 1,313 (~20%) children of the head between 9 and 17 in the THBS either because their mother did not live in the household or because she did not participate in the birth history survey.

both data sets schooling outcomes are only available for household members. Excluding observations that cannot be linked to mothers is necessary to minimize the number of children born outside the district given relatively high incidence of orphanhood in rural Tanzania. However, as a robustness check we run analogous regressions on the full sample.

The lower bound on age was based on the modal age of school enrollment, which peaks at 10 for both boys and girls. Across the entire rural THBS sample, only slightly over 50% of nine-year-olds were enrolled. Restricting the sample to ages by which most children have entered school maximizes the predictive power of our treatment variable, particularly if IDD reduces schooling attainment through grade retention as well as age of entry.[14] As a robustness check, we 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 kids 6-13.

The upper age limit in 2000 reflects the fact that oldest children in intervention districts affected by the program are 13 in 2000. In 2004, the upper age limit is driven by the fact that children in the sample leave their parents' household at high rates beginning at age 15, and reasons for leaving are likely to be systematically different for boys and girls and highly correlated with schooling attainment.[15] Because schooling data were collected only for children living in the household, and because district of birth is not available for children living outside of their birth household, it was necessary to restrict the sample to children under the age of 15 to avoid sample selection issues arising from age- and sex-specific attrition rates.

The primary THBS analysis sample contains 1785 children in 1352 households living in the 25 intervention districts that began IOC by 1992. Within-household estimates reduce this sample to 846 kids in the 413 households that have more than one family member aged 10-13. Sex-specific estimates, which

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[13] In the THBS sample, 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 kids 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 (no female head or no spouse in right age range), 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. In contrast, 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.

[14] 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.

[15] Comparing birth history data to the household roster for children of the respondent, 32% of 15-year-olds are no longer in the household. Furthermore, beginning at 15 boys are significantly more likely to have left the household than girls. At 15, they are 25% more likely and at 17 they are 64% more likely to have left. The most common reasons for leaving are likely to be marriage for girls and high school attendance for boys.

further restrict the analysis sample to households with more than one child of the same sex in this age distribution, are limited to 251 boys and 231 girls. Among children in our sample, 89% are enrolled in Standards I to VII (primary school) and 11% are not studying.[16]

The TDHS analysis sample contains 3672 children ages 10-14 in 2521 households across the country, 515 of which reside in intervention districts. Within-household estimates reduce this sample to 2160 kids in 1009 households with more than one family member aged 10 to 14, and sex-specific estimates reduce the sample to 643 boys and 534 girls. In the 2004 sample, 85.4% is enrolled in Standards 1 to 4, 4.3% is enrolled in Standards 5 to 7 and 9.76% are not studying. Since less than 5% of our 2004 analysis sample has entered high school (consistent with the modal age of high school entrance of 16), there is little ability to observe transitions to secondary school with available data.

4.3 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 380mg.[17] 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).[18] Furthermore, since brain development of the fetus takes place during the first month of pregnancy, it is believed that most of the consequences 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, 1992, 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 (≥6.5mg) 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

[16] Three 13-year-olds report enrollment in secondary school.
[17] 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).
[18] 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).

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 kids born 2-4 years after the program, requires an assumption regarding the depletion pattern of iodine from 380mg 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 323mg of iodine in the first month, after which point it is depleted hyperbolically.[19] 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 IDD remain in the body for 24 months (Eltom et al., 1985; Cao et al., 1994; Furnee, 1997). 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 an gradual and decreasing rate, which can be calculated based on our assumed depletion formula.[20] 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 (>4.2mg) at *t*=38. Figure 2 illustrates the pattern of iodine depletion implied by our assumptions, and exact probabilities for each birth month are described in Appendix A and presented in Figure 3. Importantly, the implied half-life is consistent with four studies of the approximate half-lives of urinary iodine excretion after oral oil administration to iodine-deficient human populations (Wolff, 2001).

For the analysis sample without month of birth data, 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:

Birth year - program year (x):	-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

[19] 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) for a review of the literature. Hyperbolic depletion implies a fast initial rate of depletion that slows quickly. In calculating iodine stores, we use the following simple hyperbolic discounting formula: $V = \frac{A}{1+kt}$, where k^{-1} is the half-life of iodine.

[20] Based on the recommended daily allowance for pregnant women (2 mg, 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).

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.[21] First, we introduce flexibility into the regression equation to account for 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 months (or years, when month is not available) after the point of full protection a child was born, relevant only for kids born 3-4 years after a distribution round. We also include a correction factor that accounts for the possibility that women under age 23 at the time of distribution 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. To check our assumption about the point of full protection, we also estimate the effect of IOC assuming 12 rather than 24 months of maximum coverage using an indicator of being born 1-2 years after the program. The results consistently indicate a significant effect of IOC that begins to decline after month 24, supporting the assumptions presented here.[22]

4.4 Estimation Strategy

We estimate the effect of IOC on child schooling in a regression analysis in which the primary outcome of interest is years of completed schooling. In Tanzania, as in many African countries, there is high variance in the rate at which children progress through primary school. Meanwhile, since few children drop out in the age range to which our analysis is restricted (primary school), progression is presumably a considerably more sensitive indicator of final schooling attainment than enrollment.

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, as was its intention. Relative to non-participating districts, IOC districts are more rural, have fewer households with private sources of drinking water and solid floors, have greater distance between households and secondary schools, and have lower consumption of fish, a rich source of iodine.

[21] 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 the type of iodized oil, goitre, intestinal parasites, sex, adipose tissue, cassava consumption and seasonality all influence the duration of effectiveness of IOC (Furnee, 1997).

[22] These results are available in a working paper posted on the author's website (Field, 2006).

Such differences clearly bias comparisons between participating and non-participating districts. 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 due to fever or malaria and average distance to school are significantly higher for districts in which the program started late. In contrast, the average annual consumption of durables is significantly higher in late districts, while the average number of meals and frequency of fish consumed are relatively constant across program start dates. Nonetheless, the general patterns suggest that the districts in which IOC began early were better off than late districts. Hence, program effect estimates based on comparisons across participating districts are also likely to be biased towards finding a program effect.

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

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

Here T_i is the continuous or binary likelihood child i in family f was protected from IDD during the first trimester described in the last section, A is a vector of birth year dummies, and X includes binary controls for gender and sex-specific birth order. The only difference in regression specification across the 2000 and 2004 analyses is 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 or progression. To examine whether the fetal effects of IDD are stronger for females, we also run the above regression separately by gender. The household fixed effects model minimizes the potential confounding role of unobservable cohort effects that might vary systematically with treatment and district and increases the precision of our estimates by holding family background constant. However, these estimates are necessarily restricted to the subset of households with more than one child in the relevant age range and the gender-specific regressions are run on the subset of families with at least two children of the same gender, which gives rise to potential selection issues relevant for comparison across estimates. To gauge the program effect among the larger and constant set of households, we also estimate the above regression replacing household with district fixed effects and clustering standard errors at the household level.

Since within districts and households treatment is determined entirely by age, in the above equation β_1 reflects the program effect averaged across all treated cohorts. As in all fixed effect estimates, identification of the causal effect of T requires that the error term be uncorrelated with treatment conditional on the observables contained in X and district or sibling average grade attainment (μ_f). If cohort differences in treatment are positively correlated with other trends that affect grade attainment, the estimates will overstate the true effect of iodine on schooling.

4.5 Program Effects According to Local Diet

Our analysis also makes use of anticipated variation in the impact of iodine supplementation based on district variation in baseline iodine deficiency. Given that the level of iodine provided through IOC supplements was uniform across districts, the relationship between baseline IDD rates and program impact is likely to be non-linear. In other words, we anticipate a threshold level of IDD below which rates are too low to observe a significant average treatment effect in the district population, and a second threshold above which 380 mg of iodine will be insufficient to protect against maternal iodine deficiency due to factors that raise daily requirements for iodine intake.

We test this prediction by examining variation in program effect by level of consumption of goitrogenous foods. Goitrogens – including cabbage, legumes, chaya leaves, and cassava – are foods that contain cyanogenic glycosides, which impede absorption of iodine by the thyroid gland (Bourdoux et al, 1978). Frequent consumption of such foods is one of the leading causes of IDD, and diets high in natural goitrogens can induce IDD even if the diet is rich in iodine (Gaitan 1990; Thilly 1992). Consistent with this, laboratory evidence suggests that goitrogens play a significant role in influencing biochemical events unique to the developing brain (Rao and Lakshmy, 1995).

Cassava is a staple in much of Africa and a large part of the diet in rural Tanzania. According to the 1991 THBS data, which contain detailed information on household food items consumed, cassava (either flour, dried or fresh) was the second most important food product after maize in terms of calories per day, and in 2000 was ranked third after maize and sorghum.[23] As one of the most goitrogenic food products, cassava has the potential to significantly decrease iodine absorption if not properly fermented.[24,25] Although the need for iodine increases with consumption of goitrogenous foods, so does the rate at which iodine – including that provided by the supplement – is depleted from the body by regular intake of goitrogens. Hence, in addition to serving as a central explanatory factor for baseline rates of IDD in the study population, the cyogenic effect of high cassava consumption is likely to have impaired the effect of IOC in certain districts.

The district rate of cassava *production* is used to proxy for variation in dietary intake 10 to 15 years prior. Geographic variation in local produce availability is likely to constitute a reasonable predictor of dietary differences a decade earlier, and is arguably a preferable proxy of past diet than is current diet

[23] For a description of these data, see Appendix 3. CALCULATING THE FOOD POVERTY LINE IN 2000/01 of the IFPRI document, "Analysis of the Tanzanian Household Budget Survey – Income poverty: Technical note on estimating poverty levels in Tanzania" prepared by Trudy Owens in March 2002.

[24] According to Hetzel (2000), "Although a number of other staple foods contain potential goitrogens, in contrast to cassava the goitrogens are in the inedible portions of the plants and do not contribute importantly to IDD."

[25] While the adverse effects of cassava can be countered with proper processing, there have been few efforts to train local communities in alternative processing methods and ignorance over the health effects of highly goitrogenous diets remained high in the 1990s (Bilabina et al, 1995; Delange et al, 1994). In one study in Tanzania, insufficient cassava processing was correlated with TGR (Peterson, 1994).

given recent changes in household diet. To construct this measure, a household was defined as a cassava producer if they reported consuming during the past month any fresh or dried cassava or cassava flour that was produced at home, and districts were classified according to the fraction of households in the district that reported consuming home-produced cassava. In the regressions districts are divided into terciles of cassava production. In high production regions, between 41 and 60% of households grow cassava, compared with between 11 and 40% in medium production regions and fewer than 10% of households in low productions districts.

5 Results

5.1 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 Tables 3-4. In household fixed-effects regressions from 2000 (Table 3), adequate maternal iodine in utero is associated with 0.358 years of additional schooling relative to siblings who are unprotected among children living in households with more than one child 10-13 (column 1). When the regressions are run separately by gender, the estimated effect is twice as large and statistically significant among girls but not boys in households with more than one sibling of the same sex aged 10-13. When the binary measure of program participation is used in place of the likelihood measure (columns 4-5), the results are almost identical in magnitude and retain significance. Furthermore, results are robust to the inclusion of the ~20% of children that could not be precisely matched to mothers (columns 6-7), suggesting that orphaned or fostered children are likely to live in districts where they were born.

In district fixed effects regressions that include all children 10-13 the estimated treatment effect is once again only significant for girls but considerably smaller than the household fixed effect result (columns 8-9). The difference between the two specifications appears to reflect greater vulnerability to IDD among households with many children close in age relative to smaller households, likely due to the correlation between family size and poverty. This is evident from 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 (columns 10-11).

The 2004 estimates of schooling attainment detailed in Table 4 are strikingly consistent with the 2000 estimates despite the fact that sample members were born in different years and enumeration areas from the THBS sample. Across the pooled sample of children with siblings close in age, the estimated program effect is slightly larger (0.52 years) but not statistically distinct from the Table 3 estimate, and gender-specific estimates are once again large and significant for girls but not boys. Once again the district fixed effect estimate on the full sample is half the size of the household fixed effect estimate (0.29

years), but in this case it retains significance at the 10% level. Since treatment status pertaining to kids 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 both sets of results, the coefficient estimates on the correction factors for rate of iodine depletion 3-4 years after the program are insignificant and close to zero in nearly all regressions, indicating that coverage falls at the assumed rate and that the majority of women received 380mg supplements regardless of age, as was claimed in program reports. The coefficient estimate on the indicator of young mother is only significant in the district fixed effect regression on the female sample, and the finding is not robust to the more precise measure of depletion in 2004 that makes use of birth month. The same regression yields the only high point estimate on the rate of iodine depletion in years 3 and 4. Since the average effect during years 3-4 is the weighted sum of these two coefficient estimates – which is close to zero – there is consistent evidence across all regression estimates that the depletion assumptions during years 3 and 4 are correct. More general robustness checks verify that the program effect is robust to excluding treatment interactions (Table 6, columns 1-4), and to including children as young as 6 (Appendix B, columns 1-6).

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. As described in Section 3.2, 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 (Appendix B, columns 7-10). These results provide 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 (Appendix C) provide stronger 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 3.2, 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 kids, unprotected kids 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 completed schooling operates exclusively through earlier entrance among the treated.

The measured effects underestimate the cognitive impact of IDD to the extent that not all pregnant women in a district were reached by the program. Data on program coverage rates by district (Table 1) indicate that 68% of the target population was reached in program areas between 1986 and 1990 and 65% between 1986 and 2004.[26] 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), this rate is likely to reflect true rate of treatment exposure. If the rate applies equally to pregnant women, the estimates imply an average effect of IOC of 0.52-0.76 years. However, for three reasons it may be inappropriate to inflate the baseline estimate by average coverage. First, women of childbearing age were reportedly first in the priority list for receiving IOC, so are likely to have been targeted more aggressively. Second, coverage is likely to be higher than average among pregnant women since they are more likely than men to visit village health centers where IOC were frequently distributed. Third, an evaluation of program implementation suggested that coverage rates were higher in areas with higher incidence of goiter, which also implies that *effective* coverage (coverage of those in need) was over 68% (Peterson, 2000).

One of the most striking patterns in both sets of results is the consistently higher estimated program effect on girls. In both within-sibling and within-district estimates, girls appear to benefit twice as much as boys from IOC in utero, although the difference is only significant in the district-level fixed effects regressions. In the household fixed effect model, girls gain an estimated 0.81-0.90 years of school with in utero IDD protection, which is statistically significant throughout, while boys gain an estimated 0.29-0.43 years that is not statistically distinguishable from zero. Including households with only one girl 10-13, the estimated effect of IOC is 0.36-0.42 years and again close to zero and insignificant for boys. Inflating by average coverage, this implies an effect of IOC on girls' schooling of 1.2 years.

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.

[26] Rate calculated by multiplying a district's coverage rate by district's fraction of children in our sample.

In the 2004 TDHS estimates we gauge the nature of the bias by including in the regression the interaction between program year and age to determine whether the program effect widens or narrows over time (columns 7-12). In this specification, the baseline program effects are larger and more precise in both district and household fixed effects models, while the coefficient estimate on the interaction term between age of child and IOC is strongly *negative*, suggesting that the results overestimate the effect of IOC on final schooling attainment. Furthermore, the gender difference is particularly stark – and statistically significant – when the program effect is allowed to vary by age. This indicates possible "catch-up" on the part of slower girls as those who progressed rapidly through primary school begin to drop out at the point of secondary school transition. The pattern is illustrated graphically in Figure 4, which plots average grade progression by gender and the binary indicator of treatment status. Although between ages 10 and 12 boys in program districts who received IOC have significantly lower schooling attainment than boys who did not benefit from the program, there is no significant difference in girls' rate of progression through school according to in utero IOC at the same ages. However, at ages 13 and 14, girls' schooling in program cohorts and districts falls below that of untreated girls, possibly indicating drop out at primary school completion beginning at 13.

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, a pattern observed in the 1988 Census data as well as the 2000 THBS and 2004 TDHS data. Hence, by a given age they have already reached a higher grade than their male peers. If the importance of cognitive ability on school pass rates increases with grade, as is likely to be the case, girls between 10 and 13 will benefit more from the intervention simply because they are more likely to be on the margin of influence. Although the two trends are impossible to separate without information on age of entry (unavailable from these sources), 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.

The third possible reason 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 simple way to distinguish the last story from a disproportionate improvement in female cognitive capacity. Section 6 takes a further step in this direction by utilizing information on the rates at which students pass the Primary School Leaving Examination (PSLE).

The final regression exercise divides districts in the sample according to cassava production in order to test predictions regarding heterogeneity in program effect according to local diet. Estimates in Table 5 reveal an inverted u-shaped relationship between amount of goitrogens consumed and impact of IOC on fetal development, consistent with our predictions. In areas with highly goitrogenous diets the program appears to have had little effect, suggesting that maternal iodine levels were depleted by intake of cassava. Meanwhile, in areas with relatively little cassava in the diet, the program effect is also small, which presumably reflects the fact that these were districts with the lowest TGR at the start of the program. Figure 5 splits the sample even further into five categories of cassava production and plots coefficient estimates from regressions on the separate sub-samples. These results indicate that only the extreme outliers were unaffected by the intervention. Most striking is the indication that the areas with the highest TGR appear to receive no benefit from even a program as intensive as IOC.

5.2 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 kids of the same age and district in 2000. In the results presented in Appendix D, all household fixed effect regressions yield small and insignificant estimates of the effect of program participation on 1988 schooling. In the district fixed effects estimates, due to very large samples estimated treatment effects achieve statistical significance but the point estimates are close to zero and of the opposite sign as the previous results, so cannot explain the treatment effects observed in 2000 and 2004.

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.[27] 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 6 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 (Table 7). Furthermore, the regression results indicate no measurable difference in sibling attainment gaps when both siblings are treated versus when neither is treated.

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 who were in utero or recently born at the time of the program (Table 6, columns 5-8). 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 fetal development but not late fetal 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 kids born one year after the program (Table 6, columns 9-12). 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 24-36 months.

5.3 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

[27] The asymmetry reflects the fact that most variation in program activity arises from delays rather than gaps.

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, 2002).[28] 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 kids born just after iodine depletion, contradicting the evidence in Table 7. For instance, Vitamin E supplements or nutritional supplements given to women at the same time as IOC would only 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 6) 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

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

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.[29] 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 Section 5.3 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 whether IOC operates through reducing childhood illness rather than improving cognition.

5.4 Health Effects of IOC

The patterns of results observed in Tables 3-7 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.[30] Although iodine deficiency has been demonstrated in laboratory studies to influence fetal brain

[29] 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.

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

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 health data on children in utero during the program from the 2000 and 2004 surveys. The TDHS provides information on age of death of all children born during the program years as part of each sample members' reproductive history. Using these data, we construct a new sample composed of all births to mothers of children in our sample between 1990 and 1995, and regress binary indicators of neonatal (less than 30 days), infant (30 days to 1 year), and child (1 to 15 years) mortality on the same program indicator of IDD protection in trimester 1.[31] In addition, we approximate the influence of IOC on conception and early fetal mortality by estimating the program effect on births. To do so, we construct a panel dataset in which an observation is a person-month between 1990 and 1995 and the outcome of interest is whether a woman gave birth, regressed on the probability of IDD protection 7-9 months earlier (specific to women in a given district and month).

Coefficient estimates on the variable of interest for each regression are presented in Table 8, beginning with the fertility estimates. The results indicate no effect of maternal iodine in early pregnancy on fecundity (births), fetal death rates, or infant mortality rates (columns 1-9). However, there is some indication that the program influenced child mortality after age 1 (columns 10-12). Regression results from the pooled sample imply that IOC is associated with a 10 percentage point reduction in the probability of death between ages of 1 and 15, which is independent of gender. This is strikingly high given baseline child mortality of 8.9% in treatment districts. The finding that IOC influenced mortality at older but not younger ages in inconsistent with scientific evidence on the influence of maternal thyroid hormone on offspring health in which health effects are found to fall with age. A possible explanation is that differential mortality of children reflects differences in child health inputs according to underlying cognitive status, although this is impossible to determine without more detailed information on cause of death and childhood health inputs. However, it is worth noting that observed health effects do not necessarily reflect a pure physiological effect of IOC on survival. The magnitude of the estimated effect and the fact that previous studies have only found reductions in child mortality in combination with larger improvements in neo- and peri-natal mortality suggest that this finding should be interpreted with caution.

The child mortality result not only provides new evidence of the benefits of IOC, but also has relevance for the findings on grade attainment. There are three implications worth noting. First, the result

[31] We define child mortality to include deaths occurring up to the highest age of sample members (14) rather than the standard measure of deaths under age 5 in order to gauge the program effect on attrition at any time prior to the survey. We use 30 days rather than the standard 28 to define infant mortality in order to account for clumping around one month intervals.

indicates that the education estimates may be contaminated by higher survival among children who received IOC in utero. However, even if IOC reduced mortality, it is unlikely that higher child survival would *by itself* give rise to higher grade attainment at ages 10-13 given that child mortality presumably selects out the most vulnerable. As a result of survivor bias alone, the average child who benefited from IOC in utero is likely to have lower anticipated schooling attainment in the absence of the program, biasing our estimates downward rather than upward. Furthermore there is no indication of gender differences in the effect of IOC on survival at any age, so survivor bias cannot account for these results.

However, while differences in rates of child mortality associated with IOC are not likely to bias our estimates towards finding an effect, it is possible that treatment has an inframarginal effect on health status, i.e. lower mortality among treated children corresponds to higher average health status among survivors. In this case, the program effect may operate through differences in health status rather than cognition, and could also account for the gender difference if girls' schooling is more sensitive to health status. To study this more closely, Table 9 presents results from regressions analogous to the Table 3 estimates of contemporaneous child health status and school absence due to illness on program participation. 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 due to any sickness or injury.

Regression estimates 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. This set of findings suggests that the measured program effect of IOC operates through cognitive rather than physical health improvements.

Similarly, results from tables 8 and 9 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 3.4, 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 the mortality results in Table 8 indicate a health effect only several years after the program, 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 9 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.

6 PSLE Test Scores

In order to better connect our results on schooling attainment to improvements in cognitive ability, we make use of district-level aggregate data on 2004 Primary School Leaving Examination (PSLE) pass rates by gender, available from the Ministry of Education for 93 of the 106 districts in the country.[32] PSLE data are available on the number of boys and girls in each district who take the test in 2004, and the number of boys and girls who receive each of five categories of test grade, three of which constitute passing grades. Test-takers in 2004 are likely to fall between the ages of 14 and 18, so correspond almost perfectly to the cohort of children most affected by IOC distribution, or kids aged 10-14 in the 2000 THBS data.

Our empirical analysis tests whether secondary school transition rates are higher in IOC districts conditional on district secondary school enrollment reported in the 1988 population census, and whether transition rates improved disproportionately for girls.[33] We examine the fraction of students who take the PSLE, and the fraction of test-takers who pass and therefore transition to secondary school with the following set of regressions, run separately by gender:

$$\ln(testtakers04_d) = \alpha + \beta_1 (T_d) + \beta_2 (\ln(pop04_d)) + \beta_3 (hsrate88_d) + \beta_4 (X_d) + \varepsilon_d \qquad (2)$$

$$\ln(testtpassers04_d) = \alpha + \beta_1 (T_d) + \beta_2 (\ln(testtakers04_d)) + \beta_3 (hsrate88_d) + \beta_4 (X_d) + \varepsilon_d \qquad (3)$$

In both estimates, an observation is a district. The first outcome, *testtakers04,* is the number of male or female individuals in district *d* who take the PSLE in 2004, *T* is whether there was IOC distribution in district *d* between 1986 and 1992, *pop04* is the number of males or females in district *d* between the ages of 10 and 14 in the 2002 Census (therefore the population of high-school age in 2004),

[32] PSLE data are missing for the islands of Pemba and Zanzibar, and the mainland region of Iringa, comprising 6 districts. No explanation is available from the Ministry of Education for the absence of data from these regions.

[33] Since 2002 Census data is only available in five-year age groups, the transition rate is approximated by dividing the number of boys and girls passing the exam in 2004 by the number of boys and girls between the ages of 10 and 14 in 2002, or 12 to 16 in 2004. This is slightly younger than the average age of test-takers, which is unavailable in 2004, but the population figures are unlikely to differ across cohorts close in age.

and *hsrate88* is the fraction of females or males age 21-25 in district *d* who were ever enrolled in Form 1 or above according to the 1988 Census. In the second regression, the outcome is the number of girls or boys who *pass* the 2004 PSLE (achieve a grade of C or above) controlling for the number of test-takers. In both regressions, X_d contains the following set of district-level controls: 2000/2001 Gini coefficient of income inequality and percent of population below poverty line.[34] Unfortunately, 2002 census data on school enrollment are currently unavailable, but 2004 PSLE pass rates are likely to be a reasonable proxy for the secondary school transition rate in the cohort of test-takers. As such, one caveat is that conclusions about secondary school enrollment drawn from this analysis depend on a low rate at which children who pass the test fail to enroll in secondary school, which is likely to be the case given the extreme competition for slots in Tanzanian secondary schools and low PSLE pass rates.

Results from these regressions are presented in Table 10. The estimates in the first two columns reveal that the rate at which students take the PSLE is not significantly higher for IOC districts for either gender. The point estimates are positive but small and fall short of statistical significance, which may reflect the fact that all students who have access to secondary school attempt to enter. We do, however, observe that the number of individuals passing the PSLE is significantly higher in IOC districts and particularly so for females. The point estimates of 0.24 and 0.15 on the coefficients for IOC are significant at 5% for females and males respectively. While far from conclusive, these results suggest that the IOC intervention may have positively affected the distribution of scores on the PSLE, particularly the distribution of female scores. This is also observed in a comparison of test score distributions across gender and program participation (Figure 7), which reveals little difference in the distribution of male test scores across program and non-program regions and a significantly lower fraction of scores in the lower tail of the grade distribution for girls. Corresponding regression estimates suggest that districts that participated in IOC experienced a significant decrease in the number of individuals receiving the lowest grade "F" while there is no visible difference in the proportion of individuals in the upper tail of the distribution (columns 5-8). The decrease is statistically significant for both genders but again larger in magnitude for females.

7 Impact of universal salt iodization on cross-country comparisons

The magnitude of the estimated effect of IOC on schooling in Tanzania implies that comparable reductions in iodine deficiency worldwide that have resulted from universal salt iodization (USI) over the past two decades should be visible in improvements in aggregate schooling between 1980 and 2000. Hence, partly as a robustness check, the last section of the paper examines whether cross-country

[34] Data on education come from the 2004 Ministry of Education Basic Education Statistics (MoEC) and the 2000/01 THBS, as reported in R&AWG (2005).

differences in reductions in iodine deficiency that resulted from differences in the timing and intensity of USI and differences in baseline levels of IDD are correlated with improvements in schooling attainment over the same period.

7.1 Global trends in IDD and Salt Iodization

The International Council for the Control of Iodine Deficiency Disorders (ICCIDD) was established in 1985 with the single purpose of achieving optimal iodine nutrition worldwide, and has since worked closely with UNICEF and the World Health Organization towards this objective. The resulting Universal Salt Iodization (USI) movement was based on the notion that IDD is easily and inexpensively preventable through iodized salt (Mannar, 1996). In 1990, participants in the World Summit for Children set a goal to eliminate IDD by the year 2000 through USI. 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%. Figure 8 shows current prevalence of IDD and Figure 9 current estimates of the fraction of households consuming iodized salt. 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).

7.2 Cross-country regression analysis

For the cross-country empirical analysis, data were compiled from 81 countries on the following four key variables: primary and secondary enrollment in 1980 and 2000, which spans the period during which the bulk of USI activity took place[35]; the most common indicator of iodine deficiency, total goiter rate (TGR); and a widely available indicator of recent improvements in iodine coverage, the percentage of households consuming iodized salt. All countries for which these four measures were available were included in the analysis. School enrollment information was taken from the World Bank's World Development Indicators supplemented by the Barro-Lee Educational Attainment Data for the 1980s; household consumption of iodized salt was gathered from UNICEF's Global Database on Universal Salt Iodization; and goiter rates were taken from the World Health Organization's Database on Iodine Deficiency and supplemented with Current Iodine Deficiency Status (CIDDS) database maintained by the ICCIDD.[36] To approximate the level of iodine deficiency prior to salt iodization, TGR from a year prior to

[35] The year 1980 is an appropriate pre-legislation measure of schooling for all countries that passed USI after 1975 due to the fact that children even in primary school in 1980 were born prior to the policy change.

[36] WHO data, along with a detailed description of data sources and inclusion criteria are accessible on-line at: http://www3.who.int/whosis/mn/mn_iodine/. Both sources of TGR information compile estimates from a number of government and scientific sources, and there is a great deal of overlap. However, whenever more than one estimate was available, data were taken from the WHO database given that CIDDS estimates of goiter prevalence appear to be noisier due to the variety of ways TGR is calculated (palpation vs. ultrasound; range of estimated TGR, etc).

1980 was used whenever possible, although in many cases it was necessary to include TGR measured between 1990 and 1995. Figure 10 plots IDD and degree of salt iodization for countries in Africa. As can be seen in the scatter plot, within Africa alone there is substantial variation in both baseline IDD (TGR) and policy measures taken to reduce IDD.

In the first set of estimates, we examine the impact of iodine deficiency on changes in schooling attainment over the past two decades by regressing male and female primary and secondary enrollment in 2000 on 1980 enrollment along with baseline TGR and a standard set of control variables.[37] We then test whether reductions in IDD over this period are associated with improvements in schooling attainment by adding to the regression the fraction of households consuming iodized salt in 2000. All cross-country regression results are presented in Table 11.

Three important findings emerge: First, iodine deficiency is negatively associated with improvements in female secondary school enrollment between 1980 and 2000. In particular, baseline TGR appears to have a significant adverse effect on female secondary enrollment in 2000 conditional on enrollment rates in 1980. Our results suggest that reducing TGR from 30 to 10 will increase average female secondary school participation by approximately 7%.[38] The point estimates are also negative but lower and insignificant for males. Surprisingly, the estimated effect of baseline TGR on 1980 and 2000 *primary* school participation is not significantly different from zero in any of the regressions. This is likely due to the higher degree of collinearity between TGR and 1980 primary enrollment relative to TGR and 1980 secondary enrollment. In particular, secondary school enrollment was so low in 1980 in many affected countries that IDD was less likely to pose a binding constraint.

Second, *reductions* in IDD between 1980 and 2000 appear to have had an important 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. The absence of a concomitant effect of USI on secondary enrollment is consistent with this interpretation of the estimates given that the bulk of changes in household use of iodized salt were too recent to affect the cohort of children eligible for secondary school (children above age 12 in 2000).

Third, the effect of reductions in IDD on primary school enrollment appears to be significantly larger for females. The influence of iodized salt on primary schooling enrollment is estimated to be 0.137

[37] Control variables collected primarily from the World Bank's World Development Indicators. All regressions include the following controls for earliest date available: Malaria prevalence (2001), HIV prevalence (2003), urban population as % of total population (1990), population density per square km (1990), log GDP per capita (1990), log GDP per capita squared (1990), and terms of trade (1980 export value index /import value index). Results are robust to excluding post-1990 controls.

[38] A 10% reduction in TGR is equivalent to a 3 point drop in average TGR from 30.66 in our dataset. We calculate the average increase in female secondary school participation (0.7) based on the estimated effect of TGR in Table 10 (column 5). We calculate the percentage increase in female secondary school participation (1.5%) by dividing the increase in participation (0.7) by the average participation rate of 50.71 in our dataset.

for females and significant at the 5% level (column 3). This estimate suggests that moving from the current sample average of 60% to universal salt iodization (100%) would increase female primary school participation by as much as 7%. Meanwhile, the point estimate remains positive but lower and insignificant for males (column 4), consistent with our estimated effects of IOC in Tanzania.

These findings suggest that recent increases in iodine intake have had a beneficial impact on cognitive development worldwide, particularly for females, and are consistent with the directions and magnitudes of effects found in the micro-level estimates in Tanzania. Together with the previous results, they underscore the importance of universal salt legislation for endemic regions, along with complimentary measures to ensure deeper penetration in countries, including the majority of Western Africa, for which legislation has failed to provide adequate protection.

7.3 Projections

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. Baseline levels of TGR in these countries ranged from 10% to 52%, compared with an average of 30% and range of 10-75% among districts that participated in the Tanzanian IOC program. 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, which varies from 7% in Niger to 86.1% in Nicaragua. 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.

According to our estimates, approximately 41.1 million children between the ages of 5 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. 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.[39] 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. Among all affected countries,

[39] This effect is calculated from the baseline effect (0.36 years) observed in Tanzania adjusted for an average IOC take-up rate assumed to be 78% and the average rate of maternal IDD (60%) in the target population.

the increase in average schooling due to USI amounts to 4.8%. For Central and Southern Africa, the predicted improvement in average schooling across *all* countries in the region is 7.5%.

8 Conclusions

We emphasize three conclusions from this analysis. First, our findings provide micro-level evidence of an important role of geography in economic development that operates through the influence of mineral 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.

Second, our findings 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 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 in animals should not be discounted.

Finally, 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. Our estimates indicate that at least 41 million children have been affected by these reforms, which could increase average schooling attainment in many countries by over 10%. In areas with baseline IDD comparable to districts in the middle range of our sample, 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.

However, our findings also provide evidence that universal salt iodization will not eliminate the adverse cognitive effects of fetal IDD among populations in the most afflicted settings where diets high in goitrogens require higher supplement levels or other dietary changes to overcome maternal IDD. In these areas, more intensive interventions such as IOC or introducing new methods of cassava processing are necessary to achieve current Millennium Development Goals regarding micronutrient deficiencies. Although such approaches are significantly more costly than salt iodization, the resulting gains in schooling attainment suggested by our findings indicate that expected returns well outweigh the costs.

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Figure 1: Intervention districts

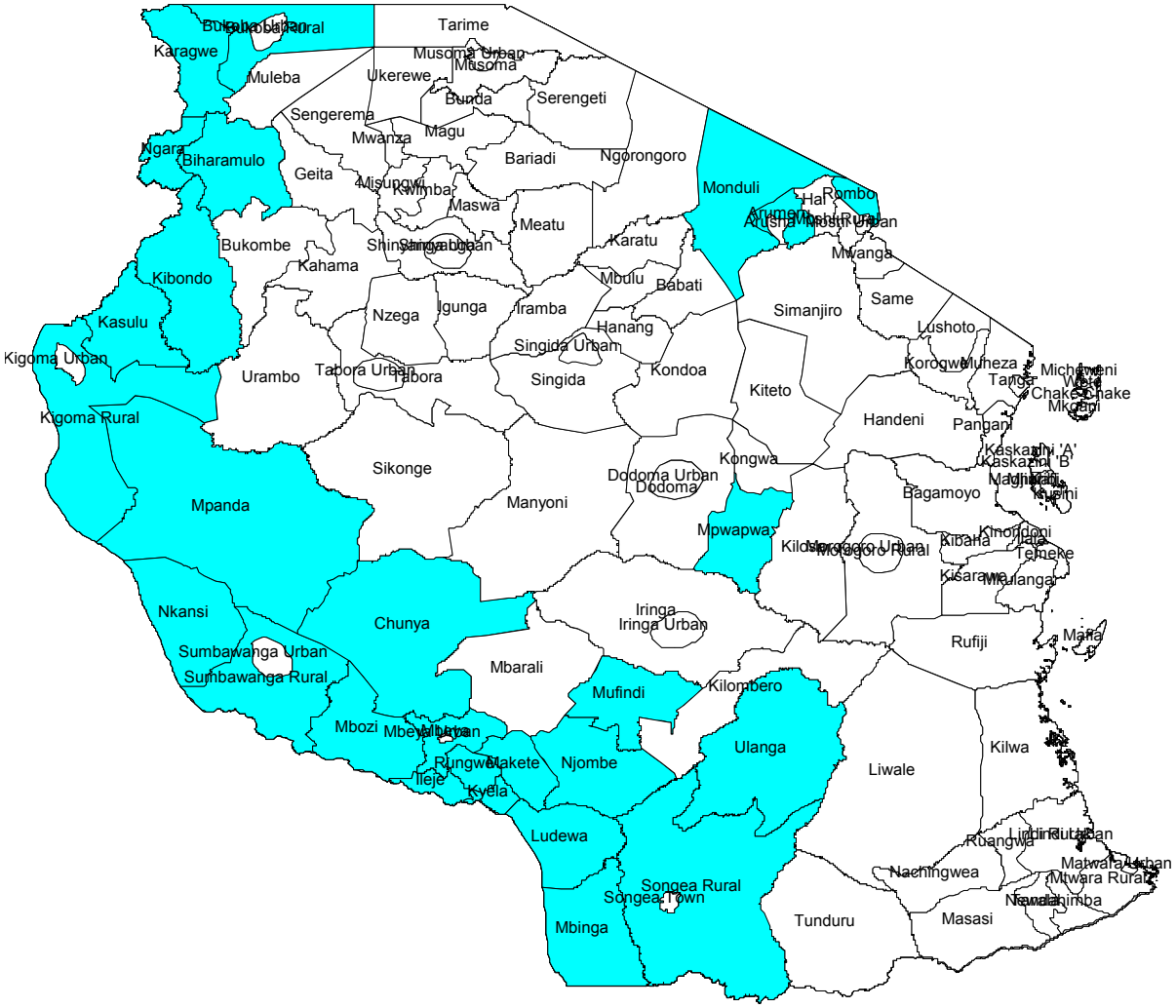


Figure 2: Iodine remaining in body after administration of 380mg iodized oil

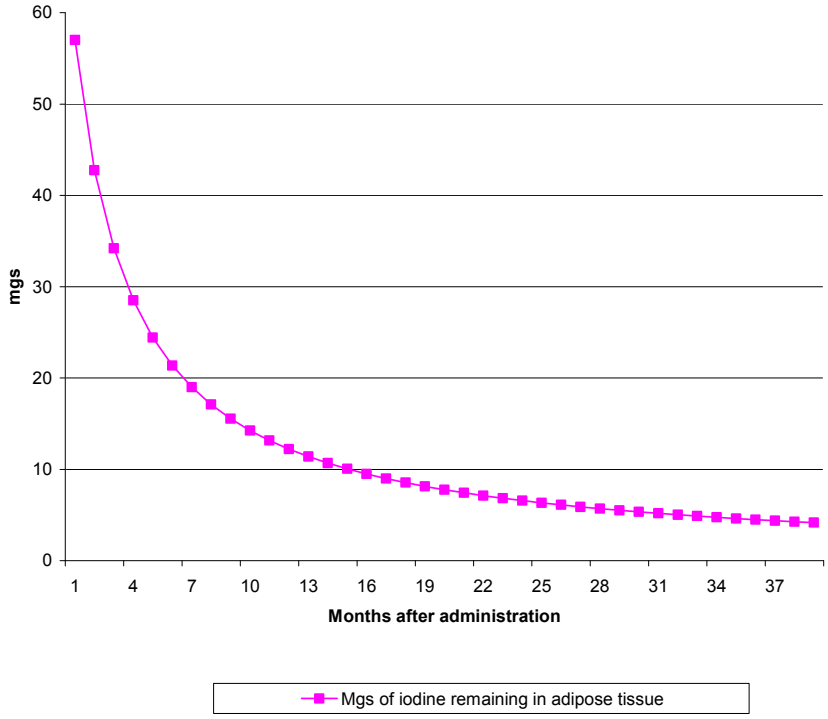


Figure 3: Child's likelihood of protection from IDD during first tri-mester

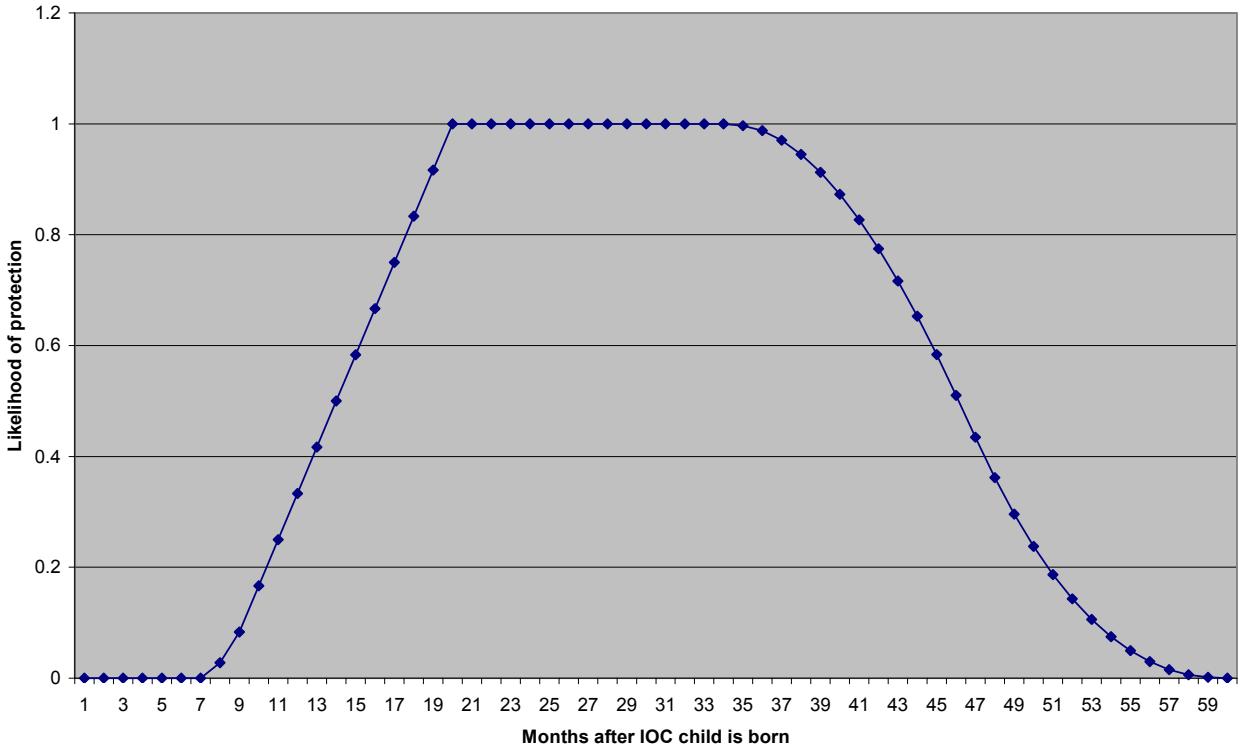
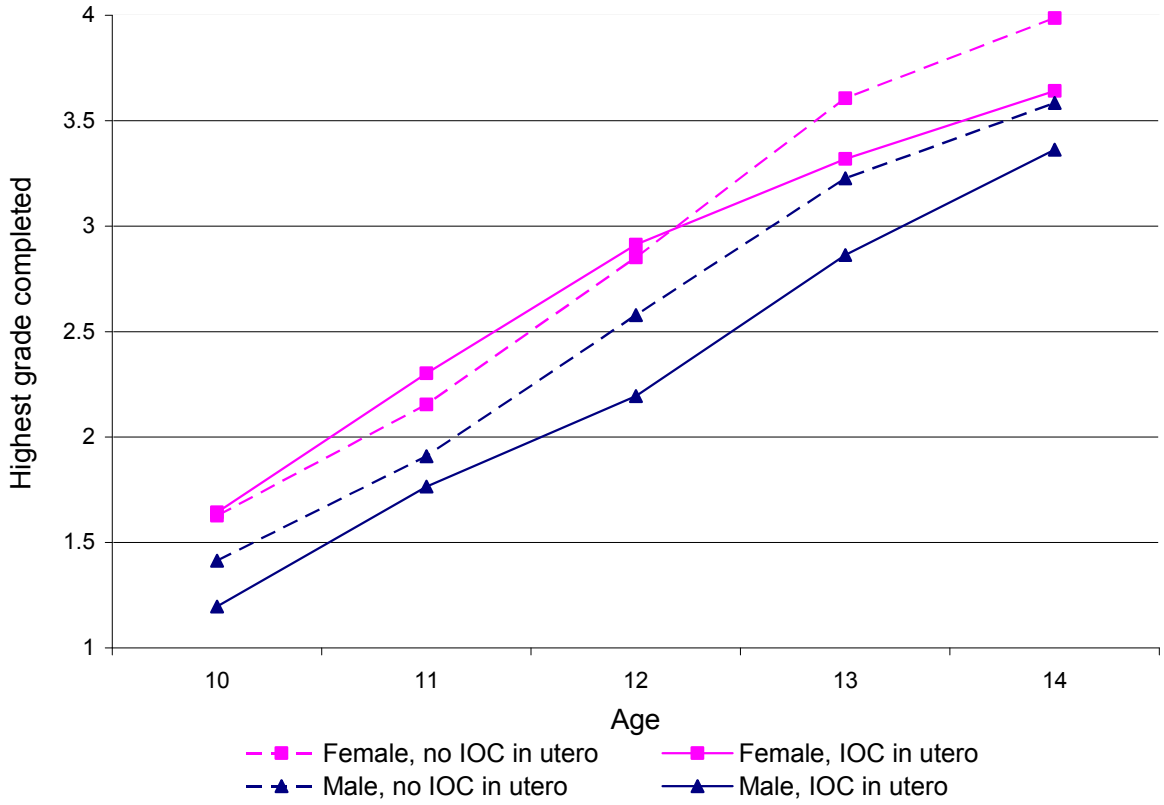
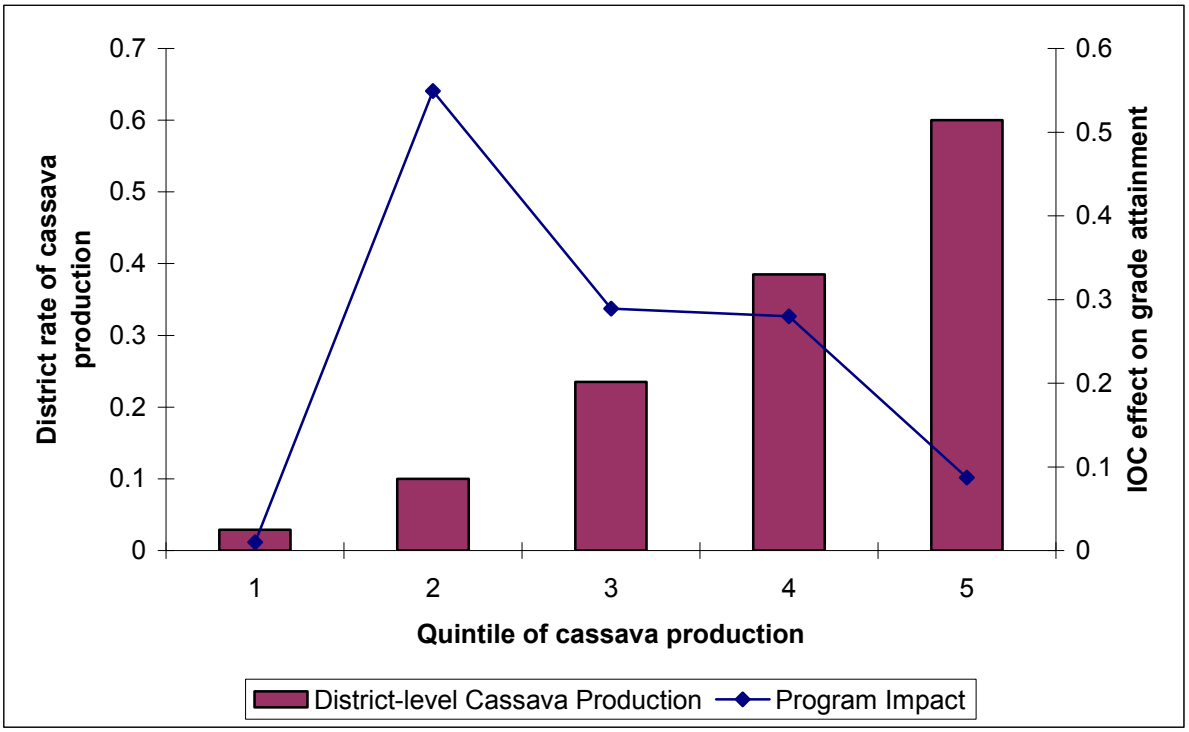


Figure 4: Grade Progression by Gender and IOC



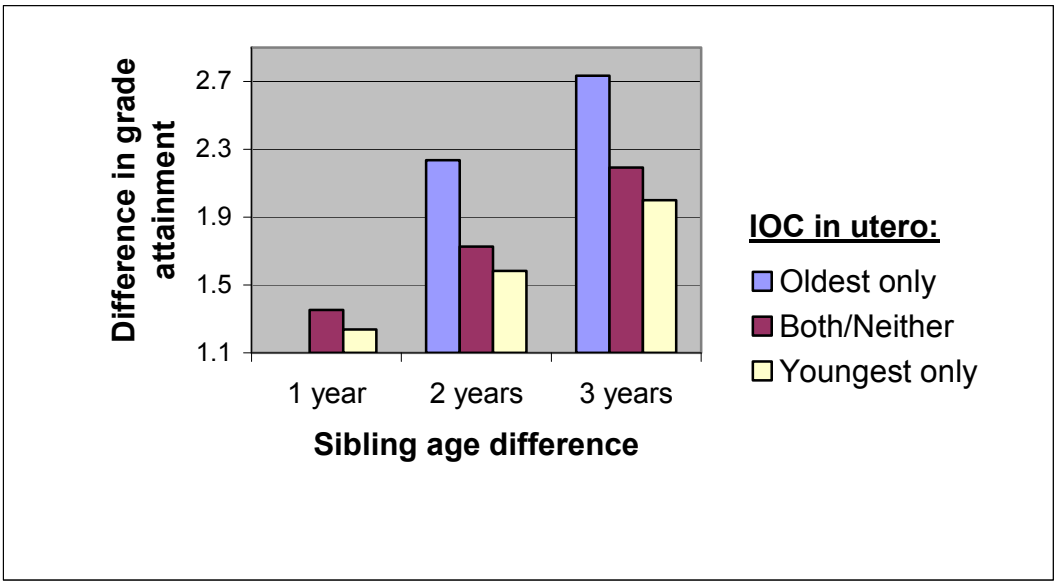
Notes: Data from the 2004 Tanzania Demographic Health Survey. 3675 observations, including all children in project districts between the ages of 10 and 14 that are children of respondents to the birth history module and who reside in the household. X-axis is child age and y-axis is completed years of schooling. IOC in utero refers to whether iodized oil capsules distributed in district of residence 1 to 3 years prior to child's year of birth.

Figure 5: Schooling Effect of IOC by District Level of Cassava Production



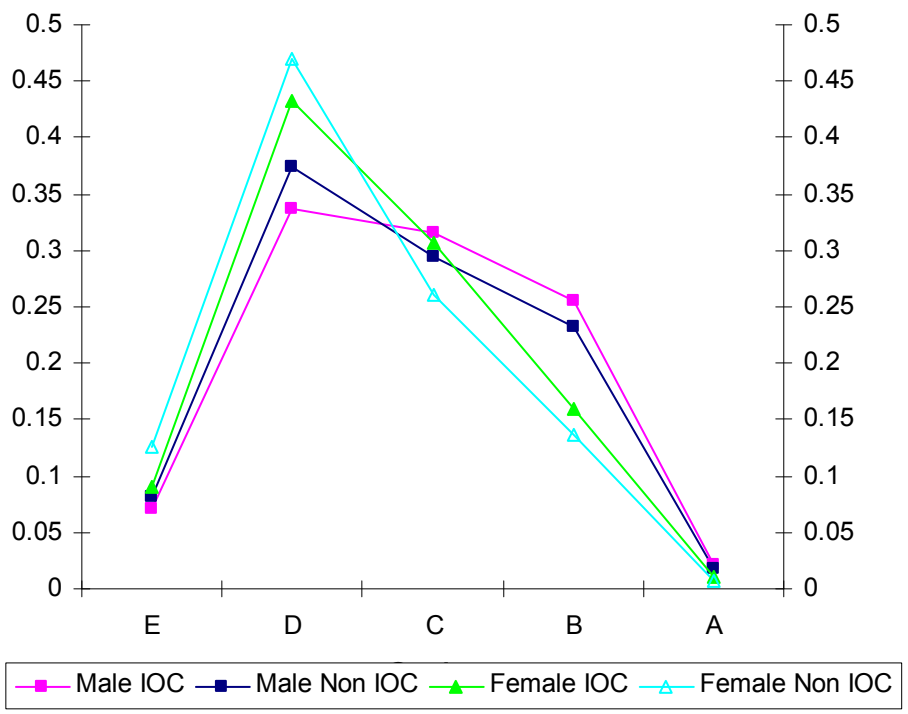
Notes: Data from the 2000 Tanzania Household Budget Survey. Includes 2277 observations are all children in project districts between the ages of 10 and 13 that are children or grandchildren of the household head or spouse. Left-hand side Y-axis is district fraction of households that grow cassava, a highly goitrogenous food; right-hand side x-axis is point estimate of coefficient on IOC in regression of grade attainment on age, gender, birth order and IOC, run separately for districts in five levels of cassava production. IOC in utero refers to whether iodized oil capsules distributed in district of residence 1 to 3 years prior to child's year of birth. Since IOC prevents maternal iodine deficiency for an estimated 24 months, IOC distributed 1-3 years before birth corresponds to higher likelihood of sufficient maternal iodine level in utero during first two trimesters of pregnancy, what is considered to be the critical intervention period.

Figure 6: 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.

Figure 7: PSLE Score Distribution by Gender and IOC participation



Note: 2004 PSLE test scores were not available for the following districts-regions: Iringa Rural-Iringa, Iringa Urban-Iringa, Njombe-Iringa, Biharamulo-Kagera, Nyamagana-Nzega, Kiteto-Arusha, Namtumbo-Newala, Mvomero-Mwanga, Mufindi-Muheza, Mbulu-Arusha, Ngara-Kagera, Nkansi-Rukwa, Sumbuwanga-Rukwa, Ludewa-Lusoto, Makete-Manyoni, Mwete-Pemba, Cheke-Pemba, Micheweni-Pemba, Mjini-Pemba, Mkoani-Pemba. The grades awarded on the PSLE range from the top grade "A" to the lowest grade "E." At least one male received the highest score "A" on the PSLE in 81 of the districts; at least one female received the highest score "A" on the PSLE in 59 districts.

Figure 8: IDD Prevalence across African countries, 2000-2005

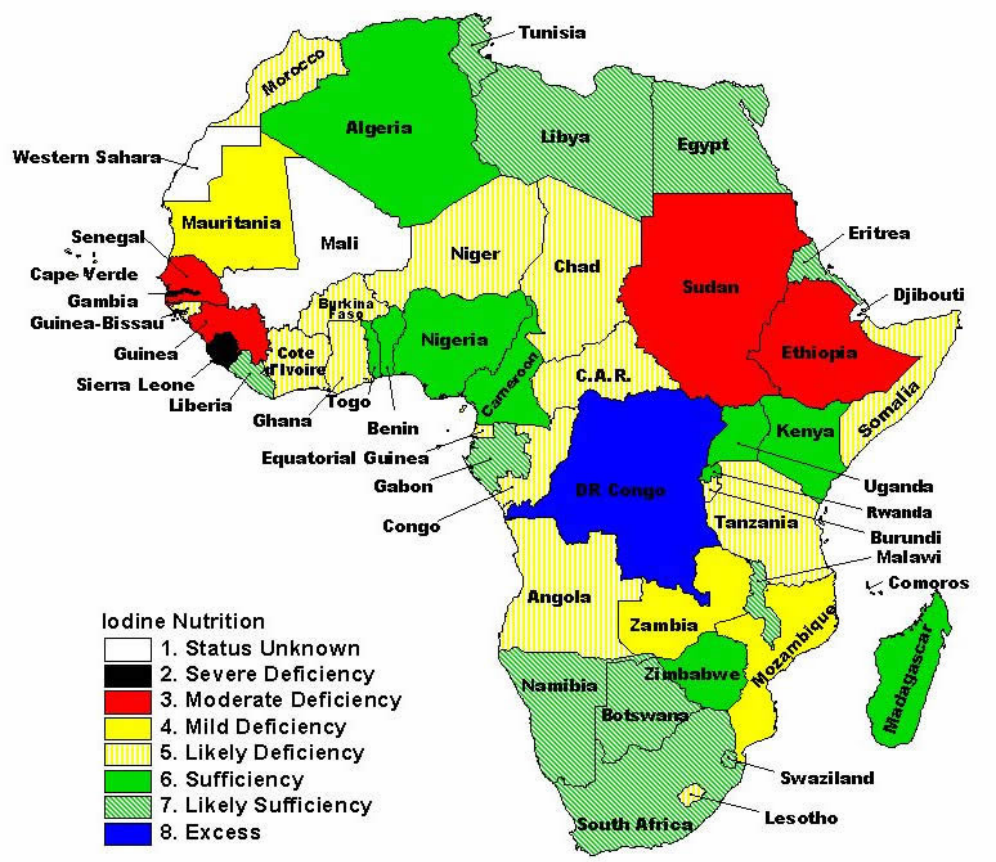


Figure 9: Fraction of households consuming iodized salt, 2000-2005

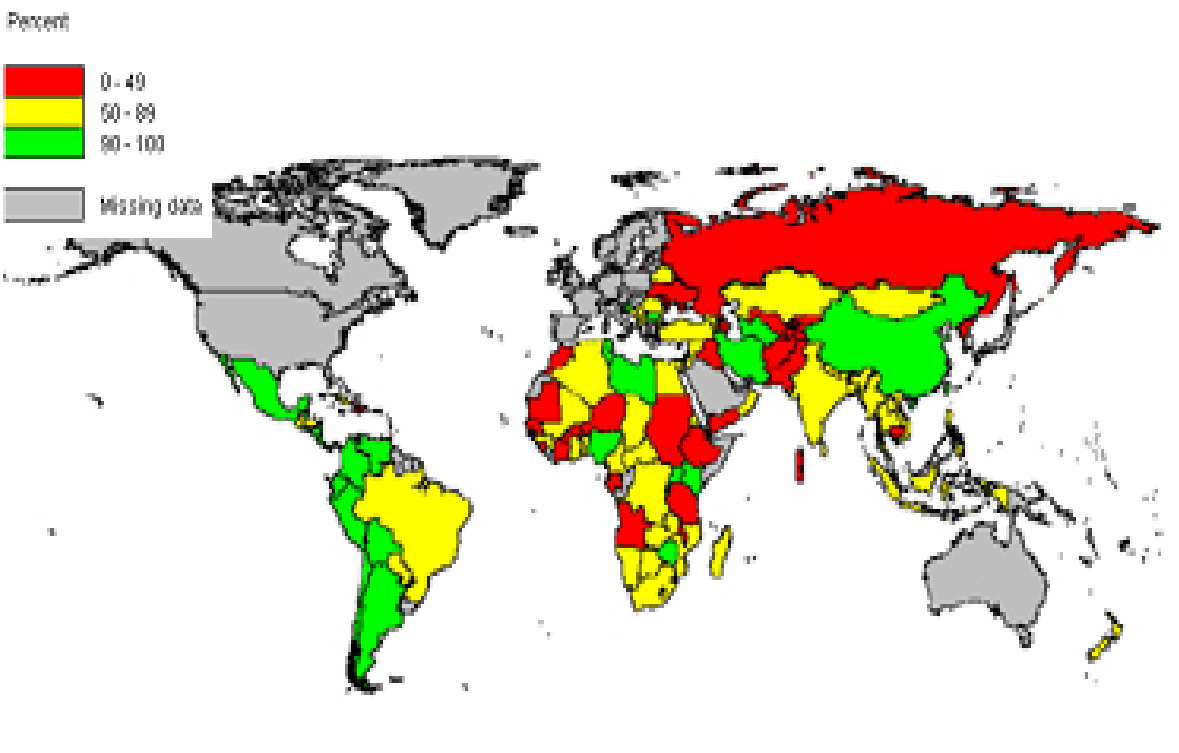


Figure 10: TGR pre-1995 and % of Households Consuming Iodized Salt, 2000-2005, by African Country

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

			Year of Intervention (Coverage - %)*					Average Frequency
	Region	District	1	2	3	4	5	(yr)
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 form 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)	
		IOC Program Timing			t_{Δ}
	No Program	1986-1987	1988-1989	1990-1995	2 - 4
Total members per household	4.86	5.02	4.58	5.08	-0.52
	(3.13)	(2.90)	(2.50)	(3.11)	
Head of household education	10.95	10.84	10.72	10.44	1.87
	(6.48)	(6.53)	(6.65)	(6.71)	
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	2.51	2.49	2.48	1.45
	(0.48)	(0.53)	(0.52)	(0.55)	
Fish per week	2.27	1.87	1.59	1.79	1.48
	(1.80)	(1.59)	(1.45)	(1.84)	
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
Observations	*17067*	*2152*	*819*	*1711*	

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, 2000

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
				Binary treatment indicator		*All kids in household*				*Universe: Households with > 1 member in sample*	
	All	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys
Pr(IOC in utero)	0.358	0.902	0.428	0.709	0.168	0.733	0.331	0.357	0.006	0.692	0.346
	[0.161]*	[0.360]*	[0.292]	[0.318]*	[0.259]	[0.327]*	[0.278]	[0.142]*	[0.194]	[0.431]	[0.389]
$Pr(IOC\ in\ utero)_{3\leq t<5}$	0.015	0.838	-0.142	0.038	-0.422	0.355	-0.088	0.478	0.032	0.754	-0.179
	[0.252]	[0.680]	[0.371]	[0.379]	[0.274]	[0.486]	[0.345]	[0.323]	[0.261]	[0.659]	[0.504]
$Pr(IOC\ in\ utero)_{3\leq t<5}$ * Young	0.168	-0.708	0.266	0.069	0.191	-0.307	0.020	-0.673	0.047	-0.756	0.443
	[0.315]	[0.740]	[0.488]	[0.407]	[0.286]	[0.603]	[0.482]	[0.335]*	[0.276]	[0.626]	[0.521]
Age 11	0.624	0.982	0.599	0.940	0.487	0.772	0.538	0.310	0.395	0.353	0.862
	[0.163]**	[0.331]**	[0.317]+	[0.352]**	[0.305]	[0.275]**	[0.283]+	[0.157]*	[0.148]**	[0.323]	[0.372]*
Age 12	1.468	1.730	1.066	1.631	0.894	1.599	1.237	1.240	1.182	1.335	1.162
	[0.134]**	[0.329]**	[0.283]**	[0.347]**	[0.290]**	[0.278]**	[0.236]**	[0.160]**	[0.151]**	[0.357]**	[0.347]**
Age 13	2.139	2.801	1.768	2.730	1.505	2.695	1.981	2.092	1.648	2.041	1.823
	[0.155]**	[0.398]**	[0.348]**	[0.448]**	[0.382]**	[0.325]**	[0.287]**	[0.177]**	[0.165]**	[0.394]**	[0.399]**
Female	0.216										
	[0.104]**										
Number boys 10-15 in HH									0.303		0.845
									[0.122]*		[0.285]**
Number girls 10-15 in HH								0.398		0.210	
								[0.138]**		[0.532]	
Fixed effects	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*District*	*District*	*District*	*District*
Observations	*846*	*231*	*251*	*231*	*251*	*300*	*335*	*865*	*920*	*231*	*251*

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. All estimates except those in columns 6 and 7 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)3≤t<4* 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 also control for binary indicators of sex-specific birth order. + significant at 10%; * significant at 5%; ** significant at 1%

Table 4: Grade Attainment and IOC Supplementation in Utero, 2004

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	All	Girls	Boys	All	Girls	Boys	All	Girls	Boys	All	Girls	Boys
Pr(IOC in utero)	0.519	0.809	0.293	0.288	0.416	0.197	0.738	1.042	0.451	0.635	0.896	0.323
	[0.191]**	[0.477]+	[0.351]	[0.165]+	[0.245]+	[0.229]	[0.238]**	[0.570]+	[0.410]	[0.208]**	[0.304]**	[0.291]
Pr(IOC in utero)$_{3\leq t<5}$ * Birth month	0.027	0.032	0.006	0.005	0.018	-0.01						
	[0.018]	[0.040]	[0.030]	[0.014]	[0.021]	[0.019]						
Pr(IOC in utero)$_{3\leq t<5}$ * Young mom	-0.043	-0.05	-0.032	0.016	0.018	0.013						
	[0.027]	[0.059]	[0.046]	[0.013]	[0.020]	[0.019]						
Pr(IOC in utero) * Age of child							-0.122	-0.13	-0.079	-0.137	-0.211	-0.035
							[0.059]*	[0.130]	[0.096]	[0.054]*	[0.079]**	[0.074]
Age 11	0.407	0.384	0.402	0.523	0.537	0.558	0.423	0.409	0.405	0.534	0.561	0.56
	[0.101]**	[0.239]	[0.189]*	[0.069]**	[0.102]**	[0.095]**	[0.101]**	[0.238]+	[0.188]*	[0.069]**	[0.102]**	[0.095]**
Age 12	1.141	0.991	1.103	1.105	1.224	0.985	1.151	0.998	1.111	1.137	1.274	0.997
	[0.089]**	[0.278]**	[0.208]**	[0.069]**	[0.103]**	[0.094]**	[0.089]**	[0.277]**	[0.207]**	[0.070]**	[0.105]**	[0.096]**
Age 13	1.637	1.867	1.209	1.721	1.901	1.526	1.672	1.905	1.217	1.766	1.967	1.543
	[0.094]**	[0.385]**	[0.285]**	[0.070]**	[0.106]**	[0.096]**	[0.096]**	[0.384]**	[0.284]**	[0.073]**	[0.110]**	[0.100]**
Age 14	2.42	2.436	2.109	2.301	2.473	2.11	2.457	2.485	2.126	2.356	2.555	2.129
	[0.096]**	[0.439]**	[0.336]**	[0.072]**	[0.112]**	[0.098]**	[0.099]**	[0.438]**	[0.334]**	[0.076]**	[0.118]**	[0.102]**
Birth month	-0.031	-0.051	-0.022	-0.038	-0.05	-0.029	-0.031	-0.052	-0.022	-0.035	-0.046	-0.029
	[0.010]**	[0.024]*	[0.017]	[0.007]**	[0.010]**	[0.009]**	[0.010]**	[0.022]*	[0.017]	[0.007]**	[0.010]**	[0.009]**
Female	0.351			0.321			0.353			0.322		
	[0.062]**			[0.068]**			[0.062]**			[0.068]**		
Number boys 10-15 in HH				-0.033		0.073				-0.031		0.075
				[0.044]		[0.063]				[0.044]		[0.063]
Number girls 10-15 in HH				-0.041	0.076					-0.041	0.079	
				[0.046]	[0.075]					[0.046]	[0.075]	
Fixed effects	*House-hold*	*House-hold*	*House-hold*	*District*	*District*	*District*	*House-hold*	*House-hold*	*House-hold*	*District*	*District*	*District*
Observations	*2160*	*534*	*643*	*3672*	*1797*	*1875*	*2160*	*534*	*643*	*3672*	*1797*	*1875*

Notes: Data from the 2004 Tanzanian Household Budget Survey, sample restricted to children ages 10-14 that reside in the household and have non-missing month of birth and education data. 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)* $_{3\leq t<4}$ 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 also control for binary indicators of sex-specific birth order. + significant at 10%; * significant at 5%; ** significant at 1%

Table 5: Variation in Effect on Schooling of IOC Supplementation in Utero

	Rate of Cassava Consumption in District		
	High (0.41-0.62)	Medium (0.10-0.40)	Low (< 0.10)
Pr(IOC in utero)	-0.037	0.750	-0.149
	[0.492]	[0.230]**	[0.305]
Age 11	0.783	0.387	0.576
	[0.323]*	[0.248]	[0.257]*
Age 12	1.951	1.353	1.066
	[0.240]**	[0.185]**	[0.206]**
Age 13	2.137	2.294	1.725
	[0.247]**	[0.226]**	[0.281]**
Female	0.47	0.243	0.105
	[0.195]*	[0.165]	[0.152]
Fixed effects	*Household*	*Household*	*Household*
Observations	529	650	606

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. Rate of cassava consumption defined as fraction of THBS households in district that report growing cassava in the 2000 survey data. 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. Regressions also control for sex-specific birth order and household fixed effects.

Table 6: Robustness Checks of Grade Attainment and IOC Supplementation in Utero, 2000

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	Full sample				**Restricted control group**				**Restricted treatment group**			
	Binary treatment indicator		*Continuous treatment indicator*		*Binary treatment indicator*		*Continuous treatment indicator*		*Binary treatment indicator*		*Continuous treatment indicator*	
	All	Girls	All	Girls	All	Girls	All	Girls	All	Girls	All	Girls
Pr(IOC in utero)	0.276	0.671	0.355	0.838	0.412	0.733	0.608	1.013	0.416	1.15	0.505	1.302
	[0.125]*	[0.276]*	[0.159]*	[0.354]*	[0.163]*	[0.384]+	[0.217]**	[0.521]+	[0.179]*	[0.484]*	[0.217]*	[0.583]*
Age 11	0.639	0.897	0.62	0.87	0.603	1.031	0.498	0.881	0.65	1.18	0.624	1.134
	[0.155]**	[0.309]**	[0.155]**	[0.308]**	[0.220]**	[0.578]+	[0.228]*	[0.584]	[0.171]**	[0.359]**	[0.172]**	[0.358]**
Age 12	1.435	1.58	1.434	1.566	1.229	1.438	1.182	1.335	1.571	1.851	1.577	1.827
	[0.118]**	[0.278]**	[0.117]**	[0.278]**	[0.171]**	[0.430]**	[0.174]**	[0.441]**	[0.153]**	[0.362]**	[0.153]**	[0.363]**
Age 13	2.111	2.654	2.116	2.654	2.11	2.326	2.124	2.308	2.137	3.251	2.128	3.166
	[0.139]**	[0.352]**	[0.140]**	[0.353]**	[0.201]**	[0.504]**	[0.200]**	[0.500]**	[0.166]**	[0.461]**	[0.165]**	[0.452]**
Female	0.244		0.242		0.274		0.276		0.321		0.318	
	[0.098]*		[0.098]*		[0.138]*		[0.138]*		[0.113]**		[0.113]**	
Fixed effects	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*
Observations	846	231	846	231	568	143	568	143	694	187	694	187

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; 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. All regressions also control for binary indicators of sex-specific birth order. + significant at 10%; * significant at 5%; ** significant at 1%

Table 7: Difference in Grade Attainment and IOC Supplementation by Birth Order

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

Table 8: Mortality and IOC Supplementation in Utero

	Fertility *(Birth in month m)*			*Neonatal mortality* *(Died within 30 days of birth)*			*Infant Mortality* *(Died within 1 year of birth)*			*Child Mortality* *(Died between ages 1 and 15)*		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	All	Girls	Boys	All	Girls	Boys	All	Girls	Boys	All	Girls	Boys
Pr(IOC in utero)	0.004	0.001	0.003	-0.026	0.027	-0.060	-0.008	-0.036	0.042	-0.101	-0.077	-0.084
	[0.003]	[0.002]	[0.002]	[0.025]	[0.064]	[0.051]	[0.037]	[0.083]	[0.076]	[0.032]**	[0.066]	[0.068]
$Pr(IOC\ in\ utero)_{3\le t<5}$ * Birth month	0.000	0.000	0.000	0.004	0.008	0.002	0.000	-0.002	0.009	-0.004	-0.007	-0.004
	[0.000]	[0.000]	[0.000]	[0.002]*	[0.003]*	[0.003]	[0.002]	[0.004]	[0.005]+	[0.002]*	[0.004]*	[0.004]
$Pr(IOC\ in\ utero)_{3\le t<5}$ * Young mom	0.000	0.000	0.000	-0.004	-0.011	0.003	0.000	0.001	-0.005	0.002	0.010	0.001
	[0.000]	[0.000]	[0.000]	[0.002]*	[0.005]*	[0.004]	[0.003]	[0.006]	[0.006]	[0.003]	[0.005]*	[0.005]
Fixed effects	*Mother*	*Mother*	*Mother*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*
Observations	*217,740*	*217,740*	*217,740*	*4352*	*1219*	*1344*	*4352*	*1219*	*1344*	*4352*	*1219*	*1344*

Notes: Data from the 2004 Tanzanian Demographic Health Survey. Analysis sample for column 1-9 regressions restricted to all births reported by TDHS sample members between 1990 and 1995, and outcomes pertain to survival of each child born. Unit of observation in analysis sample for column 10-12 regressions is a month between 1990 and 1995, and outcome is whether respondent gave birth during that month. *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 ni the mother's body to protect the fetus during month 1 of pregnancy. Precise values are given in Appendix A. *Pr(IOC in utero)* $_{3\le t<4}$ 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 distributioh, 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 birth order, sex-specific birth order, mother's birth year, and birth month. + significant at 10%; * significant at 5%; ** significant at 1%

Table 9: Effect of IOC on Reported Health Status at Ages 10-13

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	Whether any sickness last 4 weeks	*Whether fever*	*Whether diarrhea*	*Whether ear/nose/ throat condition*	*Whether eye condition*	*Whether skin condition*	*Whether dental condition*	*Whether accident-related condition*	*Whether other health problem*	*Days school/work missed due to illness*
Pr(IOC in utero)	0.046	0.071	-0.019	0.001	-0.001	0.011	-0.003	0.008	0.000	-0.030
	[0.052]	[0.044]	[0.016]	[0.018]	[0.010]	[0.015]	[0.011]	[0.008]	[0.027]	[0.064]
Age 11	-0.047	0.010	-0.009	-0.001	-0.011	0.007	-0.020	-0.001	-0.010	0.026
	[0.050]	[0.042]	[0.015]	[0.018]	[0.010]	[0.014]	[0.011]+	[0.007]	[0.026]	[0.062]
Age 12	-0.004	-0.024	-0.012	-0.012	0.006	0.021	0.009	0.011	0.000	0.056
	[0.038]	[0.032]	[0.012]	[0.013]	[0.008]	[0.011]+	[0.008]	[0.006]*	[0.020]	[0.047]
Age 13	-0.028	-0.008	-0.022	0.006	-0.006	0.013	0.005	0.000	0.024	0.004
	[0.045]	[0.038]	[0.014]	[0.016]	[0.009]	[0.013]	[0.010]	[0.007]	[0.024]	[0.056]
Female	0.008	0.016	0.006	-0.006	-0.007	-0.006	0.011	0.000	0.010	0.037
	[0.032]	[0.027]	[0.010]	[0.011]	[0.006]	[0.009]	[0.007]	[0.005]	[0.017]	[0.040]
Fixed effects	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*	*House-hold*
Observations	*1807*	*1807*	*1807*	*1807*	*1807*	*1807*	*1807*	*1807*	*1807*	*1807*

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. Regressions also control for sex-specific birth order and household fixed effects.

Table 10: Male and Female PSLE Performance by IOC Intervention

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Dependent Variable:	ln (number individuals taking PSLE) [1]		ln(number individuals with passing grade on [1]		ln (number of individuals with grade "F" on PSLE) [2]		ln (number of individuals with grade "A" on PSLE) [2]	
	Female	Male	Female	Male	Female	Male	Female	Male
IOC Intervention between 1986-1992	0.12	0.11	0.24 **	0.15 **	-0.92 ***	-0.83 ***	-0.15	-0.01
	(0.08)	(0.09)	(0.10)	(0.07)	(0.25)	(0.26)	(0.34)	(0.37)
Male secondary school attainment rate, 1988		-1.87 **		1.08		-5.44 **		-3.88
		(0.83)		(0.69)		(2.46)		(3.55)
Female secondary school attainment rate, 1988	-2.22 **		4.14 ***		-12.22 ***		-0.62	
	(1.08)		(1.24)		(3.01)		(4.22)	
ln(2002 population males 10-14)		0.53 ***						
		(0.03)						
ln(2002 population females 10-14)	0.56 ***							
	(0.03)							
ln(2004 population male test-takers)				0.92 ***		1.74 ***		1.56 ***
				(0.05)		(0.17)		(0.25)
ln(2004 population female test-takers)			0.87 ***		1.68 ***		1.18 ***	
			(0.07)		(0.16)		(0.23)	
[3] *Observations*	*93*	*93*	*93*	*93*	*93*	*93*	*93*	*93*

Notes:

[1] Dependent variable in columns 1 and 2 is the number of girls/boys that take the 2004 PSLE; dependent variable in columns 3 and 4 is the number of students who receive a passing grade of A, B, C on the PSLE. Secondry school enrollment rate in 1988 is fraction of girls/boys enrolled in form 1 or above from the 1988 Census (National Bureau of Statistics).

[2] The grades awarded on the PSLE range from the top grade "A" to the lowest grade "E." We have altered the lowest grade from "E" to "F" to match the U.S. grading system for ease of comprehension. The top grade of "A" and lowest grade of "E" were not received in several districts: no females received a top grade "A" in 28 region-districts, no males received a top grade of "A" in 11 region-districts and no males received the lowest grade of "E" in one region-district. The dependent variable was adjusted to zero [ln(0) = 0] in cases where the natural log would otherwise be undefined in the above-mentioned region-districts.

[3] The 2002 Census from the National Bureau of Statistics in Tanzania reports a total of 127 districts. A total of 18 districts were newly constructed from existing districts in the 1988 Census. These districts were merged in the 2004 PSLE data and the 2002 Census data to match the 1988 Census data. A total of 16 districts were excluded from the above analysis due to lack of information on PSLE scores: All 6 districts in the Iringa Region, all 4 districts in Pemba (North and South) and all 6 districts in Zanzibar.

* Significant at 10% ** Significant at 5% *** Significant at 1%

Sources:

Data on female/male populations age 10-14 come from the 2002 Census (National Bureau of Statistics); 2004 PSLE Examination Statistics from the National Examinations Council of Tanzania. Male/female secondary school attainment rates in 1988 were calculated from the 1988 Census (National Bureau of Statistics). The percent of population below poverty line and gini coefficient data was obtained from the "Tanzania Poverty and Human Development Report 2005."

Table 11: 2000 School Participation by Gender

Dependent Variable:	2000 Primary School Participation				2000 Secondary School Participation			
	Female (1)	Male (2)	Female (3)	Male (4)	Female (5)	Male (6)	Female (7)	Male (8)
[1] TGR	-0.01	0.11	-0.06	0.08	-0.26 **	-0.16	-0.29 **	-0.18
	(0.13)	(0.12)	(0.13)	(0.12)	(0.12)	(0.13)	(0.12)	(0.13)
[2] % Household use of adequately iodized salt			0.14 **	0.09			0.10	0.04
			(0.07)	(0.07)			(0.07)	(0.08)
1980 Female Primary School Participation	0.22 ***		0.21 ***		0.24 **		0.24 **	
	(0.08)		(0.08)		(0.10)		(0.10)	
1980 Male Primary School Participation		0.26 ***		0.25 ***		0.18 *		0.18 *
		(0.07)		(0.07)		(0.10)		(0.10)
Prevelance of Malaria (2000/01)	3.82	-8.62	6.48	-7.49	-7.47	-4.95	-5.59	-4.17
	(15.08)	(14.30)	(14.77)	(14.26)	(14.02)	(15.24)	(13.98)	(15.40)
Prevalence of HIV (2003)	0.59	0.50	0.46	0.42	0.67	0.44	0.57	0.40
	(0.35)	(0.32)	(0.35)	(0.32)	(0.31)	(0.34)	(0.32)	(0.35)
Urban Population (1990 - % of Total)	0.17	0.05	0.19	0.06	0.75	0.67	0.76	0.67
	(0.14)	(0.14)	(0.14)	(0.13)	(0.14)	(0.15)	(0.14)	(0.15)
Population Density (1990 - per sq. km)	0.02	0.02	0.02	0.02	0.00	0.00	0.01	0.01
	(0.14)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
Log GDP per capita (1990 - Constant LCU)	15.50	13.23	11.10	10.59	15.63	16.19	12.72	15.05
	(7.00)	(6.60)	(7.17)	(6.89)	(6.63)	(7.32)	(6.90)	(7.67)
Log GDP per capita [2] (1990)	-1.84	-1.40	-1.44	-1.16	-1.78	-1.79	-1.52	-1.69
	(0.81)	(0.76)	(0.82)	(0.78)	(0.77)	(0.84)	(0.78)	(0.87)
[3] Terms of Trade (1980)	5.52	3.84	6.78	4.52	5.28	4.70	6.15	5.07
	(3.07)	(2.89)	(3.06)	(2.92)	(0.35)	(3.11)	(2.90)	(3.21)
Region Effect	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
[4] TGR Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
[5] *Observations*	*81*	*81*	*81*	*81*	*81*	*81*	*81*	*81*

Notes:

1 TGR is a reported measure of TGR prior to 1995.

2 Reported % of households using adequately iodized salt in mid-1990s.

3 Terms of Trade is the ratio of the Export Value Index and Import Value Index for year 2000.

4 TGR Controls include information from the sample used to calculate TGR: minimum age, maximum age, gender (both, male, female), sample level (national, province, local, etc.), year of sample.

5 A number of countries were excluded from the above analysis do to partial or missing data: 18% of countries were missing primary school enrollment data; 14% of countries were missing secondary school participation data; and 15% were missing other variables included in the above analysis.

Sources:

Enrollment data is from the World Bank's WDI database and supplemented by data from UNESCO (United Nations Educational, Scientific and Cultural Organization) and NBER (National Bureau of Economic Research). Other national statistic data is from the World Bank's WDI database and supplemented by data from the WHO (World Health Organization) and UN (United Nations). Information on goiter rates and salt legislation years were culled from the Current Iodine Deficiency Status (CIDDS) database maintained by the International Council for the Control of Iodine Deficiency Disorders and supplemented by the WHO's Micronutrient Deficiency Information System.

Table 12: 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%**

Notes:

[1] Only countries with goiter rates similar in magnitude to Tanzania are included in the analysis. Countries with significantly lower goiter rates than Tanzania are not likely to benefit similarly from adequately iodized salt since the severity of IDD is likely to be considerably lower. Countries with significantly larger goiter rates are likely to have larger benefits if salt is adequately iodized since the severity of IDD is likely to be considerably higher. However, these countries may have lower or no benefits if salt is not properly iodized to combat the severity of IDD.

2 Population (in 1000s) is limited to children 5-9 yrs old in 2002 on the premise that this age group will be eligible for secondary school participation in 2010 at ages 13-17.

[3] This is the expected number of children (000's) that received adequately iodized salt in treatment of IDD. The rate of en utero IDD is assumed to be twice the TGR (Total Goitre Rate). The reasoning behind this assumption is that the rate of IDD is larger for women and the rate of IDD en utero occurs more quickly than adult IDD. The number of children suffering from IDD is calculated as the rate of en utero IDD times the population of children. The number of protected children is calculated by taking the number of children suffering from IDD and multiplying it times the fraction of households using adequately iodized salt.

[4] The observed increase in grade attainment (.34 yrs) in Tanzanian IOC districts is used as a baseline measure of grade attainment (yrs) for countries. This baseline is adjusted for the estimated participation of 78% in the target population of Tanzania as well as the estimated TGR level in Tanzania (30%). The total increase in grade attainment (yrs) is the product of the number of protected children times the expected average increase in years of schooling (.73 yrs).

5 This is the projected percentage increase in grade attainment among 5-9 year-olds in each country.

Sources:

Information on goiter rates and salt legislation years were culled from the Current Iodine Deficiency Status (CIDDS) database maintained by the International Council for the Control of Iodine Deficiency Disorders and supplemented by the WHO's Micronutrient Deficiency Information System. Population data is from the *Global Population Profile: 2002 report by the* International Programs Center (IPC), Population Division, U.S. Census Bureau. Baseline education information was obtained from the Barro-Lee Educational Attainment Data (1960 - 2000) available at the National Bureau of Economic Research.

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
Program year t	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
t + 1	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
t + 2	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
t + 3	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
t+ 4	0.292	0.237	0.189	0.148	0.112	0.082	0.057	0.037	0.022	0.011	0.004	0.001	0.099	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 ($\rightarrow k = 0.33\overline{3}$).

[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	0.135	0.130	0.119	0.200	0.059	0.026	0.030	0.006	0.020
	[0.063]**	[0.110]	[0.101]	[0.059]*	[0.086]*	[0.081]	[0.020]	[0.017]+	[0.019]	[0.018]
Age 7	-0.001	-0.093	0.128	0.022	0.158	-0.128	0.199	0.225	0.166	0.192
	[0.105]	[0.190]	[0.176]	[0.086]	[0.124]	[0.119]	[0.033]**	[0.025]**	[0.032]**	[0.026]**
Age 8	0.196	0.011	0.272	0.207	0.339	0.053	0.387	0.424	0.263	0.307
	[0.102]+	[0.174]	[0.182]	[0.090]*	[0.126]**	[0.129]	[0.032]**	[0.026]**	[0.031]**	[0.027]**
Age 9	0.515	0.253	0.377	0.57	0.686	0.402	0.609	0.603	0.472	0.48
	[0.099]**	[0.188]	[0.178]*	[0.088]**	[0.129]**	[0.122]**	[0.031]**	[0.026]**	[0.031]**	[0.026]**
Age 10	1.137	0.741	0.991	1.183	1.312	1.012	0.663	0.713	0.512	0.549
	[0.098]**	[0.200]**	[0.180]**	[0.083]**	[0.123]**	[0.116]**	[0.031]**	[0.025]**	[0.030]**	[0.025]**
Age 11	1.595	1.19	1.352	1.531	1.617	1.322	0.769	0.741	0.56	0.563
	[0.112]**	[0.229]**	[0.222]**	[0.095]**	[0.142]**	[0.134]**	[0.035]**	[0.028]**	[0.035]**	[0.029]**
Age 12	2.526	2.181	2.162	2.42	2.512	2.206	0.743	0.775	0.552	0.586
	[0.109]**	[0.234]**	[0.231]**	[0.089]**	[0.131]**	[0.130]**	[0.034]**	[0.026]**	[0.034]**	[0.027]**
Age 13	3.064	2.805	2.635	3.08	3.363	2.678	0.809	0.789	0.599	0.589
	[0.119]**	[0.254]**	[0.255]**	[0.097]**	[0.145]**	[0.140]**	[0.037]**	[0.029]**	[0.037]**	[0.029]**
Boys						0.062				
						[0.051]				
Girls					0.11					
					[0.054]*					
Female				0.124				0.008		0.003
				[0.043]**				[0.013]		[0.013]
Fixed effects	*House-hold*	*House-hold*	*House-hold*	*District*	*District*	*District*	*House-hold*	*District*	*House-hold*	*District*
Observations	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	0.053	0.007	0.011	0.018	0.032
	[0.018]*	[0.021]**	[0.015]	[0.019]	[0.009]+	[0.012]**
Age 7	0.052	0.048	0.002	-0.010	-0.008	0.000
	[0.026]*	[0.034]	[0.022]	[0.031]	[0.014]	[0.020]
Age 8	0.232	0.202	0.003	0.011	-0.006	-0.004
	[0.027]**	[0.033]**	[0.023]	[0.030]	[0.014]	[0.020]
Age 9	0.417	0.411	0.043	0.035	-0.002	-0.004
	[0.026]**	[0.033]**	[0.022]+	[0.029]	[0.014]	[0.019]
Age 10	0.623	0.62	0.168	0.152	0.005	-0.003
	[0.025]**	[0.032]**	[0.021]**	[0.029]**	[0.013]	[0.019]
Age 11	0.685	0.7	0.288	0.303	0.023	0.023
	[0.028]**	[0.037]**	[0.024]**	[0.033]**	[0.015]	[0.022]
Age 12	0.809	0.852	0.528	0.546	0.111	0.115
	[0.027]**	[0.036]**	[0.023]**	[0.032]**	[0.014]**	[0.021]**
Age 13	0.857	0.874	0.675	0.691	0.255	0.236
	[0.029]**	[0.039]**	[0.025]**	[0.035]**	[0.016]**	[0.023]**
Female	0.015	0.01	0.039	0.046	0.012	0.013
	[0.013]	[0.017]	[0.011]**	[0.015]**	[0.007]+	[0.010]
Fixed effects	*House- hold*	*District*	*House-hold*	*District*	*House-hold*	*District*
Observations	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]**		
Fixed effects	*House-hold*	*House-hold*	*House-hold*	*District*	*District*	*District*
Observations	*113932*	*57613*	*56319*	*113932*	*57613*	*56319*

Notes: All data except for cassava consumption 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. Cassava data from the 2000 Tanzanian Household Budget Survey. Rate of cassava consumption defined as fraction of THBS households in district that report growing cassava. In all regressions, Pr(IOC in utero) is equal to the value of the variable 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.